The Shock Trauma Manual of Operative Techniques

Thomas M. Scalea
Editor

Second Edition

Springer

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For 50 years, the Shock Trauma Center has been a leader in trauma care. For the past 22 years, I have had the privilege of being its chief. This book is dedicated to all of the men and women who are in Shock Trauma: the residents, fellows, faculty, nursing staff, support personnel, and the administration. These people in "pink scrubs" live the dream every day, dedicated to eradicating the consequences of injury. I am eternally grateful for their commitment. We all hope that this book will help further the mission.

Preface

Introduction

Operative therapy for trauma is at the center of care of injured patients. This can be for hemorrhage control and/or for repair of visceral injuries. While trauma is the quintessential team sport, the captain of the ship is the general/trauma surgeon. That individual directs the initial evaluation and resuscitation and usually performs the majority of immediate, life-saving surgical procedures. As trauma is a whole-body disease, this may involve operative procedures in the neck, chest, abdomen, retroperitoneum, vasculature, and extremities.

Years ago, operative therapy was used for diagnosis and treatment. More recently however, more sophisticated diagnostics have been able to much more precisely diagnose injuries. Nonoperative management has become the norm for many solid visceral injuries and some vascular injuries. Minimally invasive therapy such as angiographic embolization is now commonly used in situations where open therapy was once used. Thus, the need for traditional open operation is much less frequent than it once was.

The History of Care

For many years, we believed that the only test that was necessary in the abdomen was to have high suspicion that an intra-abdominal injury existed [1]. This was based on

the belief that all injuries were best diagnosed with exploration. Treatment was determined by the appearance of the injured organ.

Physical exam was the only diagnostic test available for many years. While careful physical examination can be quite helpful, even in good hands, it is only approximately 85% accurate [2]. Newer diagnostic tests emerged. In 1965, H. David Root described diagnostic peritoneal lavage. DPL allowed surgeons to accurately diagnose the presence of intra-abdominal injury [3]. However, while missing very few injuries, DPL was overly sensitive, and we soon realized that a number of injuries that had produced only a small amount of blood in the abdomen did not really require operative therapy. In the early 1980s, CT scan emerged and revolutionized the care of injured patients. As CT technology improved, we began exploring patients via CT [4, 5]. Patients with lower-grade injuries were able to be simply observed. Operative therapy was then reserved for those with proven injury requiring laparotomy and/or those who presented in shock.

As CT was able to much more accurately characterize solid visceral injuries, catheter therapy was developed and became a valuable adjunct to observation. Splenic artery embolization, first described in 1995, has been extremely useful in sparing patients the need for laparotomy, even those with high-grade injuries [6]. Catheter therapy for liver injuries can be primary hemostasis and is also quite helpful as an adjunct to operative therapy [7].

Surgical trainees and/or young faculty members may not be as experienced as surgeons were earlier. This is particularly true in the area of relatively rare injuries. However, when open operation is necessary, there is no substitute. We believe there is the need for a rapid reference that can be used on the way to the operating room, as needed to provide an overall approach to complex injury and technical tips for surgeons who may not care for trauma every day.

Operative Therapy with Personal Tips

A systematic approach to the operative therapy of patients is extremely important. When it is necessary, surgeons must be able to provide rapid care but must also be deliberate enough to do it correctly the first time. No matter where one trains or practices, it is hard to be exposed to the full gamut of injury, particularly serious injury over a short period of time. To date, simulation has simply not been good enough to provide hands-on training. The American College of Surgeons Committee on Trauma has several courses which can be quite helpful. The Advanced Trauma Operative Management Course (ATOM) provides real-life operative training [8]. ATOM requires a large animal model and is quite costly, thereby limiting its utility. The Advanced Surgical Skills for Exposure in Trauma (ASSET) Course is a cadaver-based course. ASSET allows students to become facile with operative approaches over the entirety of the body. As students share a cadaver, it is an efficient way to learn. However, it lacks the real-life flavor of a model that bleeds. Endovascular skill courses like Basic Endovascular Skills for Trauma (BEST) are available [9]. Early experience suggests that the BEST course successfully trains surgeons in a 1-day course if they have some experience with guide wires and catheters.

Shock Trauma has been providing high-volume trauma care for 50 years. Our faculty either have trained here or have been recruited from other high-volume trauma centers. We have been able to develop a very uniform practice. Thus, we conceived and wrote the first edition of *The Shock Trauma Manual of Operative Techniques*. This reference was portable and easy to understand. We tried to be sure that there were enough illustrations to make reviewing procedures easier than simply reading text. We believed that surgical trainees or young faculty members would be able to review these procedures on the way to the operating room.

Some may ask why we chose to write a second edition this soon after the first edition was published. First and foremost,

the publisher requested it. Apparently, there was enough interest in the first edition, and the publisher believed that a second edition would be wise. I was certainly grateful that the trauma community apparently thought the first edition was helpful.

The second reason is that we have had some new people join the faculty, and I thought their expertise would be quite helpful. In particular, Dr. Rosemary Kozar and Dr. David Feliciano, both international names in trauma, are now a part of our group. They both agreed to contribute to this edition.

Thirdly, we changed the content somewhat. We eliminated some chapters that seemed unnecessary. Dr. Sharon Henry, another internationally recognized surgeon, contributed a chapter on fasciotomy, a skill that all trauma surgeons must possess.

Fourthly, even though the first edition was published just a few years ago, there have been substantial changes in trauma care. In particular, the use of endovascular strategies has continued to become more common. Recent data suggest that open repair for aortic injury has essentially disappeared and has been replaced by stent grafting [10]. Several new endovascular surgeons – Dr. Jonathan Morrison and Dr. Rishi Kundi – have joined our group. They were selected to help describe these new developments. We have added an entire chapter on another endovascular innovation, Resuscitative Endovascular Balloon Occlusion of the Aorta, usually termed REBOA [11].

I fully expect this evolution in trauma care to continue. In addition, I am sure that newer techniques will evolve. While these new techniques are exciting, there will always be a need for open surgery to treat trauma. We recognize that no single book will be the answer for every question. We hope the current edition of this book aids trauma practitioners as they attempt to deal with operative challenges. Should the popularity of our new book continue, we would be delighted to provide a third edition several years from now.

Baltimore, MD, USA Thomas M. Scalea

References

1. Gohil VD, Palekar HD, Ghoghari M. Diagnostic and therapeutic laparoscopy in various blunt abdominal trauma. World J Laparosc Surg. 2009;2:42–7.
2. Shafton GW. Indications for operation in abdominal trauma. Am J Surgery. 1960:99;657–61.
3. Root HD, Hauser CW, McKinley CR, Lafave JW, Mindiola RP Jr. Diagnostic peritoneal lavage. Surgery. 1965:57;633–7.
4. Peitzman A, Makaroun MS, Slasky MS, Ritter P. Prospective study of computed tomography in initial management of blunt abdominal trauma. J Trauma. 1986:26;585–91.
5. Goldstein AS, Sclafani SJA, Kupferstein NH, Bass IS, Lewis T, Panetta T, et al. The diagnostic superiority of computerized tomography. J Trauma. 1985:25;938–43.
6. Sclafani SJA, Scalea TM, Herskowitz M, Hofer E, Kohl L, Henry S, et al. Salvage of CT-diagnosed splenic injuries: utilization of angiography for triage and embolization for hemostasis. Journal of Trauma. 1995:39;818–27.
7. 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;1032–6.
8. Jacobs L, Burns K, Luk S, Marshall WT 3rd. Follow-up survey of participants attending the Advanced Trauma Operative Management (ATOM) Course. J Trauma. 2005:58;1140–43.
9. Brenner M, Hoehn M, Pasley J, Dubose J, Stein D, Scalea T. Basic Endovascular Skills for Trauma course: Bridging the gap between endovascular techniques and the acute care surgeon. J Trauma Acute Care Surg. 2014:77;286–91.
10. Scalea TM, Feliciano D, DuBose JJ, Ottochian M, O'Connor JV, Morrison JJ. Blunt thoracic aorta injury: Endovascular repair is now the standard. J Am Coll Surg. 2019:228;605–10.
11. Brenner M, Moore LJ, DuBose JJ, Tyson GH, McNutt MK, Albarado RP, et al. A clinical series of resuscitative endovascular balloon occlusion of the aorta for hemorrhage control and resuscitation. J Trauma Acute Care Surg. 2013:75;506–11.

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Part I
Indication and Techniques for Trauma Exploration

Chapter 1
Neck: Indication and Techniques for Trauma Exploration

Laura S. Buchanan

Introduction of the Problem

Neck injury has the potential to involve critical vascular, respiratory, and digestive structures. More than 19,000 neck injuries were documented in the National Trauma Data Bank for 2016 with an overall mortality of 2.3% [1]. However, mortality for more severe injury (AIS ≥3) is 17%, greater than any other body region [1]. Neck injuries are combined with head in Centers for Disease Control and Prevention (CDC) reporting and cumulatively account for 17% of injuries requiring hospitalization and 20% of injuries treated in an emergency department [2].

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History of Interventions for Neck Trauma

Early management with expectant observation led to delayed diagnosis of injuries and high mortality. Mandatory explorations of traumatic neck wounds gained favor after World War II and resulted in decreased mortality from injury but were associated with significant rates of negative exploration with risk of surgical site morbidity. Improvement in imaging techniques and improved understanding of neck trauma have resulted in a gradual shift to selective exploration.

Treatment of neck injuries is best understood by dividing patients first by mechanism (blunt vs. penetrating) and by anatomic zones. Blunt injury most commonly results from motor vehicle collisions but may occur with strangulation, assault, and sport injuries. Seat belts and dashboards can result in injury to cerebral vessels or larynx. Cervical spine injury and spinal cord injury requiring stabilization are much more common in blunt neck trauma than in penetrating. Blunt trauma requires cervical immobilization until spine injury has been ruled out. Blunt cerebrovascular injury is rarely operative, while identified injury to aerodigestive structures typically requires repair.

Penetrating neck injury was historically treated nonoperatively until shortly after World War II. Fogelman and Steward reported the first large civilian series on penetrating neck injury and advocated mandatory exploration citing a significant improvement in mortality (6% vs. 35% with expectant management) [3]. Violation of the platysma was used to determine potential for injury. During the remainder of the twentieth century, mandatory exploration was widely accepted for penetrating injury to Zone II, with a more selective approach to Zone I and Zone III injuries. Diagnostic modalities for Zones I and III included arteriogram for diagnosis of vascular injuries, laryngoscopy and bronchoscopy for airway injuries, and contrast esophagram and esophagoscopy for digestive injuries. This combination of testing is costly and low yield and has potential complications. Improvement in multidetector computed tomography (CT) allowed for more selective approach to operative and diagnostic procedures.

The transition from mandatory neck exploration to selective operation was studied prospectively by Inaba and colleagues [4]. Including 453 patients over 3 years, they identified 9% with hard signs of injury mandating exploration. Forty-two percent had no signs of injury and were followed clinically and discharged with no missed injury. The remaining 49% of patients underwent multidetector computed tomography, which had a sensitivity of 100% and specificity of 97% in detecting injuries [4], confirming that a selective approach to exploration in neck trauma is safe and appropriate.

The Eastern Association for the Surgery of Trauma (EAST) guidelines for clinical practice (2008) advise selective management is equally safe and effective to mandatory exploration despite a paucity of prospective trials [5]. The Western Trauma Association (WTA) algorithm for penetrating trauma (2013) also advocates a selective approach [6]. Patients with hard signs (Box 1.1) of vascular or aerodigestive tract injury should undergo airway stabilization and tamponade and proceed to operative exploration. Similarly, patients who are symptomatic with Zone II injuries should undergo

Box 1.1 Hard Signs of Neck Injury as Defined in the Western Trauma Association's Algorithm

Airway compromise
Massive subcutaneous emphysema
Air bubbling through the wound
Expanding/pulsatile hematoma
Active bleeding
Shock
Neurologic deficit
Hematemesis

Used with permission of Wolters Kluwer Health, Inc., from Sperry et al. [6]

early operative exploration. In the absence of the hard signs of major injury, all Zone I and III injuries and asymptomatic Zone II injuries should undergo diagnostic evaluation.

Surgical Technique

While anatomic zones are no longer the standard to determine the need for operation, wise operative planning for injury to the neck involves understanding of specific landmarks. The neck is divided into three zones. Zone I extends from the sternal notch to the cricoid cartilage and includes the thoracic outlet vasculature, proximal carotids, and vertebral arteries, as well as the trachea, esophagus, spinal cord, thoracic duct, and cervical nerve trunks. Zone II extends from the cricoid cartilage to the angle of the mandible. The carotid arteries, internal jugular vein, vagus nerve, and upper trachea, as well as the larynx, are included in Zone II. Zone III is superior to the angle of the mandible.

Proximal and distal vascular control in Zone II can be relatively easily accomplished via a standard neck incision. This makes injuries in Zone II the most surgically accessible. Distal control in Zone I injuries can be obtained in Zone II; however, proximal control requires a thoracic incision, either a sternotomy, thoracotomy, or peri-clavicular incision. Proximal control of Zone III injuries can be obtained in Zone II; however, distal control—particularly distal vascular control—involves controlling the vascular structures within the skull.

Certainly, patients who present in shock with Zone II injuries are best treated with diagnostic exploration. Even stable patients in Zone II can be treated with operative exploration, though most prefer imaging. Unstable patients with Zone I injuries also undergo operative exploration. The thoracic incision is determined by best guess. If the incision does not provide adequate exposure, a second incision and/or third incision can be made. Incisions can be extended with attempts to gain control. Given the invasive nature of thoracic exposure, stable patients with Zone I injuries are best served by diagnostic testing. Given the same issues of difficult vascular control in

Zone III, stable patients should undergo diagnostic testing. While surgical dogma advocates operative exploration for Zone III injuries with hypotension, operative exploration can be quite time-consuming. If a catheter option is immediately available, that may be wiser, particularly in selected cases.

The relevant surface anatomy of the neck is illustrated in Fig. 1.1.

Preparation

The operative field should include the lower jaw, the bilateral neck, and the entire anterior chest. If the cervical spine is cleared preoperatively, a shoulder roll will improve neck extension. The head should be slightly turned to the contralateral side for the standard anterior sternocleidomastoid incision and midline for a collar incision.

Selection of incision is determined by location of suspected injuries. Typical incisions are depicted in Fig. 1.2. Neck exploration is typically done through an incision on the anterior border of the sternocleidomastoid. This incision allows rapid exposure and control of the vasculature and can be

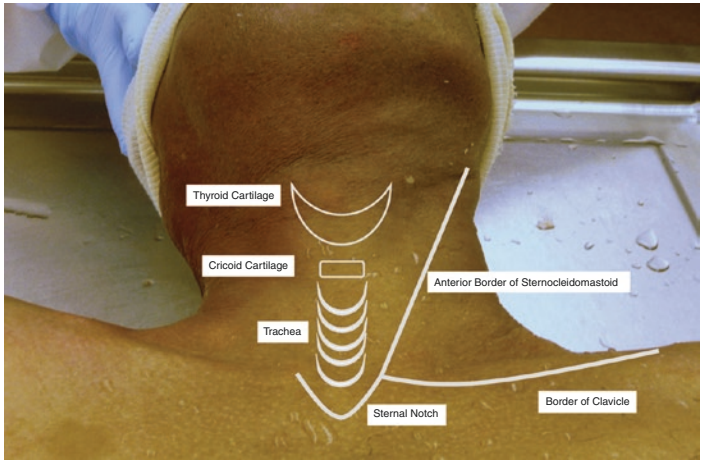

Figure 1.1 Anatomic landmarks

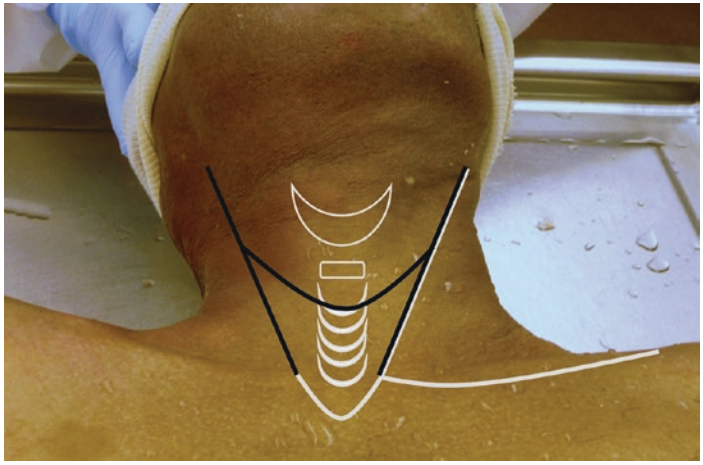

FIGURE 1.2 Typical incisions (solid black lines)

extended into a sternotomy. Bilateral exploration can be performed using bilateral standard anterior incisions or via a collar incision. A collar incision gives the best exposure for the anterior trachea. A supraclavicular incision may be indicated for exposure of Zone I and can be combined with resection of the head of the clavicle or a trapdoor sternotomy to extend exposure toward the mediastinum.

Anterior Sternocleidomastoid Neck Exploration

This standard approach starts with an incision on the side of the penetrating injury. An incision is made along the anterior border of the sternocleidomastoid extending from the clavicular head to the retromandibular area as needed. If extension is needed at the superior portion of the wound, the incision should curve posteriorly toward the mastoid process to avoid damage to the mandibular branch of the facial nerve exiting at the angle of the mandible. The platysma is divided along the same plane as the skin incision exposing the sternocleidomastoid (Fig. 1.3). The vascular sheath underlies the medial border

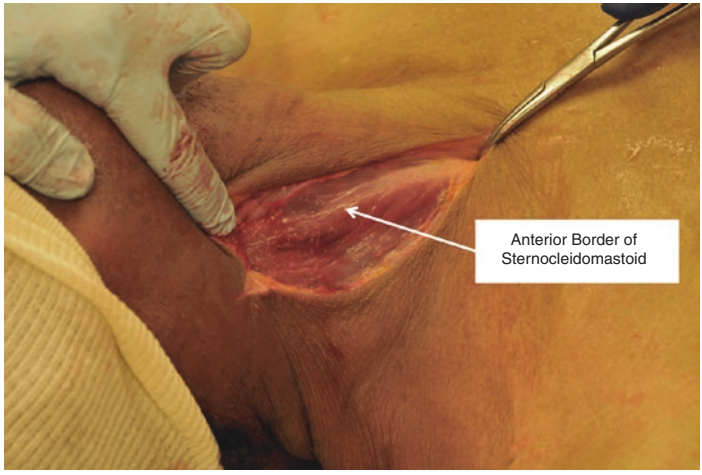

Figure 1.3 Exposure after incision of the skin and platysma

of the sternocleidomastoid. The sternocleidomastoid is retracted laterally exposing the vascular sheath. The omohyoid muscle and facial vein cross the carotid. Dividing them will allow exposure of the common carotid and its bifurcation. The internal jugular vein, vagus nerve, and carotid artery are all exposed with this technique (Fig. 1.4). The hypoglossal nerve runs perpendicular to the carotid near the level of the bifurcation and should be preserved. Damage to the hypoglossal nerve will result in deviation of the tongue toward the ipsilateral side. Unilateral hypoglossal nerve injury may be asymptomatic but can result in difficulty with mastication, speech articulation, and swallowing. Visualization of vascular structures may be difficult in cases of ongoing hemorrhage, and proximal control may require access in the chest.

The esophagus and trachea can be accessed via this same incision medial to the vascular structures (Fig. 1.5). The access shown is right sided. The esophagus can be accessed via a left- or right-side standard incision. The esophagus is a left-sided structure in Zone II, so it is best approached from the left side. The trachea and thyroid should be mobilized medially to expose the upper esophagus. Surrounding hematoma can

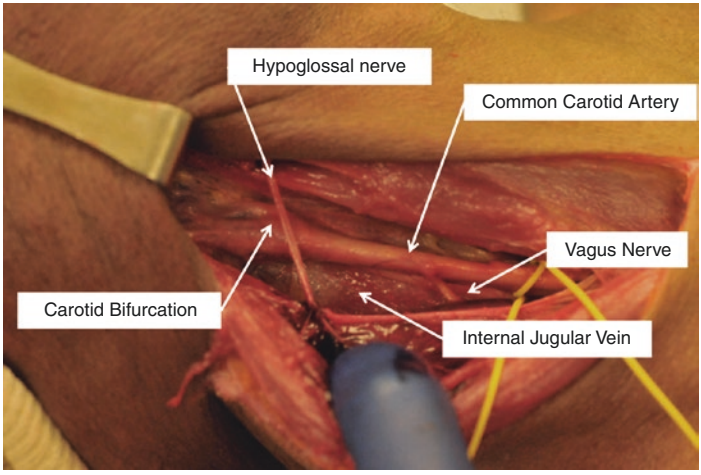

FIGURE 1.4 Structures within the carotid sheath

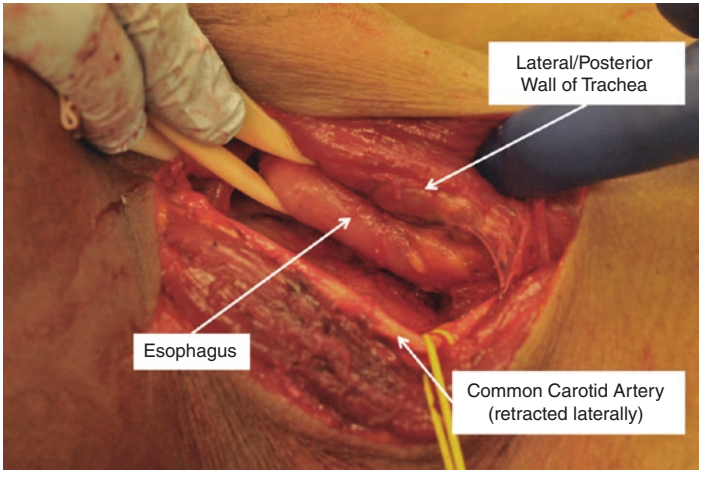

FIGURE 1.5 Lateral exposure of the trachea and esophagus

obscure structures and anatomic planes. Placement of an enteric tube can provide tactile guidance to assist in identification of the esophagus. The esophagus can be mobilized if

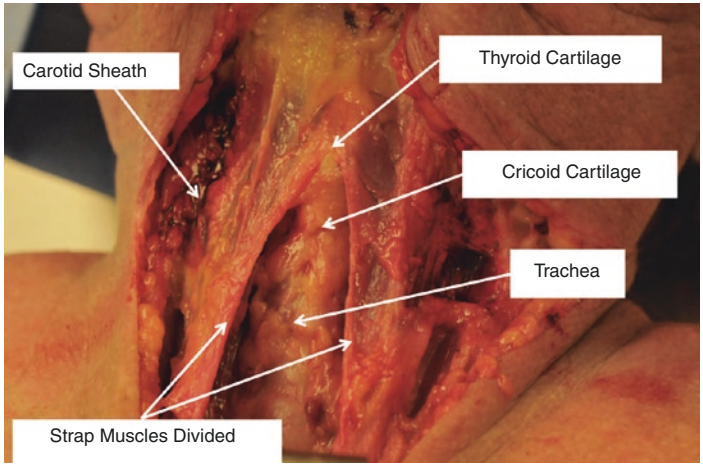

Figure 1.6 Exposure via collar incision

necessary to explore and repair injuries, as blood supply was in the wall, mobilization is safe. Mobilization should start on the posterior surface elevating the esophagus off the anterior spine. Once the esophagus is circumferentially mobile, placement of a Penrose drain will aid in retraction and exposure during any subsequent repair. Tracheal exposure via this incision gives access to the anterior and lateral tracheal wall with limited exposure to the posterior trachea.

The trachea is best accessed via a collar incision. The patient should be positioned as previously described with the head in a neutral position. The incision is placed 1–2 cm above the sternal notch and extends laterally to the medial border of the sternocleidomastoid. The platysma is divided in the same orientation, and flaps are raised superiorly and inferiorly (Fig. 1.6). Take care to ligate the anterior jugular veins. The strap muscles are divided. The thyroid isthmus is clamped and divided. This will expose the larynx and cervical trachea (Fig. 1.7). Access to the lower trachea may require partial sternotomy. Circumferential mobilization of the trachea will result in devascularization and should not be performed. The anterior and lateral surfaces can be easily reached through

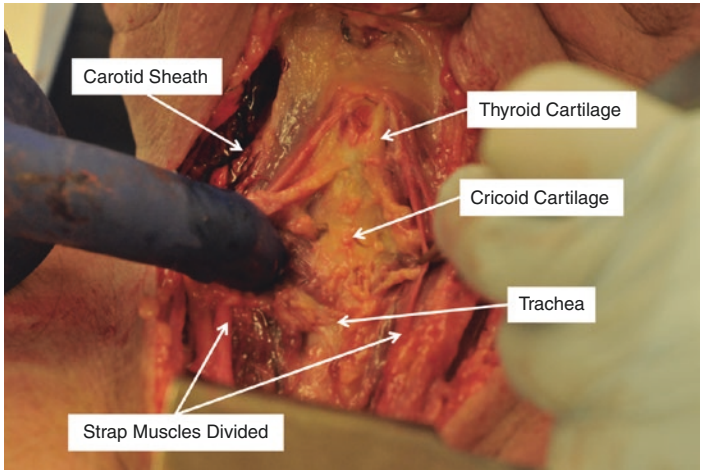

Figure 1.7 Exposure of the trachea and larynx

this incision. The posterior trachea may be easier to repair through an existing anterior tracheal wound or even opening the anterior trachea. After the posterior trachea is repaired, the anterior opening can be closed. It can also be exposed via a lateral exposure as described in the standard anterior sternocleidomastoid incision.

Surgical Exposure of the Vertebral Artery

The majority of the vertebral artery is protected by the bony canal of the cervical spine, and as a result, most intervention for injury is performed by interventional radiology. If necessary, the origin can be exposed from the subclavian artery to the transverse process of the 6th cervical vertebra. The standard anterior sternocleidomastoid incision can be used. The carotid sheath is retracted medially and the scalene fat pad retracted laterally. This exposes the anterior scalene muscle and inferior thyroid artery. The artery is divided, and the muscle is retracted laterally to expose the vertebral artery. Alternatively, a supraclavicular incision with division of the

sternocleidomastoid provides access to the proximal common carotid and the origin of the vertebral artery. Exposure of the distal vertebral artery requires subspecialty expertise.

Tips

Control of airway remains primary and should be secured before any other intervention. The preferred airway control remains endotracheal intubation followed by a standard surgical airway. Only in very rare cases when the airway is violated and easily visible in the wound should one intubate the trachea through the existing wound. When possible, obtain at a minimum a preoperative chest X-ray to evaluate for mediastinal widening suggesting proximal vascular injury and pneumothorax as these may change operative planning in an unstable patient.

Plan for the unexpected—such as the need for emergent, proximal, or distal exposure—by including the chest and jaw in the surgical field. Avoid pressure dressings as a means of temporary vascular control. Temporary vascular control is better achieved with direct digital pressure or balloon tamponade. Consider catheter therapy as adjunctive treatment, especially for injuries extending into Zone III. Techniques such as jaw dislocation, clavicular head resection, and surgical access to the distal segments of the vertebral arteries require significant time and expertise and are unlikely to benefit an unstable, actively hemorrhaging patient. A hybrid approach may be the best solution for vascular injury in these difficult-to-expose areas and may combine operative or balloon tamponade followed with catheter therapy.

Complications after treatment can relate to hematoma, pneumothorax resulting from violation of the apical pleural, infection, and iatrogenic injury to the cranial nerves in the neck. Operative drains should remain in place until output decreases. Series evaluating a mandatory exploration strategy result in 50–68% negative exploration, suggesting avoidable perioperative morbidity.

References

1. American College of Surgeons Committee on Trauma Leadership. National Trauma Data Bank. 2016 Annual Report. https://www.facs.org/~/media/files/quality%20programs/trauma/ntdb/ntdb%20annual%20report%202016.ashx.
2. Centers for Disease Control. Injury prevention & control: data & statistics (WISQARS™). http://www.cdc.gov/injury/wisqars/index.html.
3. Fogelman M, Stewart R. Penetrating wounds of the neck. Am J Surg. 1956;91:581–96.
4. Inaba K, Branco BC, Menaker J, Scalea TM, Crane S, DuBose JJ, et al. Evaluation of multidetector computed tomography for penetrating neck injury: a prospective multicenter study. J Trauma Acute Care Surg. 2012;72(3):576–83.
5. Tisherman SA, Bokhari F, Collier B, Ebert J, Holevar M, Cumming J, et al. Eastern Association for the Surgery of Trauma. Clinical practice guidelines: penetrating neck trauma. 2008. www.east.org/content/documents/peneneck.pdf.
6. Sperry JL, Moore EE, Coimbra R, Croce M, Davis JW, Karmy-Jones R, et al. Western Trauma Association critical decisions in trauma: penetrating neck trauma. J Trauma Acute Care Surg. 2013;75(6):936–40.

Chapter 2
The Chest: Indication and Techniques for Trauma Exploration

Megan T. Quintana and Jose J. Diaz

History of Interventions for Thoracic Trauma

In the contemporary era, operative therapy for thoracic trauma is considered common. However, operative cardiac intervention was previously deemed impossible [1]. Until World War II, it was believed that reinflating the lung would cause bleeding [2], thus initially limiting operative intervention for both cardiac and thoracic injuries.

Lung injuries and attempts at treatment were first reported by Homer from the Trojan War. Physicians in ancient Egypt also attempted treating chest wounds. Intervention for thoracic pathology described by Hippocrates included basic principles of treating "empyema thoracis," and he wrote

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"ibi pus ubi evacua," indicating the importance of evacuating pus from the chest. Drainage of both pneumothoraces and hemothoraces was described in the 1700s advising a metal tube be used to drain chest cavities [2]. Cardiac intervention for trauma was first performed in the late 1800s, when Daniel Hale Williams III repaired a pericardial defect [3]. Debate over whether using tubes of different materials or draining through an opening with loose bandages continued from these early decades through to World War I [4]. Mortality from thoracic injury decreased significantly from World War I to World War II. Patients who were not killed in action suffered secondary mortality from early hemorrhage, suffocation, late bleeding, erosion, and infection. Thoracic surgery was established as a specialty after World War II where established drainage methods and standardized anesthesia made thoracotomy an accepted procedure. Despite concerns about performing thoracic surgery, operative intervention for exsanguinating hemorrhage became a primary therapy [5]. World War II also contributed to the advancement of thoracic surgical tools. After the war, staplers originally developed for gastric surgery by Aladar Petz became a safe and effective way of managing lung parenchymal injury first performed by Androsov and Potechina described in 1962 [6].

As experience in chest trauma advanced, surgical aggression diminished. Minimally invasive surgical techniques were honed and applied to thoracic trauma. These operative techniques in thoracic trauma were first described in 1946, with the use of a thoracoscope to avoid a thoracotomy [7]. Thoracoscopic techniques have evolved from simple suction placement through an existing chest tube site [8] and diaphragmatic injury evaluation [9] to the currently used video-assisted thoracic surgery (VATS) [10–16].

Introduction of the Problem

Thoracic trauma is common and potentially deadly given the vital structures that reside within the chest cavity. Many deaths from thoracic trauma can be prevented with prompt

recognition and therapeutic intervention. Injuries from thoracic trauma range from rib fractures requiring only pain control to cardiac lacerations with tamponade or exsanguinating hemorrhage. Given the range of injuries, acuity and clinical presentation, multiple diagnostic modalities, and treatment options exist for thoracic trauma.

Within the overall trauma patient population, 30.6% will sustain thoracic injuries [17]. Only 34% of thoracic injuries are due to penetrating trauma, the remainder the result of blunt injury. Of these injuries, less than 10% of blunt chest trauma and only 15–30% of penetrating injuries will require operative intervention. Many patients can be definitively managed by procedural intervention.

Knowledge of injury patterns can assist with patient evaluation and treatment. Thoracic injuries such as sternal, scapular, and first rib fractures may indicate significant mechanism of injury and thus be associated with severe underlying injury. Sternal fractures are associated with mediastinal pathology, such as cardiac and great vessel injuries [18], while scapular fractures are associated with underlying thoracic cavity injuries, such as pneumothoraces and pulmonary injuries [19]. Concomitant pneumothoraces, hemothoraces, and cardiac injuries can be denoted by the presence of a first rib fracture [20].

Concurrent injuries are not limited to the thoracic cavity. Only 20% of thoracic trauma patients have isolated thoracic injuries, with extremity, abdominal, and pelvic injuries often associated with thoracic pathology [17]. Presence of extra-thoracic injuries can complicate the initial evaluation, especially in hemodynamically unstable patients, whose evaluation includes thoracic and extra-thoracic pathology [17].

Primary and Secondary Surveys

Hypoxia, hypercarbia, and compromised perfusion often result from thoracic injuries. Life-threatening injuries discovered during the primary survey should be treated before advancing to the secondary survey. Failure to address underlying pathology can result in decompensation or death,

especially in the unstable patient. Thoracic trauma can cause significant respiratory distress, as with painful rib fractures restricting chest wall movement or pneumothoraces limiting oxygenation and ventilation. Most life-threatening thoracic injuries can be treated with emergent airway control, often requiring intubation or placement of a chest tube. Airway patency and air exchange should be assessed.

The chest is inspected and auscultated. Hemodynamically unstable patients with signs of hemothorax or pneumothorax on exam require urgent chest tube insertion at this step in the primary survey. If an open chest wound is present, a three-sided occlusive dressing should be placed to prevent air from entering the thoracic cavity. A flail chest with overt paradoxical chest wall motion may be appreciated on inspection of the chest wall which can disrupt pulmonary mechanics. Patients with flail chest may commonly require urgent endotracheal intubation for respiratory insufficiency. The chest is inspected to note location of wounds, with heightened attention paid to wound proximity or trajectory indicating injury to major vascular structures or the mediastinum. Injuries entering "the box" (bordered by the clavicles, midclavicular lines, and costal margins) [21] may portend an underlying mediastinal injury (Fig. 2.1).

When assessing circulation, the patient's pulse should be evaluated for quality, rate, and regularity. Unequal upper-extremity pulses suggest a possible subclavian arterial injury. Heart sounds are auscultated to evaluate for pericardial effusion and underlying cardiac injury. Beck's triad occurs in only 10% of cardiac injuries [22] and is not relied upon for diagnosis in trauma. If there is bleeding from a chest wall wound, this should be controlled with direct pressure.

A minority of thoracic trauma results in isolated chest pathology [17]. The secondary survey is especially important in hemodynamically unstable patients. Hemodynamically unstable thoracic trauma patients may have concurrent extrathoracic injuries requiring emergent intervention.

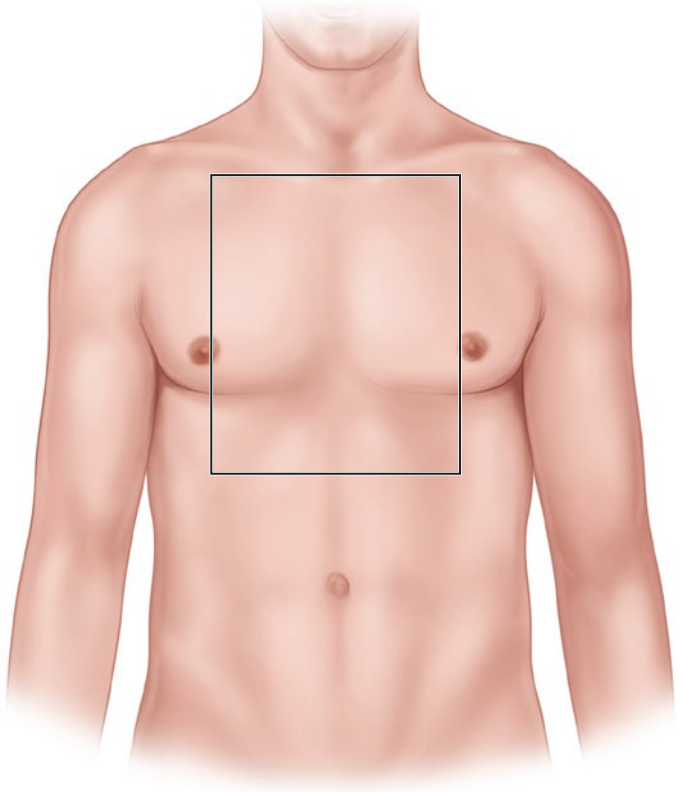

FIGURE 2.1 "The box" is bordered by the clavicles superiorly, left and right midclavicular lines laterally, and costal margins inferiorly

Indications for operative intervention encountered during the primary and secondary survey include those for an ED thoracotomy, an initial chest tube output greater than 1500 cc, a chest tube with a massive air leak, a concern for tracheobronchial injury, and an open chest wall wound with compromising hemodynamic instability. In all other patients, further radiologic evaluation should be undertaken as hemodynamic stability allows.

Diagnostic Tests

Focused assessment with sonography for trauma (FAST) and chest X-ray (CXR) are both rapid and repeatable bedside tests and help identify indications for immediate operative intervention. CXR and FAST are performed in all patients who sustain thoracic trauma. After CXR and FAST, subsequent diagnostic evaluation of thoracic trauma patients may include a variety of noninvasive and invasive testing to determine the need for operative intervention.

Chest X-Ray

CXR displays a global image of the chest, with imaging of the mediastinum, bony structures, soft tissues, and both thoracic cavities. While ongoing research continues to develop the ideal targeted thoracic imaging algorithm [23, 24], an initial CXR is the main modality in screening for and diagnosing thoracic injuries in trauma patients. A CXR should be obtained in all thoracic trauma patients and reviewed by the trauma team [25]. Radiographic evidence of a hemothorax or pneumothorax should prompt chest tube placement and subsequent monitoring of the chest tube and its output for operative indications. An abnormally wide mediastinum or pneumomediastinum on CXR should prompt further imaging with computed tomography (CT) in the hemodynamically stable patient. Massive pneumothorax with significant subcutaneous emphysema may be a sign of a tracheobronchial injury.

Ultrasound

In the chest, FAST allows visualization of the pericardial sac. FAST has also been "extended" to evaluate for pneumothorax through the "eFAST": extended focused assessment with sonography in trauma [26]. In combination with CXR, FAST is helpful in the hemodynamically unstable polytrauma

patient. It can help determine the need for abdominal or thoracic exploration, or both, for hemorrhage control. Early reports evaluating pericardial eFAST in penetrating cardiac wounds had a 0% false-negative rate [27]. However, false-negative pericardial eFAST exams have now been reported, potentially due to hemopericardium decompressing into the pleural space through a disrupted pericardium. Mortality is 40% for cardiac injuries missed with eFAST [28]. eFAST is routinely used in blunt trauma despite the rarity of underlying cardiac injury [29], as mortality of undiagnosed hemopericardium is high [28]. eFAST may be an excellent test for pneumothorax detection for both blunt and penetrating trauma [30]. In detection of intrathoracic fluid, the eFAST has a specificity and sensitivity of over 90%. Ultrasound can detect amounts of fluid as small as 20 ml, whereas CXR requires upward of 200 ml to detect similar pathologies. The eFAST is often helpful in unstable patients to evaluate for pneumothorax when CXR is not rapidly available.

A positive pericardial FAST is an indication for exploration, either with a pericardial window or sternotomy, depending on patient stability. In penetrating trauma with concern for cardiac injury, a negative FAST for pericardial fluid does not negate the need for operative exploration of the pericardium, especially with a concurrent hemothorax. A positive eFAST in a hemodynamically unstable patient should prompt immediate chest tube insertion.

If a significant amount of subcutaneous emphysema is present, false-negative examinations can occur. False-negative exams can also occur if the amount of fluid is too little to detect in an emergency and time-limited situation. They can also occur when fluid was present prior to the traumatic injury [31, 32].

Computed Tomography (CT)

In blunt and penetrating thoracic trauma, CT can add clinically significant information. In one review, 14% of patients had an occult hemothorax or pneumothorax seen on CT but

not CXR; 62.8% required intervention [33]. Importantly, missed injury on CXR identified on CT also included aortic injuries [34, 35]. CT angiogram is beneficial in penetrating thoracic trauma to evaluate for transmediastinal trajectory and subsequent mediastinal injuries. Traditional work-up for transmediastinal gunshot wounds included angiography, bronchoscopy, and esophagoscopy to evaluate the major mediastinal structures [36]. These have been replaced by less-invasive screening CT angiogram without increasing the risk of missed injuries [37].

It is imperative to understand that a patient with penetrating thoracic trauma resulting in hemodynamic instability requires a rapid evaluation, including CXR and FAST, followed by operative intervention. There is no role for additional imaging such as CT in this patient.

Thoracic Intervention

Preoperative Preparation

Success of thoracic exploration can be optimized with preoperative preparation. Most thoracotomies are performed for hemorrhage control [38]. Preoperative preparation should thus include obtaining sufficient blood products and potentially activating a massive transfusion event or protocol. A Cell Saver® (Haemonetics Corp., Braintree, MA, USA) can be a useful adjunct as it allows suctioning of hemothorax alongside providing for autotransfusion back to the patient. If there is concern for intrathoracic vascular injury, the central venous catheter used for blood transfusion and medication infusion should be placed on the contralateral side to the vascular injury so as to not infuse through the injured vessel. An arterial line should be placed for continuous blood pressure monitoring. The arterial line should be placed in the upper extremity as femoral waveforms will be lost if the aorta is cross-clamped.

If pericardial tamponade is present or a possibility, the patient should be prepped and draped prior to intubation if

possible. These patients are preload dependent, and loss of vasomotor tone can occur with induction resulting in cardiovascular collapse. In this situation, an emergency exploration is required to release the tamponade and address the injury.

In the acute setting, intubation should be performed with a single-lumen endotracheal tube. For more chronic thoracic pathology, such as empyema or retained hemothorax, a double-lumen tube can improve operative exposure by allowing single-lung ventilation. If a double-lumen tube is not available, a single-lumen tube can be placed with a bronchial blocker to impede ventilation, or the single-lumen tube can be placed in a mainstem bronchus for the same purpose. A bronchoscope should be available and can be used preoperatively or intraoperatively to assess tracheobronchial injury and provide airway clearance of secretions and blood and can help to confirm endotracheal tube placement.

Specific instruments should be available for every thoracic exploration. A sternal saw should be accessible for all cases should a sternotomy be required. A Lebsche knife and mallet should also be available, as it can be used to perform a sternotomy if the sternal saw malfunctions or to extend an anterolateral thoracotomy across the sternum into a clamshell thoracotomy. A Finochietto retractor can be used for either an anterolateral thoracotomy or sternotomy, but a specialized sternal retractor is preferred for a sternotomy. Internal paddles should be on the field and attached to a defibrillator. Sarot bronchus clamps, Semb lung retractors, and Davidson pulmonary vessel clamps can be invaluable tools for various lung injuries. Vascular clamps of various sizes should also be available, with vessel loops, various ties, and stitches loaded for rapid vascular ligation if and when needed. The various sutures used are the individual surgeon's choice. Having them ready, however, is essential.

Indications for Operation

There are multiple indications for acute thoracic exploration. In patients with FAST or CT evidence of a pericardial effu-

sion, cardiac exploration is always indicated, regardless of clinical stability. Initial chest tube output greater than 1500 cc or 150–200 cc/h over several hours is an indication for exploration [39]. Other markers for acute exploration include unexplained hemodynamic instability with known thoracic injury, persistent bloody chest tube output despite not meeting initial chest tube output requirement for thoracotomy, large air leak with concern for airway injury, and known airway, esophageal, or vascular injury (Box 2.1). It is vital to obtain a CXR after placing a chest tube. Blood in the chest often clots and does not come out the tube. This risks placing the surgeon in to a false sense of security. A large retained hemothorax after chest tube placement is often an indication for operation, particularly in an unstable patient. In stable patients, operative intervention is targeted to specific injuries as seen on radiologic evaluation.

Pericardial Window

Pericardial window is the gold standard for diagnosing hemopericardium. It is performed under general anesthesia with the patient supine. A 5-cm vertical midline incision is made from the lower sternal border and carried inferior to the xiphoid process (Fig. 2.2). Excising the xiphoid will improve exposure. Dissection is carried down through the fascia, but the peritoneum is left intact. The heart should be evident on palpation, and blunt dissection is undertaken through the substernal connective tissue. Hemostasis must be meticulous prior to incising the pericardium to avoid a false-positive result. The pericardium is then grasped between two clamps and sharply incised. A negative pericardial window has return of clear fluid, and the myocardium should be visible. Return of bloody fluid or no fluid return should prompt median sternotomy. If the window is negative for hemopericardium, the pericardium can be closed if desired. The fascia, subcutaneous tissues, and skin are closed.

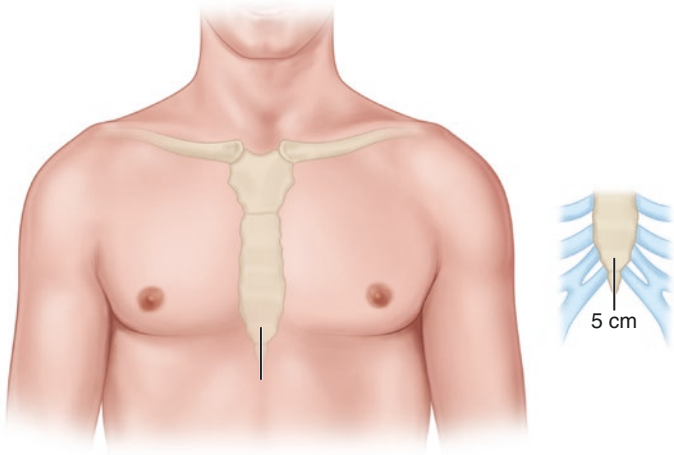

FIGURE 2.2 A pericardial window incision extends from the lower sternal border to below the xiphoid process

If a laparotomy is performed and a pericardial window is indicated, the pericardium can be approached through the central tendon of the diaphragm. To perform a transdiaphragmatic pericardial window, the central tendon of the diaphragm is first grasped with clamps and sharply divided. It may be necessary to mobilize the left lobe of the liver to get good exposure. Inspection of the heart and pericardial fluid proceeds as in a subxiphoid window. When evaluation is complete, the diaphragm is closed in two layers. Thoracoscopic pericardial evaluation has also been reported in stable patients [40] but has not gained widespread use. This is due to the low rate of complications seen with subxiphoid pericardial window [41] and modern use of FAST in the initial cardiac evaluation.

Although pericardiocentesis is often used in the management of medical pericardial effusions, it has no role in trauma. In trauma, pericardiocentesis has been associated with an 80% false-negative rate, as the pericardium is often filled

with clot [42]. Unless there are extenuating circumstances, such as the inability to perform a pericardial window, pericardiocentesis should not be performed.

Operative Exposure

No single incision or exposure will allow access to all compartments and structures of the thoracic cavity. The incision must provide adequate exposure, be performed rapidly, and be versatile. The presence of hemodynamic instability will also influence the choice of incision. Finally, the operative approach will depend on operative intention of exploration or definitive organ repair.

The combination of mechanism, location of wounds, physical exam, hemodynamics, CXR, and FAST results will dictate the operative approach. Hemodynamically unstable patients require rapid access to the chest and mediastinum. Anterolateral thoracotomy and extension into the contralateral chest with a clamshell incision are rapid and afford adequate exposure. Trauma surgeons and most general surgeons are familiar with this incision. Surgeons familiar with sternotomy can perform it quickly in the unstable patients; however, in most emergent circumstances, the clamshell will be the incision of choice [43].

With trauma, concomitant extra-thoracic injuries can mandate exploration of other body cavities. A sternotomy can be extended onto the abdomen. If an anterolateral thoracotomy was initially chosen, this can be extended onto the abdomen, or a separate abdominal incision is made. If there is concern for thoracoabdominal injury, the patient is positioned in a "hailing cab" position, exposing the anterolateral aspect of the chest and abdomen on the side of the thoracic injury (Fig. 2.3). The chest and abdomen are can then be explored. A midline laparotomy is made and can be extended to a thoracoabdominal incision at the subxiphoid and extending it onto the 5th and 6th intercostal space.

When operating on a stable patient for a specific, predefined injury, the choice of incision depends on preopera-

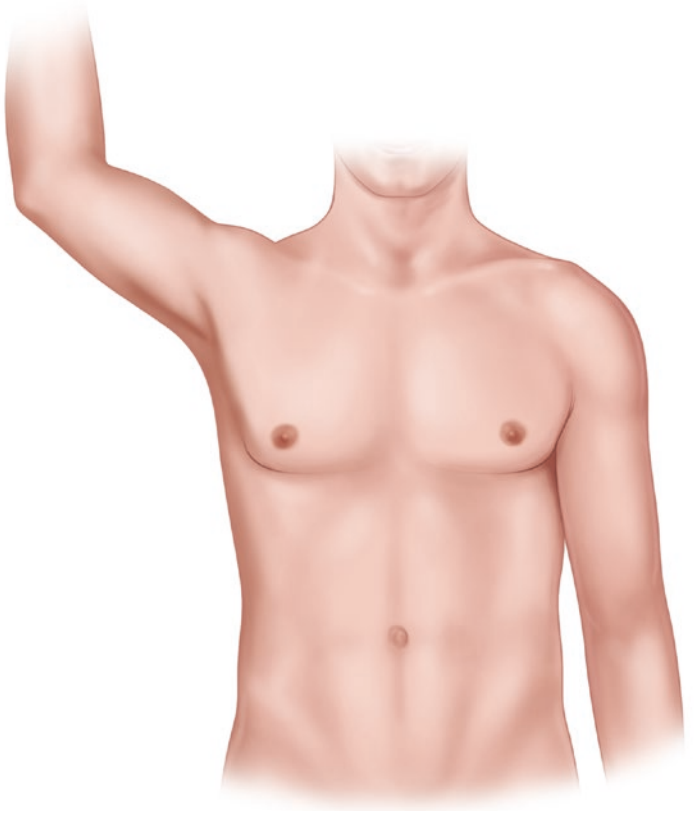

FIGURE 2.3 The "hailing cab" position is used for thoracoabdominal injuries

tive imaging and optimal exposure of the injury (Box 2.2). Injuries to the thoracic esophagus, azygos and hemiazygos veins, trachea, and right lung are best approached using a right posterolateral thoracotomy. Similarly, left-sided injuries are approached through a left posterolateral thoracotomy. Exposure of esophageal injuries depends on the location of the injury; cervical injuries are exposed through the left neck and distal injuries through a left posterolateral

thoracotomy. A posterolateral thoracotomy is the preferred incision for elective thoracic surgery, including patients who have been injured.

However, a posterolateral thoracotomy is not suitable in unstable patients as hemodynamic decompensation may occur with lateral positioning. Also, a posterolateral thoracotomy is not a versatile incision since it cannot be extended. If additional exposure is needed, the chest must be temporarily closed and the patient placed supine. Finally, the lateral decubitus position risks aspiration into the "good" lung that is now in the dependent position and an increased risk of atelectasis. For these reasons, the anterolateral thoracotomy is the most frequently used incision in trauma for rapid access to the left or right hemithorax.

Anterolateral Thoracotomy

The anterolateral thoracotomy is frequently used in unstable patients and is the most common incision in penetrating trauma [44]. A left anterolateral thoracotomy allows access to the descending aorta should cross-clamping be necessary, left lung, pulmonary hilum, and pericardium but allows limited cardiac exposure. It can provide limited access to the left subclavian vessels but does not provide ideal access to great vessels, mediastinal structures, or the entire heart. The right anterolateral thoracotomy allows access to the right lung and pulmonary hilum but has the same disadvantages as a left anterolateral thoracotomy. The anterolateral thoracotomy is versatile, however, and can be extended as a clamshell incision for increased exposure. An anterolateral thoracotomy can also be extended as a trapdoor thoracotomy, created by adding a sternotomy and a supraclavicular incision (Fig. 2.4). A trapdoor thoracotomy can be used for access to the proximal left subclavian artery, though we favor using a sternotomy to expose the proximal left subclavian. Our group rarely uses a trapdoor.

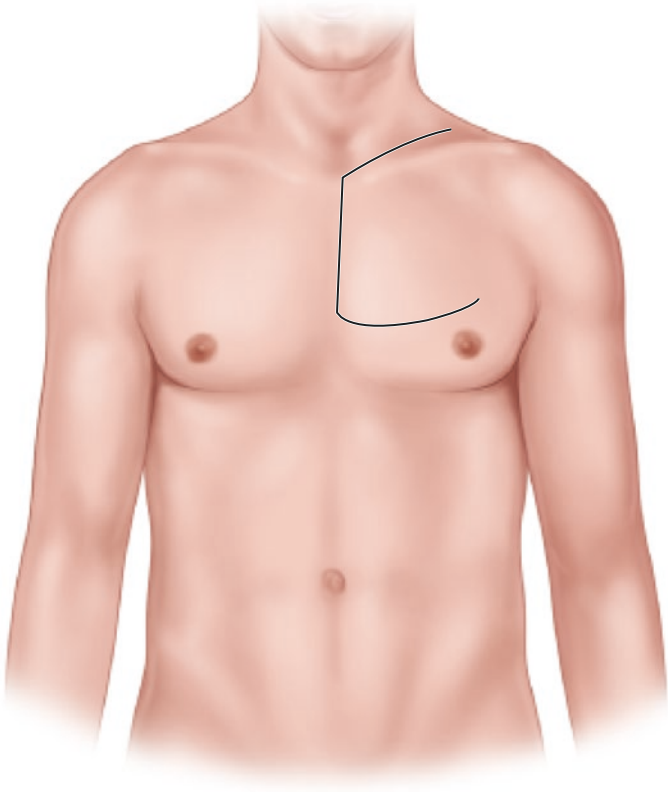

FIGURE 2.4 The trapdoor thoracotomy can be created for subclavian artery access

When performing an anterolateral thoracotomy, enhanced exposure is achieved by placing a 20–30° bump under the back, extending the ipsilateral arm, and carrying the incision to the axilla. This is assuming there is no concern for a spine injury. The incision is placed in the inframammary fold and follows the curve of the ribs (Fig. 2.5). The subcutaneous tissue is incised, and the muscles are divided. The intercostal muscles should be incised on the superior border of the rib to

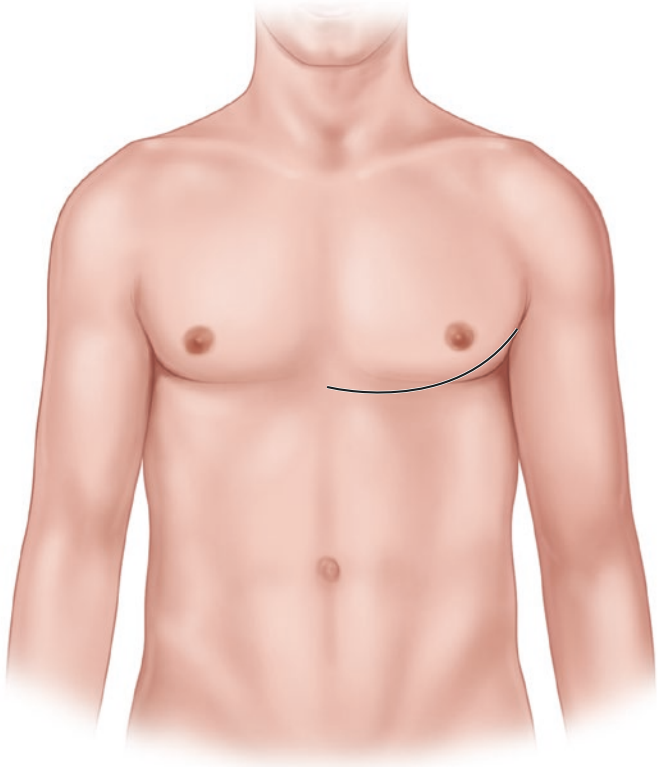

FIGURE 2.5 The anterolateral thoracotomy is used for rapid access to the chest in unstable patients

avoid injury to the neurovascular bundle. A Finochietto rib retractor is placed with the retractor handle toward the floor so as to not block the exposure. If additional exposure is necessary, the anterolateral thoracotomy can be extended to the contralateral side as a clamshell incision. This incision can also be extended onto the abdomen in the case of associated abdominal injury.

If damage control is used in the chest, closure is carried out in a similar manner to abdominal damage control procedures with placement of a negative pressure dressing but should include placement of chest tubes. With standard closure of the incision, two chest tubes are positioned at the posterior

base and apex of the thoracic cavity. The ribs are reapproximated with interrupted pericostal sutures with each encircling two ribs. A Bailey's rib approximator can assist with closure by taking tension off the chest wall. The muscles are closed in layers incorporating the fascia. The subcutaneous tissue and skin are then closed. If the patient is hemodynamically unstable, severely acidotic, hypothermic, or coagulopathic, a damage control thoracotomy can be performed [45]. The chest can be left open placing a temporary negative pressure dressing and can be formally closed when the patient's condition has stabilized.

Posterolateral Thoracotomy

A posterolateral thoracotomy should be reserved for the hemodynamically stable patients requiring a thoracotomy. A left posterolateral thoracotomy provides exposure to the left pulmonary hilum, lung, distal esophagus, and descending aorta. The incision is made two fingerbreadths below the scapula tip and follows the curved rib contour (Fig. 2.6). The muscles are divided as in an anterolateral thoracotomy, but muscle-sparing techniques can be used. The latissimus dorsi can be retracted posteriorly or divided for increased exposure if needed. The serratus anterior should be retracted and/or split along the fibers, and not transected. The pleura is entered, and a Finochietto retractor is placed. If necessary, a rib can be transected to improve exposure. Chest wall closure is performed in a similar manner to that of an anterolateral thoracotomy.

Clamshell Thoracotomy

The clamshell thoracotomy provides enhanced exposure and access to the right and left pleural spaces, heart, and mediastinum. A clamshell thoracotomy can be rapidly performed and is the exposure of choice in the unstable patient with concern for transthoracic injury. Despite its advantages for surgical exposure, a clamshell thoracotomy is a morbid

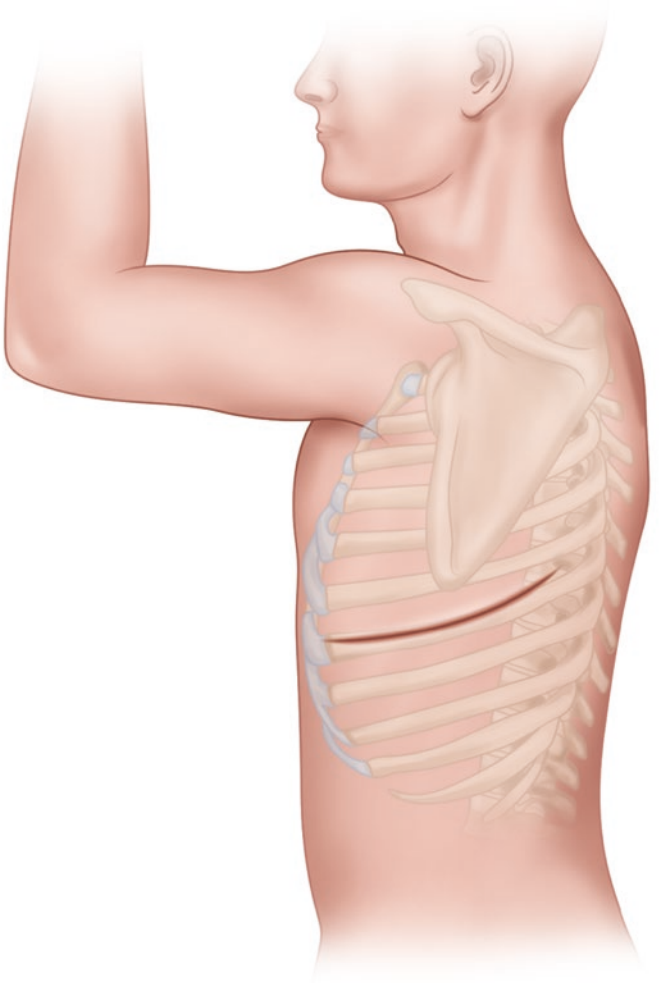

FIGURE 2.6 The posterolateral thoracotomy provides excellent access to the lung, hilum, trachea, and esophagus

incision, with more wound complications, potential for sternal overriding, and chronic pain when compared with sternotomy [46].

A clamshell thoracotomy incision is started as an anterolateral thoracotomy. The incision begins at the inframammillary grove which correlated to the 4th–5th intercostal space. It is extended across the midline to the contralateral thorax (Fig. 2.7). Care must be taken to extend over the midportion of the bony sternum, rather than the costal margin inferiorly.

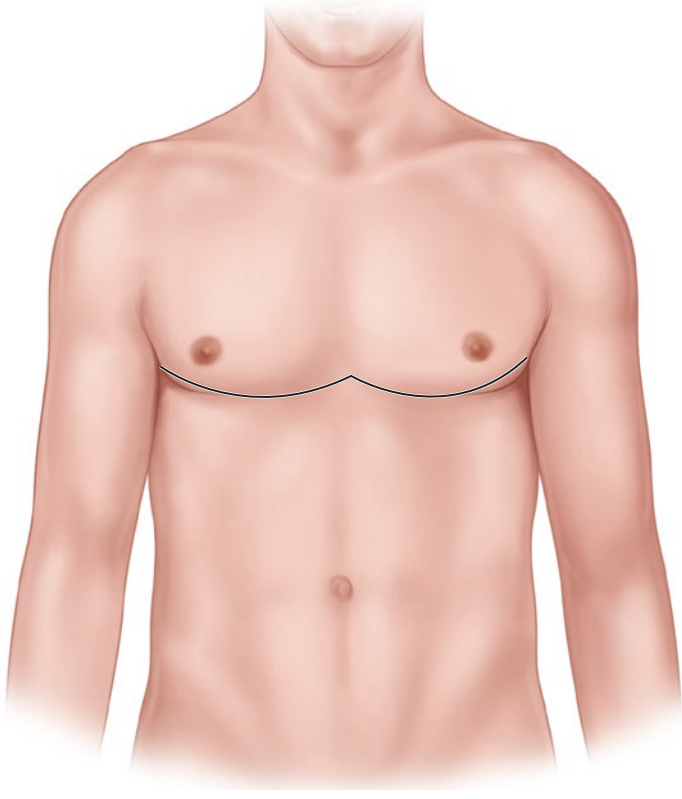

Figure 2.7 The clamshell thoracotomy provides rapid exposure to both thoracic cavities and the mediastinum

If the initial incision is too low, it can be bridge to a level above on the opposite side of the sternum. The sternum is divided transversely using a Lebsche knife and mallet, both internal mammary arteries are ligated, and a chest retractor is positioned. In the hypotensive patient, the internal mammary arteries may not initially bleed but once normal blood pressure is achieved can be a source of significant bleeding. If the incision was placed correctly, excellent exposure of the heart, great vessels, and bilateral pleural spaces will be achieved. Closure of the clamshell is performed in a similar fashion to an anterolateral thoracotomy, with sternal wires used to reapproximate the sternum.

Sternotomy

Sternotomy is the incision of choice for cardiac injury as it provides optimal exposure of the heart and the great vessels in the anterior mediastinum. It has an underappreciated role in thoracic trauma. It can be rapidly performed by surgeons familiar with the technique, it is versatile, and it provides outstanding mediastinal exposure. Access to the lung parenchyma can be achieved by widely excising the pleura. Exposure of the subclavian or carotid vessels can be obtained by extending the sternotomy as a pericostal or neck incision (Fig. 2.8). The neck extension is performed along the anterior border of the sternocleidomastoid. The clavicular extension is performed along the superior border of the clavicle, with resection of the clavicle if needed.

To perform a sternotomy, the patient is placed supine with arms extended. A skin incision is made from the sternal notch to the xiphoid. The xiphoid process is excised. Blunt dissection is performed posterior the sternum, superiorly and inferiorly, ensuring a free retrosternal plane. The sternum is divided in the midline using a sternal saw, applying upward tension on the sternum to minimize potential for injury to underlying structures. A sternal retractor is then placed. If needed, the pericardium is

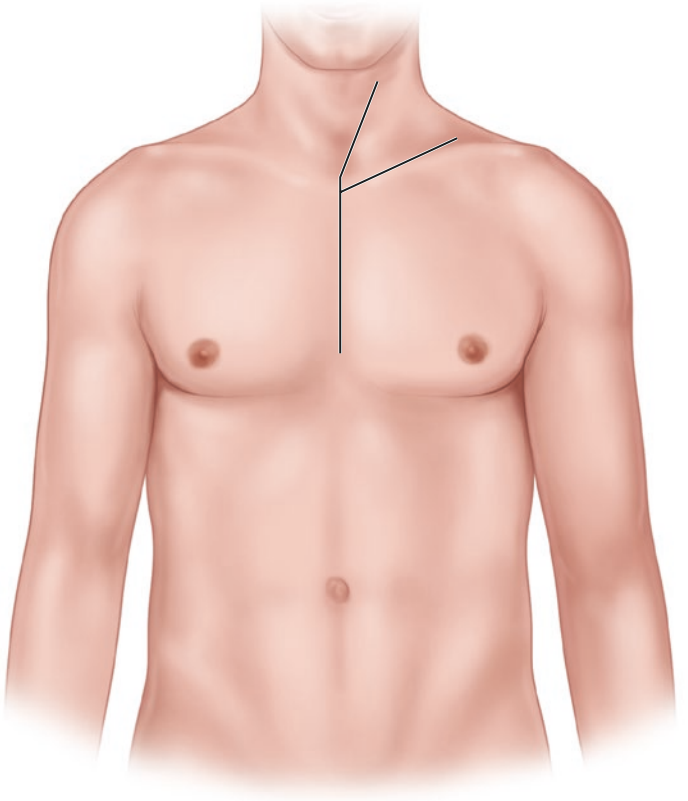

FIGURE 2.8 A neck or clavicular extension can be added to the median sternotomy for increased access to the great vessels

incised, and a pericardial sling is constructed, tacking the pericardial edges up to the chest wall or the sternal retractor. At closure, mediastinal drains are placed, and the pericardium is generally left open but can be closed if closure does not compromise cardiac function. The sternum is then closed with wires. If the pleura was opened, a chest tube is placed in that hemithorax. The subcutaneous tissue and skin are closed in the usual fashion.

Video-Assisted Thoracic Surgery (VATS)

The role of video-assisted thoracic surgery (VATS) in trauma is expanding; as more are performed, the number of thoracotomies is decreasing [47]. For retained hemothoraces, the rate of utilization of VATS was 33.5%, compared to 22.2% for thoracotomy [48]. The early experience with VATS suggested the procedure should be performed within 7–10 days of injury [49]. More recent studies suggest successful evacuation of retained hemothoraces beyond this time frame [15, 47, 50]. VATS has been successful in the acute post-injury setting as well [14].

Although VATS has been commonly used for removal of retained hemothorax, it is also been used to address persistent pneumothorax or air leak and empyema [13, 15, 51]. Expanded uses of VATS include control of intercostal arterial bleeding, pulmonary resection, bronchoplasty, thoracic duct ligation, pericardial window creation, foreign body removal, evaluation and repair of diaphragmatic injury, evaluation of esophageal injury, and chest wall repair [14, 47]. Despite the broad spectrum of interventions reported with VATS, underlying thoracic pathology often influences the decision to perform a thoracotomy as opposed to a VATS [48]. The diaphragm can be evaluated from the abdomen using laparoscopy, which allows assessment for other intra-abdominal injuries.

VATS is performed in the lateral decubitus position as for a posterolateral thoracotomy. Single-lung ventilation is achieved with a double-lumen endotracheal tube. Appropriately positioned ports are essential to thoroughly examine the pleural space and perform an uncomplicated procedure. The first port is placed in the anterior axillary line inferiorly but above the costal margin and diaphragm, typically in the 7th intercostal interspace. Once the optimal site for the first port is chosen, the skin is incised, and dissection to the chest wall is carried out in a similar fashion to chest tube placement. The tract should be superior to the rib avoiding injury to the intercostal neurovascular bundle. The VATS port is passed through the tract. The camera is placed first, and subsequent ports are placed under direct visualization. In general, the remaining two working ports are placed on the

posterior axillary line adjacent to the scapular tip and on the anterior axillary line (Fig. 2.9). The working ports are placed on the potential posterolateral thoracotomy incision, as these

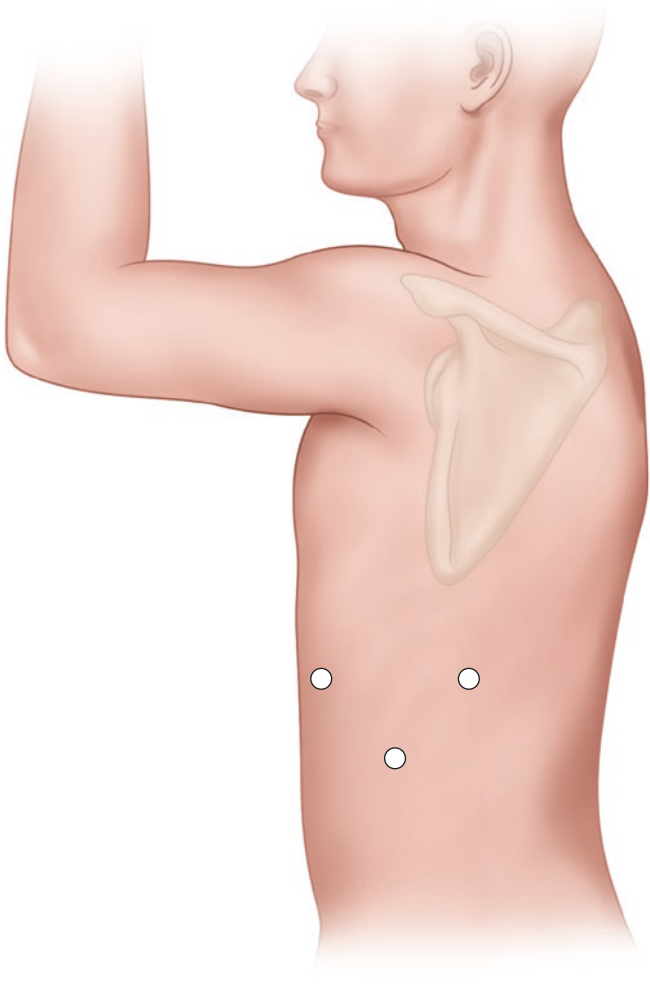

FIGURE 2.9 Initial port placement for VATS in trauma utilizes three ports

port incisions can be included in the incision if conversion to thoracotomy is required. At VATS completion, the port sites can be used for chest tube placement.

Outcomes

The majority of thoracic trauma is treated with nonoperative management and is successful in 85% of patients, particularly after blunt trauma [17]. However, procedural or operative intervention is sometimes required, often for severe or complicated injuries in hemodynamically unstable patients. Intervention is more often required in penetrating as opposed to blunt trauma [38]. Multiple procedures are occasionally necessary, as some patients will fail initial chest tube management and require subsequent operative intervention for hemothorax, empyema, or air leak [15].

Overall quantification of outcomes for patients with thoracic trauma can be difficult, given the frequency of polytrauma in this population. Specifically for operative thoracic interventions, chest tubes are typically removed in 3–5 days depending on the operative approach [52] and drain output (<200 cc/day). Overall length of stay varies widely from 6 to 30 days [52–54] and is longer in patients with concurrent injuries and associated morbidities [53]. The majority of patients have a good long-term functional outcome, with 60% of thoracotomy patients and 81% of VATS patients returning to usual activities after 2 years [52].

Technical success of VATS likely depends on the underlying injury. Overall failure rate of VATS ranges from 3.4% to 73% [15, 52, 54, 55]. The implications of technical failure of VATS extend beyond the need to convert to an open thoracotomy; it is also associated with empyema development [15]. Multiple factors predict a successful VATS, such as administration of antibiotics with first chest tube insertion, VATS done after 5 days [15], and smaller retained hemothorax volume [48]. Specifically for diaphragm evaluation, success with VATS has been excellent, with some reports of no missed injuries [56].

Adjunct Operative Maneuvers

After making the incision, important maneuvers in the management of thoracic injuries include aortic cross-clamping, pulmonary hilar cross-clamping and hilar vessel control, complete inflow occlusion (Schumacher maneuver), and control of the pulmonary arteries and pulmonary veins. The aorta should be encircled digitally after bluntly dissecting the vessel before it enters the abdominal cavity. The esophagus should be identified anterior to the aorta prior to placing a Crafoord-DeBakey aortic cross-clamp. The same instrument can be used to cross-clamp the pulmonary hilum if there is active bleeding or suspected injury in this area. The hilum can be approached with blunt and sharp dissection allowing for the digital encirclement of each structure. In the event that a cardiac injury is present in the area of the lateral atrium or cavo-atrial junction, the Schumacher maneuver allows for placement of the cross-clamp on the intrapericardial portion of the superior and inferior vena cava as they enter the right atrium. This allows immediate obstruction of preload, and the heart will empty immediately. Control of the pulmonary artery or vein can be approached extrapleural at the hilum or intrapericardial [57].

Complications

Thoracic trauma has an overall morbidity of 25.2%. Although overall chest injury is common, as few as 5–15% requires acute operative intervention. However, a significant number of thoracic operations are performed for complications that occur after thoracic injury. The most common complication is atelectasis, occurring in 14.6% of patients. The more deadly pulmonary complications are less frequent, with respiratory failure occurring in 5.5% of patients, acute respiratory distress syndrome (ARDS) in 3.2%, and bronchopleural fistula in 0.3% [38]. Empyema is a common complication in thoracic trauma [58]. Specifically, for patients with retained hemotho-

races, empyema develops in 26.8% [51]. Overall mortality with thoracic trauma is high, ranging from 9.3% to 20% [17, 38], and increases with associated injuries [38].

Morbidity varies by surgical approach and underlying pathology. Morbidity ranges from 14.6% for thoracotomy within the first day after injury [53], to 33% with evacuation of empyema, and to 11% with VATS evacuation of empyema [55]. Reoperation is common, with 2.5–12% of patients requiring reoperation [53, 54]. Similar to morbidity, mortality varied by surgical approach, with no mortality after VATS in many series [13, 14, 52, 54] versus 10.8–29% mortality after thoracotomy [44, 53, 59]. This difference in mortality between the operative approaches is likely due to a discrepancy in underlying injury severity, with the more seriously injured, unstable patients undergoing thoracotomy. Extent of resection was also associated with mortality, with pulmonary repair having the lowest mortality and lobectomy and pneumonectomy the highest [44, 53, 59]. Thoracic bleeding was the cause of 48–54% mortality after thoracotomy [44], and 70% of patients who died had an intraoperative death [53].

Conclusion

Overall, thoracic trauma is a morbid and mortal condition. Early diagnosis of injuries and immediate intervention can significantly improve patient outcomes. Evaluation and operative options for thoracic trauma continue to evolve, especially as our ability to identify injury burden preoperatively improves. Unstable patients should proceed emergently to the operating room for exploration. Stable patients can undergo a more detailed work-up, with identification of the injuries preoperatively, allowing for a more targeted intervention. Choice of incision ultimately depends on the injury pattern, patient hemodynamics, concurrent injuries, and surgeon preference.

Box 2.1 Indications for Acute Operative Intervention in the Thoracic Trauma Patient

1. Cardiopulmonary arrest meeting ED thoracotomy guidelines
2. Impending cardiopulmonary arrest upon arrival to the ED
3. Greater than 1500 cc of initial chest tube output or 150–200 cc/h over 2–4 h after chest tube placement [39]
4. Tension hemothorax
5. Persistent bloody chest tube output
6. Large retained hemothorax
7. Massive air leak from chest tube with concern for airway injury
8. Known or suspected esophageal, tracheobronchial, or vascular injury
9. Evidence on imaging of pericardial effusion

Box 2.2 Choice of Incision for Thoracic Intervention in the Hemodynamically Stable Patient

Sternotomy

1. Cardiac injury
2. Great vessel and ascending aorta injury

Right posterolateral thoracotomy

1. Azygos vein injury
2. Tracheal injury
3. Mid-esophageal injury
4. Isolated right pulmonary injury, hilar injury, retained hemothorax, or persistent pneumothorax

Left posterolateral thoracotomy

1. Descending aortic injury not amenable to endovascular therapy
2. Distal esophageal injury
3. Isolated left pulmonary injury, hilar injury, retained hemothorax, or persistent pneumothorax

VATS

1. Evaluation for diaphragmatic injury
2. Drainage of retained hemothorax

References

1. Weisse AB. Cardiac surgery: a century of progress. Tex Heart Inst J. 2011;38(5):486–90.
2. Wagner RB, Slivko B. Highlights of the history of nonpenetrating chest trauma. Surg Clin North Am. 1989;69(1):1–14.
3. Gordon RC. Daniel Hale Williams: pioneer Black surgeon and educator. J Investig Surg. 2005;18(3):105–6. Cover
4. Molnar TF, Haase J, Jeyasingham K, Rendeki MS. Changing dogmas: history of development in treatment modalities of traumatic pneumothorax, hemothorax, and posttraumatic empyema thoracis. Ann Thorac Surg. 2004;77(1):372–8.
5. Moynehan B. Surgical experiences in the present war. Surg Gynecol Obstet. 1917;25(6):583–612.
6. Kittle CF. The history of lobectomy and segmentectomy including sleeve resection. Chest Surg Clin N Am. 2000;10:105–30.
7. Martins Castello Branco J. Thoracoscopy as a method of exploration in penetrating injuries of the thorax. Dis Chest. 1946;12:330–5.
8. Jones JW, Kitahama A, Webb WR, McSwain N. Emergency thoracoscopy: a logical approach to chest trauma management. J Trauma Inj Infect Crit Care. 1981;21(4):280–4.

9. Jackson AM, Ferreira AA. Thoracoscopy as an aid to the diagnosis of diaphragmatic injury in penetrating wounds of the left lower chest: a preliminary report. Injury. 1976;7(3):213–7.
10. Freeman RK, Al-Dossari G, Hutcheson KA, Huber L, Jessen ME, Meyer DM, et al. Indications for using video-assisted thoracoscopic surgery to diagnose diaphragmatic injuries after penetrating chest trauma. Ann Thorac Surg. 2001;72(2):342–7.
11. Ahmed N, Chung R. Role of early thoracoscopy for management of penetrating wounds of the chest. Am Surg. 2010;76(11):1236–9.
12. Meyer DM, Jessen ME, Wait MA, Estrera AS. Early evacuation of traumatic retained hemothoraces using thoracoscopy: a prospective, randomized trial. Ann Thorac Surg. 1997;64(5):1396–400; discussion 1400–1.
13. Carrillo EH, Schmacht DC, Gable DR, Spain DA, Richardson JD. Thoracoscopy in the management of posttraumatic persistent pneumothorax. J Am Coll Surg. 1998;186(6):636–9; discussion 639–40.
14. Goodman M, Lewis J, Guitron J, Reed M, Pritts T, Starnes S. Video-assisted thoracoscopic surgery for acute thoracic trauma. J Emerg Trauma Shock. 2013;6(2):106–9.
15. Smith JW, Franklin GA, Harbrecht BG, Richardson JD. Early VATS for blunt chest trauma: a management technique underutilized by acute care surgeons. J Trauma Inj Infect Crit Care. 2011;71(1):102–5; discussion 105–7.
16. Villavicencio RT, Aucar JA, Wall MJ Jr. Analysis of thoracoscopy in trauma. Surg Endosc. 1999;13(1):3–9.
17. LoCicero J 3rd, Mattox KL. Epidemiology of chest trauma. Surg Clin North Am. 1989;69(1):15–9.
18. Oyetunji TA, Jackson HT, Obirieze AC, Moore D, Branche MJ, Greene WR, et al. Associated injuries in traumatic sternal fractures: a review of the National Trauma Data Bank. Am Surg. 2013;79(7):702–5.
19. Baldwin KD, Ohman-Strickland P, Mehta S, Hume E. Scapula fractures: a marker for concomitant injury? A retrospective review of data in the National Trauma Database. J Trauma Inj Infect Crit Care. 2008;65(2):430–5.
20. Richardson JD, McElvein RB, Trinkle JK. First rib fracture: a hallmark of severe trauma. Ann Surg. 1975;181(3):251–4.
21. Nagy KK, Lohmann C, Kim DO, Barrett J. Role of echocardiography in the diagnosis of occult penetrating cardiac injury. J Trauma Inj Infect Crit Care. 1995;38(6):859–62.

22. Demetriades D, van der Veen BW. Penetrating injuries of the heart: experience over two years in South Africa. J Trauma. 1983;23(12):1034–41.
23. Rodriguez RM, Hendey GW, Marek G, Dery RA, Bjoring A. A pilot study to derive clinical variables for selective chest radiography in blunt trauma patients. Ann Emerg Med. 2006;47(5):415–8.
24. Lopes JA, Frankel HL, Bokhari SJ, Bank M, Tandon M, Rabinovici R. The trauma bay chest radiograph in stable blunt-trauma patients: do we really need it? Am Surg. 2006;72(1):31–4.
25. Aukema TS, Beenen LF, Hietbrink F, Leenen LP. Initial assessment of chest X-ray in thoracic trauma patients: awareness of specific injuries. World J Radiol. 2012;4(2):48–52.
26. Kirkpatrick AW, Sirois M, Laupland KB, Liu D, Rowan K, Ball CG, et al. Hand-held thoracic sonography for detecting post-traumatic pneumothoraces: the extended focused assessment with sonography for trauma (EFAST). J Trauma. 2004;57(2):288–95.
27. Rozycki GS, Feliciano DV, Ochsner MG, Knudson MM, Hoyt DB, Davis F, et al. The role of ultrasound in patients with possible penetrating cardiac wounds: a prospective multicenter study. J Trauma Inj Infect Crit Care. 1999;46(4):543–51; discussion 551–2.
28. Ball CG, Williams BH, Wyrzykowski AD, Nicholas JM, Rozycki GS, Feliciano DV. A caveat to the performance of pericardial ultrasound in patients with penetrating cardiac wounds. J Trauma Inj Infect Crit Care. 2009;67(5):1123–4.
29. Press GM, Miller S. Utility of the cardiac component of FAST in blunt trauma. J Emerg Med. 2013;44(1):9–16.
30. Nandipati KC, Allamaneni S, Kakarla R, Wong A, Richards N, Satterfield J, et al. Extended focused assessment with sonography for trauma (EFAST) in the diagnosis of pneumothorax: experience at a community based level I trauma center. Injury. 2011;42(5):511–4.
31. Alrajab S, Youssef AM, Akkus NI, Caldito G. Pleural ultrasonography versus chest radiography for the diagnosis of pneumothorax: review of the literature and meta-analysis. Crit Care. 2013;17(5):R208.
32. Sauter TC, Hoess S, Lehmann B, Exadaktylos AK, Haider DG. Detection of pneumothoraces in patients with multiple blunt trauma: use and limitations of eFAST. Emerg Med J. 2017;34(9):568–72.

33. Mollberg NM, Wise SR, De Hoyos AL, Lin FJ, Merlotti G, Massad MG. Chest computed tomography for penetrating thoracic trauma after normal screening chest roentgenogram. Ann Thorac Surg. 2012;93(6):1830–5.
34. Exadaktylos AK, Sclabas G, Schmid SW, Schaller B, Zimmermann H. Do we really need routine computed tomographic scanning in the primary evaluation of blunt chest trauma in patients with "normal" chest radiograph? J Trauma. 2001;51(6):1173–6.
35. Self ML, Blake AM, Whitley M, Nadalo L, Dunn E. The benefit of routine thoracic, abdominal, and pelvic computed tomography to evaluate trauma patients with closed head injuries. Am J Surg. 2003;186(6):609–13; discussion 613–4.
36. Shanmuganathan K, Matsumoto J. Imaging of penetrating chest trauma. Radiol Clin N Am. 2006;44(2):225–38, viii.
37. Grossman MD, May AK, Schwab CW, Reilly PM, McMahon DJ, Rotondo M, et al. Determining anatomic injury with computed tomography in selected torso gunshot wounds. J Trauma Inj Infect Crit Care. 1998;45(3):446–56.
38. Demirhan R, Onan B, Oz K, Halezeroglu S. Comprehensive analysis of 4205 patients with chest trauma: a 10-year experience. Interact Cardiovasc Thorac Surg. 2009;9(3):450–3.
39. Mowery NT, Gunter OL, Collier BR, Diaz JJ Jr, Haut E, Hildreth A, et al. Practice management guidelines for management of hemothorax and occult pneumothorax. J Trauma Inj Infect Crit Care. 2011;70(2):510–8.
40. Morales CH, Salinas CM, Henao CA, Patino PA, Munoz CM. Thoracoscopic pericardial window and penetrating cardiac trauma. J Trauma Inj Infect Crit Care. 1997;42(2):273–5.
41. Duncan AO, Scalea TM, Sclafani SJ, Phillips TF, Bryan D, Atweh NA, et al. Evaluation of occult cardiac injuries using subxiphoid pericardial window. J Trauma Inj Infect Crit Care. 1989;29(7):955–9; discussion 959–60.
42. Demetriades D. Cardiac wounds. Experience with 70 patients. Ann Surg. 1986;203(3):315–7.
43. O'Connor JV, Scalea TM. Penetrating thoracic great vessel injury: impact of admission hemodynamics and preoperative imaging. J Trauma. 2010;68(4):834–7.
44. Karmy-Jones R, Jurkovich GJ, Nathens AB, Shatz DV, Brundage S, Wall MJ Jr, et al. Timing of urgent thoracotomy for hemorrhage after trauma: a multicenter study. Arch Surg. 2001;136(5):513–8.

45. Halonen-Watras J, O'Connor J, Scalea T. Traumatic pneumonectomy: a viable option for patients in extremis. Am Surg. 2011;77(4):493–7.
46. Macchiarini P, Ladurie FL, Cerrina J, Fadel E, Chapelier A, Dartevelle P. Clamshell or sternotomy for double lung or heart-lung transplantation? Eur J Cardiothorac Surg. 1999;15(3):333–9.
47. Milanchi S, Makey I, McKenna R, Margulies DR. Video-assisted thoracoscopic surgery in the management of penetrating and blunt thoracic trauma. J Minim Access Surg. 2009;5(3):63–6.
48. DuBose J, Inaba K, Demetriades D, Scalea TM, O'Connor J, Menaker J, et al. Management of post-traumatic retained hemothorax: a prospective, observational, multicenter AAST study. J Trauma Acute Care Surg. 2012;72(1):11–22.
49. Heniford BT, Carrillo EH, Spain DA, Sosa JL, Fulton RL, Richardson JD. The role of thoracoscopy in the management of retained thoracic collections after trauma. Ann Thorac Surg. 1997;63(4):940–3.
50. Oosthuizen GV, Clarke DL, Laing GL, Bruce J, Kong VY, Van Staden N, et al. Introducing video-assisted thoracoscopy for trauma into a South African township hospital. World J Surg. 2013;37(7):1652–5.
51. DuBose J, Inaba K, Okoye O, Demetriades D, Scalea T, O'Connor J, et al. Development of posttraumatic empyema in patients with retained hemothorax: results of a prospective, observational AAST study. J Trauma Acute Care Surg. 2012;73(3):752–7.
52. Ben-Nun A, Orlovsky M, Best LA. Video-assisted thoracoscopic surgery in the treatment of chest trauma: long-term benefit. Ann Thorac Surg. 2007;83(2):383–7.
53. Onat S, Ulku R, Avci A, Ates G, Ozcelik C. Urgent thoracotomy for penetrating chest trauma: analysis of 158 patients of a single center. Injury. 2011;42(9):900–4.
54. Morrison CA, Lee TC, Wall MJ Jr, Carrick MM. Use of a trauma service clinical pathway to improve patient outcomes for retained traumatic hemothorax. World J Surg. 2009;33(9):1851–6.
55. Scherer LA, Battistella FD, Owings JT, Aguilar MM. Video-assisted thoracic surgery in the treatment of posttraumatic empyema. Arch Surg. 1998;133(6):637–41; discussion 641–2.
56. Ochsner MG, Rozycki GS, Lucente F, Wherry DC, Champion HR. Prospective evaluation of thoracoscopy for diagnosing diaphragmatic injury in thoracoabdominal trauma: a preliminary report. J Trauma. 1993;34(5):704–9; discussion 709–10.

57. Asensio JA, Trunkey DD, editors. Current therapy of trauma and surgical critical care. 2nd ed. Philadelphia: Elsevier; 2016.
58. O'Connor JV, Chi A, Joshi M, DuBose J, Scalea TM. Post-traumatic empyema: aetiology, surgery and outcome in 125 consecutive patients. Injury. 2013;44(9):1153–8.
59. Thompson DA, Rowlands BJ, Walker WE, Kuykendall RC, Miller PW, Fischer RP. Urgent thoracotomy for pulmonary or tracheobronchial injury. J Trauma. 1988;28(3):276–80.

Chapter 3
Emergency Department Thoracotomy

Jay Menaker

Introduction of the Problem

Emergency department thoracotomy (EDT), sometimes described as the "last chance" for survival, is perhaps the most dramatic procedure performed on injured patients [1]. Thus, there is great interest in attaining the skills necessary to perform it. Rapid decision-making and skilled performance are essential to maximize survival. However, EDT remains one of the most controversial procedures, as there are no absolute clear indications for its use. Even if applied to the patients most likely to live, survival is far less than 50%.

History of Thoracotomy

Schiff, in 1874, was the first to suggest thoracotomy as a resuscitative procedure in order to perform open cardiac massage [2]. In 1882, Block introduced the concept of suture repair

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when he performed experiments on rabbits [3]. Although initially shunned by his colleagues [4, 5], the practice continued, and in 1897, Rehn reported the first successful left thoracotomy for a cardiac injury in a human [6]. The first half of the twentieth century saw widespread use of EDT. This continued until 1960 when Kouwenhoven and colleagues introduced the practice of closed cardiac massage for cardiac arrest [7], essentially eliminating the need for open cardiac massage. During the 1960s, Beall and colleagues renewed the interest in EDT with multiple publications supporting its use for patients in extremis following penetrating chest trauma [8–10].

As interest in performing a resuscitative thoracotomy increased, so did the controversy surrounding it. As most hospitals do not see a sufficient number of patients who are candidates for EDT, the concern for adequate training for both emergency medicine and surgical residents arose. However, the learning to perform this lifesaving bedside procedure must be balanced against the potential abuse and in inappropriate patient selection. As a result, ethical issues of performing an EDT on patients that are clearly non-survivable in order to "practice" became a concern.

Indications

One of the biggest challenges in creating an algorithm of indications for EDT is that most published reports are retrospective in nature, as a prospective randomized trial would not be feasible. Most cite mechanism of injury, location of injury, and presences of signs of life (SOL)/vital signs as the key determinants of whether or not to perform an EDT. Signs of life/vital signs include pupillary response, spontaneous ventilation, presence of a carotid pulse, measurable or palpable blood pressure, extremity movement, or cardiac electrical activity [11–13]. In 2000, Rhee and colleagues reviewed the literature on EDT from the previous 25 years [14]. The authors made three general recommendations for performing an EDT:

1. *Indications*: "patients with penetrating thoracic injuries with SOL in the field who do not respond to fluids and are losing vital signs in the resuscitation bay"
2. *Relative indications*: "patients with penetrating abdominal injury with at least one clear SOL in the field. Blunt trauma patients who lose SOL in the hospital or immediately before"
3. *Contraindications*: "patients without any SOL in the field either from penetrating or blunt trauma" [14]

In 2001, the American College of Surgeons—Committee on Trauma established guidelines for performing an EDT [15]. The committee stated that an EDT is best applied to those with penetrating cardiac injuries arriving to the hospital with SOL. Additionally, they recommend that EDT should be performed for noncardiac thoracic and exsanguinating abdominal vascular injuries; however, both these patient populations have low survival rates. Furthermore, blunt trauma patients who suffer a cardiac arrest should rarely have an EDT performed due to low survival and poor neurological outcome. Only patients who arrive to the hospital with vital signs and then have a witnessed cardiac arrest should have an EDT following blunt trauma.

In 2004 Powell and colleagues published indications for EDT based on 26 years of consecutive data [12]. The authors concluded that EDT is indicated when there is penetrating trauma and <15 minutes of prehospital cardiopulmonary resuscitation (CPR); asystole if pericardial tamponade is the cause; or witnessed blunt trauma and <5 minutes of prehospital CPR. Furthermore, they concluded that EDT was contraindicated in patients with penetrating trauma and >15 minutes of prehospital CPR and no SOL, asystole without pericardial tamponade, and blunt trauma with >5 minutes of prehospital CPR and no SOL [12].

More recently both the Eastern Association for the Surgery of Trauma (EAST) and the Western Trauma Association (WTA) have both published guidelines for patient selection for an emergency department thoracotomy [11, 16]. Although they differ slightly in the specifics, the

theme remains similar between the two guidelines, as well as to those suggested by Rhee and colleagues in 2000 [14]. It is recommended that patients with penetrating thoracic injury with or without signs of life should have an EDT performed. Those with extra-thoracic penetrating wound have a conditional recommendation, and those who present pulseless following blunt trauma should not have an EDT performed.

Procedural Technique

EDT should be performed as part of the initial resuscitation process in patients that present without any vital signs/SOL. It should occur simultaneously with securing the airway, establishing adequate intravenous access and volume resuscitation. For those patients that arrive with any vital signs/SOL, an ultrasound to evaluate for pericardial fluid (tamponade) should be performed immediately. If hemopericardium is identified, and patients have any vital signs/SOL, they should be immediately transported to the operating room (OR) prior to intubation. When cardiac tamponade is present, cardiovascular collapse—from the loss of vasomotor tone in preload-dependent patients—on induction of anesthesia is a significant risk. Thus, it is preferable to intubate these patients in the OR after the patient is prepped and draped. However, if the patient is marginal and/or the OR is a distance away, intubation and EDT in the resuscitation bay are probably wisest.

Prior to performing an EDT, the clinical team should, if possible, notify the operating room, anesthesia staff, and the blood bank to initiate a massive transfusion. Additionally, universal precautions including protective eyewear for all team members are essential. All emergency departments/resuscitation bays should have an established EDT tray that is always available with all necessary equipment (Fig. 3.1). A left anterolateral thoracotomy is the incision of choice when performing EDT (Fig. 3.2). The advantages of the incision include rapid access, ability to perform in a supine patient,

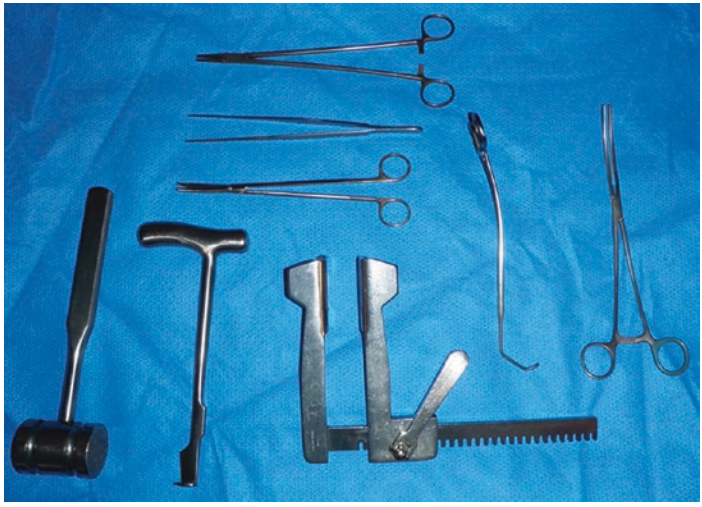

FIGURE 3.1 Basic components of an emergency thoracotomy tray. Bottom left: Lebsche knife and mallet for crossing sternum. Bottom center: Finochietto retractor. Bottom right: atraumatic vascular clamps, a Satinsky clamp on the left, and a DeBakey aortic occlusion clamp on the right. Top center: long-handled needle driver, tissue forceps, and Metzenbaum scissors. Not illustrated: scalpel with #10 or #20 blade and Mayo scissors

and ability to be extended to the right hemithorax (clamshell) or laparotomy if needed.

The patient should be positioned supine with the left arm extended above the head (Fig. 3.3), and the entire region should be prepped with antiseptic solution prior to the incision. The incision should begin to the right of the sternal border (Fig. 3.4) and progress along the fifth intercostal space toward the left axilla in a curved fashion (Fig. 3.5). This will facilitate sternal transection and extension to a clamshell thoracotomy if needed. Anatomically, the fifth intercostal space corresponds to the inferior border of the pectoralis major and is just below the patient's nipple. In women, the inframammary fold should be the anatomical landmark. The breast may need to be retracted superiorly in order to allow

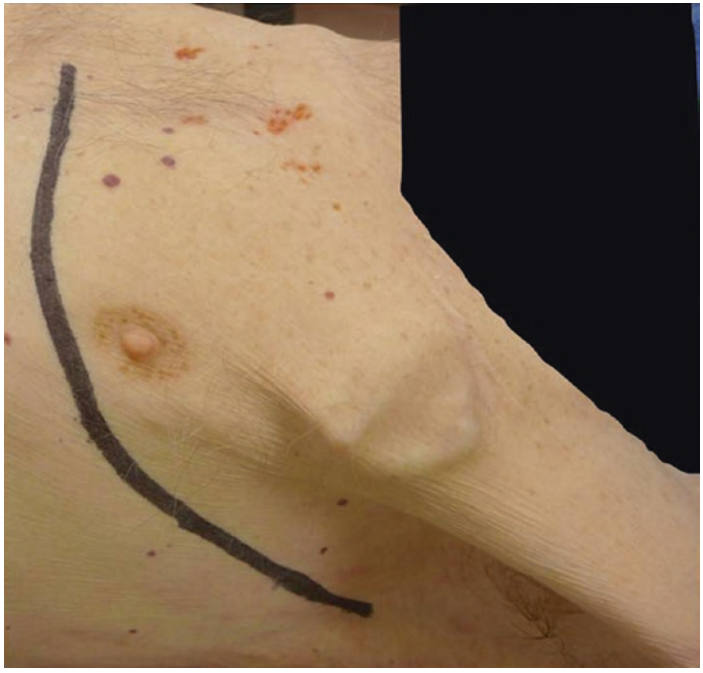

FIGURE 3.2 A left anterolateral thoracotomy is the incision of choice for an EDT

better access to the proper landmarks. Once at the level of the nipple, begin to curve toward the left axilla, and follow the rib cage (Fig. 3.6). Transverse incisions should be avoided.

The skin, subcutaneous tissue, and chest wall muscle are quickly divided. Intercostal muscles can then be cut with a scalpel or curved scissors. This should be done on the superior side of the rib in order to avoid injuring the intercostal neurovascular bundle. Careful attention by the providers is essential to avoid injuring the heart and lungs as the chest cavity is entered. Once the chest cavity has been entered, a Finochietto retractor is placed, with the handle toward the axilla (Fig. 3.7), and the ribs are spread. To facilitate exploring the mediastinum and right chest, the incision can be extended

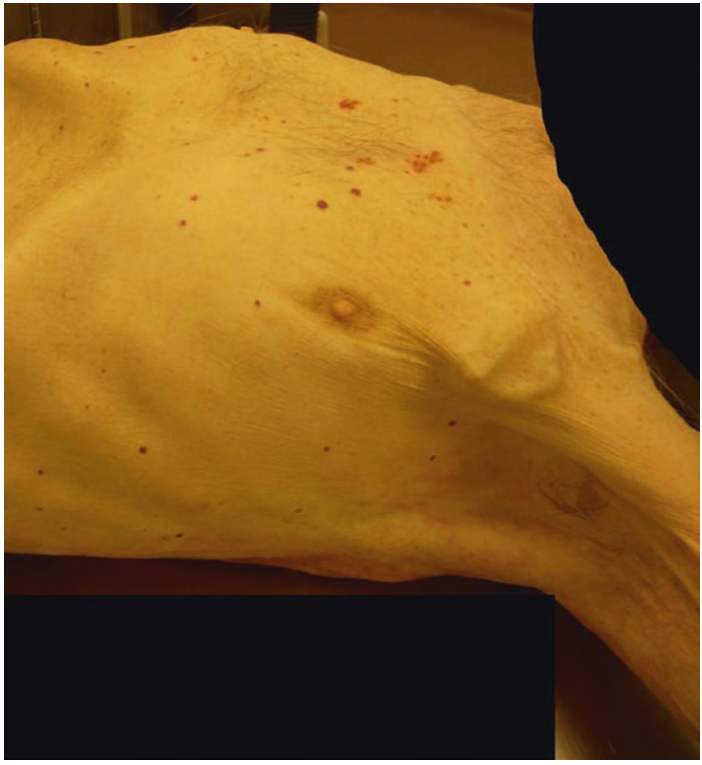

FIGURE 3.3 Patient should be in a supine position with left arm above head

as a clamshell. This is accomplished by dividing the sternum transversely using a Lebsche knife (Fig. 3.8) and extending the incision as a right anterolateral thoracotomy (Fig. 3.9). If the sternum is divided and perfusion is restored, the internal mammary arteries will need to be ligated.

We routinely perform a clamshell thoracotomy when doing an EDT. We believe the additional exposure is well worth any added morbidity from the additional incision. This allows exploration and control of any injuries in the right hemithorax. Visualizing and repairing any structures, particularly the heart, is much easier with both chest cavities open.

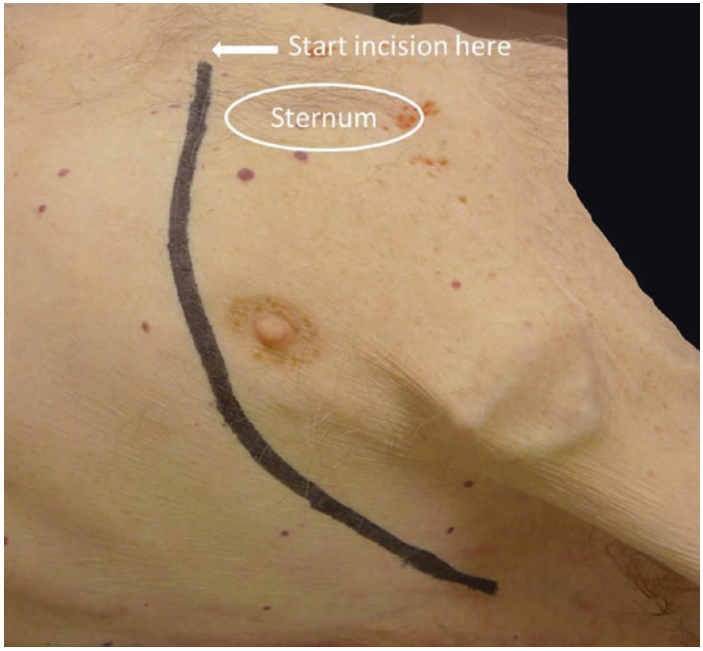

FIGURE 3.4 Begin incision to the right of the sternum

Exposure can be increased, when needed by using two Finochietto retractors. We place one at the level of the sternal edges and the other laterally in the left chest.

Pericardiotomy

The pericardium should be opened following all traumatic arrest. Cardiac tamponade should not be ruled out visually, and wide incision of the pericardium is required. If the pericardial sac is not tense, one may pick up the pericardium with toothed forceps and make an incision from the apex to the aortic root. The incision should be parallel and anterior to the phrenic nerve (Fig. 3.10). If the pericardial sac is filled with blood, a knife may be needed to make the initial incision and

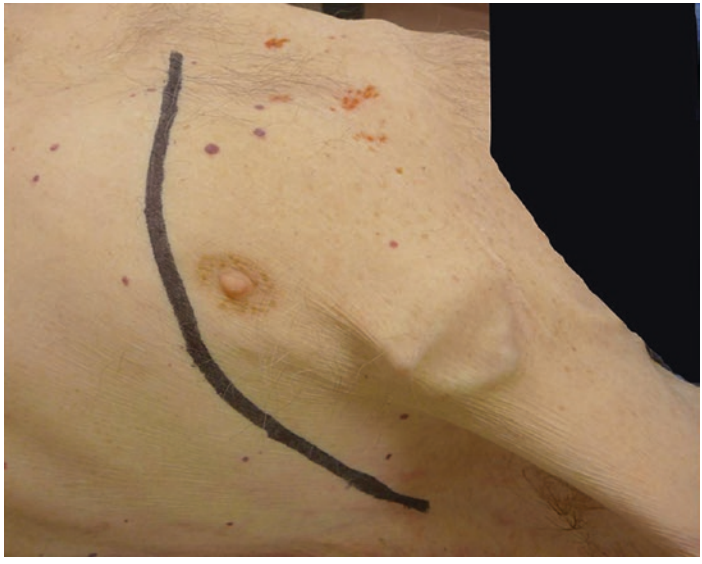

FIGURE 3.5 Incision along the fifth intercostal rib space toward the left axilla in a curved line

then the pericardium incised as described previously. Once the pericardium is widely opened, the heart should be delivered and all blood clots removed allowing full inspection of the myocardium. Any bleeding should initially be controlled with digital pressure while preparing for a more definitive repair. Any injury should be rapidly repaired by using either temporary sutures or staples. Although historically thought to be a valuable tool, the use of a Foley catheter as a temporizing measure for cardiac injuries may actually worsen the injury. We virtually never use this manuever. An alternative option for repair includes using sequentially stacked intestinal Allis clamps, similar to what has been described for retroperitoneal venous injury [17]. Atrial and great vessel injuries may be controlled with a partial occlusion (side-biting) vascular clamp. One should be careful when suturing the ventricle not to include any of the coronary vessels in the repair causing ischemia.

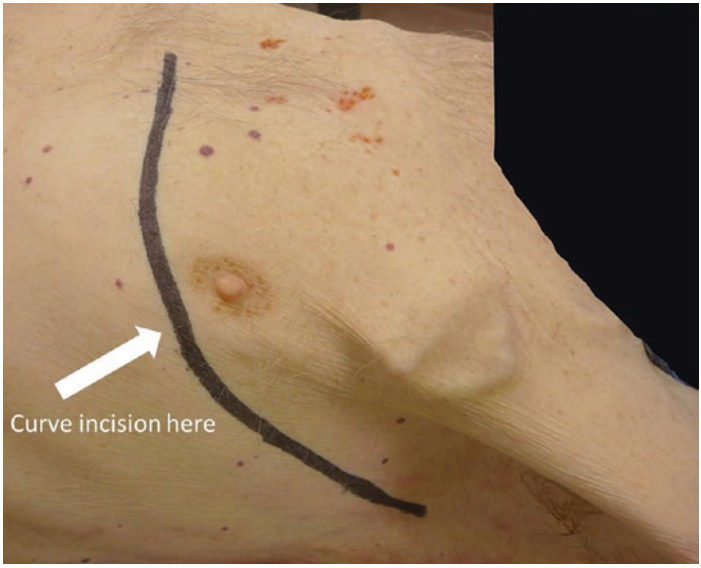

FIGURE 3.6 Begin curving incision at the level of the nipple

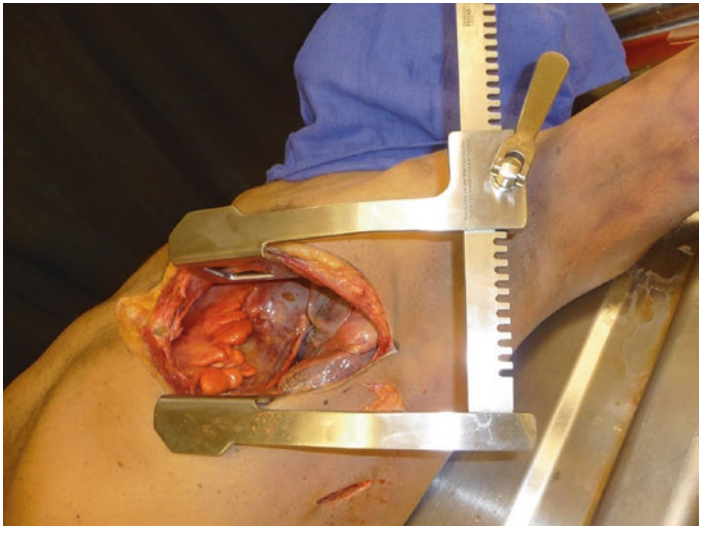

FIGURE 3.7 Retractor with handle down (away from midline)

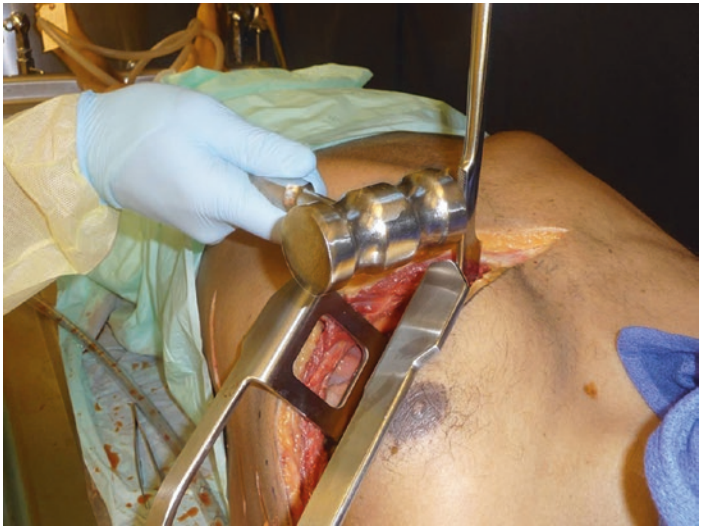

FIGURE 3.8 Lebsche knife to extend into right hemithorax

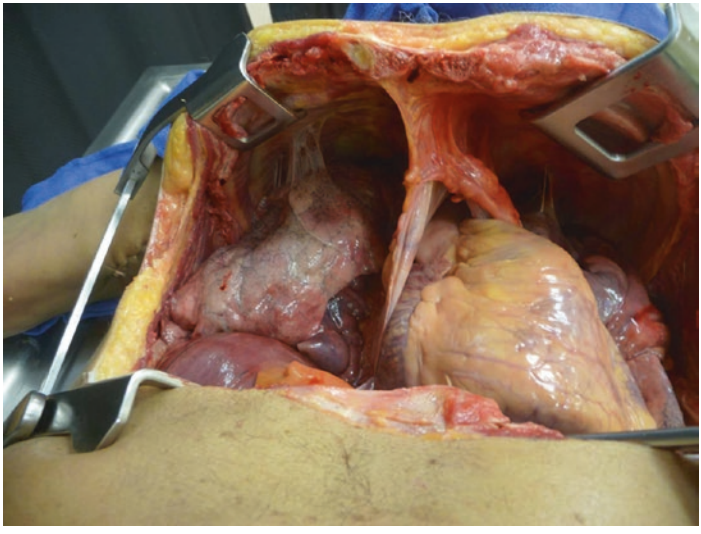

FIGURE 3.9 Clamshell thoracotomy

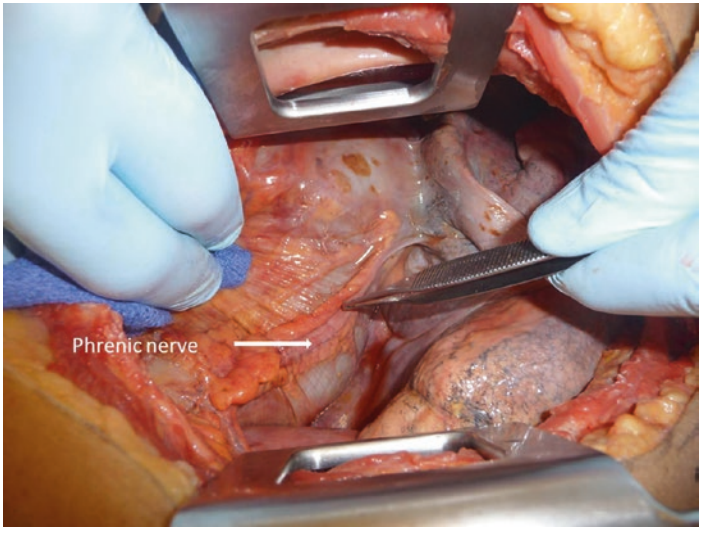

Figure 3.10 Pericardiotomy should be medial to phrenic nerve

Aortic Cross-Clamping

Aortic cross-clamping is often described as a simple procedure, but, in reality, accessing the aorta is very challenging, particularly in the hypovolemic, hypotensive patient in whom the aorta is collapsed. The aorta, which lies anterior to the vertebrae, is best exposed by retracting the lung anteriorly and superiorly. At this point, the inferior pulmonary ligament can be taken down for better exposure, but one must be diligent as to not cause injury to the inferior pulmonary veins. Ideally one should dissect under direct vision; however, this is often not possible. When one cannot directly incise the mediastinal pleura and bluntly separate the aorta and esophagus, blinded blunt dissection with one's thumb and fingertips can be used. Once the aorta is separated from the esophagus, the clinician's left hand encircles the aorta, and a clamp is placed with the right hand (Fig. 3.11). Placing a nasogastric tube may help differentiate the aorta from the esophagus and facilitate proper clamp placement. If isolating the aorta still remains a

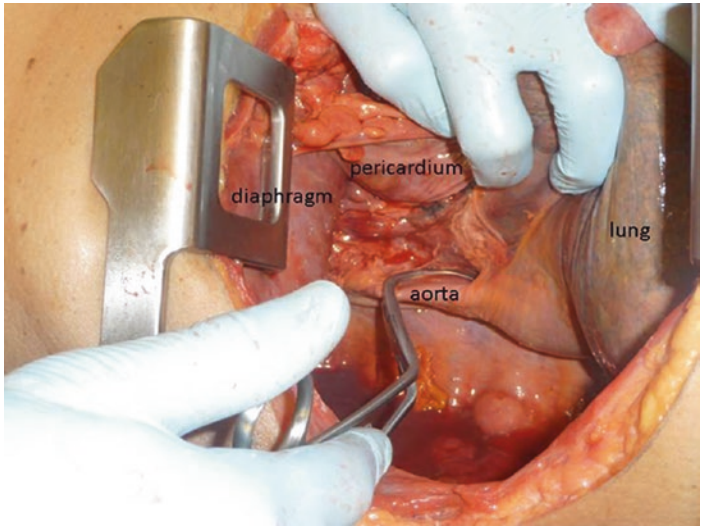

FIGURE 3.11 Cross-clamping the aorta

challenge, then simply providing digital pressure against the spine can be performed as a temporizing measure.

The goal of aortic cross-clamping is to increase blood flow to the brain and coronary arteries. However, total aortic occlusion can have significant negative effects as well. Distal ischemia can exacerbate tissue acidosis and oxygen debt, both of which may affect multiple organ failure [18]. The safe duration of aortic cross-clamping remains unknown. Extrapolating data from traumatic aortic injuries, more 30 min of cross-clamp time significantly increases complication rates [19, 20]. Furthermore, removal of the aortic cross-clamp can cause rapid reperfusion of previously ischemic tissue resulting in worsening acidosis. This "de-clamp shock" can cause hemodynamic instability and even cardiac arrest [21, 22, 23].

Although taught as a key component of the EDT, cross-clamping of the aorta is not routinely performed at this author's institution. Even though increased cardiac and brain perfusion is desired, placing an aortic cross-clamp significantly

increases the afterload on an already failing heart. Additionally, when the aorta is crossed-clamped, aggressive volume resuscitation can lead to further cardiac failure due to acute ventricular dilatation.

Cardiac Massage

Following an EDT, if cardiac arrest occurs or persists, bimanual internal cardiac massage should begin immediately (Fig. 3.12). The bimanual technique is preferred to a one-hand technique (Fig. 3.13) because it is more effective and has less potential for causing damage to the heart [13, 22]. A "hinged clapping" motion from the palms to the fingertips compressing the heart from the apex to the aortic root is preferred [13, 22].

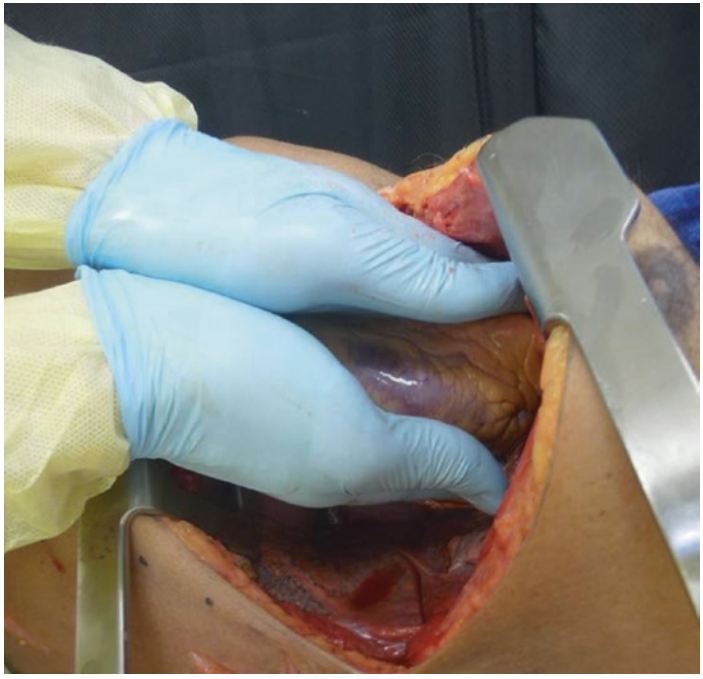

FIGURE 3.12 Two-hand internal cardiac massage

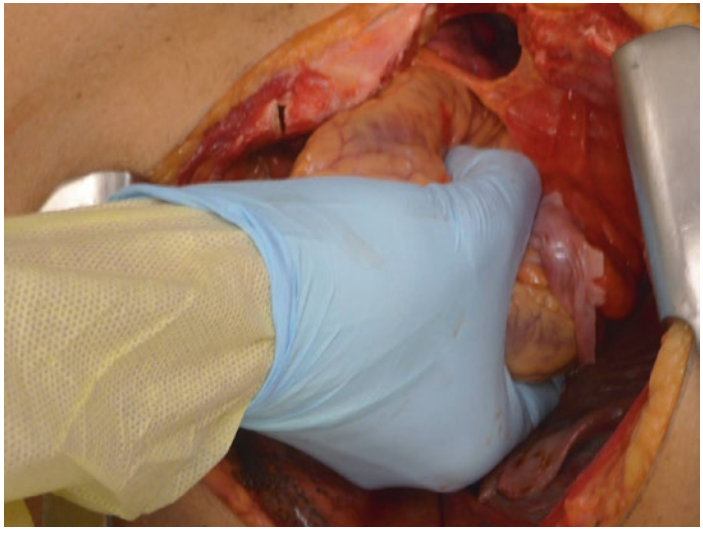

FIGURE 3.13 One-hand internal cardiac massage

Controlling Noncardiac Hemorrhage

Injuries to the great vessels, lung parenchyma, or central pulmonary vessels are other possible causes of massive hemorrhage. Great vessel injuries are typically lethal; however, if patients arrive to the ED in a timely manner and an EDT is performed quickly, these injuries are best initially managed with digital pressure, while resuscitation continues, and the patient is transported to the OR. Adequate repair of a mediastinal great vessel is very difficult in the emergency department/resuscitation bay and is best achieved in the operating room.

Lung parenchyma injuries are usually managed with tube thoracostomy alone, and an EDT is typically not needed. In cases of massive pulmonary parenchymal bleeding, simply applying a vascular clamp proximal to the source of bleeding should be sufficient. More extensive dissection and definitive hemorrhage control should be performed in the OR. In contrast to parenchymal injuries, pulmonary hilar injuries often

present with massive hemorrhage requiring rapid control. Options for hemorrhage control include digital pressure, placing a vascular clamp over the hilum, or twisting the lung on its hilum.

Outcomes

Much of the debate over the utility of the EDT centers on the patient selection and outcome of the procedure. Simply measuring "survival" may not be the best endpoint. Survival with significant neurologic deficit should not be considered a success. Outcomes reported in the literature vary widely but in general are associated with mechanism of injury, pattern of injury, and the presence of SOL. Very simply, an isolated stab wound has better survival than multiple stab wounds. Stab wounds tend to have better outcomes than gunshot wounds, and blunt injuries typically have the lowest survival.

The 2000 review by Rhee and colleagues looking at 25 years of EDT and survival rates remains one of the most comprehensive reviews of the subject [14]. The authors only included thoracotomies performed in the ED or resuscitation bay and excluded those performed in the OR. More than 4600 patients were included in the study. The overall survival following EDT was 7.4%, ranging from 1.8% to 27.5%. When survival was analyzed by mechanism of injury, penetrating injuries had a rate of 8.8%, while blunt trauma had just 1.4%. When stratifying penetrating injuries, the authors showed survival rate for stab wounds was 16.8%, while that for gunshot wounds was 4.3%. When survival was evaluated from the perspective of the major site of injury, "cardiac" had the highest rate, 19.4%, as compared to noncardiac thoracic, abdominal, or multiple sites of injury (10.7%, 4.5%, 0.7%, respectively). Those with SOL in the hospital had an almost fivefold increase in survival rates as compared to those with no SOL on arrival (11.5% vs. 2.6%). Those with SOL during transport had a sevenfold increased rate of survival than those with no SOL in the field (8.9% vs. 1.2%). Finally, 92.4%

of patients discharged from the hospital were reported to have normal neurological outcome.

In the 2001 American College of Surgeons—Committee of Trauma practice management guidelines, 42 studies were reviewed for outcome following EDT [15]. These studies included all but one of the studies in Rhee and colleagues 2000 review, and as one would expect, the results were essentially identical. Overall survival following EDT was 7.83% (7.4% Rhee [14]). Following penetrating injury, the survival rate was 11.16% (8.8% Rhee [14]), while that for blunt was 1.6% (1.4% Rhee [14]).

More recently there has been a trend toward a more selective rather than obligatory use of EDT [16]. As a result, outcomes reported have changed over time. According to the 2012 WTA algorithm for resuscitative thoracotomy, the success rate approximates 35% for those with penetrating cardiac injury that arrive in shock [16]. Blunt trauma patients that arrive in shock have 2% survival rate, while those who arrive with no SOL following blunt trauma have less than a 1% rate of survival. In 2015, the EAST practice management guidelines analyzed over 10,000 who underwent EDT [11]. Patients presenting pulseless with any SOL after penetrating thoracic injury had 21% survival, but only 12% were neurologically intact. Those without SOL on arrival had 4% neurologically intact survival rate. Neurologically intact outcomes following EDT in pulseless bunt trauma with and without SOL were 2.4% and 0.1%, respectively.

Complications

As is with any surgical or invasive procedure, there exist numerous potential inherent complications to performing an EDT. First and foremost, injury to a member of the care provider team is possible. While this is often a chaotic situation, all providers should be careful while using needles and scalpels. That is also why universal precautions including protective gown, mask, and eyewear are imperative for all team

members. If there is any question regarding possible needle stick, body fluid exposure, or any break in skin barrier, the hospital's infection control or "STIK" hotline (may vary from one hospital to another) should be contacted immediately.

Technically, there is potential for damage to other structures while performing an EDT including the ribs, intercostal vessels, lacerating the lung parenchyma, phrenic nerve, and even the heart including the coronary arteries. If a clamshell thoracotomy is performed, one must remember to ligate the internal mammary arteries if circulation/perfusion is restored. Post-procedural complications include infection, bleeding, and postpericardiotomy syndrome.

The Future

It is clear from the literature that patients without a direct penetrating cardiac/thoracic injury do poorly after an EDT. In these patients, EDT is performed to allow for crossing clamping of the aorta and direct open cardiac massage. Once believed to be superior, studies have demonstrated that open cardiac massage following traumatic arrest does not lead to better outcomes than traditional closed-chest compressions [24, 25].

Cross-clamping the thoracic aorta for non-thoracic injury, although effective, may not be the best decision. Opening the thoracic cavity can cause additional bleeding in what is most likely an already coagulopathic patient. Additionally, the open chest cavity allows for a significant amount heat loss from the body worsening potential hypothermia. Thus, for patients who present in extremis with non-thoracic cavity bleeding (abdomen, pelvis), alternative techniques to EDT should be implemented [26]. Recently, the use of the resuscitative endovascular balloon occlusion of the aorta (REBOA) has become prevalent in many trauma centers. It can be very effective in hemorrhage control for non-compressible torso and pelvic bleeding. However, specialized training is required in order to perform the procedure correctly. As with any inva-

sive procedure, complications occur that must be managed appropriately. See Chap. 20.

Conclusion

The emergency department thoracotomy remains an important procedure and, when rapidly performed in the appropriate patient population, saves lives. A number of guidelines have been published outlying indications for EDT. Patients with a stab wound to the heart have the highest rates of survival, while those with blunt trauma have the lowest. As we go forward, less invasive techniques are emerging as an alternative for patients with low rates of survival following EDT, thus providing an opportunity to improve survival following injury.

References

1. Seamon MJ, Fisher CA, Gaughan JP, Kulp H, Dempsey DT, Goldberg AJ. Emergency department thoracotomy: survival of the least expected. World J Surg. 2008;32:604–12.
2. Hermreck AS. The history of cardiopulmonary resuscitation. Am J Surg. 1988;156:430.
3. Block: Verhandlungen der Deutschen Gesellschaft für Chirurgie, Elften Congress. Berlin, 1882 part 1:108.
4. Jeger E. Die Chirurgie der Blutgefässe und des. Herzens. 1913:295.
5. Riedinger F. Verletzungen und Chirurgische Krankheiten des thorax und seines. Inhaltes. 1888;42:189.
6. Rehn L. Ueber Penetrierende Herzwunden und Herznaht. Arch Klin Chir. 1897;55:315–9.
7. Kouwenhoven WB, Jude JR, Knicekerbocker GG. Closed-chest cardiac massage. JAMA. 1960;173:1064–7.
8. Beall AC Jr, Ochsner JL, Morris GC Jr, Cooley DA, DeBakey ME. Penetrating wounds of the heart. J Trauma. 1961;1:195–207.
9. Beall AC Jr, Diethrich EB, Crawford HW, Cooley DA, De Bakey ME. Surgical management of penetrating cardiac injuries. Am J Surg. 1966;12:686–92.

10. Beall AC Jr, Diethrich EB, Cooley DA, DeBakey ME. Surgical management of penetrating cardiovascular trauma. South Med J. 1967;60:698–704.
11. Seamon MJ, Haut ER, Van Arendonk K, Barbosa RR, Chiu WC, Dente CJ, et al. An evidence-based approach to patient selection for emergency department thoracotomy: a practice management guideline from the Eastern Association for the Surgery of Trauma. J Trauma Acute Care Surg. 2015;79:159–73.
12. Powell DW, Moore EE, Cothren CC, Ciesla DJ, Burch JM, Moore JB, et al. Is emergency department resuscitative thoracotomy futile care for the critically injured patient requiring prehospital cardiopulmonary resuscitation? J Am Coll Surg. 2004;199:211–5.
13. Spoerke NJ, Trunkey DD. Emergency department thoracotomy. In: Cameron JL, Cameron AM, editors. Current surgical therapy. 10th ed. Philadelphia: Elsevier Saunders; 2011.
14. Rhee PM, Acosta J, Bridgeman A, Wang D, Jordan M, Rich N. Survival after emergency department thoracotomy: review of published data from the past 25 years. J Am Coll Surg. 2000;190:288–98.
15. Working Group, Ad Hoc Subcommittee on Outcomes, American College of Surgeons, Committee on Trauma. Practice management guidelines for emergency department thoracotomy. J Am Coll Surg. 2001;193:303–9.
16. Burlew CC, Moore EE, Moore FA, Coimbra R, McIntyre RC Jr, Davis JW, et al. Western Trauma Association critical decisions in trauma: resuscitative thoracotomy. J Trauma Acute Care Surg. 2012;73:1359–63.
17. Henry SM, Duncan AO, Scalea TM. Intestinal Allis clamps as temporary vascular control for major retroperitoneal venous injury. J Trauma. 2001;51:170–2.
18. Oyama M, McNamara JJ, Suehiro GT, Suehiro A, Sue-Ako K. The effects of thoracic aortic cross-clamping and declamping on visceral organ blood flow. Ann Surg. 1983;197:459–63.
19. Katz NM, Blackstone EH, Kirklin JW, Karp RB. Incremental risk factors for spinal cord injury following operation for acute traumatic aortic transection. J Thorac Cardiovasc Surg. 1981;81:669–74.
20. Fabian TC, Richardson JD, Croce MA, Smith JS Jr, Rodman G Jr, Kearney PA, et al. Prospective study of blunt aortic injury: multicenter trial of the American Association for the surgery of trauma. J Trauma. 1997;42:374–80.

21. Kralovich KA, Morris DC, Dereczyk BE, Simonetti V, Williams M, River EP, et al. Hemodynamic effects of aortic occlusion during hemorrhagic shock and cardiac arrest. J Trauma. 1997;42:1023–8.
22. Cothren CC, Moore EE. Emergency department thoracotomy. In: Feliciano DV, Mattox KL, Moore EE, editors. Trauma. 6th ed. New York: McGraw-Hill; 2008.
23. Perry MO. The hemodynamics of temporary abdominal aortic occlusion. Ann Surg. 1968;168:193–200.
24. Bradley MJ, Bonds BW, Chang L, Yang S, Hu P, Li HC, et al. Open chest cardiac massage offers no benefit over closed chest compressions in patients with traumatic cardiac arrest. J Trauma Acute Care Surg. 2016;81:849–54.
25. Endo A, Shiraishi A, Otomo Y, Tomita M, Matsui H, Murata K. Open-chest versus closed-chest cardiopulmonary resuscitation in blunt trauma: analysis of a nationwide trauma registry. Crit Care. 2017;21:169.
26. Teeter W, Romagnoli A, Wasicek P, Hu P, Yang S, Stein D, et al. Resuscitative endovascular balloon occlusion of the aorta improves cardiac compression fraction versus resuscitative thoracotomy in patients in traumatic arrest. Ann Emerg Med. 2018;72:354–60.

Chapter 4
Indications and Techniques for Trauma Laparotomy

Paulesh K. Shah and William C. Chiu

History of Care of Penetrating Abdominal Trauma

Prior to World War I, penetrating abdominal trauma was managed nonoperatively, with accompanying high mortality rates. Toward the end of World War I and afterward, a policy of routine abdominal exploration for penetrating trauma was adopted. Mortality decreased over the following decades with earlier operative intervention, faster transport times, better understanding of fluid resuscitation, use of blood transfusion, and the advent of antibiotics. The

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concept of selective nonoperative management of penetrating abdominal trauma was revisited in the 1960s and later. Several investigators observed that many patients who sustained abdominal stab wounds ultimately were diagnosed with no significant injury at laparotomy [1–4]. These findings were later extended to gunshot wounds as well, although the incidence of intra-abdominal injury requiring surgical repair is higher with gunshot wounds than with stab wounds [5–7]. As advanced imaging including ultrasonography and CT scanning was integrated into the trauma workup, management algorithms incorporating these diagnostic techniques began to be developed. However, through all of this, a few principles remained constant. The physical examination remains a necessary component of the trauma workup. The presence of peritonitis on physical examination or the presence of hemodynamic instability referable to intra-abdominal bleeding almost always mandates operative exploration.

Within the last 20 years, it has been appreciated that for the most severely injured patients, correction of the physiologic derangements associated with trauma is more important than definitive anatomic reconstruction, especially in the setting of profound hemorrhagic shock complicated by hypothermia, acidosis, and coagulopathy. This realization gave rise to the concept of trauma damage control, which employs a staged approach to treating injuries [8]. Damage control begins with limited surgery to address sources of major hemorrhage and GI contamination, followed by rewarming, resuscitation, and correction of metabolic abnormalities in the ICU. Definitive operative management should occur when the patient is not as physiologically compromised and in better shape to tolerate surgery. While these principles have changed the care of the most critically injured patients, and have been extended to other settings besides trauma, they are also associated with their own set of challenges and complications.

Techniques

Preoperative Evaluation

The critical points to be addressed during the evaluation of an injured patient include presence of abdominal injuries and whether those injuries require operative management. The mainstay of the evaluation is physical examination. The presence of peritonitis in the setting of blunt or penetrating trauma is an indication for operative exploration. Likewise, hemodynamic instability, in the absence of another explanation for it, is generally an indication for laparotomy. Adjuncts to the physical exam, such as chest and pelvis X-rays and FAST, can be very helpful to narrow down the potential location of bleeding, particularly in blunt trauma, and can guide operative decisions. In general, hemodynamic instability and a positive FAST are indications for laparotomy. For the hemodynamically stable penetrating trauma patient, CT scan may be a helpful adjunct to physical exam, particularly for selected gunshot wounds or back/flank stab wounds [9–11]. While CT scan is very good at accurately characterizing solid organ injuries, and guiding decisions for observation versus angiographic and/or operative intervention [12, 13], it is notably less helpful with respect to hollow viscus injuries. The most frequent finding with hollow viscus injuries after blunt trauma is unexplained free fluid, but this is quite nonspecific [14, 15]. If this is found, then further observation, examination, lab studies, and/or imaging may be warranted. Of course, physical examination may be limited by intoxication, severe head injury, and/or need for intubation and sedation. The decision on whether or not to operate must take into account the ability of the physician to obtain accurate serial abdominal exams.

Operative Techniques

The optimum operative care of the critically injured patient begins prior to incision. The surgeon must utilize all

information gleaned in the workup in order to anticipate the suspected injuries, establish operative priorities and contingency plans, and guide the intraoperative resuscitative effort. Communication between the operative team and the anesthesia team is critical. The patient should generally be positioned supine, unless there is a plan to perform sigmoidoscopy or other procedure requiring perineal or perianal access in which case lithotomy may be chosen. Generally, prep should extend from nipples to mid-thigh, extending higher if thoracoabdominal injury is suspected, and from table to table laterally (Fig. 4.1). Preventing hypothermia with forced air blankets and by keeping the room warm is important as well.

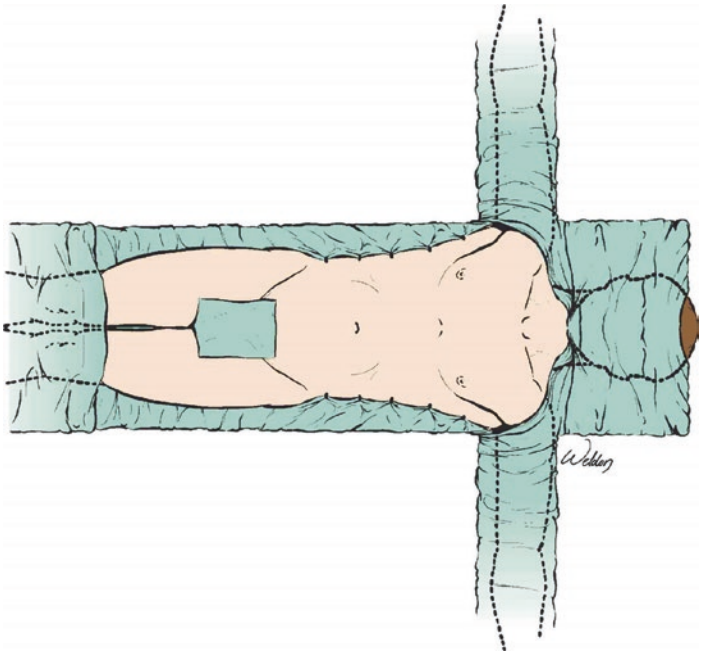

Figure 4.1 Positioning and prep area for standard trauma laparotomy. (Used with permission of McGraw-Hill from Hirshberg [16])

Exploring the Abdomen

The abdomen is explored almost always via a midline laparotomy incision (Fig. 4.2). This provides adequate exposure to most of the abdomen and may easily be extended if necessary. The incision should be long enough to provide the exposure required without struggling, but does not necessarily have to involve the entire length of the abdomen. In fact, there are several circumstances where excessive inferior extension of the incision may ultimately prove troublesome. For example, if the abdomen is to be left open at the end of the case, as in a damage control procedure (more on that later), it may be difficult to secure the vacuum dressing if the incision terminates very low. Also, a midline incision that extends very low may interfere with placement of a low transverse incision for preperitoneal pelvic packing, if that is being considered in the setting of pelvic fracture bleeding. Finally, if a pelvic vascular injury is suspected, usually by absence of one femoral pulse, leaving a soft tissue envelope for a femoral-femoral bypass is wise.

Once the abdomen is entered, the immediate priority is rapid temporary hemostasis. The hemoperitoneum is evacuated quickly (having two suctions set up is handy), the small bowel is eviscerated, and the abdomen is packed in all four quadrants. Packing may also be guided by imaging results if available. We spend a few seconds and divide the falciform ligament routinely to facilitate exploring and if necessary packing the right upper quadrant. Effective packing relies on mechanical compression of bleeding sources; merely placing sponges on top of blood is insufficient (Fig. 4.3). Packs should utilize the abdominal wall to provide the necessary compression. Manual compression or clamping of a vessel may also be necessary, such as with a large liver laceration or shattered spleen. Once temporary hemostasis has been achieved, allow some time for the anesthesia team to catch up on volume resuscitation, replacement of blood, and correction of coagulopathy and metabolic derangements.

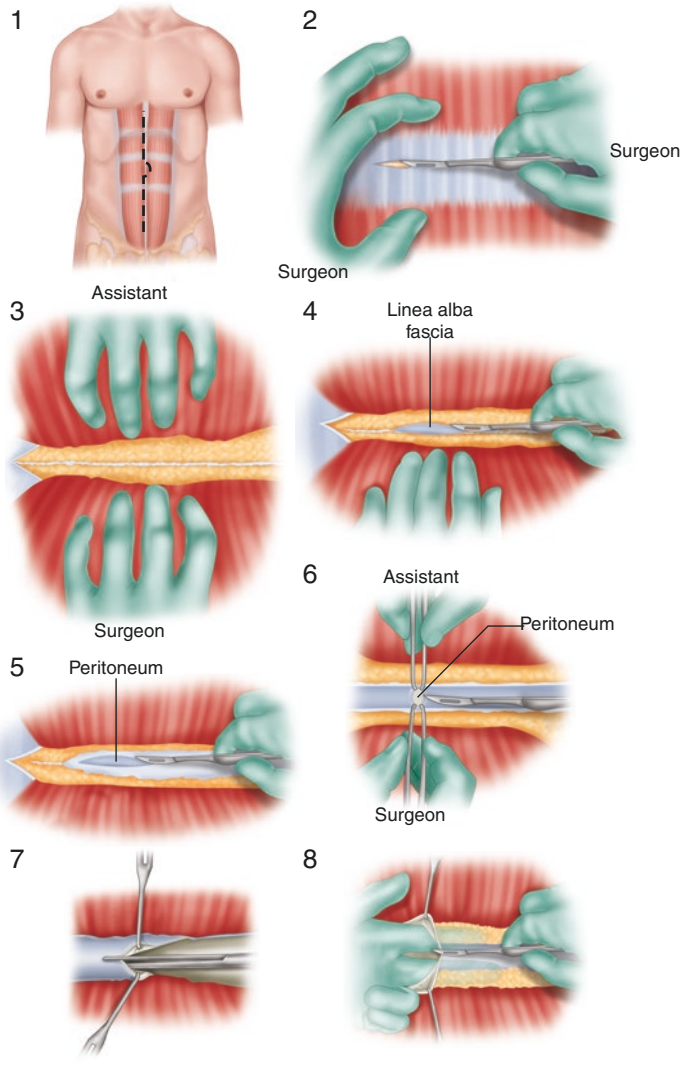

FIGURE 4.2 Technique for midline laparotomy. (Used with permission of McGraw-Hill from Zollinger and Ellison [17])

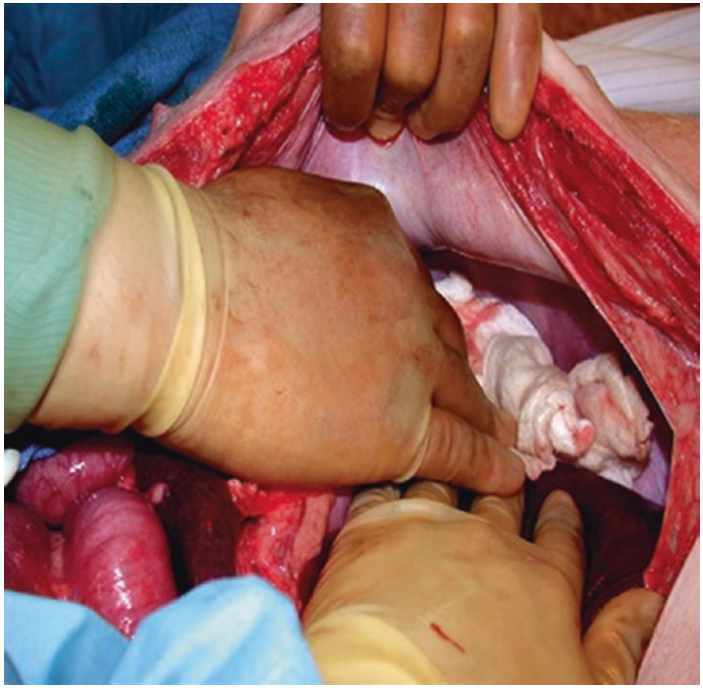

FIGURE 4.3 Technique of liver packing. (Used with permission of the American College of Surgeons from Jacobs and Luk [18])

Systematically exploring the abdomen is necessary to avoid missing injury. The most common method is used to explore the area of least suspicion first, working ones way back to the area where injury is most likely to be found. This is certainly a reasonable way to proceed, but not the only way. However, it is vitally important to utilize the same method every time. This avoids forgetting to do a thorough exploration and potentially missing an injury.

Hemorrhage from the abdomen and/or retroperitoneum comes from one of four sources, the liver, the spleen, the mesentery, or the retroperitoneal vasculature. Rapid exploration of the liver or spleen with the hand should identify major injury. The mesentery can be fanned out quickly to allow

visual inspection. Finally, with the small bowel lifted up cephalad, the surgeon can get a good look at all zones of the retroperitoneum.

The injured liver and spleen must be completely mobilized in order to fully explore them and to make further decision about further operative therapy. The spleen is mobilized by releasing all of the lateral ligament attachments. The spleen should be mobilized up onto the anterior abdominal wall to allow full visualization and operative therapy. The liver must be mobilized by completely taking the falciform ligament down to the vena cava. The triangular ligaments must be completely divided as well. This allows for mobilization of the right lobe onto the anterior abdominal wall. The liver is then suspended only on its vasculature.

The mesentery of the small bowel should be carefully inspected for injury. Similarly, the mesentery of the right colon, transverse colon, and the left colon should be examined. Major hemorrhage from any of these sources should be controlled before further exploration is undertaken.

The next priority is temporary control of ongoing contamination from hollow viscus injury. The entire GI tract should be quickly but thoroughly inspected, with particular attention paid to those areas at highest risk of injury, for example, based on evaluation of the trajectory of a penetrating injury. Injuries should be controlled with atraumatic clamps, such as Babcock's, rather than being definitively repaired at this time. Care should be taken to completely evaluate for through-and-through injuries by appropriate mobilization and exploration, e.g., opening the lesser sac and evaluating the posterior wall of the stomach when there is an anterior penetrating wound.

Once bleeding and contamination have been temporarily controlled, the abdomen and retroperitoneum can be assessed for contained injuries, such as hematomas, and decisions made about exploring or observing those injuries. This is also the time at which a decision needs to be made about whether the current operation is to be the definitive one or whether the abdomen is to be managed via a damage control approach. If the patient does not have serious metabolic derangements, definitive care is best. Repair and/or resection

of bleeding solid organs proceeds as appropriate, followed by repair and/or resection of the hollow viscus injuries. Once all identified injuries have been definitively managed and hemostasis is satisfactory, the abdomen is irrigated and the fascia is closed if there is no significant tension. If there is significant bowel edema precluding fascial closure without undue tension and concern for abdominal compartment syndrome, then the abdomen may be left open and managed with a negative pressure wound dressing.

Exploring the Retroperitoneum

The retroperitoneum is generally divided into three zones (Fig. 4.4). Zone 1 is the central portion. Zone 2 is the perinephric spaces and Zone 3 is the pelvis. Conventional teaching is that all Zone 1 hematomas must be surgically explored to evaluate the central vasculature. Zone 2 hematomas are explored for penetrating trauma but are observed for blunt trauma unless there is pulsatile hemorrhage or they are rapidly expanding. Another indication for exploring a Zone 2 hematoma would be proven injury within the kidney or ureter requiring surgical therapy. Zone 3 hematomas are generally explored for penetrating injury but observed in blunt trauma.

This conventional dogma, however, has more recently been questioned. The average general surgeon spends little time in the retroperitoneum, thus may not be experienced with exploration in Zone 1. If there is significant suspicion of injury, calling a more senior colleague may be prudent. For small hematomas, further exploration with a combination of CT and/or angiography may be preferable to surgical exploration.

Preoperative imaging is now fairly common in stable patients, even those with penetrating trauma. Thus, patients who have had preoperative imaging and who either have no injury or minor injury in Zone 2 likely do not require full exploration. Finally, extraperitoneal pelvic packing is now used relatively commonly to obtain hemostasis following pelvic fractures. This should be accomploshed via a separate incision, not via the laparotomy. Thus, the dictum of never opening a pelvic hematoma after blunt trauma no longer holds true.

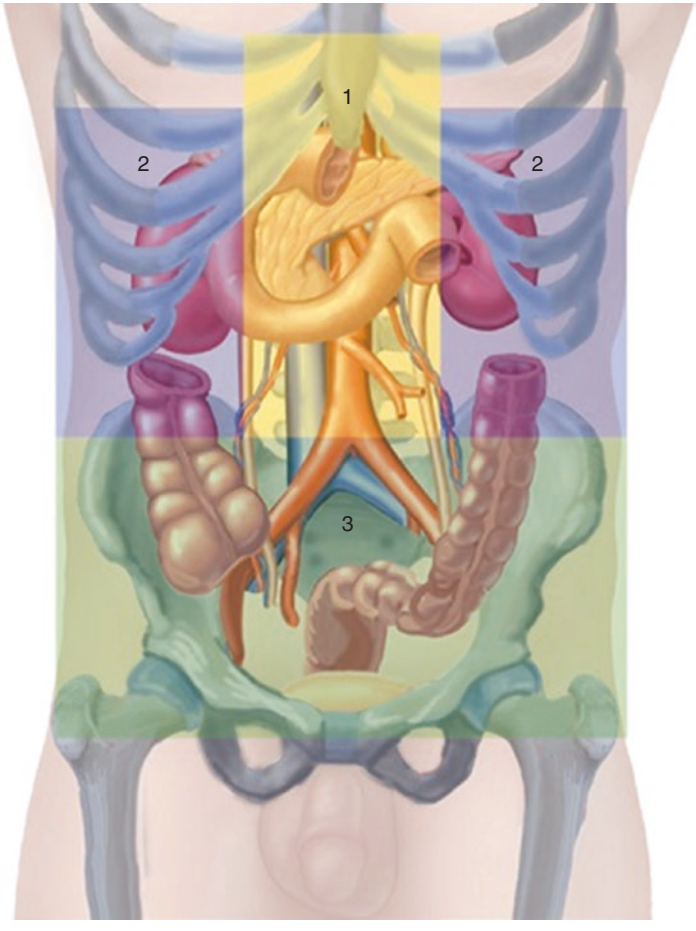

FIGURE 4.4 Vascular zones of the retroperitoneum. (Used with permission of the American College of Surgeons from Jacobs and Luk [18])

Full exploration of the retroperitoneum is generally accomplished with a medial visceral rotation. This is done similarly whether on the right side, termed the Cattell-Braasch maneuver, or on the left side, often called the Mattox maneuver. Regardless of the side mobilized, it is wise to fully divide the white line of Toldt. In order to be complete, the mobilization

should be brought around either the splenic or hepatic flexure onto the transverse colon. If the colon is grasped in one hand, and pressure put at the base of the mesentery with a lap pad, the colon can be rapidly mobilized. This gives wonderful exposure to the vena cava on the right side, as well as all of its branch vessels. On the left side, the aorta will be encountered at the base of the mesentery. All branch vessels of the aorta come out either at 12:00 or 3:00. The plane between them is open; thus, dissection can come up the aorta relatively rapidly.

On the right side, fully mobilizing the duodenum using a Kocher maneuver gives visualization of the vena cava up to the level of the retrohepatic vena cava. The duodenum should be completely mobilized up off of the cava. Superiorly, the dissection should go to the level of the porta. The second and third portion of the duodenum should be completely mobilized (Fig. 4.5). This gives access to the vasculature, particularly the kidney on the right side.

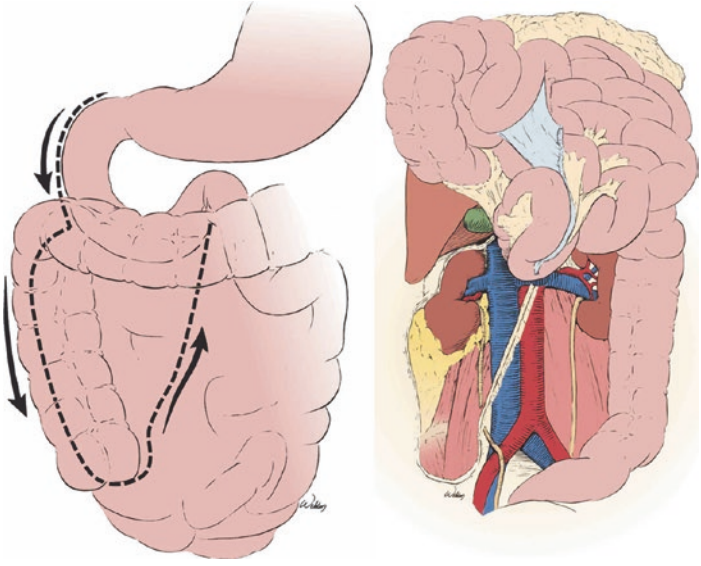

FIGURE 4.5 Right medial visceral rotation for wide retroperitoneal exposure. (Used with permission of McGraw-Hill from Hirshberg [16])

A full left-sided medial visceral rotation often involves mobilizing the kidney. Our preference is to leave the kidney in situ, believing that full mobilization of the kidney is only necessary if there is high suspicion of injury to the kidney requiring surgical therapy (Fig. 4.6). The spleen and tail of the pancreas can be mobilized, giving access to the aorta at the level of the diaphragm.

When a central retroperitoneal hematoma is encountered, one question is whether a right- or left-sided approach is the best option. In those cases, we generally utilize a right-sided approach. The small bowel mesentery can be mobilized up off of the aorta, giving access to the infrarenal aorta and vena cava. Visualization from the left side may not be ideal, particularly in the pelvis, as the mesentery of the sigmoid colon will be in the way.

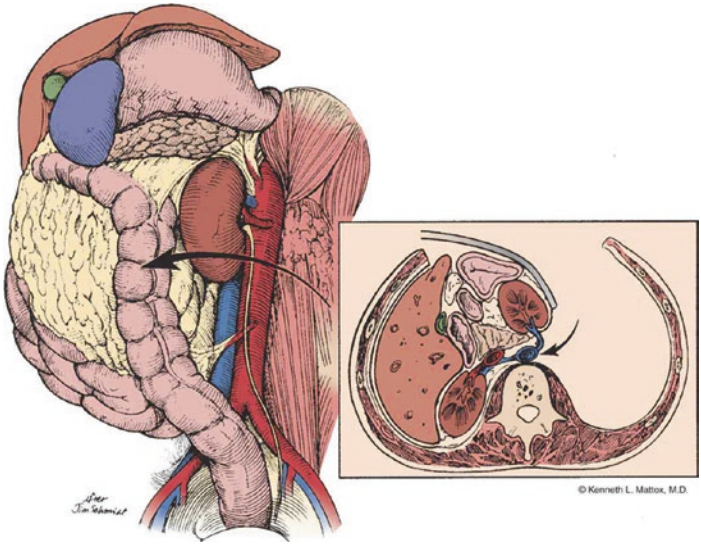

Figure 4.6 Left medial visceral rotation for exposure of the aorta, especially the suprarenal segment. In the modified version, the kidney is left in place. (Used with permission of McGraw-Hill from Hirshberg [16])

Exposure of the superior mesenteric artery and celiac axis can be problematic. In fact, there is no good way to expose the very proximal portion of either of those blood vessels. Widely opening the lesser sac by dividing the gastrocolic omentum gives access to the body of the pancreas. The superior mesenteric artery is often best exposed through a combination of a left-sided medial visceral rotation and opening the lesser sac (Fig. 4.7). Widely opening the lesser omentum, combined with a left-sided medial visceral rotation, often gives the best access to the proximal celiac axis (Fig. 4.8).

Absolute inflow control for abdominal or retroperitoneal injury can be obtained by controlling the aorta at the level of the diaphragm. An aorta occluder or sponge stick may provide temporary control until more definitive steps are taken. Several approaches exist to control the aorta at the diaphragm. The most common is a full left-sided medial visceral rotation. The spleen and tail of the pancreas must be

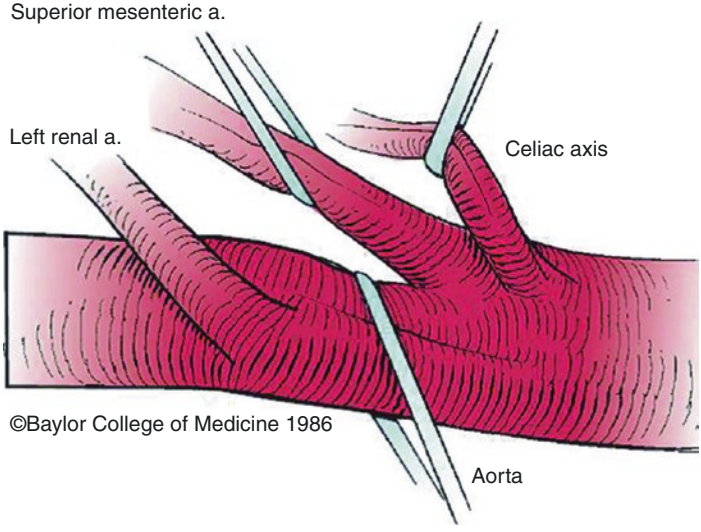

Figure 4.7 Exposure of celiac axis and superior mesenteric artery. (Used with permission of McGraw-Hill from Dente and Feliciano [19])

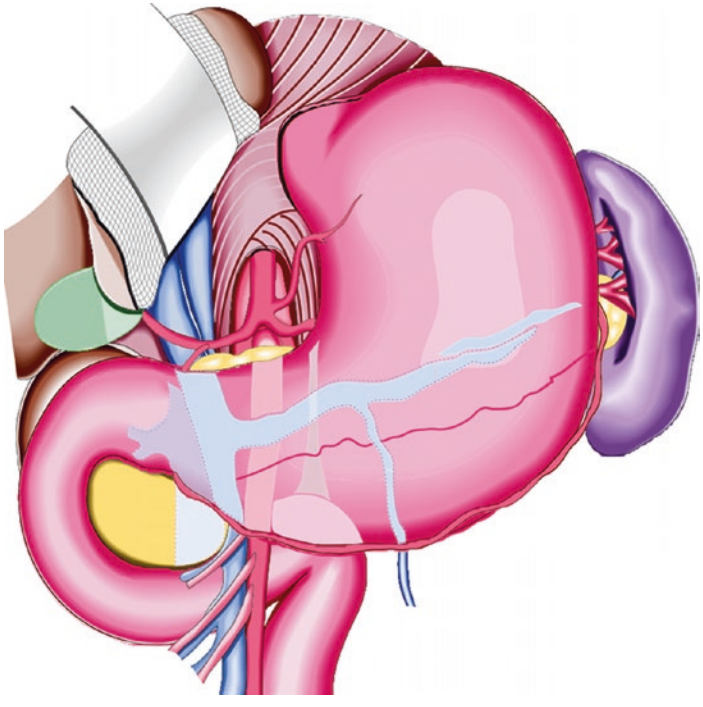

Figure 4.8 Exposure of celiac axis via opening the lesser omentum. (Used with permission of Springer Nature from Desai and Gewertz [20])

mobilized. The esophagus can be differentiated from a flaccid aorta if a nasogastric tube is placed. The lower diaphragmatic crura generally have to be divided to get access to the supraceliac aorta. We prefer to mobilize the esophagus to positively identify the aorta and prevent iatrogenic esophageal injury. The aorta must be fully mobilized dividing the posterior retroperitoneal tissues. We encircle it from the patient's left side using the index finger of the left hand. The posterior attachments are bluntly dissected and the aorta completely encircled prior to placing the clamp. Blindly placing a clamp without full mobilization usually results in the clamp falling off. Recently, another option has been using an internal aortic clamp via a transfemoral balloon catheter (REBOA).

Damage Control

Damage control is a philosophy where operative therapy is staged, with emphasis on control of major physiologic insults early and definitive anatomic reconstruction at a later time when the patient has been stabilized. This came out of experience in the late 1980s and early 1990s. Penetrating trauma with high-velocity weapons became much more common in the United States that patients did not tolerate prolonged operative therapy, particularly when they presented with hypotension. Damage control allows for control of immediately life-threatening injuries only on the night of admission. Injuries not requiring immediate therapy, such as gastrointestinal injuries, are temporarily controlled. Definitive care is deferred until the patient is physiologically more stable. The seminal article on Damage Control by Rotondo and colleagues in 1993 described the three separate and distinct phases: (I) surgical control of hemorrhage and contamination, (II) ICU resuscitation, and (III) re-exploration and definitive surgical management [21].

Damage control should be used early in appropriate patients. If one waits until the patient has developed the lethal triad of coagulopathy, acidosis, and hypothermia, patients are likely to die, regardless of therapy selected. Thus, early use of damage control is wise. However, one must control all means of bleeding. Packing major vascular injuries is doomed to failure. The judgment as to when to "bail out" often requires senior consultation. Junior surgeons should ask for consultative help early on. Excessive use of damage control increases costs and worsens outcomes. Good judgment is key.

When using damage control, no thoughts should be given to preserving organs amenable to resection. Injured spleens and even kidneys should be resected, not repaired. Major vascular injuries must, of course be controlled. Complex repair should be deferred. Intraluminal shunting can be a very valuable technique to preserve distal perfusion. Any conduit suffices. It need only be a good size match to the vessel. The aorta can be shunted with a chest tube. The

superior mesenteric artery can be shunted with IV tubing. As patients are often coagulopathic, no anticoagulation is necessary to preserve flow. Virtually all veins in the abdomen or retroperitoneum can be ligated. While ligation of the superior mesentery vein can be done, these patients virtually all develop serious mesenteric venous hypertension and intestinal ischemia. Thus, we prefer to shunt the SMV, rather than ligate it. In addition, the suprarenal vena cava cannot be ligated. One must at least obtain temporary control, preferably by direct suture repair of these injuries. The location makes shunting problematic.

One should also recognize that direct surgical therapy may not be the best idea for a number of injuries such as retroperitoneal vascular injuries located deep in the pelvis or deep injuries to the right lobe of the liver. Temporary packing and catheter therapy can be lifesaving in these types of injuries. In the past, this has required transfer of the patient to the angiography suite. However, the advent of the hybrid operating room is ideal for these patients, as catheter therapy and direct surgical therapy can occur at the same time.

One should consider use of the REBOA as a means of temporary inflow occlusion for such critically injured patients. The REBOA can provide inflow control and keep patients alive while catheter therapy is planned. We have had great success using it for serious liver injuries, as well as exsanguinating hemorrhage from pelvic fracture bleeding.

Temporary Abdominal Closure

After surgical control of hemorrhage and contamination, phase I of damage control laparotomy is typically concluded by temporary abdominal closure [21]. A temporary closure method is desirable because these patients often have marked visceral edema, retroperitoneal hematoma, and/or intra-abdominal packing. Primary fascial closure at this juncture may be impossible or may result in intra-abdominal hypertension or abdominal compartment syndrome (ACS). Many

techniques for temporary closure have been reported, but a few favorites have evolved. The ideal temporary closure appliance should maintain sterility, contain and protect the bowel, avoid fascial injury, and not leak intraperitoneal fluid externally. This appliance should be rapidly applied, be durable during the ICU resuscitation phase, and then be easily removed at operative re-exploration.

Towel Clip Closure

The fastest technique of temporary abdominal closure is the towel clip closure method [22–25]. Beginning at either end of the laparotomy wound, the skin edges are held together in an everted fashion as penetrating towel clips are applied at 1–2 cm intervals (Fig. 4.9). Only the skin is closed with no attempt to include the fascia. This closure reportedly can be performed in as little as 60 s. The wound and towel clips are then covered with a sterile towel and sterile adhesive barrier.

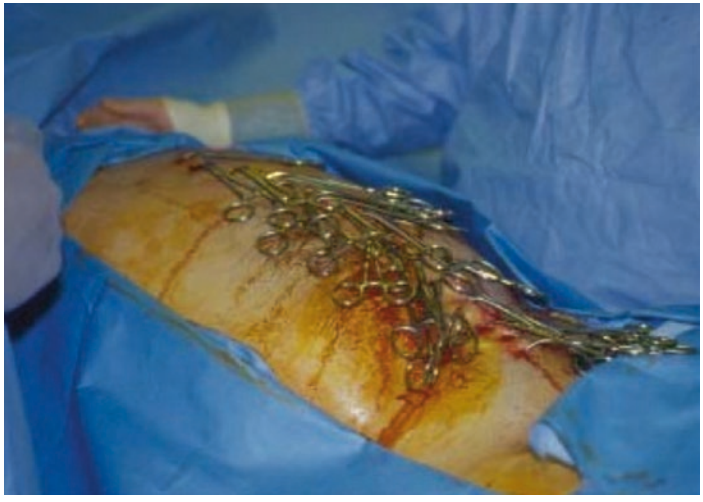

FIGURE 4.9 Towel clip closure of the abdominal skin. (Used with permission of Elsevier from Burlew [25], p. 829)

The drawbacks of this closure include the risk of ACS and the metal clips obscure the imaging view if angiography is needed.

Skin Suture Closure

Another expedited technique for temporary closure utilizes a large-diameter nonabsorbable monofilament suture such as nylon or polypropylene. The suture is applied to the skin in a running continuous fashion. The skin closure can be advanced from one end of the wound to the other if there is not undue tension or begun at both corners and tied at the midportion. A sterile dressing is then applied. This technique avoids the imaging problem with metal towel clips but has similar risk for ACS.

Silo Closure

The silo method is an accepted technique for the management of ventral hernia from gastroschisis or omphalocele. Its application in trauma is most commonly referred to as the "Bogota bag" or "bolsa de Bogota," named after Bogota, Colombia, and the phrase was coined by Dr. David Feliciano [22]. This technique was first used by Dr. Oswaldo Borraez in 1984, who was a second-year surgery resident, on a septic trauma patient with liver injury in which visceral edema prevented abdominal wound closure [26]. He used a plastic intravenous fluid bag sutured to the fascia to cover the open wound and is credited as the first ever to use the silo technique in a trauma patient (Fig. 4.10).

Vacuum Pack Closure

The vacuum pack technique of temporary abdominal closure was popularized by Dr. Donald Barker from University of Tennessee in Chattanooga [27]. This technique has become

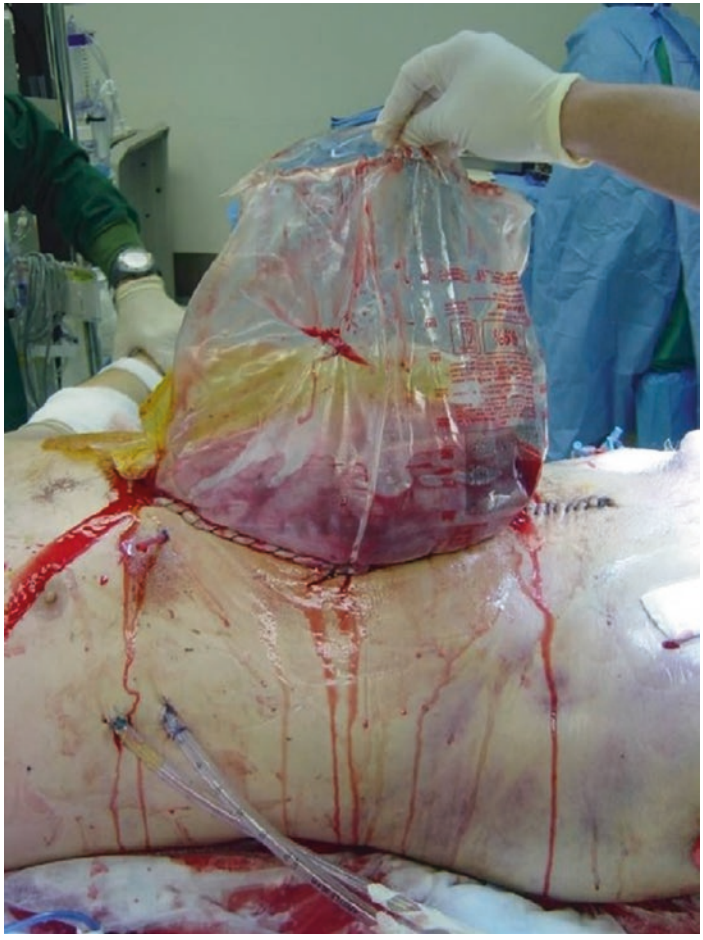

FIGURE 4.10 Bogota bag closure of the abdomen. (Used with permission of Elsevier from Burlew [25], p. 830)

the most popular method of rapid temporary abdominal closure. Following damage control laparotomy, prepare a sterile polyethylene drape by cutting multiple perforations with scissors. We commonly use the Steri-Drape™ Large Towel Drape 1010 (3M Health Care, St. Paul, MN, USA). This film is

60 cm × 45 cm in size, and the adhesive strip on one edge may be cut off with scissors. The drape is then placed over the abdominal viscera and inserted beneath the abdominal wall, creating a nonadherent barrier over the bowel with the fenestrations allowing for fluid penetration (Fig. 4.11a–c).

Next, we like to place a sterile surgical towel over the sterile drape, also tucked beneath the abdominal wall. The towel seems to contain the bowel and reduce the risk of postoperative evisceration. Two drains are then placed over the towel. The original description utilizes silicone drains (Jackson-Pratt or Blake). We commonly use nasogastric suction tubes because the larger lumen allows for more reliable continuous suction. We usually place a second towel over the suction tubes to better maintain the tubes in position. The remainder of the abdominal wall subcutaneous space is filled with unrolled Kerlix gauze. The skin surrounding the wound is then dried as much as possible, and the entire apparatus is covered using a large adhesive polyethylene incise drape, such as Ioban™2 (3M Health Care, St. Paul, MN, USA). The suction tubes are connected with a T-adaptor and connected to a suction source at approximately 125 mmHg negative pressure.

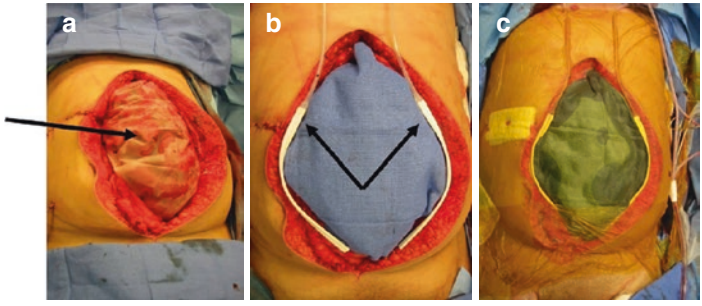

Figure 4.11 (**a**–**c**) Vacuum pack closure technique. (**a**) It begins with placement of a fenestrated drape over the bowel. (**b**) The drape is covered with a towel, and two suction drains are placed. (**c**) The subcutaneous space is filled with gauze and then covered with an adhesive drape. (All: Used with permission of Elsevier from Burlew [25], p. 830)

Commercial Negative Pressure Therapy Systems

The Barker's vacuum pack technique (described above) offers a rapid and lower-cost option for negative pressure wound closure, but newer commercial dressing devices may have advantages. The cumbersome layers of towels, suction tubes, and gauze can be substituted using a commercial polyurethane foam product, such as the V.A.C.® GranuFoam Dressing (KCI, San Antonio, TX, USA) (Fig. 4.12).

The polyethylene drape nonadherent barrier would still be required to interface between the abdominal viscera and the polyurethane foam. A newer system is the ABThera™ Open Abdomen Negative Pressure Therapy Dressing (KCI, San Antonio, TX, USA). The ABThera dressing incorporates all of the necessary components of visceral protective barrier and perforated foam into one large drape that is easily

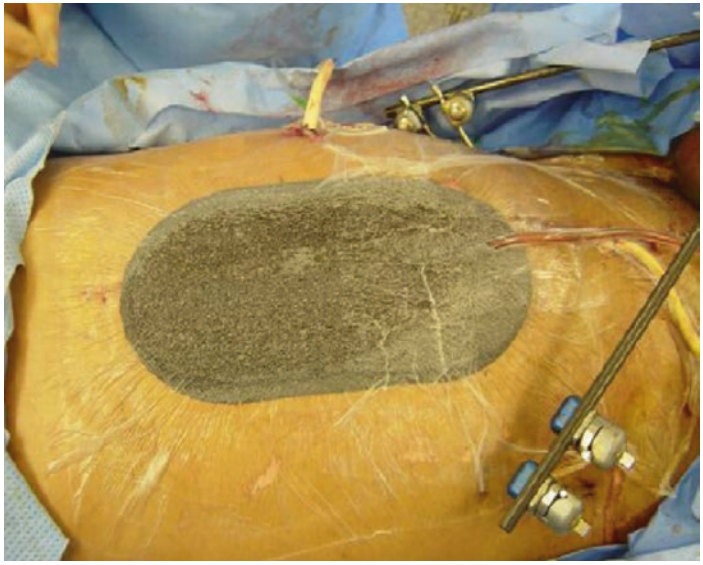

FIGURE 4.12 Black V.A.C. foam used for temporary abdominal negative pressure wound closure. (Used with permission of Elsevier from Burlew [25], p. 832)

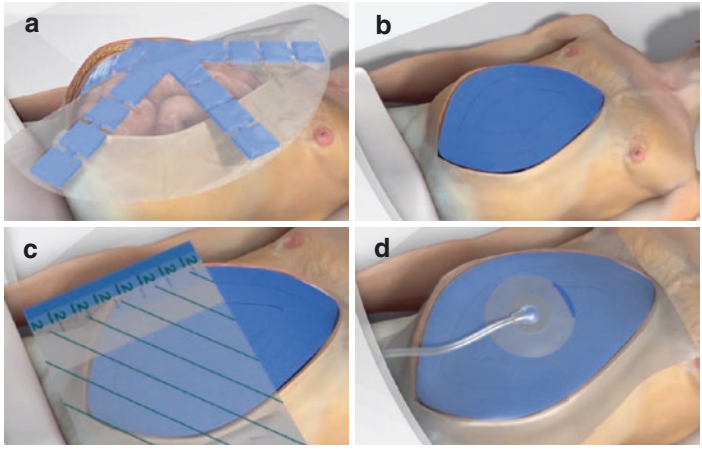

Figure 4.13 (**a**–**d**) Application of the ABThera™ Open Abdomen NPT System. (**a**) Place the ABThera drape. (**b**) Size and position the perforated foam. (**c**) Drape and seal the dressing. (**d**) Connect and start negative pressure suction. (All: Used with permission. Courtesy of KCI, an Acelity Company; makers of V.A.C.® Therapy.)

applied (Fig. 4.13a–d). Studies have shown that the ABThera system may be cost-effective because the medial tension may increase the success of later fascial closure [28].

Complications

The specific complications following laparotomy depend greatly on what injuries were found at the time of the initial operation and accordingly what operative repairs were performed. However, general complications of laparotomy may be divided arbitrarily into early, median, and late categories. Early complications (<4–5 days after index operation) frequently require an unplanned return to the operating room for management and include postoperative hemorrhage, abdominal compartment syndrome, fascial dehiscence or evisceration, or early anastomotic leak. Later complications (7–14 days) include wound infections, fascial dehiscence, and

fistula formation. During this phase, evolving intra-abdominal inflammation and increasing acute adhesions make formal operative exploration increasingly hazardous. Management of these complications at this stage frequently involves operative debridement, wound management, and external drainage of fistulas, with formal repair reserved for several months post-op to allow the intra-abdominal adhesions to mature. Late complications (>14 days) include standard long-term complications of laparotomy, including incisional hernia and bowel obstruction.

Inability to close the fascia is an unfortunate sequela of damage control surgery. Persistent visceral and/or retroperitoneal edema may render primary fascial closure impossible in the acute setting. Prompt recognition of the inability to close the abdomen and expedient visceral soft tissue coverage is the priority in this case, in order to reduce the risk of fistula formation [29]. Frequently, it is possible to close the skin over the bowel and reconstruct the fascia several months later. In certain circumstances, neither the skin nor the fascia is able to be closed. Our preference in this case is to cover the bowel with a double layer of woven Vicryl mesh sutured to the fascia or to the skin if the fascia is of extremely poor quality or tightly adherent to the underlying viscera. A negative pressure dressing is used to cover the wound and may usually be changed at the bedside. Once adequate granulation tissue forms, a split-thickness skin graft is applied to the granulation tissue overlying the abdominal viscera. As above, reconstruction of the abdominal wall is planned when the intra-abdominal adhesions have matured, usually several months later.

References

1. Shaftan GW. Indications for operation in abdominal trauma. Am J Surg. 1960;99:657.
2. Nance FC, Wennar MH, Johnson LW, Ingram JC Jr, Cohn I Jr. Surgical judgment in the management of penetrating wounds of the abdomen: experience in 2212 patients. Surgical judgment in

the management of penetrating wounds of the abdomen: experience in 2212 patients. Ann Surg. 1974;179:639.
3. Demetriades D, Rabinowitz B. Indications for operation in abdominal stab wounds. Ann Surg. 1987;123:1141.
4. Shorr RM, Gottlieb MM, Webb K, Ishiguro L, Berne TV. Selective management of abdominal stab wounds. Arch Surg. 1988;123:1141.
5. Velmahos GC, Demetriades D, Foianini E, Tatevossian R, Cornwell EE 3rd, Asensio J, et al. A selective approach to the management of gunshot wounds of the back. Am J Surg. 1997;174:342.
6. Demetriades D, Velmahos GC, Cornwell E 3rd, Berne TV, Cober S, Bhasin PS, et al. Selective nonoperative management of gunshot wounds of the anterior abdomen. Arch Surg. 1997; 132:178.
7. Velmahos GC, Demetriades D, Toutouzas KG, Sarkisyan G, Chan LS, Ishak R, et al. Selective non-operative management in 1856 patients with abdominal gunshot wounds: should routine laparotomy still be the standard of care? Ann Surg. 2001;234:395.
8. Nicholas JM, Rix EP, Easley KA, Feliciano DV, Cava RA, Ingram WL, et al. Changing patterns in the management of penetrating abdominal trauma: the more things change, the more they stay the same. J Trauma. 2003;55:1095.
9. Demetriades D, Gomez H, Chahwan S, Charalambides K, Velmahos G, Murray J, et al. Gunshot injuries to the liver: the role of selective nonoperative management. J Am Coll Surg. 1999;188:343.
10. Himmelman RG, Martin M, Gilkey S, Barrett JA. Triple-contrast CT scans in penetrating back and flank trauma. J Trauma. 1991;31:852.
11. Chiu WC, Shanmuganathan K, Mirvis SE, Scalea TM. Determining the need for laparotomy in penetrating torso trauma: a prospective study using triple-contrast enhanced abdominopelvic computed tomography. J Trauma. 2001;51:860.
12. Shanmuganathan K, Mirvis SE, Boyd-Kranis R, Takada T, Scalea TM. Nonsurgical management of blunt splenic injury: use of CT criteria to select patients for splenic arteriography and potential endovascular therapy. Radiology. 2000;217:75.
13. 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;216:418.

14. Fakhry S, Watts D, et al. Diagnosing blunt small bowel injury (SBI): an analysis of the clinical utility of computerized tomography (CT) scan from a large multi-institutional trial. J Trauma. 2001;51:1232.
15. Livingston DH, Lavery RF, Passannante MR, Skurnick JH, Baker S, Fabian TC, et al. Free fluid on abdominal computed tomography without solid organ injury after blunt abdominal injury does not mandate celiotomy. Am J Surg. 2001;182:6.
16. Hirshberg A. Trauma laparotomy: principles and techniques. In: Mattox KL, Moore EE, Feliciano DV, editors. Trauma. 7th ed. New York: McGraw-Hill; 2013.
17. Zollinger RM, Ellison EC. Zollinger's atlas of surgical operations. 9th ed. New York: McGraw-Hill; 2011.
18. Jacobs LM, Luk SS. Advanced trauma operative management. 2nd ed. Inc.: Cine-Med Publishing; 2010.
19. Dente CJ, Feliciano DV. Abdominal vascular surgery. In: Mattox KL, Moore EE, Feliciano DV, editors. Trauma. 7th ed. New York: McGraw-Hill; 2013.
20. Desai TR, Gewertz B. Bypass procedures for mesenteric ischemia. In: Lumley JSP, Hoballah JJ, editors. Vascular surgery. Berlin, Heidelberg: Springer; 2009.
21. Rotondo MF, Schwab CW, McGonigal MD, Phillips GR III, Fruchterman TM, Kauder DR, et al. "Damage control": an approach for improved survival in exsanguinating penetrating abdominal injury. J Trauma. 1993;35:382.
22. Feliciano DV, Burch JM. Towel clips, silos, and heroic forms of wound closure. In: Maull KI, editor. Advances in trauma and critical care, vol. 6. Chicago: Mosby Year Book; 1991.
23. Smith PC, Tweddell JS, Bessey PQ. Alternative approaches to abdominal wound closure in severely injured patients with massive visceral edema. J Trauma. 1992;32:16.
24. Burch JM, Ortiz VB, Richardson RJ, Martin RR, Mattox KL, Jordan GL. Abbreviated laparotomy and planned reoperation for critically injured patients. Ann Surg. 1992;215:476.
25. Burlew CC. The open abdomen: practical implications for the practicing surgeon. Am J Surg. 2012;204:826.
26. Feliciano DV, Borraez Gaona OA. Origin of the Bogota bag and its application. In: Duchesne J, Inaba K, Khan MA, editors. Damage control in trauma care: an evolving comprehensive team approach. Cham, Switzerland: Springer International Publishing AG; 2018.

27. Barker DE, Kaufman HJ, Smith LA, Ciraulo DL, Richard CL, Burns RP. Vacuum pack technique of temporary abdominal closure: a 7-year experience with 112 patients. J Trauma. 2000;48:201.
28. Frazee RC, Abernathy SW, Jupiter DC, Hendricks JC, Davis M, Regner JL, et al. Are commercial negative pressure systems worth the cost in open abdomen management? J Am Coll Surg. 2013;216:730.
29. Bradley MJ, Dubose JJ, Scalea TM, Holcomb JB, Shrestha B, Okoye O, et al. Independent predictors of enteric fistula and abdominal sepsis after damage control laparotomy: results from the prospective AAST open abdomen registry. JAMA Surg. 2013;148:947.

Chapter 5
Indications and Techniques for Vascular Exploration

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Patient Assessment

All trauma patients should be assessed in a systematic manner which identifies and treats life-threatening problems first. Advanced trauma life support (ATLS) is the most recognized and practiced resuscitation pathway. It is of critical importance not be distracted by dramatic, non-life-threatening injuries.

Vascular injury can largely be considered in two categories: ischemia and/or hemorrhage. The latter is often dramatic and can present with hemorrhagic shock and significant physiological derangement which should be identified during the primary survey. Obvious external hemorrhage should be immediately controlled with direct pressure, balloon tamponade, tourniquets, and/or clamps in rare cases. Ischemia is often more subtle and can be more challenging to identify,

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requiring an appropriate level of suspicion. This can require a more nuanced approach during the secondary survey.

The signs and symptoms of vascular injury can be divided into hard and soft signs. Hard signs that are highly diagnostic of vascular injury include active/pulsatile bleeding, shock not explained by other injuries, expanding or pulsatile hematoma, absent peripheral pulse, audible bruit, palpable thrill, or evidence of regional ischemia (pain, pallor, paresthesia, paralysis, pulselessness). While care must be individualized, in general, patients with hard signs of vascular injury require emergent operative control and repair.

Soft signs that are suggestive but not diagnostic of vascular trauma include mild shock, stable hematoma, slow bleeding, injury in proximity to a major neurovascular tract, peripheral nerve injury, and diminished pulses. Patients with soft signs or orthopedic injuries associated with potential vascular injuries should undergo further evaluation. Figure 5.1 shows the Western Trauma Association (WTA) algorithm for the workup of peripheral vascular injury.

For injured extremities, the injured extremity index should be obtained. This involves obtaining the Doppler-determined arterial pressure in the injured extremity, divided by the pressure of an unaffected limb using a manual blood pressure cuff. An index above 0.9 excludes injury, while a value of less than 0.9 warrants further investigation such as multidetector computed tomography angiography (CTA), formal angiography, or operative exploration [2, 3].

Assessing distal pulses may be difficult in a patient in shock or who is hypothermic; therefore, these patients should be resuscitated in the usual fashion, and once improved, palpation and distal assessment with noninvasive testing should be performed [1].

Decision-Making, Prioritization, and Adjuncts

While the principle that patients presenting with hard signs are explored and soft signs undergo further imaging is conceptually simple, patients are frequently more complex.

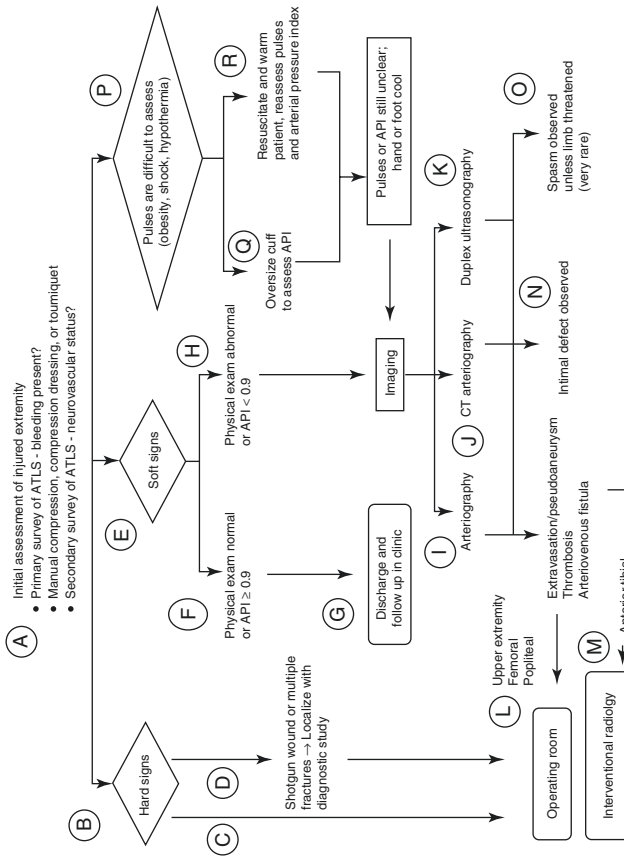

FIGURE 5.1 The WTA developed an algorithm for the workup of peripheral vascular injury. (Used with permission of Wolters Kluwer Health, Inc, from Feliciano et al. [1])

Decision-making needs to take into account competing priorities, associated injuries, and institutional resources.

Active hemorrhage should be prioritized above other injuries. While this generally involves definitive care for the vascular injury, if physiological derangement is substantial, temporary damage control maneuvers should be utilized, such as shunting or ligation. Critical neurosurgical intervention should be prioritized once hemorrhage control is achieved. Where extremities have significant bony and soft tissue destruction associated with the vascular injury, real thought needs to be applied as to whether limb salvage should be attempted. In the setting of non-salvageable injury, limited resources, and/or other injuries, a swift amputation may be the best management.

A further consideration is the application of endovascular techniques either as definitive care or combined with open exploration, which may or may not be an option depending on resources. Such techniques can be complimentary to open exploration, specifically in junctional regions where classical surgical approaches are often involved and are associated with significant morbidity. For example, exposure of the left subclavian artery is challenging and may require multiple incisions for proximal and distal control. The placement of an endovascular balloon for proximal control may obviate the need for a chest incision but requires fluoroscopy, wires, and balloon catheters. Such hybrid approaches are attractive but add a layer of complexity to the procedure. These cases often take additional time and require operative resources be available at all times.

Where patients are unstable, there is very limited role for "precision" endovascular interventions that involve formal imaging. However, resuscitative endovascular balloon occlusion of the aorta (REBOA) is an endovascular adjunct that can be used for proximal aortic control of the abdominal and/or pelvic vasculature. This can be placed from the femoral artery without imaging and will be discussed in detail in a later chapter.

Finally, in any extremity with a vascular injury, consideration must be given to the need for fasciotomy. Patients that are "high risk" for developing early compartment syndrome after trauma include hypotension in the field; delay in treatment, especially without arterial inflow for 4–6 h; ongoing hypotension during resuscitation or operation; evidence of crush injury combination of arterial and venous injury, especially the popliteal artery and vein; and need for arterial or venous ligation or early thrombosis and repair of either [4].

Compartment pressures can be measured intraoperatively or postoperatively if a fasciotomy was not performed. Devices such as the Stryker Intracompartmental Pressure Monitor System (Stryker Instruments, Kalamazoo, MI, USA) or arterial line with slit/side port catheter can be used. There is ongoing debate about absolute pressures versus delta P (compartment perfusion pressure (CPP) = mean arterial pressure – compartment pressure) on who needs a fasciotomy. Absolute pressures of 30–35 mmHg or delta P of less than 30 mmHg should warrant fasciotomy [4]. The operative technique for fasciotomy is discussed elsewhere in this book.

Operative Technique

Taking time to mentally rehearse your operative approach and plan is important, especially when assessing a complex vascular injury. Consider what you might have to do rather than what you are going to do. For example, in the case of carotid injury that you believe is accessible via the neck, is there a chance you might need to perform a sternotomy? In which case, do you have a sternal saw in the room? Have you told the scrub team that this may be required?

A similar thought process is required for any hybrid maneuver – is the patient on the correct table that can support imaging? Who will drive the imaging equipment? Is the correct inventory of sheaths/wires/catheters in the room? Planning is as important as execution when hybrid maneu-

vers are used. Efficiency will be lost if circulating staff are continually out of the OR looking for specialist equipment.

Always review any imaging in detail. Pay attention to the absence of the normal, as well as the presence of the abnormal. This is especially important in the case of aberrant anatomy. If you have the luxury of a preoperative CTA, the identification of pitfalls such as retroaortic renal veins, duplicate IVCs, and high bifurcating femoral or brachial arteries optimizes your operative plan. Such awareness is the strength of preoperative imaging.

The affected area should be widely prepped and draped for adequate exposure. This prep should include proximal areas outside of the zone of injury, in case vascular control must be gained at that location. For instance, in patients that have neck or upper extremity vascular injury, the chest should be included in the prep, in case a sternotomy, thoracotomy, or a clavicular incision is needed for more proximal control. With patients that have lower extremity injury, the abdomen should be included in the prep, in case access is needed to the external iliac arteries. If manual pressure is required to maintain hemostasis, the hand should be prepped into the field until another source of control can be obtained. The lower extremity, groin, and thigh should always be included, as saphenous vein from an uninjured leg may be needed as conduit.

Proximal and distal control of the injured vessel is the basic principle of vascular surgery. If the injury is to an extremity and it is distal to the groin or axilla, a sterile pneumatic tourniquet can serve as inflow control, until the vessel can be properly dissected out. Once identified, control can be obtained with vascular clamps, bulldogs, or vessel loops and the tourniquet taken down. For the upper extremity, proximal vascular control can be obtained on the brachial artery, axillary artery, subclavian artery, or great vessel control via sternotomy, depending on the location of the injury.

For the lower extremity, superficial femoral artery, common femoral artery, or external iliac artery control may be needed, depending on the location of the injury. The external

iliac can be accessed either transperitoneally or through a lower abdominal oblique incision into the preperitoneal space, typically used in transplant surgery. Distal control should also be performed outside the area of hematoma or hemorrhage. If this is difficult, direct control can be performed and internal balloon tamponade with a Fogarty and a three-way stopcock can be used. Trying to control a vascular injury locally often causes the injury hematoma to rupture which can precipitate large-volume bleeding. Obtaining initial control, using vascular clamps far proximally and distally, and then walking them in toward the wound can provide excellent control and avoid extra hemorrhage rather than trying to attack these wounds in close proximity to the injury.

Once the vessel is isolated both proximally and distally, vessel loops are used for inflow and outflow control. The injury should be assessed as to whether a simple or complex repair is needed. Additional injuries and the patient's hemodynamic status must be considered. In the case of complex injury and/or if the patient is unstable, damage control with shunting or ligation should be used; see shunt section in this chapter.

The vessel should have the edges debrided to normal, healthy tissue. Inflow and back bleeding should be noted. A Fogarty catheter can be run proximally and distally to clear the vessel of any thrombus. Distally, the catheter should be used until no thrombus is returned on two consecutive passes. Appropriate sizes for the catheter include a #6 for the common and external iliac arteries, a #4 to #5 for the common femoral artery, a #4 for the superficial femoral artery, a #3 to #4 for the popliteal artery, and a #3 for the other arteries of the leg. Balloon catheters are never passed in venous injuries because these will disrupt the veins [5].

Systemic heparinization should be considered in stable patients with isolated extremity/vascular injury. 5000 (50–75 units/kg) units IV should be given. Systemic heparin should be avoided in patients with torso or head injuries. Local administration of heparinized saline should be injected both proximally and distally before any repair to aid in pre-

venting local thrombosis using approximately 20–25 mL per site (50 U/mL).

If the injury to the artery only has minimal loss, a primary repair can be used depending on the luminal diameter. When closing, the last stitch or two is left loose until proximal and distal flushing are performed. Care is made not to cinch down the last knot or knots too tightly to avoid constricting the anastomosis. For partial arterial injuries of smaller vessels (brachial, SFA, popliteal), patch angioplasty can be used so the resulting luminal diameter is not too narrow.

For a complete transection, the injured vessel must be debrided to healthy tissue at both ends. Some length can be gained by sacrificing some of the branches of the vessel depending on its location. This maneuver may gain up to 3 cm of total advancement of both ends. If this is not feasible or the defect is too large, an interposition graft is necessary [5]. Choice of conduit will depend on size match and location of the injured vessel. Greater saphenous vein from the uninjured leg is typically the first choice for conduit. As the injured artery is typically vasoconstricted, using a slightly larger conduit than one would expect is often wise.

For suturing into the arterial wall, the key maneuver is a perpendicular pass of the needle. Most vascular trauma is to young healthy arteries, so although formal vascular training teaches "inside out" on the artery to prevent intimal flaps, this is less worrisome in vascular trauma. For the repair, it is often easier to perform the distal anastomosis first, especially if it is in a difficult location to allow for better visualization of the posterior suture line. Figure 5.2 shows the WTA algorithm for operative decision-making in vascular injury.

Conduit Options

Saphenous Vein

The ideal conduit for extremity vascular injury is contralateral saphenous vein. The saphenous vein can be used as the

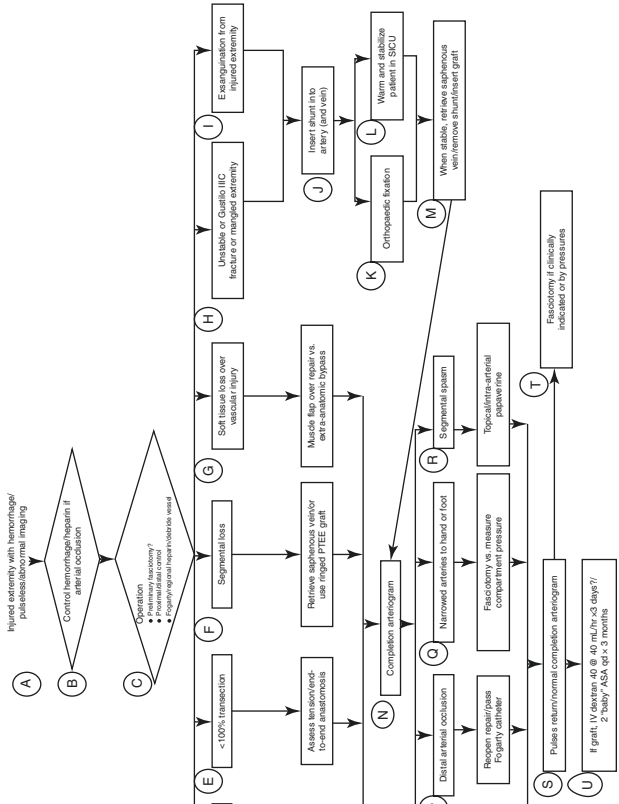

FIGURE 5.2 The WTA II flow chart shows one algorithm for the operative decision-making for vascular injury. (Used with permission of Wolters Kluwer Health Inc, from Feliciano et al. [5])

primary conduit, patch, or spiraled for reconstruction of larger vessels. The amount of vein harvested should be longer than the defect so that it may be trimmed down prior to the final anastomosis. During vein harvest, small branches should be ligated with 2-0 or 3-0 silk. After the vein is harvested, it should be reversed and an olive-tip catheter secured to the distal end with silk suture.

Using heparinized saline, the vein should be dilated while closing the other end to look for any leaks. These leaks can be controlled with ties or 6-0 Prolene sutures. One side of the vein is marked using a sterile marker, so as to not twist the conduit when performing the anastomosis. Once the conduit is satisfactory, it is placed in a heparinized saline bath. If the greater saphenous vein is absent, diseased, has a poor diameter, or is the only venous outflow for an injured extremity, other options include the lesser saphenous or the cephalic or brachial vein [5].

Polytetrafluoroethylene (PTFE)

Due to the size of injured vessels ideally, PTFE should only be placed proximally to the axilla and/or to the knee. If the injury is distal to this location, vein should be used because the diameter of the injured vessel is small and smaller PTFE will thrombose. PTFE has shown improved patency (70–90% short term) and rare infection, even in contaminated wounds [6, 7]. It is clear that patency with PTFE is equivalent to that of a vein for injuries proximal to the popliteal artery. PTFE grafts smaller than 6 mm should be avoided.

PTFE and vein grafts must be covered with soft tissue to prevent hemorrhage from desiccation of the vein, with subsequent autolysis or breakdown of the anastomosis [8]. Patients in whom PTFE is used should be put on ASA of 162–325 mg daily × 3 months postoperatively [9]. The ASA recommendation is extrapolated from aortosaphenous bypass from CABG data as well as from bypass from peripheral vascular disease.

Biological

An emerging field of study is the development of biological conduits which permit some level of recipient tissue incorporation postimplantation. The basic design is that of an acellular collagen scaffold that supports recipient tissue ingrowth. The origin of that collagen varies – this can be derived from bovine carotid tissue (e.g., Artegraft®, Artegraft, North Brunswick, NJ, USA) or human stem cells (e.g., Humacyte®, Humacyte, Durham, NC, USA), which are ultimately decellularized.

These types of conduit promise a number of advantages: an off-the-shelf vascular conduit, which is infection resistant, is durable, and handles well. Clinical trials are ongoing, so the long-term outcomes of these novel grafts in trauma are unknown at this time. Recipients will require close monitoring to ensure issues such aneurysm formation or thrombosis are recognized early if they occur.

Damage Control Options

Shunts

Shunts are the primary damage control modality for vascular injury. In an unstable situation, a shunt can be placed to re-establish blood flow, and the patient can be resuscitated in the OR or ICU. A formal repair can be performed when the patient is stable. It is vitally important that the shunt be a good size match with the artery. Various commercial shunts are available in assorted sizes and configurations. If commercial shunts are not available, anything from IV tubing to pediatric feeding tubes all the way to chest tubes can be used for conduits, depending on the size of the injured vessel. We use Argyle shunts because they are straight, providing direct flow. The shunt size should be the largest diameter that will fit comfortably into the injured vessel. Other shunts can loop and become dislodged, so we typically do

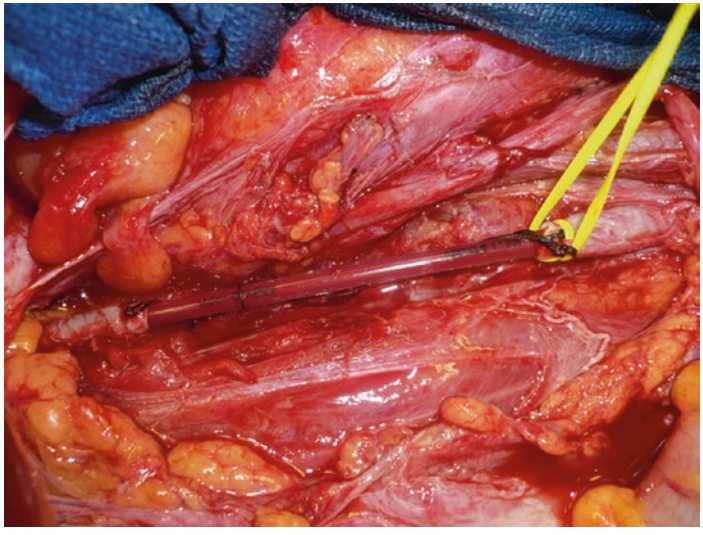

FIGURE 5.3 Shunt in place

not use them. Venous shunting can be considered. However, in true exsanguination, ligation may be used for almost any venous injury.

For arterial shunting, once proximal and distal control is obtained, Fogarty catheters should be used to clear the thrombus. Local heparin (10 units/1 mL) can be instilled, and the shunt can be inserted into the injured vessel. The distal end is inserted first. Back bleeding must be confirmed. Heparinized saline is then instilled in the shunt as it is inserted proximally to prevent an air embolism. The shunt is then secured to the injured vessel with 2-0 silk suture as close to the edge of the injured vessel as possible, as this portion of the artery will need to be resected for the definitive repair (Fig. 5.3). We typically leave the vessel loops in place loosely, with the end secured by a clip. Patency of the shunt should be confirmed with palpation of a pulse, an angiogram, or with Doppler wave flow. If possible, muscle is loosely approximated over this or a moist Kerlix/towel used. Dry dressings are then placed and covered with Ioban or another occlusive

dressing. For venous shunting, larger tubes may be needed such as small chest tubes (16–24 F) depending on the site. Continued pulse checks should occur in the ICU and prompt re-exploration performed if there is a loss of pulse or loss of Doppler signal. Once the patient is more clinically stable, the shunt should be removed and a formal repair performed.

Ligation

This is the ultimate damage control maneuver which should not be forgotten as an option in vascular injury for patients in extremis. This applies to both arterial and venous injuries with a number of caveats. The suprahepatic IVC, superior mesenteric vein, and portal vein injuries must all be repaired as ligation would be fatal. Likewise, the supraceliac aorta cannot be ligated, and most would consider ligation between the infrarenal aorta and iliac system unwise. In the face of a devastating injury to those vascular regions, ligation with extra-anatomical bypass can be an option to consider.

Specific Access and Repair Options

Axillary Artery Exposure

The subclavian artery becomes the axillary artery as it crosses beneath the first rib. The pectoralis minor muscle divides the artery into three sections. This artery becomes the brachial artery as it courses across the lower border of teres major.

The incision begins at the inferior edge of the center of the clavicle and runs in the deltopectoral groove (Fig. 5.4). A self-retaining retractor is placed after the skin incicion is performed. The pectoralis major can be retracted or split in the level of its fibers (if hemodynamically stable) or divided 2 cm from its insertion into the humeral head (if unstable). We reposition the self-retaining retractor and then divide the pectoralis minor by placing an army navy retractor underneath the pectoralis minor and dividing the muscle using

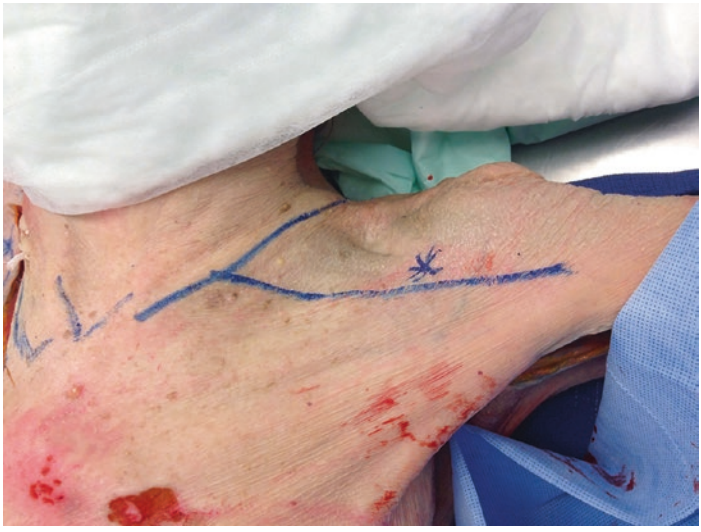

FIGURE 5.4 Skin markings for axillary exposure. The sternal notch and clavicle should be delineated. At approximately proximal 1/3 mark of the clavicle, a lateral incision in the deltopectoral groove should be carried out. If the injury is to the distal axillary artery, the incision can be carried into the beginning of the confluence of the biceps and triceps for proximal brachial artery exposure

cautery (Fig. 5.5). This provides access to the second portion of the axillary artery. The brachial plexus is just inferior to the artery, and the axillary vein typically runs with the artery, just inferior as well (Fig. 5.6) [10].

Options for repair include shunting or ligation with fasciotomy, if the patient is in extremis. Since there are abundant collaterals around the shoulder, ligation is an option. Reconstruction with vein or graft is preferred in the stable patient.

Brachial Artery

The brachial artery is subcutaneous in its course as it runs between the biceps groove between the biceps and triceps of

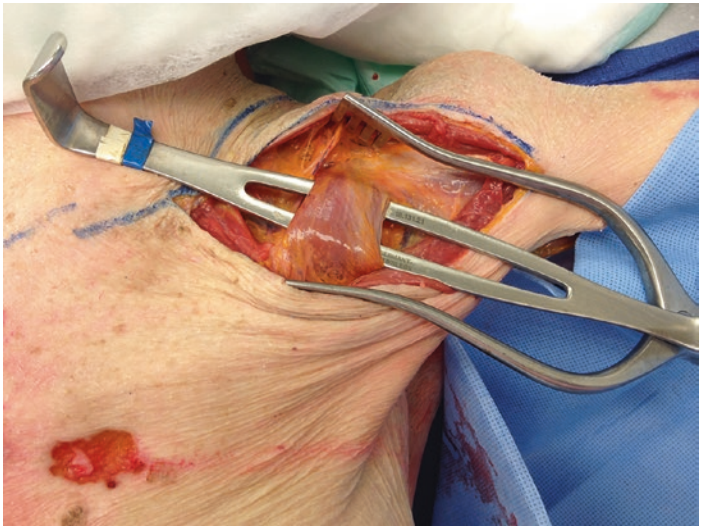

FIGURE 5.5 Dissection of pectoralis minor prior to division

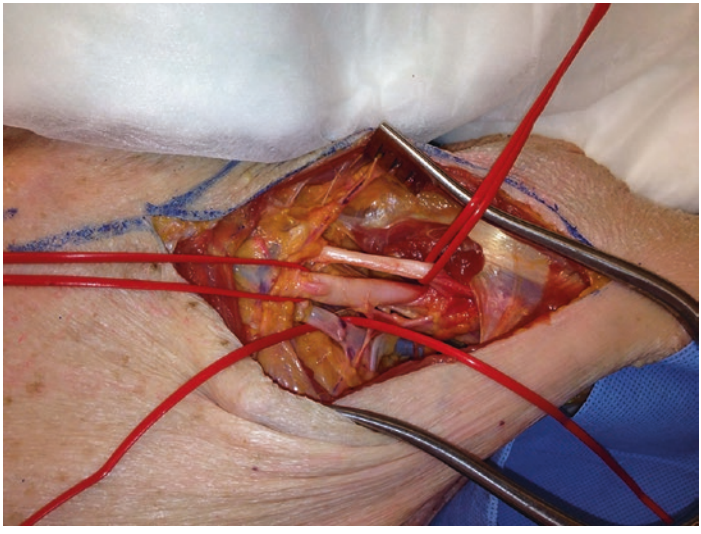

FIGURE 5.6 Axillary artery (middle), vein (inferior), and associated nerve (superior) with vessel loops

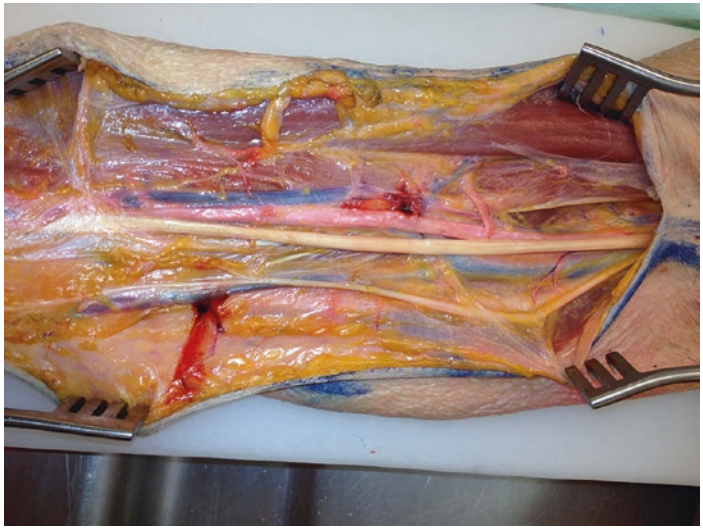

FIGURE 5.7 Brachial artery exposure. Median nerve anterior and inferior in middle of picture. Brachial artery is just posterior to this nerve

the medial arm. It veers laterally at the antecubital fossa and collaterals arise around the elbow. The artery is accompanied by two venae comitantes, typically on either side of the artery. The median nerve runs anteriorly and the ulnar nerve posteriorly. Halfway down the arm, the median nerve crosses the artery and runs along the posteromedial side of the artery (Fig. 5.7).

The incision is carried out in the groove between the triceps and biceps muscle bellies. It can be extended obliquely across the antecubital fossa laterally if exposure of the bifurcation is necessary. A self-retaining retractor is placed. Sometimes, an injured brachial or basilic vein can be used as a conduit; therefore, care should be taken not to injure these in dissection. If the bifurcation of the brachial artery needs to be exposed, the bicipital tendon must be divided. This tendon lies just beneath the median cubital vein [10]. Repair of these injuries are either simple repair, plus or minus vein patch, or interposition vein graft.

Proximal Aorta and IVC

For aortic control, consideration should be made for an endovascular balloon for aortic occlusion versus open occlusion.

When the upper portion of zone 1 has a hematoma or hemorrhage, injury to the aorta, celiac axis, SMA, or renal arteries should be considered and proximal control should be performed. Proximal aortic control can be obtained via a left sided medial visceral rotation as described in a previous chapter or directly via a lesser sac exposure. The lesser omentum should be incised vertically, creating a window between the distal esophagus and liver. The hepatophrenic ligament may need to be divided. The left crus can be split along its fibers or divided. A finger is used to bluntly dissect off loose tissue around the aorta. The aorta can be clamped or occluded with a sponge stick, "occluder" device, or clamp. This should be released when more specific control can be obtained. The aorta must be encircled to bensure complete aortic control. The tips of the clamp should contact the spine and paraspinous muscles to ensure complete control. It is helpful to invert the clamp so the handles are going up toward the thoracic cavity and not as much in the way. This can be tethered up with an umbilical tape and clamped to the drape. Another option can be intraluminal control with an aortic balloon occluder.

Injuries to the right retroperitoneum (zone 1/2) can be accessed by a medial visceral rotation (Cattell-Braasch maneuver). The IVC and renal vasculature can be visualized and controlled in this fashion. The parietal peritoneum at the white line of Toldt is taken down from the base of the cecum to the hepatic flexure. This can be started with cautery or scissors and extended in the same manner or bluntly. The colon is mobilized medially and superiorly, exposing the iliac vessels, IVC, and renal vessels. This can be accomplished by lifting the colon and pushing it away from the retroperitoneum with a lap pad. If more proximal exposure is needed, the duodenum can be mobilized using a standard Kocher maneuver.

For IVC injuries, sponge sticks provide great means for compression to obtain proximal and distal control. If a simple

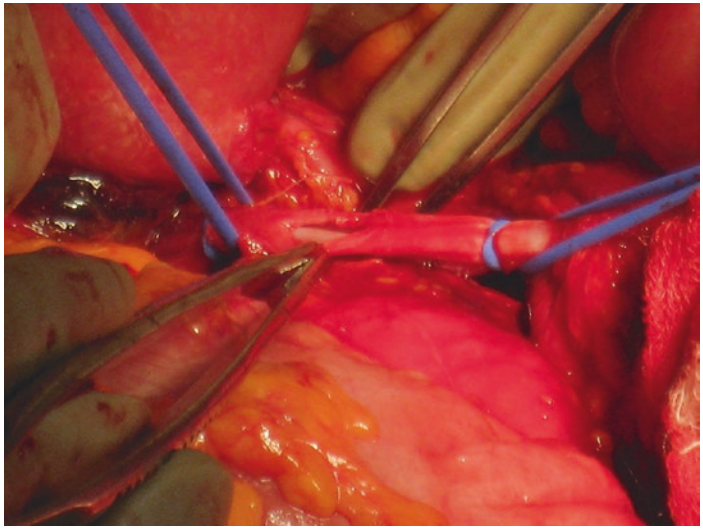

Figure 5.8 Anterior IVC injury

laceration is noted anteriorly, a Satinsky clamp can be used to clamp the injured side while it is repaired in a running fashion. Allis clamps can also be used to bring the edges together while repairing the vein. Depending on the injury mechanism and trajectory, a posterior injury should also be considered (Figs. 5.8, 5.9, 5.10, and 5.11). The IVC can be rotated carefully to inspect this area and repair any other injury, if identified. The posterior injury can also be fixed through the anterior injury, if necessary. It is almost always necessary to extend the anterior injury cephalad and caudead to get adequate exposure of the posterior injury for the repair. If a larger wound is noted, attempts can be made for primary end-to-end repair. Otherwise, consideration can be used for a panel graft or ligation. If the infrarenal IVC is to be ligated, it is recommended to ligate it right next to the renal veins so that there is no excess IVC to form a clot infrarenally.

Our preferred method to control the IVC is using intestinal Allis clamps. The injury is first controlled with digital pressure. Proximal and distal control can be obtained with

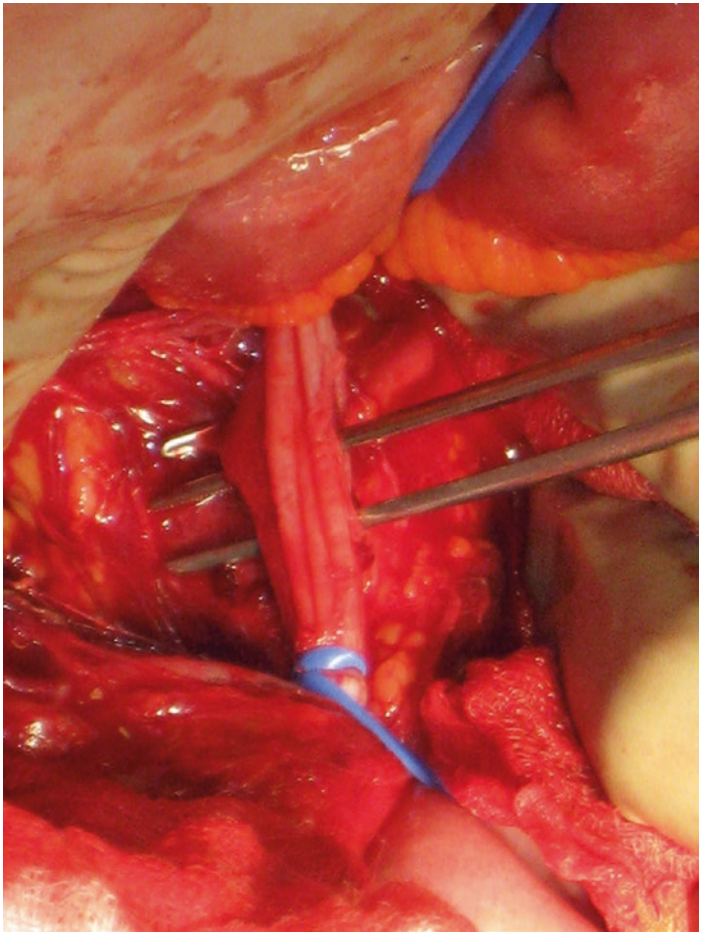

FIGURE 5.9 Through-and-through IVC injury with forceps delineating the injury

either vessel loops or from pressure from sponge sticks. The first clamp is applied in a controlled fashion. Adjacent clamps can then be placed to approximate the wound. When the injury is completely controlled, the clamps are lifted, thus improving venous return. The injury is then repaired under

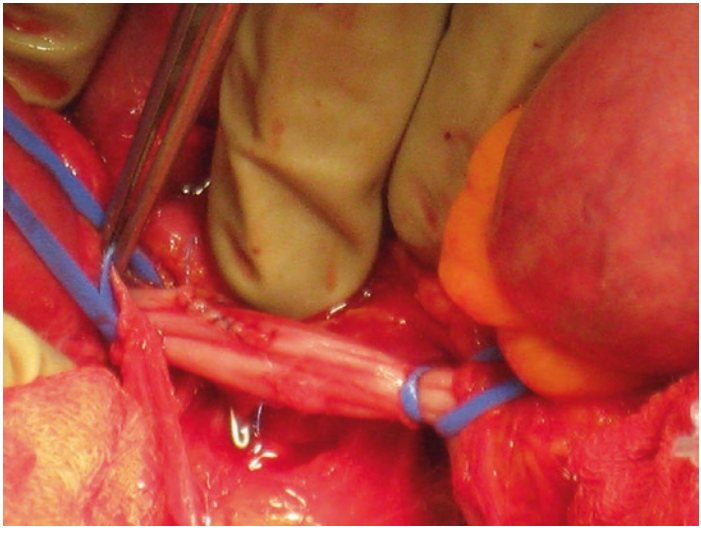

FIGURE 5.10 IVC repair with proximal and distal flow controlled with vessel loops

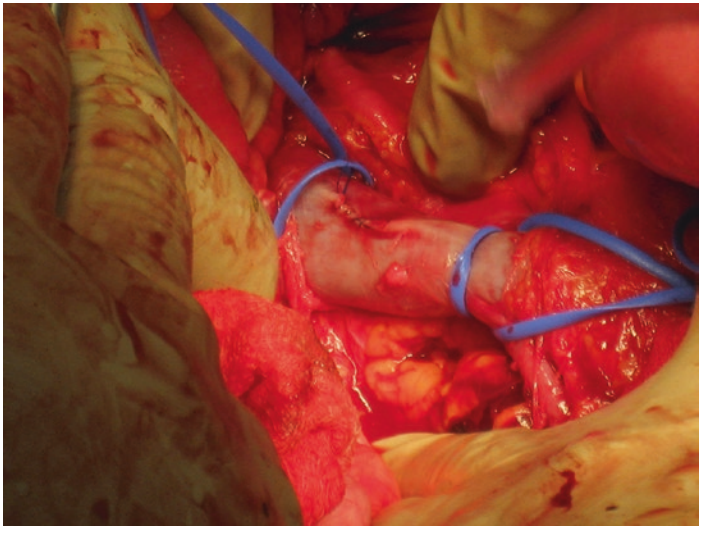

FIGURE 5.11 IVC repair with vessel loops loose, assessing for venous flow

the clamps, sequentially removing them as the repair is completed. This control can also be performed with a Satinsky clamp and repaired in a similar fashion.

Distal Aorta and Iliac Vessels

With hemorrhage in zone 3, a vascular injury to the distal aorta or IVC or common/internal/external iliac artery or vein must be ruled out. Due to the proximity, the ureter should also be evaluated. A visceral medial rotation should be performed to expose this area. The common iliac veins are densely adherent to the back wall of the common iliac arteries; therefore, dissection should be done carefully. The right common iliac artery crosses over the distal bifurcation of the IVC and the confluence of iliac veins, so this may need to be divided to obtain control of underlying structures. The artery can be reanastomosed once the venous injury is handled. For a distal aortic injury, the proximal aorta should be controlled for inflow and the iliac arteries for outflow. Ideally, this is performed with vessel loops. Temporary maneuvers can include lap pads, sponge sticks, or digital compression.

The mid-aorta can be controlled via a left-sided medial visceral rotation. The IVC is best approached from the right side. If there is doubt, we prefer a right-sided approach. The mesentery of the small intestine can be lifted off the retroperitoneum, giving access to the IVC and infrarenal aorta, as well as the proximal iliac vasculature on both sides.

Iliac arterial injuries can be isolated by vessel looping the common iliac near the aortic bifurcation and the distal external iliac artery by the inguinal ligament. The internal iliac artery can be identified by lifting these vessel loops and placing one around the internal iliac artery.

Hypogastric venous injuries can be difficult to expose. The vein runs behind the artery and the main vessel is quite short. It then branches into many smaller vessels. Dividing the hypogastric artery aids in exposure. The artery may be dissected off the vein quickly, greatly improving exposure.

Iliac arterial repairs will depend on the extent of the injured vessel, degree of contamination, as well as the overall disease burden of the patient. If the patient is in extremis, shunting is a viable option. For extreme situations, the iliac artery can be ligated. A fasciotomy should be performed, and if the patient survives, a femoral-femoral bypass can be considered. Interpositions grafts can be used in a contaminated field; however, if it is severely contaminated, ligation with extra-anatomic bypass should be considered.

External Iliac Artery

The external iliac can be exposed via a transperitoneal approach, but it is typically more expedient to approach it from a retroperitoneal approach above the ligament. A "hockey stick" incision can be carried out from the groin to above the inguinal ligament in a lateral direction above the iliac crest. The external and internal obliques are separated and the retroperitoneal space is entered. A self-retaining retractor can be placed and a lap pad used to rotate all of the peritoneum medially to expose the psoas muscle and vessels (Fig. 5.12). The inguinal ligament can be divided if the femoral artery needs to be accessed. Care must be taken not to injure the ureter that can be found at the common iliac bifurcation [10].

Common Femoral Artery

The common femoral artery lies just distal to the inguinal ligament until it divides into the SFA and profunda. It is found between the anterior superior iliac spine and the pubic tubercle, typically about 2 fingerbreadths lateral to the pubic tubercle. The incision should be started just above the inguinal ligament and carried out longitudinally along the medial border of the sartorius. A self-retaining retractor should be placed. Dissection should start laterally as the vein and lymph channels are more medial. The femoral sheath can be opened staying anterior to the artery. Occasionally bridging veins will

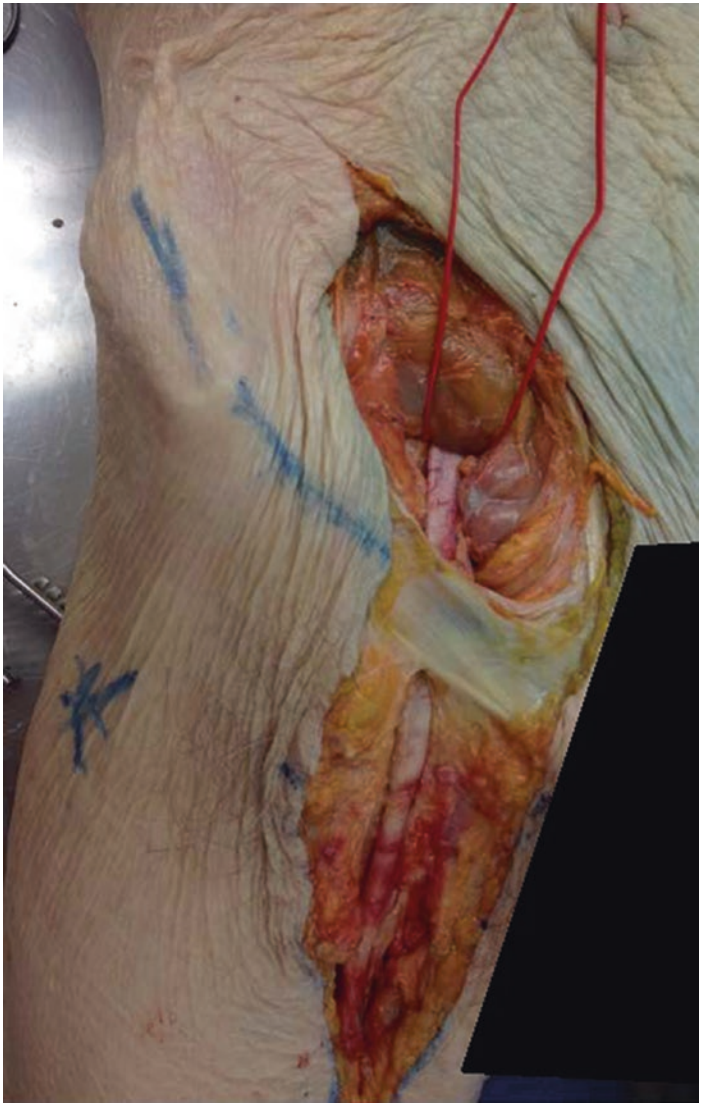

FIGURE 5.12 External iliac artery control above the inguinal ligament. The vessel loop is around the distal external iliac artery above the inguinal ligament

be encountered and these can be ligated. Exposure can be carried out to the bifurcation [10]. If there is difficulty exposing the proximal artery due to bleeding, it is wise to divide the inguinal ligament or use an extraperitonal approach to access the external iliac artery.

Proximal SFA and Profunda

The profunda is typically found 4–6 cm inferior to the inguinal ligament coursing posterolaterally. Once dissection of the common femoral bifurcation is performed as above, typically the takeoff of the profunda can be seen by a decrease in the diameter of the common femoral artery. Upward traction of the common femoral and superficial femoral artery can be obtained with vessel loops to aid in finding the origin of the profunda. Profunda control is performed by passing a vessel loop under the common femoral and then the SFA to avoid damaging the lateral circumflex vein [10].

Distal SFA

The SFA is a continuation of the common femoral on the anterior aspect of the thigh, subsartorial, until it enters the popliteal (Hunter's) canal. The adductor hiatus (Hunter's canal) is fascial-lined cleft located medially to the vastus muscles and lateral to the adductor muscles in the mid-thigh. Distal to the hiatus is the suprageniculate popliteal artery. Full exposure of the SFA is most easily achieved through an incision that parallels the lateral border of the sartorius. The sartorius is retracted medially to expose the roof of the canal to allow entry to the vessels.

In a large hematoma, more proximal control may be necessary as the anatomy may be distorted. The sartorius is a good coverage option for vascular repairs. Venous injury often accompanies arterial injury, so visualization with repair or ligation must occur if an injury is identified [10]. If soft tissue

coverage is needed for the vascular repair, a sartorius flap can be mobilized in this position.

Popliteal Artery

The popliteal artery is located between the adductor hiatus and the lower border of the popliteus muscle. Medially, the semitendinosus and its confluence with gracilis and sartorius and the semimembranosus cover the popliteal fossa and vessels. The veins are typically adherent to the artery. The tibial nerve is loosely attached to the vessel sheath within the popliteal fossa. The distal end of the popliteal artery is located at the hiatus by the origin of the soleus.

The knee should be flexed at 30° and laterally rotated. A bump or a rolled-up sheet can aid in this. The skin incision should be between vastus medialis and sartorius. The saphenous vein should be maintained and ideally left with the posterior skin flap. The incision can be carried distal to the knee, 1 cm posteriorly to the tibia. For full exposure, it is often necessary to divide the semimembranosus, semitendinosus, gracilis, and sartorius 2–3 cm from the insertion and tag each of these to reapproximate later. For the distal portion, the medial head of the gastrocnemius will need to be divided [10].

Postoperative Care

Postoperatively, the patients should be monitored for frequent neurovascular and pulse checks. The most immediate complication of vascular repair is early thrombosis. This can be suspected with pulse changes and signs of distal ischemia. Prompt identification of thrombosis is critical and operative re-exploration with revision is needed to salvage the limb. If ischemic signs are not present, revascularization may be delayed or further evaluated by angiography [11]. Any changes in pulse examination or Doppler signals

necessitate investigation, either with imaging or operative exploration [12].

Postoperative anticoagulation is not generally indicated following repair, unless previous condition exists such as hypercoagulable state, mechanical valve, etc. [11]. Antiplatelet therapy can be considered for below-knee bypasses, extrapolated from elective vascular disease.

Reperfusion injury may occur, producing hyperkalemia, hypoxia, and acidosis. These must be aggressively corrected. Any changes in pulse examination or Doppler signals necessitate investigation, either with imaging or operative exploration [12].

If a fasciotomy was not done at the initial operation, monitoring for signs of compartment syndrome is vital. If fasciotomy was performed, it is still critical to monitor vitals, pain, lab values, and renal function to assure that ongoing rhabdomyolysis is not present from a missed or inadequately released compartment. With worsening acidosis, CPK, or myoglobin, or worsening renal failure, re-exploration is needed to assure all compartments are fully released.

Infection at the site of vascular repair may lead to thrombosis, false aneurysm, or anastomotic disruption with hemorrhage. Close monitoring of the wounds must take place, and appropriate antibiotics and drainage are indicated if infection is suspected. If the anastomosis is infected, the vessel must be ligated and resected and bypass through a clean field performed [11].

At discharge, a daily walking program should be encouraged to improve flow to the local extremity. Smoking cessation should also be advocated. Aspirin administration of 162 mg for 3 months is encouraged. Due to poor patient follow-up with trauma patients, it is unknown what the courses of these vascular repairs are, but we offer patients follow-up at 1, 3, 6, and 12 months with duplex ultrasound by a vascular technician in a specialist vascular trauma clinic.

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References

1. Feliciano DV, Moore FA, Moore EE, West MA, Davis JW, Cocanour CS, et al. Evaluation and management of peripheral vascular injury: part 1. Western Trauma Association/critical decisions in trauma. J Trauma. 2011;70:1551–6.
2. Fox N, Rajani RR, Bokhari F, Chiu WC, Kerwin A, Eastern Association for the Surgery of Trauma, et al. Evaluation and management of penetrating lower extremity arterial trauma: an Eastern Association for the Surgery of Trauma practice management guideline. J Trauma Acute Care Surg. 2012;73(5 Suppl 4):S315–20.
3. Johansen K, Lynch K, Paun M, Copass M. Non-invasive vascular tests reliably exclude occult arterial trauma in injured extremities. J Trauma. 1991;31(4):515–9. discussion 519–22
4. Dente CJ, Wyrzykowski AD, Feliciano DV. Fasciotomy. Curr Probl Surg. 2009;46(10):779–839.
5. Feliciano DV, Moore EE, West MA, Moore FA, Davis JW, Cocanour CS, et al. Western Trauma Association critical decisions in trauma: evaluation and management of peripheral vascular injury, part II. J Trauma Acute Care Surg. 2013;75(3):391–7.
6. Feliciano DV, Mattox KL, Graham JM, Bitondo CG. Five-year experience with PTFE grafts in vascular wounds. J Trauma. 1985;25:71–82.
7. Martin LC, McKenney MG, Sosa JL, Ginzburg E, Puente I, Sleeman D, et al. Management of lower extremity arterial trauma. J Trauma. 1994;37:591–8.
8. Lau JM, Mattox KL, Beall AC Jr, DeBakey ME. Use of substitute conduits in traumatic vascular injury. J Trauma. 1977;17:541–6.
9. Feliciano DV. Management of peripheral arterial injury. Curr Opin Crit Care. 2010;16(6):602–8.
10. American College of Surgeons COT: ASSET—advanced surgical skills for exposure in trauma. 2010.
11. Feliciano D, Mattox K, Moore E. Trauma. 6th ed. New York: McGraw-Hill; 2008. p. 941–70.
12. Starnes BW, Beekley AC, Sebesta JA, Andersen CA, Rush RM Jr. Extremity vascular injuries on the battlefield: tips for surgeons deploying to war. J Trauma. 2006;60(2):432–42.

Part II
Techniques for Diagnosis and Resuscitation

Chapter 6
Intubation, Cricothyrotomy, Tube Thoracostomy, Diagnostic Peritoneal Lavage, and Local Wound Exploration

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Intubation

Introduction

Airway is the first concern after injury. The Eastern Association for the Surgery of Trauma (EAST) defines endotracheal intubation (ETI) as necessary in patients with airway obstruction, severe hypoxemia, hypoventilation, Glasgow Coma Scale ≤8, severe hemorrhagic shock, and cardiac arrest. EAST also emphasized the need for ETI in patients with smoke inhalation with evidence of airway obstruction, major cutaneous burns (>40% BSA), moderate to severe facial burns, or airway injury appreciated on endoscopy [1]. Other patient who will benefit from ETI include patients with facial or neck injury, persistent combativeness and concern for brain injury, and cervical spine injury with evidence of respiratory insufficiency.

Technique

Rapid sequence intubation (RSI) is used for emergent intubation in the critically ill trauma population. RSI is the simultaneous administration of both an induction agent and a paralytic to facilitate ETI. The bag valve mask should be applied to the face, and the patient should be passively preoxygenated with 100% oxygen using a jaw thrust or chin lift to open the airway. Active bagging should be avoided to prevent gastric distention. When done properly, RSI decreases the risk of complications including vomiting and aspiration while increasing the rate of successful intubation to approximately 98% [2].

The most common technique utilized in the trauma patient is direct laryngoscopy with cervical spine precautions. The laryngoscope is placed into the mouth, sweeping the tongue to the left of the oral cavity and inserting the tip of the laryngoscope blade into the posterior pharynx. The vocal cords and arytenoids should be seen. If the epiglottis is seen overhanging

the larynx, the laryngoscope should be further advanced into the vallecula to reveal the vocal cords. The endotracheal tube is introduced, passing directly through the cords and advanced to approximately 21–24 cm from the teeth. The balloon should be inflated and the endotracheal tube secured. Color capnography should be attached to the endotracheal tube, and color change should be demonstrated on six successive breaths. The oxygen saturation probe should be properly attached to the patient and continuously monitored. If present, end-tidal CO_2 should be monitored. The intubator should auscultate breath sounds and should observe the chest wall to visualize chest rise and fall with ventilation. A chest radiograph should ensure proper location of the endotracheal tube in the trachea [3].

For patients in which there is concern for, or a known, unstable cervical spine fracture, intubation should be preferably performed by an anesthesiologist or other skilled operators using a fiber-optic scope to prevent fracture displacement and/or worsening spinal cord injury.

Outcomes

Intubation is a well-tolerated and successful procedure if performed by an experienced individual.

Complications

Peri-intubation emesis and aspiration are likely the most common complications of intubation. Patients who are properly medicated prior to intubation have decreased complications including vomiting, aspiration, airway trauma, and death [4].

The difficult airway can often be handled using adjunctive airway devices like the gum elastic bougie to facilitate endotracheal tube placement or a laryngeal mask airway (LMA). The LMA is not considered a definitive airway, but can oxygenate and ventilate patients, while a definitive or surgical airway can be established.

The difficult airway may require placement of an emergent surgical airway. Transtracheal jet ventilation, needle cricothyroidotomy, and other emergent, invasive airway rescue techniques can be used to rescue the difficult airway, but these methods require highly skilled and well-versed physicians to be successful. Cricothyroidotomy is the preferred intervention for failed endotracheal intubation [5].

Cricothyrotomy

Introduction

Cricothyrotomy is the preferred procedure for establishing an emergent surgical airway. When attempts at orotracheal intubation fail, and a patient cannot be adequately ventilated by alternate means, the immediate solution is a cricothyrotomy. Emergent surgical airways are rarely needed in one study accounting for 0.3% of urgent and emergent airways [6].

The landmarks for cricothyrotomy are easily palpable in an adult, making the procedure one, which can be performed entirely using tactile input. The risk of esophageal injury is minimal, as the posterior wall of the airway is completely encased by the cricoid cartilage. The risk of injury to blood vessels or the recurrent laryngeal nerve is also minimal, as the lateral aspects of the cricothyroid membrane are bounded by the inferior horns of the thyroid cartilage. The cricothyroid membrane is superior enough to be away from the thyroid isthmus or superior thyroid arteries and veins.

A number of prepackaged kits exist for the purpose of performing percutaneous cricothyrotomy. These kits utilize a Seldinger technique of first introducing a needle, then a wire, and then a series of dilators, ultimately followed by a kit-specific airway device, through the cricothyroid membrane. The open surgical technique has advantages over these percutaneous kits and has been demonstrated to be superior in the simulated environment [7].

Technique

As time and patient stability allow, prep and drape the anterior neck. Grasp the patient's larynx between the thumb and middle finger of the nondominant hand. This "laryngeal handshake" will provide a continuous reference to midline, throughout the procedure. Identify the thyroid and cricoid cartilages, as well as the cricothyroid membrane between them. Make a vertical incision extending from over the mid-thyroid cartilage to over the mid-cricoid cartilage. In larger individuals, a larger incision may be required to define the anatomy. With the blade oriented horizontally, insert the scalpel blade straight through the middle of the cricothyroid ligament, penetrating 2–3 mm. Incise the membrane to the lateral margins where it is bounded by the cartilaginous structures of the thyroid cartilage (Fig. 6.1). Dilate the opening with a finger or the handle of the scalpel. Insert the tracheal hook, and retract the laryngeal cartilage, anterior and cephalad (Fig. 6.2). Insert the airway

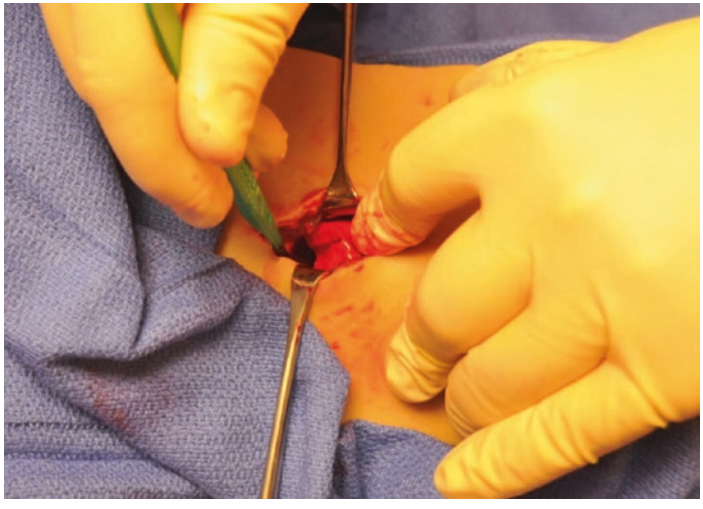

FIGURE 6.1 Incise the cricothyroid membrane laterally in both directions to the margins defined by the thyroid cartilage, and then dilate the opening with the handle of the scalpel or with your finger

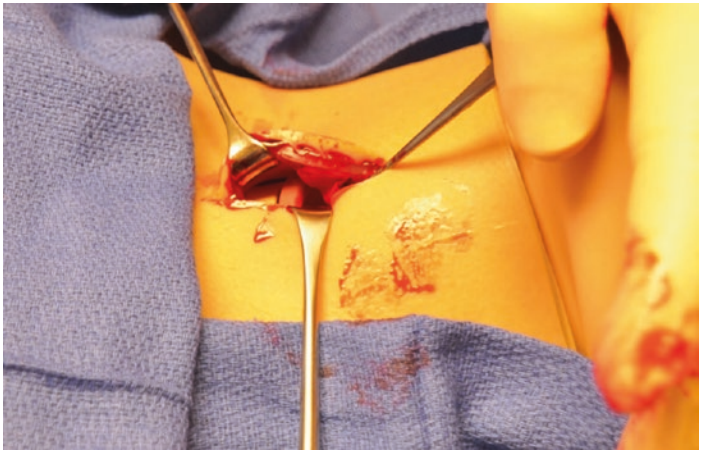

FIGURE 6.2 Insert a tracheal hook and lift the thyroid cartilage cephalad and anterior

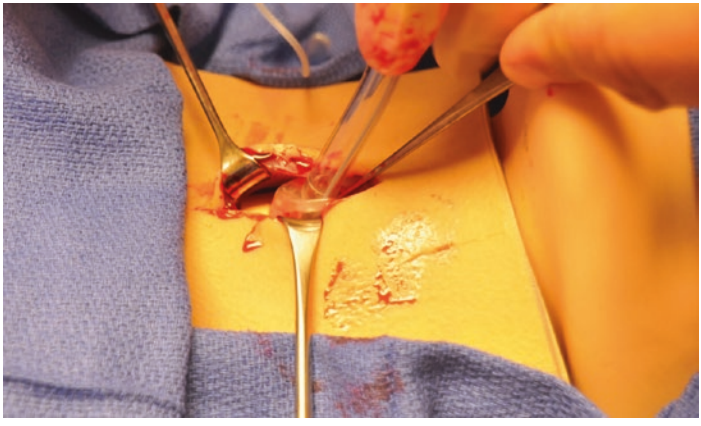

FIGURE 6.3 Insert the airway

(Fig. 6.3). Here we use a 6.0 ETT, but a 6.0 internal diameter Shiley or similar tracheostomy device will also work. We prefer the ETT as the operator can customize the curve of the tube, likely making insertion easier. Once the airway is through the cricoid cartilage into the trachea, remove the tracheal hook. Ventilate and confirm airway position with end-tidal CO_2.

Complications

The worst complication of a cricothyrotomy is failure to appropriately cannulate the trachea, usually by creating a false tract when attempting to place the airway device. Immediate confirmation of airway position by the use of end-tidal CO_2 detection is imperative in order to immediately identify the mal-placed airway.

Bleeding is an inevitable complication to some degree. Practice performing the cricothyrotomy using only tactile input beforehand. It is rare, given the confines of the cricothyroid cartilaginous cage, to encounter hemodynamically significant hemorrhage if the midline and lateral margins of the operative field are defined with a firm "laryngeal handshake."

Because the cricoid ring is a closed circumferential ring around the airway, prolonged use of a cricothyrotomy may lead to scarring and stenosis at the cricoid level. Cricothyrotomies should be converted to tracheostomies in a controlled environment within 3 days of placement.

Critical Take-Home Points

- Be decisive about recognizing the need and proceeding with a cricothyrotomy. The biggest risk is failing to perform it quickly when needed.
- Establish control of the larynx and define the midline using a "laryngeal handshake."
- Once the cricothyroid membrane is entered, always have something inserted in the tract to maintain it: the scalpel, your finger, a tracheal hook, etc.
- Practice this procedure so you will not hesitate to perform it when required.
- Practice performing this procedure using only tactile input, as visual references will rapidly disappear with even minimal incision bleeding in the trauma bay.

Tube Thoracostomy

Introduction

After ensuring an adequate airway, correction of hemodynamic compromise, inadequate oxygenation, or inadequate ventilation due to hemothorax or pneumothorax is the most immediately correctable cause of trauma mortality during a primary survey. This is done in the resuscitation bay by means of placement of a tube thoracostomy (TT). TT is the procedure of choice for the initial management of traumatic hemothorax or pneumothorax.

Indications for tube thoracostomy in the trauma patient include clinical suspicion for a tension pneumothorax or massive hemothorax, any identified hemothorax on CXR or CT scan [8], and/or any pneumothorax visualized on CXR. While some debate exists, the current EAST Practice Management Guidelines states: "Occult pneumothorax, those not seen on chest radiograph, may be observed in a stable patient regardless of positive pressure ventilation (Level 3)" [8]. For the acute trauma patient in the resuscitation bay, there are no absolute contraindications to tube thoracostomy. Relative contraindications in the more stable patient include known pleural disease and coagulopathy.

History

While the use of tubes to aspirate empyema goes back as far as Galen and Celsus in the Roman age [9], the first modern published account of continuous pleural space drainage was published by Dr. F. Cresswell Hewett, A.M.D., in 1876 [10]. Significant reductions in the rate of posttraumatic empyema were shown during the Franco-Prussian War of 1870–1871 using Hewett's technique when applied to empyema [11] and were widely endorsed as the preferred alternative to open management after the US experience in World War I [9]. This approach was further cemented during World War II, where

TT with an underwater seal became the standard of care for simple traumatic hemothorax and pneumothorax.

Technique

Patient Prep and Positioning

If possible, place the patient in a lateral recumbent position with the arm extended over their head. If this is not safe, placing the ipsilateral arm as far over the patient's head as possible will help expand the lateral chest wall and define anatomic landmarks, as well as provide a reasonable degree of exposure. Unless the patient is in extremis, the time should be taken to thoroughly prep widely around the site of insertion and place a maximal sterile barrier drape.

Make sure all requisite equipment is in place, including the following: the chest tube; two large clamps, one for occluding the chest tube and the other for guiding the tip of the tube during placement; a large curved Kelly clamp for bluntly dissecting through the intercostals and parietal pleura; suture for securing the chest tube; and water-seal or equivalent suction apparatus setup and ready for use.

Tube thoracostomy with inadequate or, worse, no analgesia in a conscious patient is one of the more brutal procedures performed in the trauma bay. While the use of fentanyl or other systemic opioid analgesics is helpful, it is unlikely to be adequate except in large doses. Thorough regional anesthetic use is requisite. The ease of performing tube thoracostomy is inversely proportional to the degree of patient discomfort.

Choice of Tube

Large chest tubes have traditionally been used for the drainage of hemothorax in trauma patients, having less the chance to clot and becoming occluded. No advantage is seen in the use of very large (36–40 Fr) chest tubes over the use of more moderate (28–32 Fr) tubes in the multi-trauma patient [12]. Use of tubes smaller than 28 Fr is not wise.

Incision and Finger Thoracostomy

A routine trauma chest tube should be placed in the fifth or sixth intercostal space (Fig. 6.4). This will roughly correspond to the intercostal space at or just superior to the level of the inframammary crease. Extending the ipsilateral arm of the patient over their head will often reveal a rectangular area of relatively accessible ribs lying along the fifth or sixth intercostal space posterolateral to the pectoral groove at the anterior axillary line and anterior to the soft tissue bulk associated with the latissimus dorsi, which often extends as far forward as the midaxillary line.

Place the incision immediately over the rib inferior to the intercostal space you plan to enter. Bluntly dissect through the subcutaneous tissue and intercostal muscles, spreading the tissue with the tips of a large curved Kelly clamp (Fig. 6.5). Creating a tract through the subcutaneous tissue slightly cephalad and posterior from the skin incision will help direct the tube in the cephalad and posterior direction as it enters the chest.

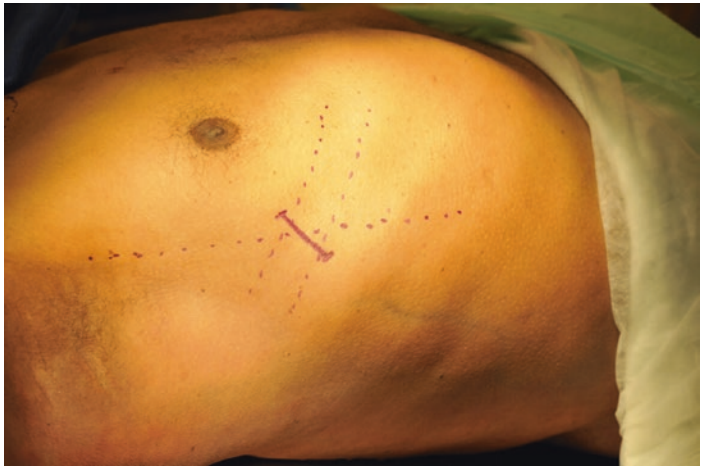

Figure 6.4 External landmarks. The dashed lines represent the anterior axillary line and the superior and inferior margins of the 6th rib. The solid line is the location for our incision

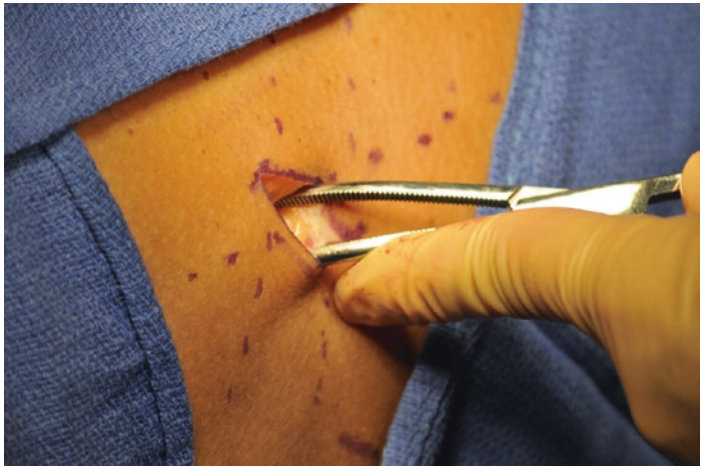

Figure 6.5 Blunt dissection through subcutaneous tissue

Place the tip of the closed Kelly clamp against the rib, grasp it approximately 2 cm from the skin surface, and firmly push the clamp over the rib and through the intercostal muscles just superior to the rib (Fig. 6.6). There should be a "pop" or give as the underlying parietal pleura is punctured. Spread the Kelly clamp, actively parting the intercostal muscles just superior to the rib until you can gain entry to the thoracic space with your index finger.

With your finger now in track. Next, hook your finger against the parietal pleura, and turn your finger completely around the thoracostomy, ensuring the lung and chest wall are separated and free of adhesions. If the lung is adhered to the chest wall, consider an alternate site for chest tube insertion [11].

Tube Insertion

Clamp the end of the chest tube to prevent blood spilling on the floor. Slide the tube along the palmar aspect of the finger you are keeping in the thoracic cavity (Fig. 6.7). Pass the tube posterior and superior to the lung. Advance the tube to the

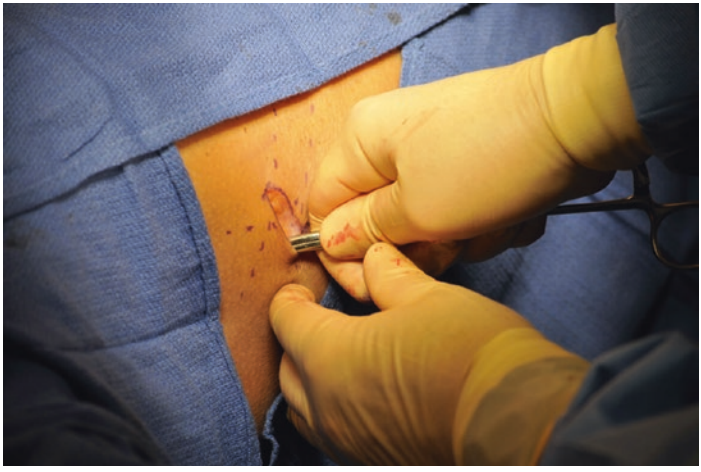

FIGURE 6.6 Entering the pleura. Note the Kelly clamp held firmly 1–2 cm from the skin to prevent overinsertion

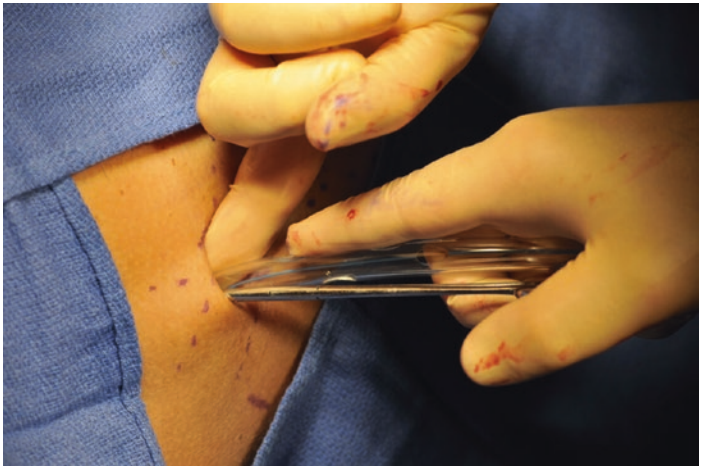

FIGURE 6.7 Inserting the chest tube. Slide the tube along the palmar aspect of your index finger, replacing your finger with the tube in the pleural space

depth far enough to ensure the most proximal side port of the tube is well within the thoracic space. Verify the tube is within the thoracic space by feel. Connect the tube to suction or water seal.

Place a purse-string mattress suture around the tube, and close the skin snugly against the tube, leaving long trailing suture on either end of your knot. Then wrap each end of the suture two or three times around the tube in opposing directions and pull tight. The suture should be pulled tight enough to slightly indent the plastic of the tube; otherwise, the tube will be free to slide in and out of the suture "collar." We entwine the tube with Xeroform gauze and snug this up against the insertion site followed by gauze.

Complications

Complication rates following tube thoracostomy have been reported as being as high as 25%. By far, the most common complication is malposition [13]. Tube malposition is common and has been reported to be as high as 20% [14]. Incorrect placement can result in a lack of adequate or any drainage from the pleural space or damage to internal organs.

Intraparenchymal placement occurs when the tube is advanced toward a preexisting defect in the lung parenchyma. This can be avoided by fastidious efforts to direct the tube along the chest wall during insertion. Placement in the fissure occurs when the tube is advanced medially. Overinserted tubes are often found kinked or bent as the tip of the tube abuts either the apex of the pleural space or the mediastinum. Underinserted tubes, unless recognized while an entire sterile field is still in place, must be replaced, as advancing a tube will track nonsterile material into the chest cavity.

Empyema rates following TT for trauma have been reported as high as 10%. Empyema following TT is almost always caused by skin flora introduced at the time of inser-

tion. The best prevention of this complication is fastidious adherence to aseptic technique throughout tube insertion.

The most likely cause of new hemorrhage as a complication of tube thoracostomy is damage to the intercostal vessels. This can be best avoided by using a blunt dissection technique and by ensuring access is gained immediately over the top of the rib.

Diagnostic Peritoneal Lavage

Introduction

Although falling out of practice in modern-day trauma care secondary to the widespread use of ultrasound and CT scans, diagnostic peritoneal lavage (DPL) remains an important procedure. Ultrasound is insensitive to visceral organ injury, and it is within this population of patients, which DPL may continue to have a role.

History

First introduced by H. David Root in the mid-1960s, DPL is used to diagnose occult intra-abdominal hemorrhage in a small population of patients. In his paper, Root demonstrated 100% accuracy among 28 patients in identifying intra-abdominal injuries [15]. Several important practices were also outlined in Root's paper that would improve the reliability of the procedure, namely, minimizing skin bleeding to prevent contamination of the intra-abdominal contents, peritoneal aspiration prior to lavage to evaluate the presence of gross blood, rocking the patient after lavage and prior to withdrawal to ensure good mixing of the fluid, and lavaging/removing as much as possible. It has been used ever since as a technique to determine the presence of intraperitoneal hemorrhage.

Within the past 10 years, bedside ultrasound has become prevalent in trauma resuscitation bays and has essentially supplanted the use of DPL in the diagnosis of potential

hemoperitoneum. The Focused Assessment with Sonography for Trauma (FAST) exam can be done rapidly and safely identifying as little as 100 mL of blood in the abdomen when done by an experienced practitioner [16].

Technique

Primarily used in the setting of hemodynamically unstable trauma patients with blunt abdominal trauma, DPL is a straightforward procedure. There are two primary approaches to DPL, percutaneous or open. Infraumbilical approaches are preferred in all patients except those with pelvic fractures and pregnant women in which the supraumbilical approach is safer to avoid entering the pelvic hematoma or injuring the gravid uterus. Regardless of the technique used, a Foley catheter and nasogastric tube should be placed to decompress the bladder and stomach, respectively, prior to starting the procedure. All supplies should be ready at the bedside.

For the percutaneous procedure: The abdomen is prepped and draped in a sterile fashion. A needle is passed in the midline through the abdominal wall into the peritoneal cavity (Fig. 6.8) in a caudad direction through which a guidewire is advance (Fig. 6.9). The needle is removed and a small incision is made in the skin where the guidewire enters the abdomen. The catheter is then placed over the guidewire and inserted into the peritoneal cavity, and the guidewire is removed (Fig. 6.10). A 10 mL syringe is attached to the catheter and aspirated (Fig. 6.11). Aspiration of 10 mL of blood or bloody fluid is considered a positive result. If there is an absence of blood, or less than 10 mL of blood is aspirated, 1 L of lactated ringers or normal saline is infused into the peritoneal cavity. To improve the sensitivity, the patient can be tilted or rolled or the abdomen otherwise maneuvered to encourage distribution of the fluid. Once the fluid is infused, the bag is placed on the ground, and the fluid is siphoned back into the IV fluid bag. The fluid is sent for laboratory analysis.

The open procedure: Again, the abdomen is prepped and draped in normal sterile fashion. Local anesthetic is used. A

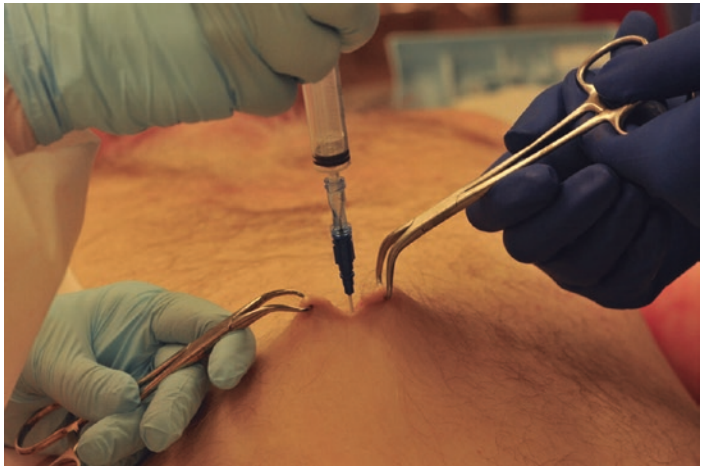

FIGURE 6.8 A needle is advanced through the abdominal wall into the peritoneum

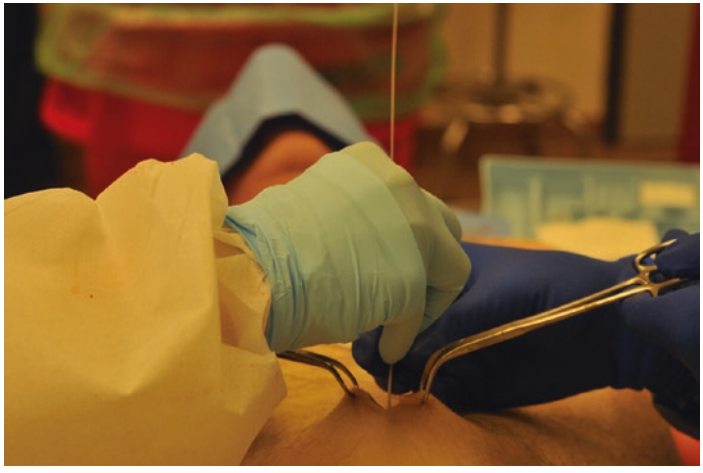

FIGURE 6.9 Guidewire advanced through needle into the peritoneum

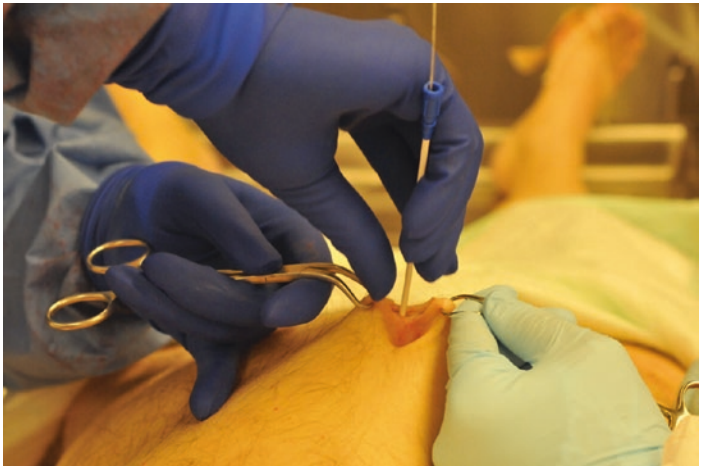

FIGURE 6.10 A catheter is placed over the guidewire, and the wire is removed

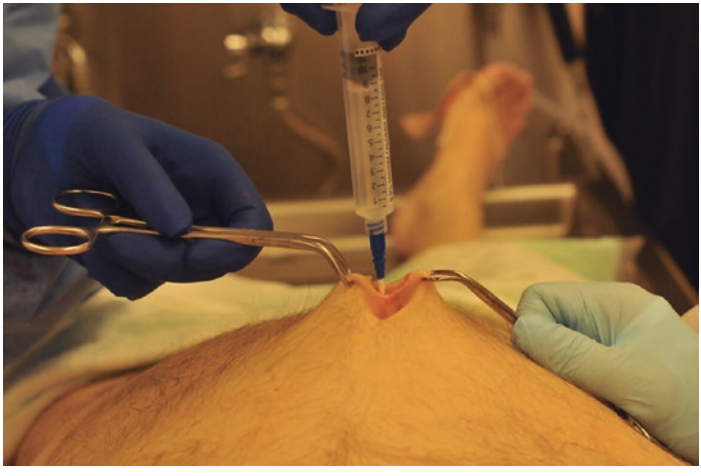

FIGURE 6.11 Aspirate off the catheter with a 10 mL syringe

midline abdominal incision is made approximately one-third of the way between the umbilicus and the symphysis pubis down to the fascia. The fascia is then incised exposing the peritoneal cavity. A catheter is then placed through the fascia into the peritoneum, and the following steps are the same as in the percutaneous technique.

Outcomes

Interpretation of the results is based on immediate return of 10 mL of gross blood upon initial aspiration or a threshold of red blood cells noted in the lavage fluid after laboratory evaluation; see Table 6.1 for laboratory result interpretation.

Currently, the only contraindication to DPL is a previously determined need for laparotomy. Relative contraindications exist, but in general, DPL is well tolerated with few complications. There is no difference in outcome or complications associated when comparing the percutaneous to open techniques [17].

Complications

The most common complications that occur with DPL include local wound infections or dehiscence and bowel,

Table 6.1 Diagnostic peritoneal lavage—red blood cell criteria

	Positive	**Indeterminate**
Stab wounds		
Anterior abdomen	100,000	20,000–100,000
Flank	100,000	20,000–100,000
Back	100,000	20,000–100,000
Low chest	5000–10,000	1000–5000
Blunt abdominal trauma	100,000	20,000–100,000
Gunshot wounds	5000–10,000	1000–5000

bladder, or vascular injury. Bleeding from the incision can result in a false-positive lavage. Overall complications occur in less than 1% of patients undergoing this procedure [18].

Local Wound Exploration

Introduction

Severity of penetrating abdominal trauma can be difficult to evaluate. Specifically, determining if a hemodynamically stable patient with stab wounds to the anterior abdomen requires an exploratory laparotomy or CT scan can be difficult. Local wound exploration (LWE) at the bedside can help clarify the depth of injury and guide further management. LWE should be used only for anterior abdominal wall stab wounds and not for wounds in the flank or back.

History

In 1977, Erwin Thal published the first paper evaluating LWE in combination with DPL for anterior lower chest and abdominal stab wounds. His data were based on 123 patients who had a combination of physical exam, LWE, and DPL and found a reduction in unnecessary laparotomies (69.9% of patients with negative LWE and DPL were spared) [19].

Technique

The goal of local wound exploration is to fully evaluate the depth of penetrating wounds to determine if the posterior peritoneal fascia has been violated. The use of local anesthetic containing epinephrine should be considered to help maintain a bloodless field for exploration. Each wound should be thoroughly examined at every layer of tissue, and if necessary, the wound can be extended to improve exposure, which can include the use of retractors or even extending the wound. Once it has been determined that no penetration of

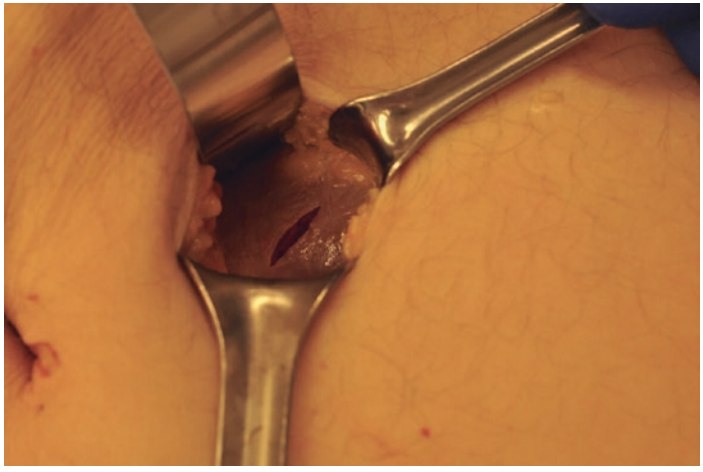

Figure 6.12 Visualization of a violated peritoneal fascia

the posterior peritoneal fascia has occurred, the patient can be safely discharged home assuming they have no other conditions that require inpatient management. If posterior fascia has been violated, then the next management steps are variable (Fig. 6.12). In a stable patient, CT scan of the abdomen utilizing triple contrast to maximize sensitivity or serial exams is an option.

Outcomes

In a hemodynamically stable patient with abdominal wound that can be evaluated with LWE and for which it can be determined there has been no posterior fascial penetration, serial abdominal exams are likely the sufficient management. In patients in whom full exploration of the wound is impossible due to depth, tracking, or poor hemostasis, DPL or imaging with CT scanning should be pursued to rule out an intraperitoneal involvement.

Local wound exploration has no role in the hemodynamically unstable patient with penetrating abdominal wounds.

References

1. Mayglothling J, Duane TM, Gibbs M. Emergency tracheal intubation immediately following traumatic injury. J Trauma. 2012;73(5):S333–40.
2. Reynolds SF, Heffner J. Airway management of the critically ill patient: rapid sequence intubation. Chest. 2005;127:1397.
3. Marx JA, Hockberger RS, Walls RM, editors. Rosen's emergency medicine: concepts and clinical practice, vol. 1. 6th ed. St. Louis: Mosby; 2006.
4. Li J, Murphy-Lavoie H, Bugas C, Martinez J, Preston C. Complications of emergency intubation with and without paralysis. Am J Emerg Med. 1999;17(2):141–3.
5. Salvino CK, Dries D, Gamelli R, Murphy-Macabobby M, Marshall W. Emergency cricothyroidotomy in trauma victims. J Trauma. 1993;34(4):503–5.
6. Stephens CT, Kahntroff S, Dutton RP. The success of emergency endotracheal intubation in trauma patients: a 10-year experience at a major adult trauma referral center. Anesth Analg. 2009;109(3):866–72.
7. Heard AMB, Green RJ, Eakins P. The formulation and introduction of a "can't intubate, can't ventilate" algorithm into clinical practice. Anesthesia. 2009;64:601–8.
8. Mowery NT, Gunter OL, Collier DO, Diaz JJ, Haut E, Hildreth A, et al. Practice management guidelines for management of hemothorax and occult pneumothorax. J Trauma Inj Infect Crit Care. 2011;70(2):510–8.
9. Molnar TF, Hasse J, Jeyasingham K, Reneki S. Changing dogmas: history of development in treatment modalities of traumatic pneumothorax, hemothorax, and posttraumatic empyema thoracis. Ann Thorac Surg. 2004;77(1):372–8.
10. Hewett FC. Thoracentesis: the plan of continuous aspiration. Br Med J. 1876;1(793):317.
11. Fitzgerald M, Mackenzie CF, Marasco S, Hoyle R, Kossman T. Pleural decompression and drainage during trauma reception and resuscitation. Injury. 2008;39(1):9–20.
12. Inaba K, Lustenberger T, Recinos G, Georgio C, Velmahos GC, Brown C, et al. Does size matter? A prospective analysis of 28-32 versus 36-40 French chest tube size in trauma. J Trauma Acute Care Surg. 2012;72(2):422–7.

13. Kesieme EB, Dongo A, Ezemba N, Irekpita E, Jebbin N, Kesieme C. Tube thoracostomy: complications and its management. Pulm Med. 2012;2012, article ID 256878.
14. Huber-Wagner S, Korner M, Ehrt A, Key MV, Pfeifer KJ, Mutschler W, et al. Emergency chest tube placement in trauma care – which approach is better? Resuscitation. 2007;72(2):226–33.
15. Root HD, Hauser CW, McKinley CR, Lafave JW, Mendiola RP Jr. Diagnostic peritoneal lavage. Surgery. 1965;57:633–7.
16. Dolich MO, McKenney MG, Varel JE, Compton RP, McKenney KL, Cohn SM. 2,576 Ultrasounds for blunt abdominal trauma. J Trauma. 2001;50(1):108–12.
17. Hodgson NF, Stewart TC, Girotti MJ. Open or closed diagnostic peritoneal lavage for abdominal trauma? A meta-analysis. J Trauma. 2000;48:1091.
18. Fabian TC, Mangiante EC, White TJ, Patterson CR, Boldreghini S, Britt LG. A prospective study of 91 patients undergoing both computed tomography and peritoneal lavage following blunt abdominal trauma. J Trauma. 1986;26:602.
19. Thal ER. Evaluation of peritoneal lavage and local exploration in lower chest and abdominal stab wounds. J Trauma. 1977;17:642–8.

Chapter 7
Ultrasound for Point-of-Care Imaging: Performing the Various Exams with Technical Tips

Daniel J. Haase and Sarah B. Murthi

Introduction

Ultrasound (US) use has increased dramatically in trauma patients over the last 30 years. It has uses in heavily resourced as well as remote, underdeveloped regions. Ultrasound is being studied everywhere, even on the international space station [1]. A good working knowledge of ultrasound is essential when caring for trauma patients.

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Focused assessment with sonography for trauma (FAST) is an important innovation in ultrasound, not because it is a technological advancement but because it redefines who can use ultrasound and when [2, 3]. The FAST establishes the safety and feasibility of using US to diagnose and manage trauma in real time, thus creating the concept of point-of-care ultrasound (POCUS). Currently, POCUS includes both diagnostic imaging, like the FAST, and ultrasound for procedural guidance, including central lines and percutaneous drains. More advanced Doppler capabilities make US useful as physiologic measurement tool as well. A variety of cardiac exams can be used to guide complex resuscitation from the trauma bay to the ICU [4–7]. Taken together, the US is a portable stethoscope, CT scanner, and pulmonary artery catheter in one.

History

Ultrasound has been used in medicine since the 1940s, but the technology was understood long before. In the 1700s, an Italian physicist, Lazzaro Spallanzani, first recognized that bats used echolocation. With the improved understanding of ultrasound physics and the discovery of the piezoelectric effect (the coupling of mechanical and electrical forces allowing the interpretation of ultrasound waves), the ability to use US for diagnostics was born. In 1942, Austrian Neurologist Karl Dussik used an "ultrasonic apparatus" to diagnose brain tumors. In 1948, George Ludwig, an American naval officer, first used US to diagnose gallstones. In 1958, Scottish physician Ian Donald pioneered the use of ultrasound for obstetrics. Through the 1950s, US technology advanced from large machines requiring submersion of the patient in fluid, to smaller devices, to modern handheld ultrasounds [8]. With the increasing access and availability, as well as the decreasing cost, US has spread across medical specialties. With telesonography and virtually guided sonograms, this market will only continue to grow [9].

Focused Assessment with Sonography for Trauma

Physical exam after trauma is frequently unreliable as 20–40% of patients with intra-abdominal bleeding will have a normal exam [10, 11]. Prior to the use of FAST, diagnostic peritoneal lavage (DPL) or surgical exploration was the primary assessment of intra-abdominal bleeding. In most institutions, the FAST exam has essentially replaced the DPL [12]. In the current 10th edition of ATLS, DPL has become an optional skill station [13], while ultrasound is considered an adjunct to the primary survey. Early investigators realized that fluid is easy to see on ultrasound and that FAST could be used to rapidly, noninvasively detect blood in the pericardium and abdomen [14–16].

The traditional FAST has four views: right upper quadrant (RUQ) or Morrison's pouch, left upper quadrant (LUQ), pelvis (P) or pouch of Douglas in women, and subxiphoid (SX) pericardium. The extended FAST (eFAST) adds anterior lung windows to evaluate for PTX [17]. Some centers have added functional evaluation of the heart and the inferior vena cava (IVC) to the eFAST [18, 19]. The eFAST is a reliable and repeatable exam, with a compressed learning curve. While experience yields improved accuracy, the ability to perform an adequate exam is obtained after only a few exams [20, 21].

In hypotensive patients with blunt abdominal trauma, FAST is 100% specific and sensitive for blunt abdominal trauma [14, 22]. In a blunt trauma patient with hypotension, a FAST showing fluid is an indication for operative exploration. Absence of fluid makes it unlikely that intra-abdominal, thoracic, or pericardial bleeding is the cause of hypotension. Furthermore, in stable patients with blunt trauma, FAST will miss up to 30% of injuries, as it misses isolated visceral injury without significant hemorrhage [23–25]. These patients should get further evaluation—usually a CT scan or serial abdominal exams.

In hypotensive patients with penetrating trauma, the FAST is 100% specific and 99.3% sensitive for precordial wounds [14, 22]. For all penetrating torso injuries, the FAST exam was highly specific (94.1–100%), but not adequately sensitive (28.1–100%) [26] for intra-abdominal injury [26]. The authors concluded that a positive FAST was adequate to prompt exploratory laparotomy, even in hemodynamically stable patients. A negative FAST exam does not rule out significant intra-abdominal injuries and should prompt further diagnostic imaging.

In 2004, Kirkpatrick coined the term eFAST and found that the eFAST was comparable in specificity to CXR but had superior sensitivity (48.8% vs. 20.9%) in diagnosing PTX. Other series confirm that US is superior to CXR to diagnose PTX but can fail to detect a small PTX [27, 28].

At the Shock Trauma Center, we perform an eFAST on almost all trauma patients. We believe that a positive FAST in a stable patient with blunt injury can result in more rapid workup and better triage. It may also provide important diagnostic information should a patient deteriorate prior to further diagnostic workup. A positive abdominal FAST in a stable patient with penetrating torso injury should likely prompt an exploratory laparotomy in almost every case. Furthermore, we believe that routine performance of the eFAST exam is necessary for trainees to obtain and maintain ultrasound skills.

General Tips for Learning Ultrasound

Regardless of the exam being performed, the following tips are suggested:

1. *Get to know your machine*: This is the most important factor in safely and reliably using ultrasound.
2. *Clean the transducer and keyboard*: Infection control and patient safety are key points that are often overlooked but are critical to patient safety.

3. *Turn the machine on*: All machines will have an on/off or power button. It is useful to keep the machine plugged in during and between uses. Machines left on may run low on battery power, and start-up can take longer.
4. *Enter the patient data*: Learn how to enter patient data and save the study. All ultrasound machines require a patient identifier to save images, even if it is a temporary identifier.
5. *Select the proper transducer*: The appropriate transducer for the desired exam should be selected.
6. *Select the proper exam type (preset)*: Generally, the machine will default to the last exam. Most machines can be set to start in a particular exam type that will optimize your imaging. Again, it is important to know your machine!
7. *Find the gain and depth controls*: Manipulate depth and gain to optimize images.
8. *Develop patience:* BE PATIENT! Even with the eFAST exam, slow and steady scanning through more than one plane is very important.

Which Transducer to Select for the eFAST?

There is much debate about preferred transducer selection for the eFAST exam. While there is no definitively correct answer, there are benefits and drawbacks of each of the transducers. In general, low-frequency transducers (commonly the curvilinear [C] and phased array [PA]) are best for deeper abdominal or thoracic imaging. High-frequency (HF) transducers (most commonly the linear) are best for imaging superficial structures, such as the pleural line or blood vessels for central line insertion. See Fig. 7.1a–c.

The phased array (frequently called the "cardiac") transducer may be best for imaging between the ribs in the RUQ and LUQ due to its small footprint. In the RUQ and LUQ, the curvilinear (frequently called the "abdominal") transducer may have difficulty maintaining contact with the skin on thinner patients with less subcutaneous tissue. The curvi-

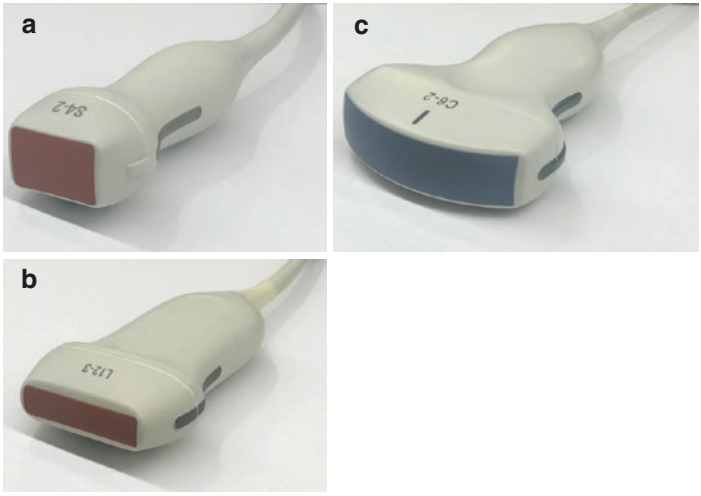


FIGURE 7.1 (**a**–**c**) Common ultrasound transducer types: (**a**) phased array, (**b**) linear, (**c**) curvilinear

linear transducer is best for imaging transabdominal structures such as the aorta, bladder, or gallbladder.

For pneumothorax detection, the sonographer is only trying to visualize pleural line. Thus, use of a high-frequency transducer may be most appropriate. In larger patients in whom the pleural line is several centimeters deep, a low-frequency transducer may be preferred.

In general, we suggest taking the time to use the optimal transducer for each of the six areas on the eFAST exam. This may mean taking several seconds to change transducers. However, we believe that improved image quality leads to more precise decision-making in critically ill trauma patients.

Pathology in the eFAST

Diagnosis of Hemorrhage

Fluid will appear black and collect in dependent areas. It is assumed that "fluid" is blood in a trauma patient; however, it

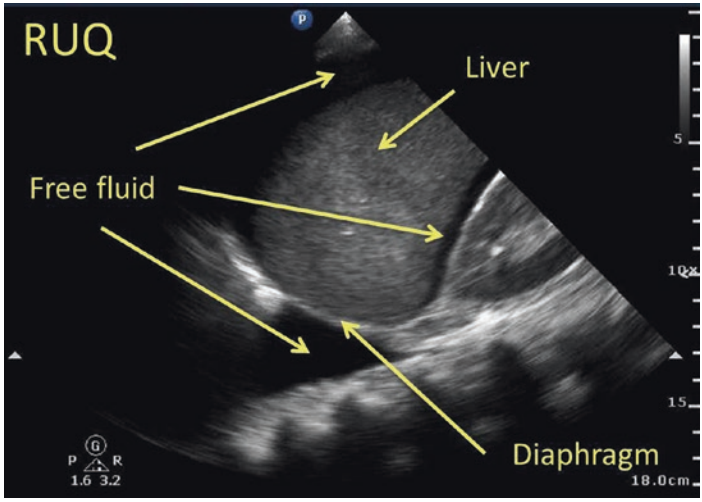


FIGURE 7.2 Free fluid in the right thorax and RUQ

could be ascites, free bladder rupture, or bowel contents in the abdomen (Fig. 7.2). In older patients, pericardial and pleural effusions are more common and must be considered as a possibility. In addition, clotted blood may have a similar echogenicity (or brightness) to solid organs and can mimic the appearance of those organs.

Diagnosis of Pneumothorax

Diagnosis of a PTX has a steep but short learning curve. A normal lung on the contralateral side makes comparative diagnosis easier. US cannot penetrate air, so the finding is a *loss* of signal in the potential space between the parietal and visceral pleura of the chest. Normally, the parietal and visceral pleural interface "slides," creating a sparkle or dancing effect, termed *sliding*. If pleural surfaces are intact, transmitted cardiac pulsations can be seen in the pleural line – this is called lung pulse. When a PTX is present, lung sliding and lung pulse are lost. A *lung point* occurs when the transition

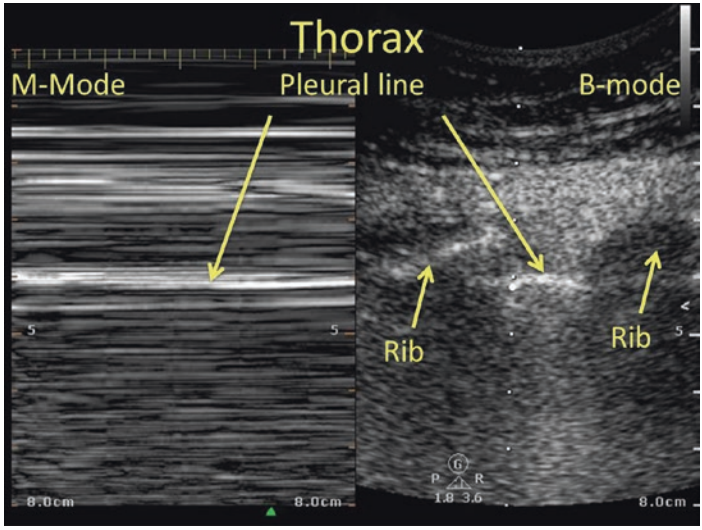


FIGURE 7.3 B-mode on right with cursor placed in between adjacent ribs through the pleural line. M-mode imaging on left with "barcode sign"

from sliding to no sliding can be clearly seen; it is 100% specific for the diagnosis of PTX [29]. Lack of lung sliding alone can be caused by other processes including pneumonia, bullous disease, and other pleural pathologies.

M-mode can help confirm the PTX diagnosis and may be very helpful when initially learning to detect it, but the assessment can be entirely made from 2D imaging. On M-mode, a normal pleural interface will show the bright line of pleura with a smattering of signal returning to the probe from the lung parenchyma, called *seashore sign.* If air is present from a PTX, it will stop the signal, and instead of seashore sign, there will be a series of horizontal lines sometimes called *barcode* or *stratosphere sign* (Figs. 7.3 and 7.4).

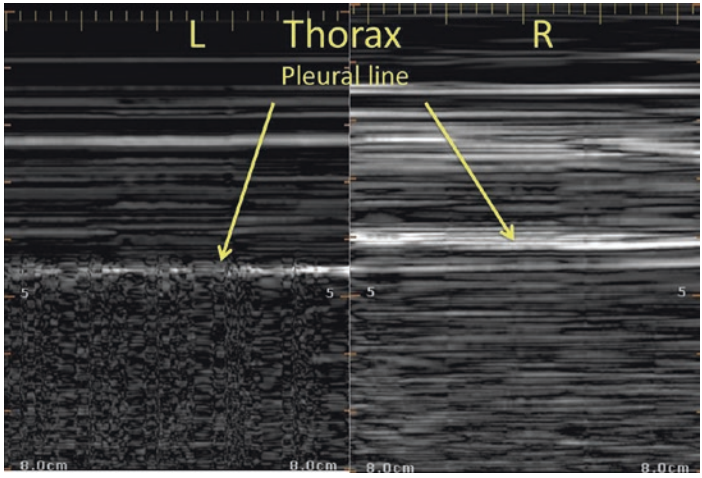


FIGURE 7.4 M-mode on right with "barcode sign" versus a normal M-mode image of lung sliding on left

eFAST Views

Hepatorenal Space or Right Upper Quadrant (RUQ)

The probe is placed along the ribs at the midaxillary line, oriented vertically as in the LUQ view. The right kidney is more anterior than the left. The liver appears as a homogenous gray, and the fascia of Glisson's capsule is a bright white line. Once the kidney is identified, fanning across the area can reveal any free fluid. Visualizing at the caudal tip of the liver maximizes sensitivity. As with the LUQ, the probe is angled cranially to view above the diaphragm to look for free fluid in the thorax. See Figs. 7.5, 7.6, and 7.7.

Critical Image Imaging down to the caudal tip of the liver (or spleen in the LUQ) improves the sensitivity of identification of free fluid (Fig. 7.8).

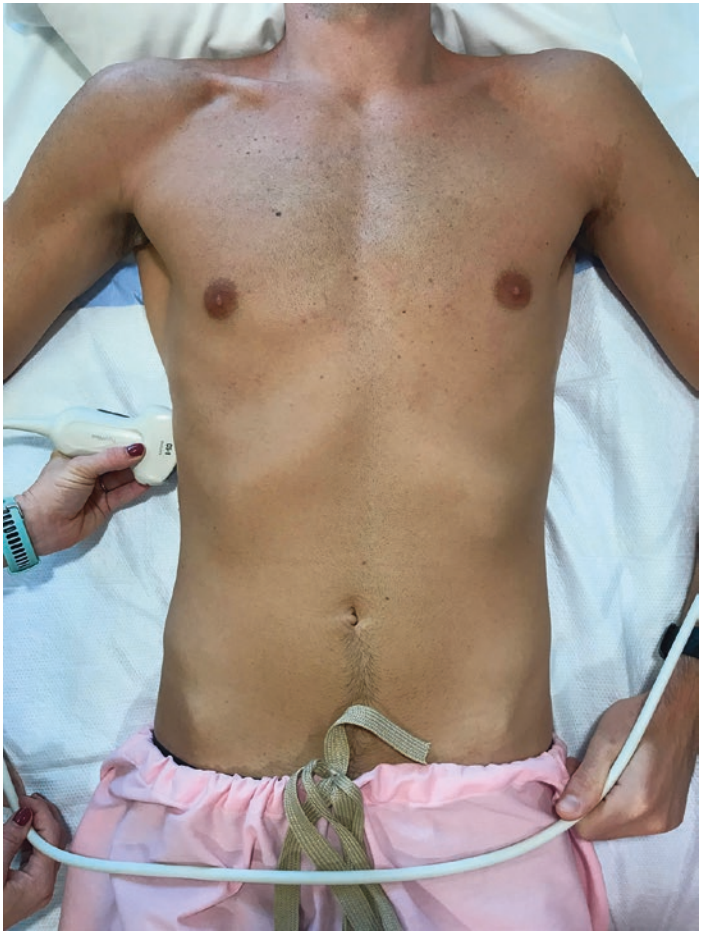


FIGURE 7.5 Curvilinear transducer positioned in the midaxillary line to view the hepatorenal space

Splenorenal Space or Left Upper Quadrant (LUQ)

The transducer is moved to the posterior axillary line, below the costal margin on the left side. It may be necessary to press your knuckles into the gurney and angle the transducer anteriorly, as the view can be very posterior. Start at the

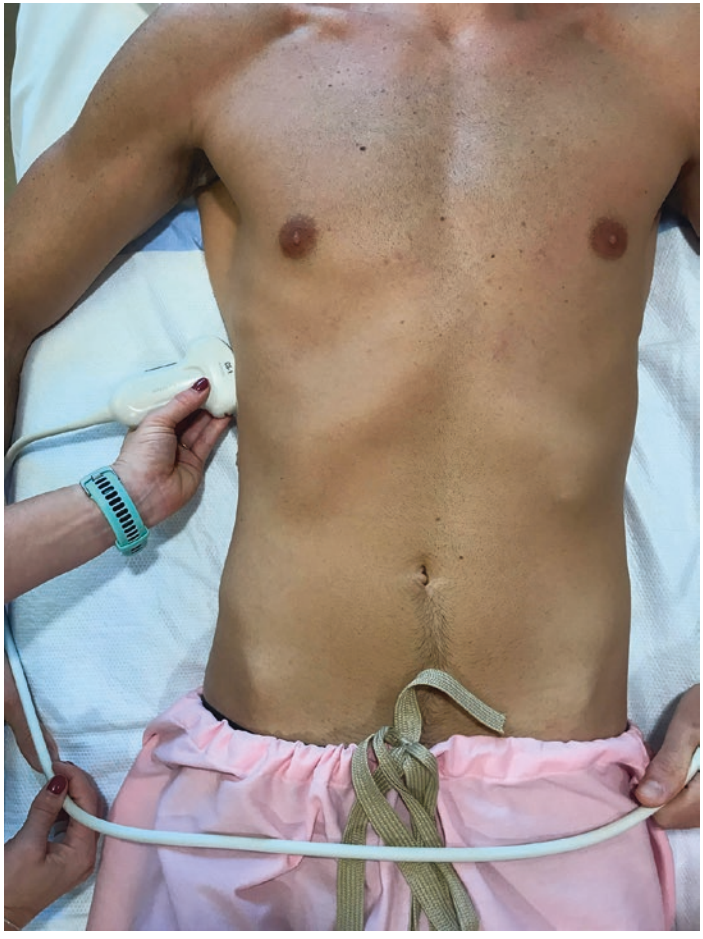


FIGURE 7.6 Transducer now angled up to view diaphragm and possible presence of hemothorax in the right thorax

costophrenic angle and then move caudally until the kidney comes into view. The probe should be fanned across the entire kidney. The space between the spleen and diaphragm should be viewed to avoid missing fluid above it. A simple cephalic sweep allows visualization of posterior diaphragmatic recess to assess for hemothorax. See Fig. 7.9.

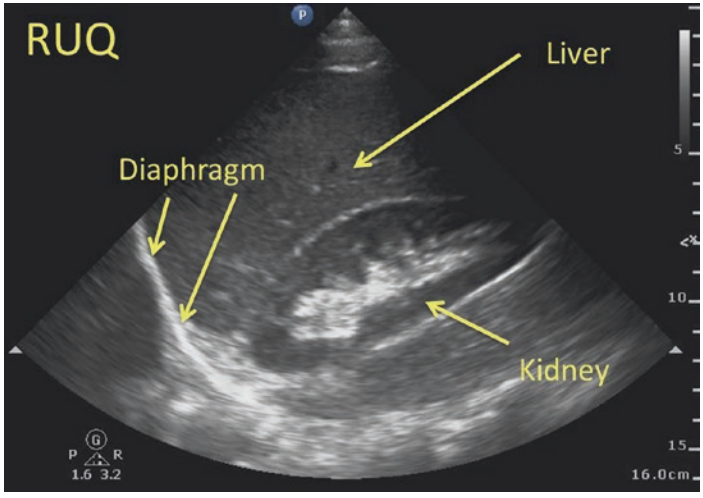


FIGURE 7.7 Negative imaging of the RUQ with views of both R thorax and hepatorenal space

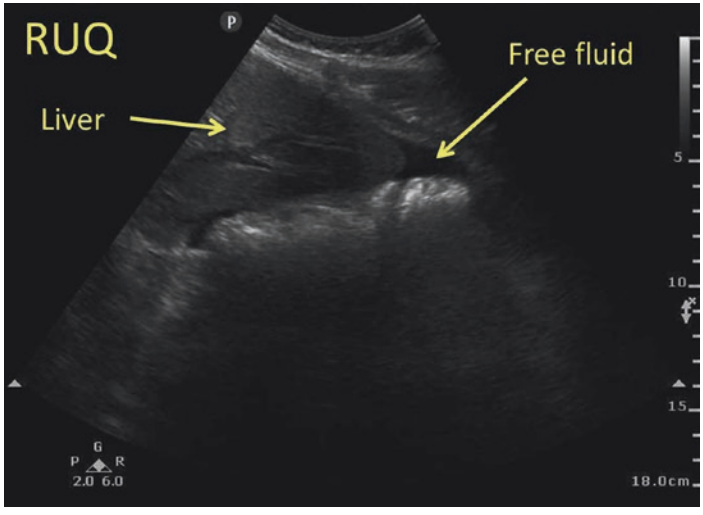


FIGURE 7.8 Imaging down to the caudal tip of the liver (or spleen in the LUQ) improves the sensitivity of identification of free fluid

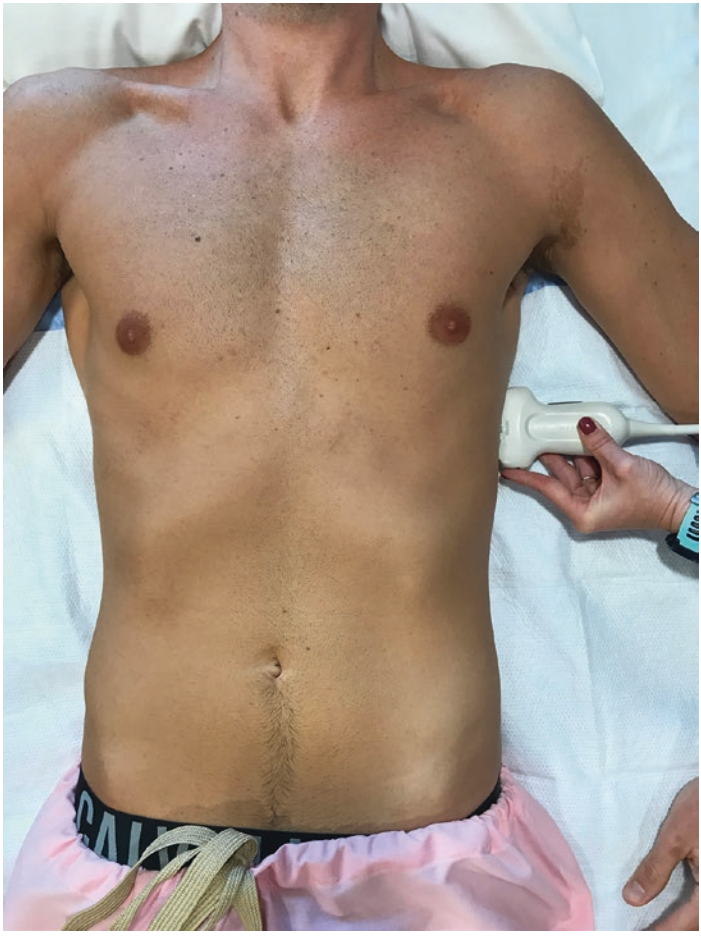


FIGURE 7.9 The curvilinear transducer is slightly more superior and posterior when compared with the RUQ

Critical Images Free fluid present in both the splenorenal space and above the spleen (Fig. 7.10). Left hemothorax present in the LUQ (Fig. 7.11).

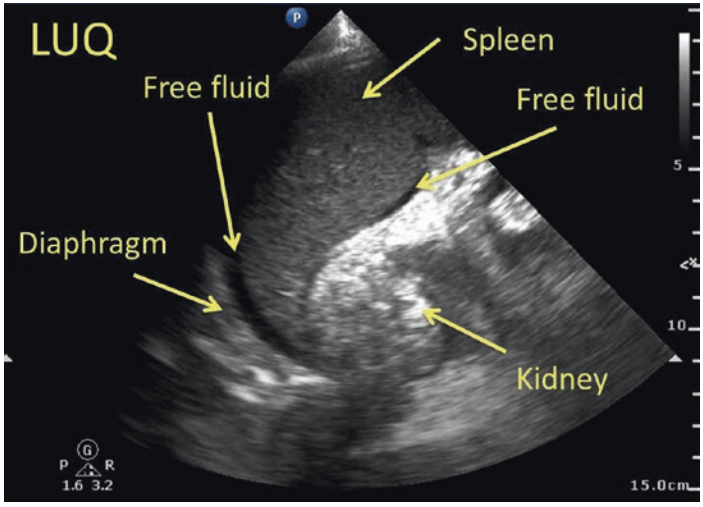


FIGURE 7.10 Free fluid present in both the splenorenal space and above the spleen

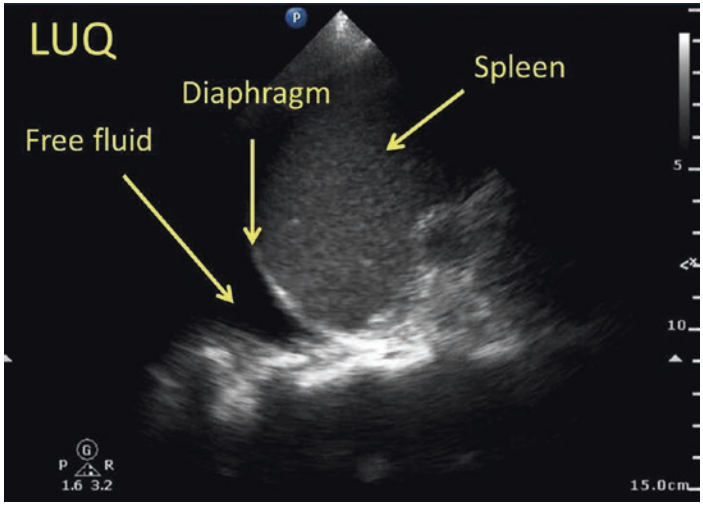


FIGURE 7.11 Left hemothorax present in the LUQ

Pelvis (P)

The transducer is placed just above the pubic symphysis along the midline abdomen, angled toward the feet. The indicator is oriented to the patient's right. The fluid-filled bladder is identified (appearing more square in shape), and a caudal-cranial sweep is performed to obtain adequate transverse views. Longitudinal views can be added to improve sensitivity. The transducer is rotated 90° so the probe marker is now toward the patient's head. A lateral (left to right) sweep is then performed. The goal is to evaluate for fluid in the retrovesical space in men (between the pelvic floor and bladder) or pouch of Douglas in women. See Figs. 7.12, 7.13, 7.14, and 7.15.

Critical Image Positive pelvic FAST with fluid in pouch of Douglas, posterior to the uterus (Fig. 7.16). Positive longitudinal view of the bladder with free fluid posteriorly. Note the Foley balloon in the bladder (Fig. 7.17).

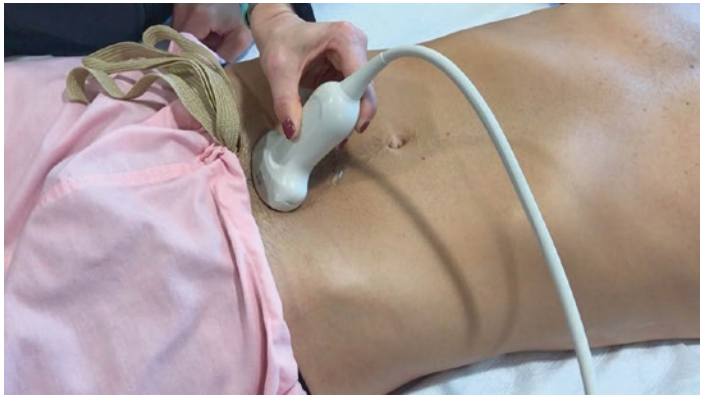


FIGURE 7.12 Curvilinear transducer with transverse view of bladder. Note how the transducer is angled down into the pelvis

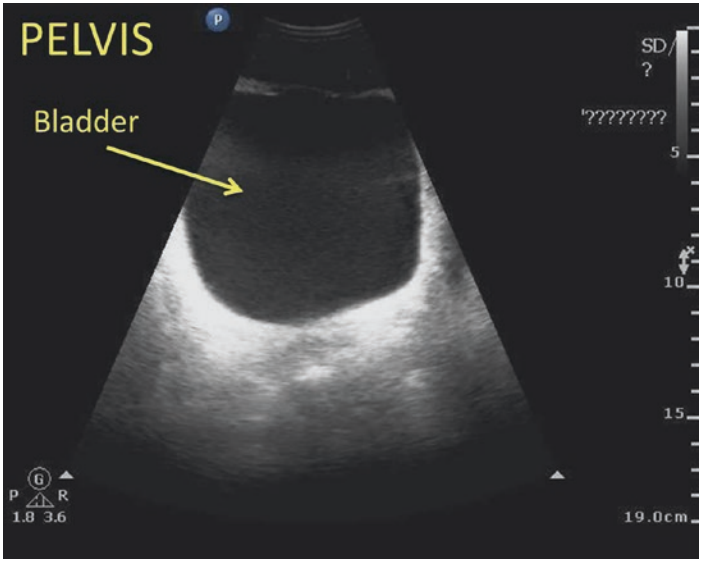


FIGURE 7.13 Transverse view of the pelvis with urine-filled bladder

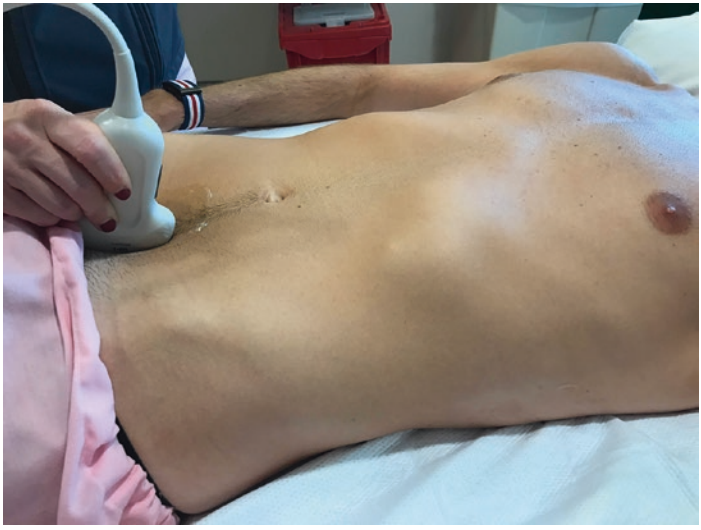


FIGURE 7.14 Transducer now with indicator toward the patient's head for a longitudinal view of the bladder. The transducer remains angled into the pelvis

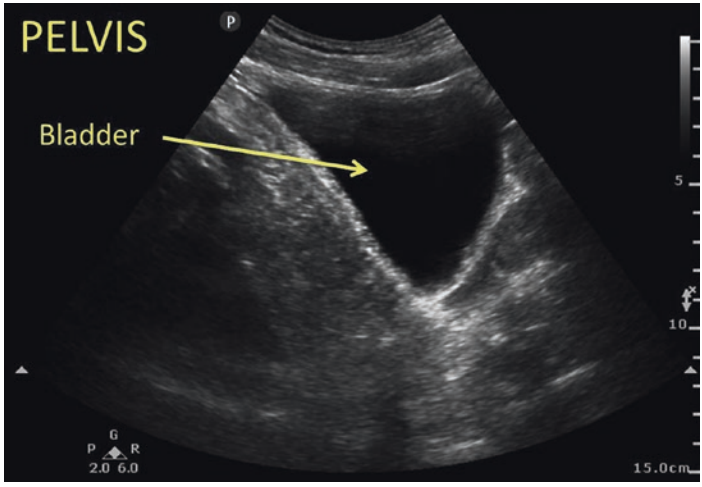


FIGURE 7.15 Negative longitudinal view of bladder

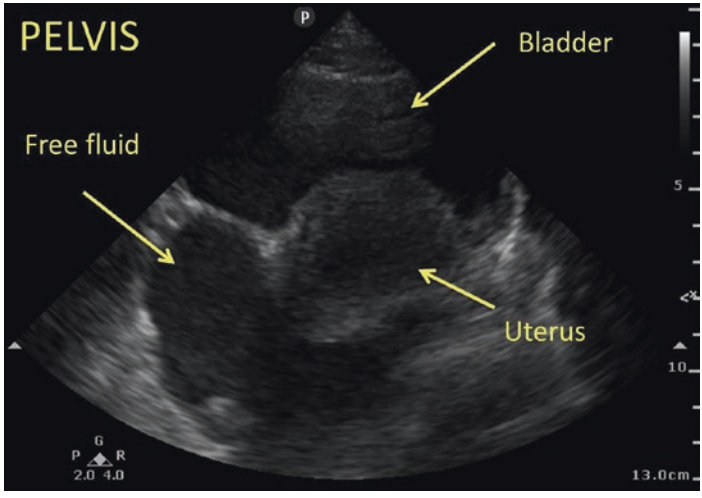


FIGURE 7.16 Positive pelvic FAST with fluid in pouch of Douglas posterior to the uterus

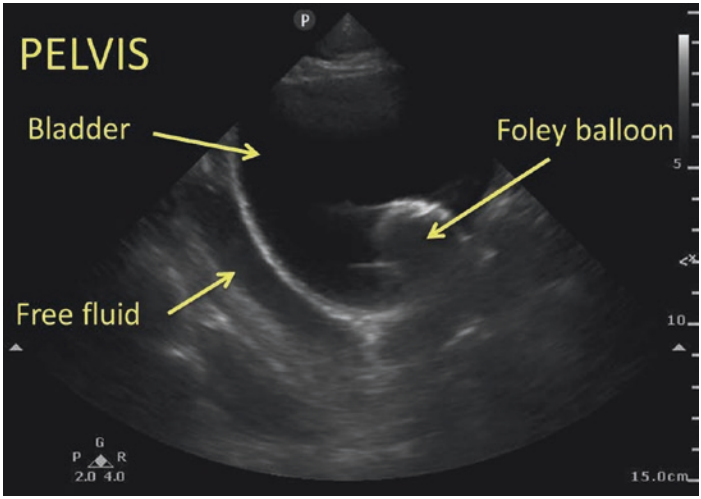


FIGURE 7.17 Positive longitudinal view of the bladder with free fluid posteriorly. Note the Foley balloon in the bladder

Pitfall The transducer should be angled caudally, down toward the patient's feet. The pelvic structures cannot be seen if the transducer is angled perpendicular to the abdomen.

Note Multiple view of the pelvis improves sensitivity. Small amounts of fluid in women can be physiologic. This window is best obtained with a full bladder. An empty bladder can mimic free fluid. Identification of landmarks (bladder/uterus) is necessary to determine a "negative" pelvic FAST.

Pericardial Space or Subxiphoid (SX)

Classically, the transducer is placed about 2–3 cm below the xiphoid, with the indicator to the right, and pointed to the left shoulder, attempting to look "under" the xiphoid to the heart.

The transducer should be nearly flat against the abdomen, when compared with a more perpendicular angle in the other views.

When SX imaging is difficult due to bowel gas, the transducer should actually be moved toward the RUQ under the costal margin, using the liver as an acoustic window. See Figs. 7.18 and 7.19.

Critical Image In the space between the right ventricle and the liver, blood will appear as a black stripe below the pericardial bright white strip (Fig. 7.20).

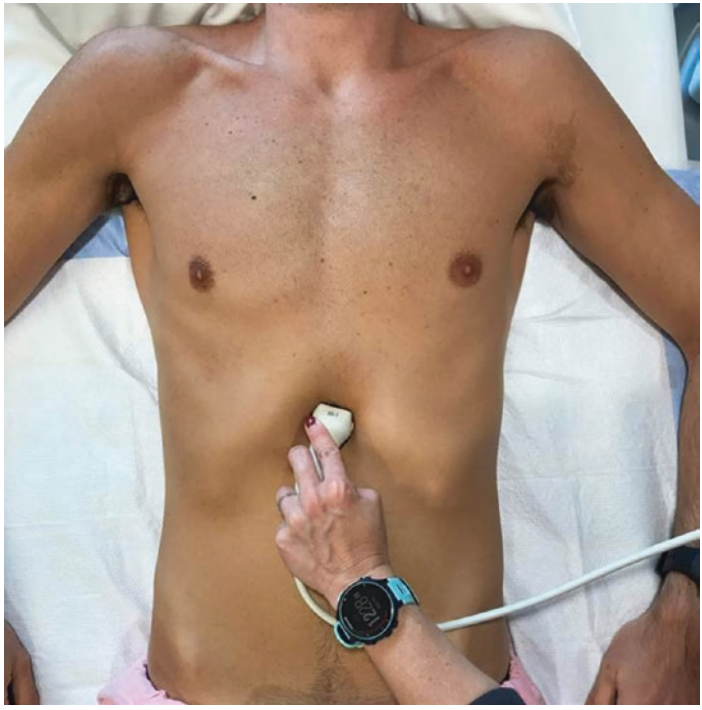


Figure 7.18 SX with phased array transducer

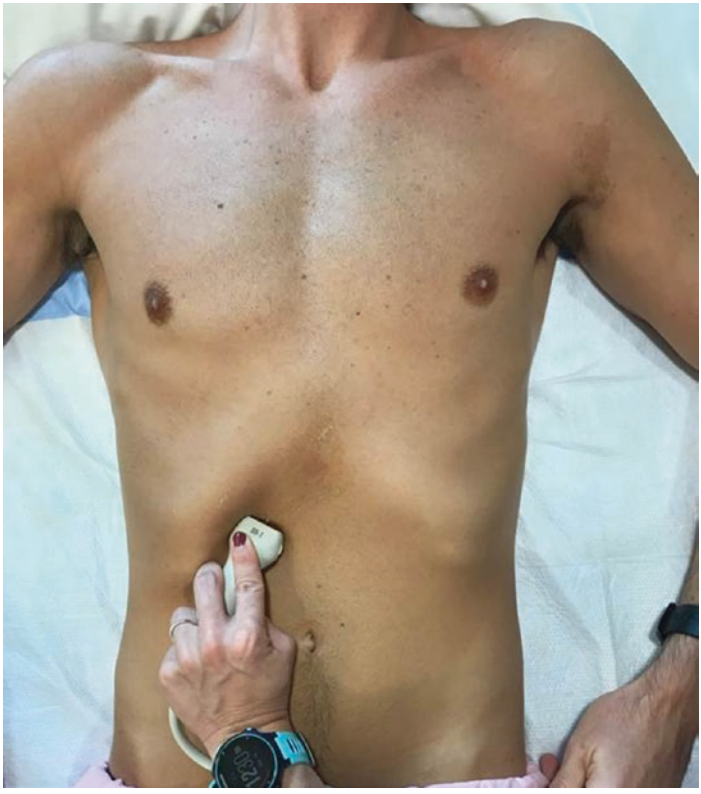


FIGURE 7.19 SX with transducer moved to RUQ to use liver as acoustic window

Pitfall When performing a SX analysis after the abdominal views, the depth on the US machine needs to be increased to see the beating heart.

Parasternal Long (PSL)

Frequently, a SX view is difficult or impossible due to patient body habitus, bowel gas, or abdominal guarding. A PSL view

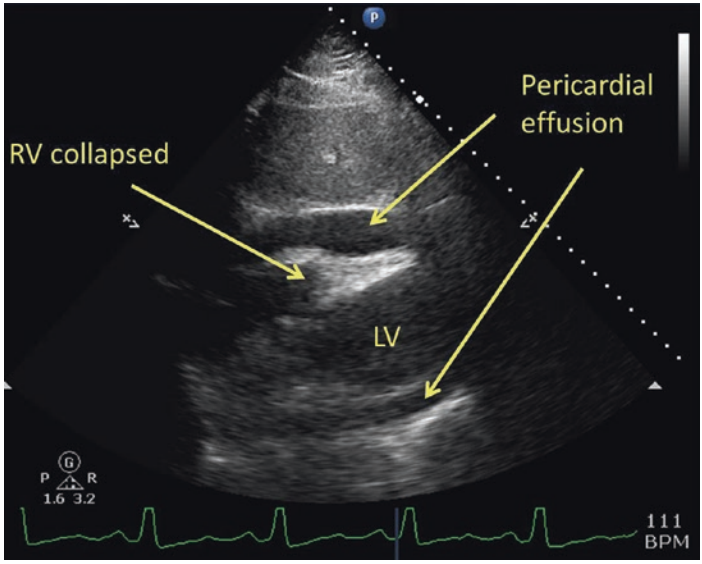


FIGURE 7.20 In the space between the right ventricle and the liver, blood will appear as a black stripe below the pericardial bright white strip

may be obtained by placing the phased array transducer just left of the sternum at the patient's 3rd–4th intercostal space with the indicator pointed toward the patient's left shoulder. Fluid will appear black and should accumulate posteriorly between the left ventricle and pericardium. In older, more obese patients, epicardial fat may appear dark gray and is typically only anterior. Do not confuse epicardial fat with pericardial fluid. See Fig. 7.21.

Critical Image PSL view with posterior pericardial effusion anterior to descending thoracic aorta (Fig. 7.22). PSL with pleural effusion that is posterior to descending thoracic aorta (Fig. 7.23).

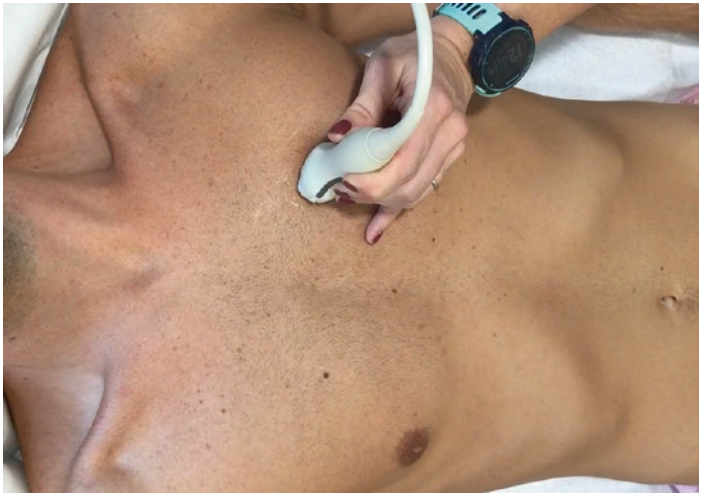


FIGURE 7.21 PSL view with phased array transducer

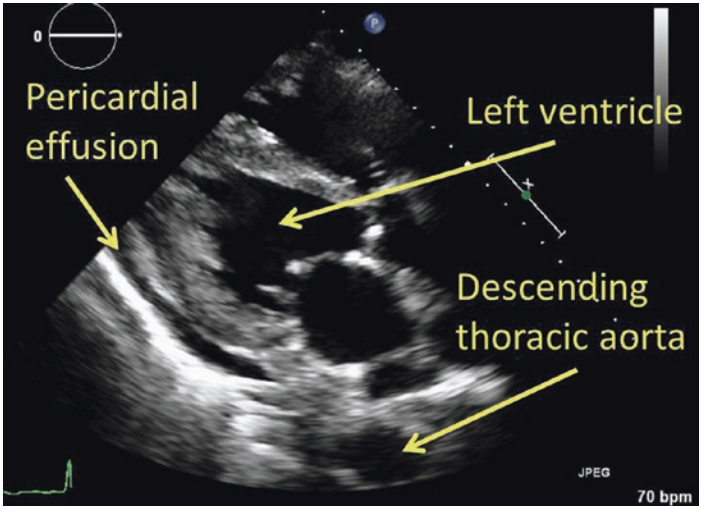


FIGURE 7.22 PSL view with posterior pericardial effusion anterior to descending thoracic aorta

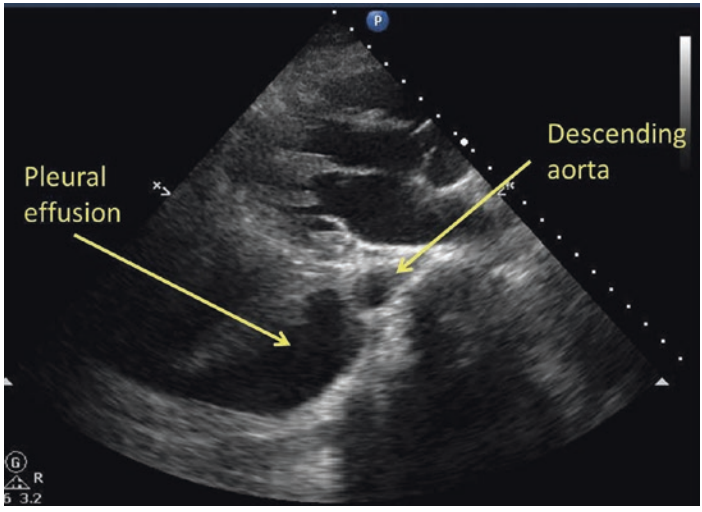


FIGURE 7.23 PSL with pleural effusion that is posterior to descending thoracic aorta

Pitfall In the case of penetrating cardiac trauma, blood can decompress into the chest rendering the pericardial window negative. In this case, thoracic ultrasound (or chest X-ray) will show a hemothorax. Suspicion of a cardiac injury must remain high.

Right and Left Anterior Thorax

The transducer is moved to the least dependent area of the left and right chest. In a supine patient, this is usually the midclavicular line of the second or third rib. A HF probe can be used, but it is not a requirement. The probe is oriented transversely across the rib. An acoustic shadow artifact makes the rib easy to see. Just below the rib, a shiny horizontal line representing the interface of the parietal and visceral pleura can be seen. M-mode can be applied to confirm 2D imaging if available.

Critical Image The pleural line of the most anterior aspect of both chests (Figs. 7.2 and 7.3).

What Is the Appropriate Order of the eFAST Exam?

When evaluating patients with blunt trauma, the clinical situation dictates whether to start with chest or abdominal imaging. In patients with hypoxia or significant chest pain or shortness of breath, whether hemodynamically stable or unstable, we recommend starting with PTX assessment because chest tube insertion can be performed immediately in the ED and while the sonographer proceeds to abdominal imaging. Otherwise, in patients without significant chest complaints or findings, we prefer the following series of imaging:

RUQ → LUQ → Pelvis → Pericardium → R Thorax → L Thorax

Again, we recommend that the sonographer assess the posterior diaphragmatic recess for hemothorax while imaging the hepatorenal and splenorenal spaces in the abdomen.

In general, we recommend a systematic approach to abdominal imaging (RUQ → LUQ → Pelvis), starting with the RUQ because it is the most commonly "positive" area in the abdomen and is most sensitive for hemoperitoneum [30].

For penetrating thoracoabdominal trauma, we recommend starting with pericardial imaging (subxiphoid and/or parasternal long axis) to assess for cardiac tamponade and effusion. We then recommend proceeding to PTX assessment with the HF transducer on both sides of the thorax. The sonographer should then proceed to the abdominal views.

Pericardium → R Thorax → L Thorax → RUQ → LUQ → Pelvis

Pearls and Pitfalls of the FAST

- Should be able to be performed in 3–4 minutes
- Low-frequency probe (3–5 MHz), curvilinear probe (C), or phased array (PA)
- High-frequency probe for PTX detection (pleural line imaging) is preferred.
- Ultrasound should be performed during the primary survey in an unstable patient and the secondary survey in a stable patient. It should never interfere with resuscitation. The patient is more important than the eFAST.
- A negative abdominal FAST does not rule out clinically significant injury. It may frequently miss mesenteric, bowel, retroperitoneal, or diaphragmatic injury.
- The abdominal FAST is poor at detecting fluid volumes less than 400 mL.
- The eFAST is more sensitive and specific than CXR in the diagnosis of PTX.
- Repeated eFAST exams improve sensitivity and should be repeated with clinical changes.

IVC Ultrasound in the FAST

The IVC can be easily visualized from the SX window, by simply fanning over the liver and finding the IVC/RA junction. The probe is then rotated so the IVC is in long axis. Both the diameter of the IVC (< 1 cm, 1–2 cm, and >2 cm or FLAT vs. FAT) and its respiratory variations are reflective of volume status. M-mode can be used to better quantify both measures. The measurement is relatively easy and has utility in initial fluid management. If the patient is on positive pressure ventilation, it is unreliable.

Cardiac Ultrasound

Background

The assessment of volume status, responsiveness to fluids, and cardiac function is difficult, especially in the intubated patient. Focused cardiac ultrasound (FOCUS), like the FAST, is performed by the treating physician to answer specific questions. With a 1-day course and a limited number of proctored exams, practitioners can become competent in diagnosing severe LV and RV cardiac dysfunction [6, 31].

The focused rapid echocardiographic evaluation (FREE) is a hybrid between a formal echo and a bedside cardiac ultrasound. The FREE incorporates measurements of cardiac function and volume status, with clinical information, and characterizes hemodynamics [5]. The majority of the time, one can place left ventricular function into "depressed," "normal," or "hyperdynamic" categories. The right ventricle can occasionally be assessed as well as "full" or "empty" [6, 32].

Cardiac Views

There are four standard windows: the parasternal long axis (PSL), parasternal short axis (PSS), apical four chamber (A4C), and SX. Echo-based presets will generally orient the groove on the right, which is the opposite of the abdominal presets. Familiarity with all windows is important, as each window provides different information. If performing the exam in abdominal or FAST presets, the probe would need to be turned 180°. Here are the standard cardiac views:

1. *PSL*: The transducer is placed to the left of the sternum from the second to the fourth interspace in non-intubated patients and fourth to sixth interspace in mechanically ventilated patients (Fig. 7.21). The groove is oriented to the patient's right shoulder, and the transducer is gently rocked under the sternum. The LV can be seen in long axis (Fig. 7.24).

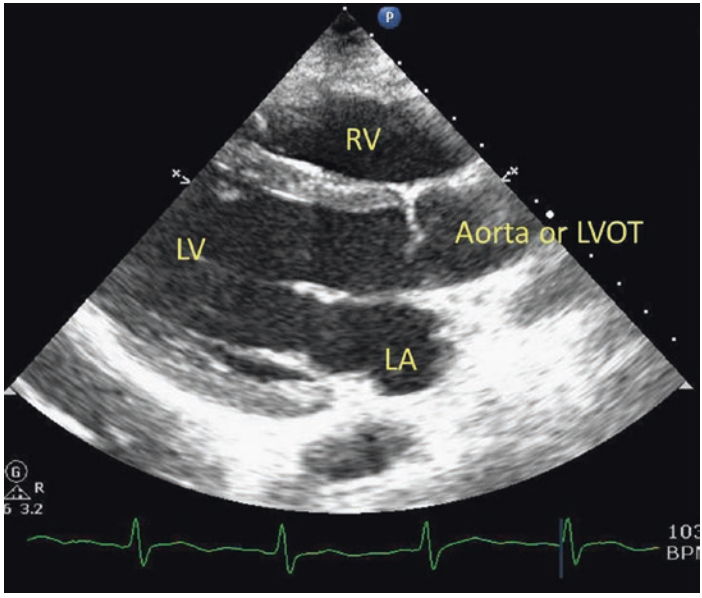


FIGURE 7.24 Parasternal long axis (PSL) cardiac view: LV can be seen in long axis

2. *PSS*: The transducer is rotated 90° so that the indicator points toward the left shoulder (Fig. 7.25). The LV and RV are seen in cross section. The transducer is rocked up to visualize the aortic valve and then down to see the LV at the mitral, papillary muscles, and the apex (Fig. 7.26).
3. *A4C*: The transducer is moved to the apex of the heart. This is usually located between the sixth and eighth interspace of the left chest (Fig. 7.27). It is generally lateral (at anterior axillary line) in non-intubated patients and more medial and inferior in intubated patients. The groove is oriented to the patient's left. Propping the right side of the patient up can improve the view. The LV and RV are visualized. If the transducer is rocked up, the aortic valve can be seen. This view is the best view for comparing the LV and RV (Fig. 7.28).

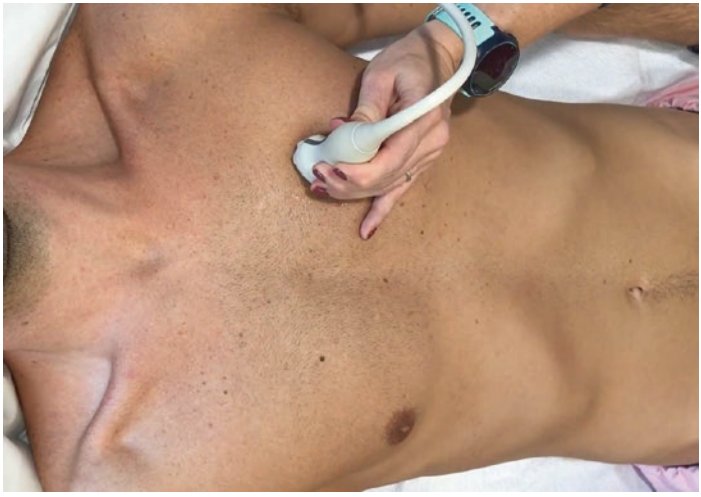
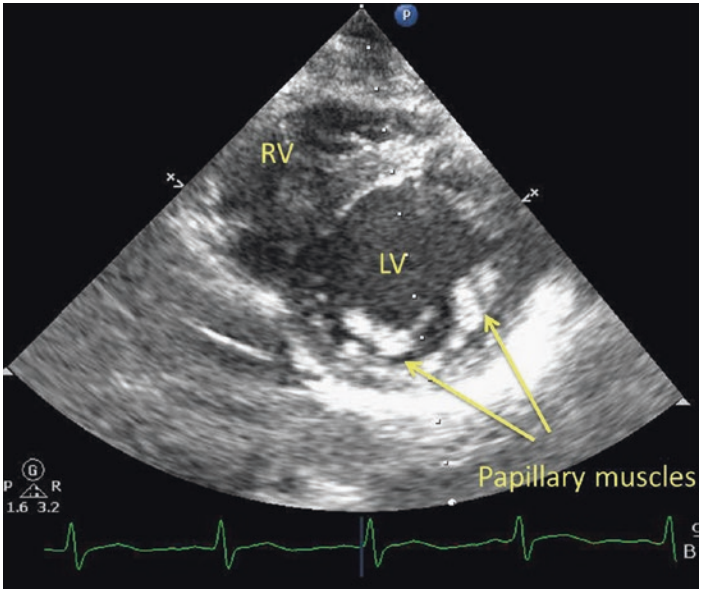


FIGURE 7.25 Parasternal short axis (PSS) cardiac view: The transducer is rotated 90° so that the indicator points toward the left shoulder



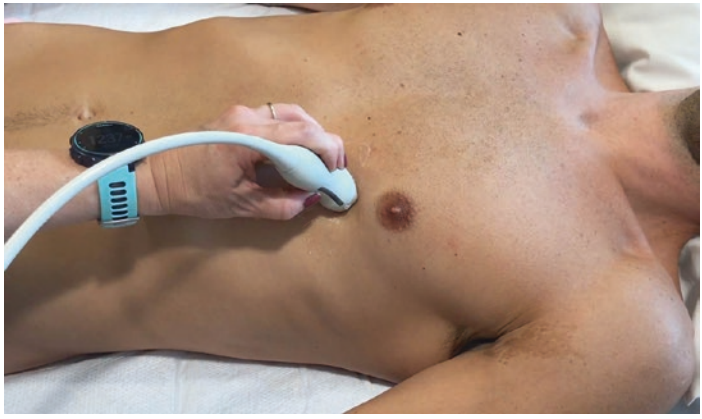


Figure 7.27 Apical four-chamber (A4C) cardiac view: The transducer is moved to the apex of the heart. This is usually located between the sixth and eighth interspace of the left chest. It is generally lateral (at anterior axillary line) in non-intubated patients and more medial and inferior in intubated patients. The groove is oriented to the patient's left. Propping the right side of the patient up can improve the view

4. *SX*: The transducer is moved 2–4 cm below the xiphoid, and the groove is oriented to the left. The transducer is rocked up and looking under the xiphoid (Fig. 7.18). The RV and LV can be seen (Fig. 7.29). The ventricles are often foreshortened and can appear globular. If this is the case, the transducer should be flattened more against the abdomen. Pericardial fluid can be seen superficial to the RV. The groove is then rotated toward the head and angled slightly toward the liver to see the IVC in long axis (Fig. 7.30a, b).

Figure 7.26 Parasternal short axis (PSS) cardiac view: The LV and RV are seen in cross section. The transducer is rocked up to visualize the aortic valve and then down to see the LV at the mitral, papillary muscles, and the apex

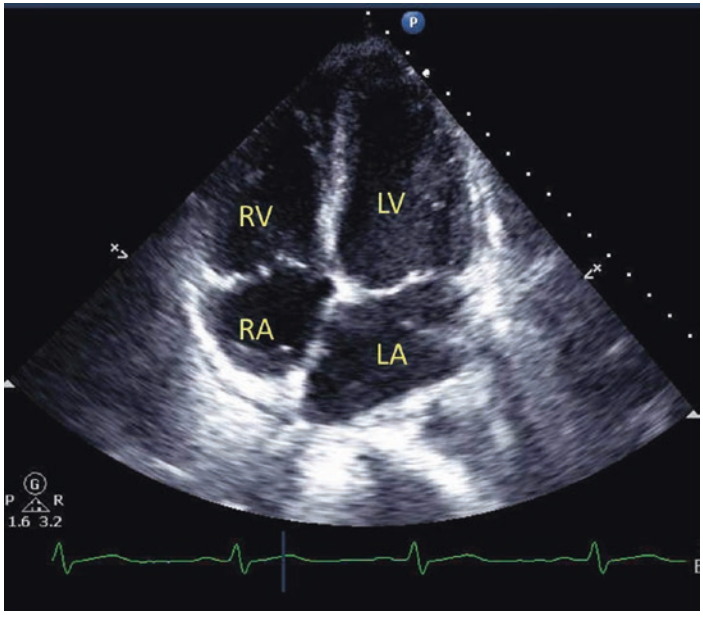


Figure 7.28 Apical four-chamber (A4C) cardiac view: The LV and RV are visualized. If the transducer is rocked up, the aortic valve can be seen. This view is the best view for comparing the LV and RV

Future Directions

There may soon be a role for the US in the bedside evaluation of solid organ injury (especially with the use of IV contrast), in the initial workup of traumatic brain injury (optic nerve sheath diameter may predict ICP), diagnosis of pelvic fracture, diagnosis and treatment of pneumothorax, confirmation of endotracheal tube placement, and assessment of long bone fractures. Ultrasound has been studied as a tool for

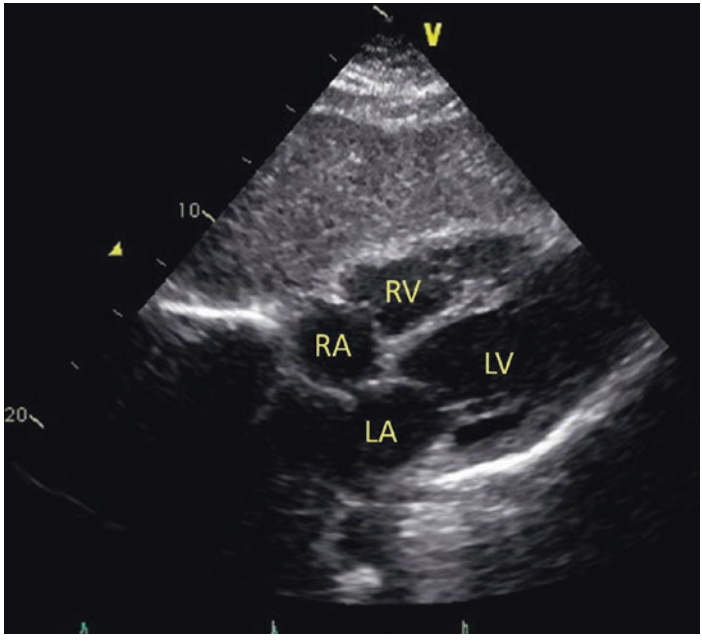


FIGURE 7.29 Pericardial space or subxiphoid cardiac view. The RV and LV can be seen. The ventricles are often foreshortened and can appear globular. If this is the case, the transducer should be flattened more against the abdomen. Pericardial fluid can be seen superficial to the RV

mass casualty triage, as a triage tool in the prehospital environment, and for providing portable diagnostics in the battlefield or humanitarian environments. US is the only diagnostic modality available on the international space station. With cost pressures, convenience, and risk of ionizing radiation, it seems that US is poised to change the face of diagnostic imaging.

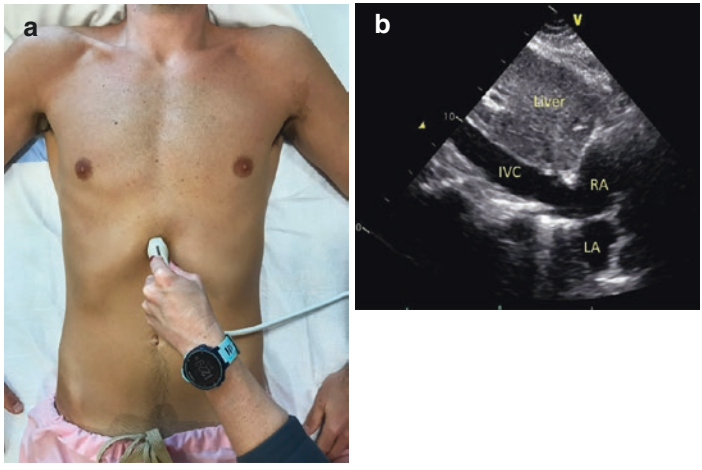


FIGURE 7.30 (**a**, **b**) Pericardial space or subxiphoid cardiac view: The groove is then rotated toward the head and angled slightly toward the liver (**a**) to see the IVC in long axis (**b**)

References

1. Sargsyan AE, Hamilton DR, Jones JA, Melton S, Whitson PA, Kirkpatrick AW, et al. FAST at MACH 20: clinical ultrasound aboard the international space station. J Trauma. 2005;58(1):35–9.
2. 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.
3. Moore CL, Copel JA. Point-of-care ultrasonography. N Engl J Med. 2011;364(8):749–57.
4. Perera P, Mailhot T, Riley D, Mandavia D. The RUSH exam: rapid ultrasound in SHock in the evaluation of the critically III. Emerg Med Clin North Am. 2010;28(1):29–56. vii
5. Murthi SB, Hess JR, Hess A, Stansbury LG, Scalea TM. Focused rapid echocardiographic evaluation versus vascular catheter-based assessment of cardiac output and function in critically ill trauma patients. J Trauma Acute Care Surg. 2012;72(5):1158–64.
6. Ferrada P, Evans D, Wolfe L, Anand RJ, Vanguri P, Mayglothling J, et al. Findings of a randomized controlled trial using limited

transthoracic echocardiogram (LTTE) as a hemodynamic monitoring tool in the trauma bay. J Trauma Acute Care Surg. 2014;76(1):31–8.
7. Ferrada P, Murthi S, Anand RJ, Bochicchio GV, Scalea T. Transthoracic focused rapid echocardiographic examination: real-time evaluation of fluid status in critically ill trauma patients. J Trauma. 2011;70(1):56–62. discussion 62-4
8. Newman PG, Rozycki GS. The history of ultrasound. Surg Clin North Am. 1998;78(2):179–95.
9. http://www.jsonline.com/business/ge-sees-strong-future-with-its-ultrasound-business-uj8mn79–190533061.html#ixzz2xIlP84cx.
10. Schurink GW, Bode PJ, van Luijt PA, van Vugt AB. The value of physical examination in the diagnosis of patients with blunt abdominal trauma: a retrospective study. Injury. 1997;28(4):261–5.
11. Rodriguez A, DuPriest RW Jr, Shatney CH. Recognition of intra-abdominal injury in blunt trauma victims. A prospective study comparing physical examination with peritoneal lavage. Am Surg. 1982;48(9):457–9.
12. Sheng AY, Dalziel P, Liteplo AS, Fagenholz P, Noble VE. Focused assessment with sonography in trauma and abdominal computed tomography utilization in adult trauma patients: trends over the last decade. Emerg Med Int. 2013;2013:678380.
13. American College of Surgeons Committee on Trauma. Advanced trauma life support (ATLS). 9th ed. Chicago: American College of Surgeons; 2012.
14. Rozycki GS, Ballard RB, Feliciano DV, Schmidt JA, Pennington SD. Surgeon-performed ultrasound for the assessment of truncal injuries: lessons learned from 1540 patients. Ann Surg. 1998;228(4):557–67.
15. Rozycki GS, Ochsner MG, Jaffin JH, Champion HR. Prospective evaluation of surgeons' use of ultrasound in the evaluation of trauma patients. J Trauma. 1993;34(4):516–26. discussion 526-7
16. Rozycki GS, Ochsner MG, Schmidt JA, Frankel HL, Davis TP, Wang D, et al. A prospective study of surgeon-performed ultrasound as the primary adjuvant modality for injured patient assessment. J Trauma. 1995;39(3):492–8. discussion 498–500
17. Kirkpatrick AW, Sirois M, Laupland KB, Liu D, Rowan K, Ball CG, et al. Hand-held thoracic sonography for detecting post-traumatic pneumothoraces: the extended focused assessment with sonography for trauma (EFAST). J Trauma. 2004;57(2):288–95.

18. Ferrada P, Anand RJ, Whelan J, Aboutanos MA, Duane T, Malhotra A, et al. Qualitative assessment of the inferior vena cava: useful tool for the evaluation of fluid status in critically ill patients. Am Surg. 2012;78(4):468–70.
19. Ferrada P, Vanguri P, Anand RJ, Whelan J, Duane T, Aboutanos M, et al. A, B, C, D, echo: limited transthoracic echocardiogram is a useful tool to guide therapy for hypotension in the trauma bay–a pilot study. J Trauma Acute Care Surg. 2013;74(1):220–3.
20. Gracias VH, Frankel HL, Gupta R, Malcynski J, Gandhi R, Collazzo L, et al. Defining the learning curve for the focused abdominal sonogram for trauma (FAST) examination: implications for credentialing. Am Surg. 2001;67(4):364–8.
21. Shackford SR, Rogers FB, Osler TM, Trabulsy ME, Clauss DW, Vane DW. Focused abdominal sonogram for trauma: the learning curve of nonradiologist clinicians in detecting hemoperitoneum. J Trauma. 1999;46(4):553–62. discussion 562-4
22. Rozycki GS, Newman PG. Surgeon-performed ultrasound for the assessment of abdominal injuries. Adv Surg. 1999;33:243–59.
23. Kluger Y, Soffer D. Abdominal injuries without hemoperitoneum: a potential limitation of focused abdominal sonography for trauma (FAST). J Trauma. 1997;43(4):728.
24. Shanmuganathan K, Mirvis SE, Sherbourne CD, Chiu WC, Rodriguez A. Hemoperitoneum as the sole indicator of abdominal visceral injuries: a potential limitation of screening abdominal US for trauma. Radiology. 1999;212(2):423–30.
25. Chiu WC, Cushing BM, Rodriguez A, Ho SM, Mirvis SE, Shanmuganathan K, et al. Abdominal injuries without hemoperitoneum: a potential limitation of focused abdominal sonography for trauma (FAST). J Trauma. 1997;42(4):617–23. discussion 623-5
26. Quinn AC, Sinert R. What is the utility of the focused assessment with sonography in trauma (FAST) exam in penetrating torso trauma? Injury. 2011;42(5):482–7.
27. Ma OJ, Mateer JR. Trauma ultrasound examination versus chest radiography in the detection of hemothorax. Ann Emerg Med. 1997;29(3):312–5. discussion 315-6
28. Nandipati KC, Allamaneni S, Kakarla R, Wong A, Richards N, Satterfield J, et al. Extended focused assessment with sonography for trauma (EFAST) in the diagnosis of pneumothorax: experience at a community based level I trauma center. Injury. 2011;42(5):511–4.

29. Lichtenstein D, Meziere G, Biderman P, Gepner A. The “lung point”: an ultrasound sign specific to pneumothorax. Intensive Care Med. 2000;26(10):1434–40.
30. Rozycki GS, Ochsner MG, Feliciano DV, Thomas B, Boulanger BR, Davis FE, et al. Early detection of hemoperitoneum by ultrasound examination of the right upper quadrant: a multicenter study. J Trauma. 1998;45(5):878–83.
31. Murthi SB, Frankel HL, Narayan M, Lissauer M, Furgusen M, Fatima SH, et al. Making the financial case for a surgeon-directed critical care ultrasound program. J Trauma Acute Care Surg. 2014;76(2):340–4. discussion 344-6
32. Ferrada P, Anand RJ, Whelan J, Aboutanos MA, Duane T, Malhotra A, et al. Limited transthoracic echocardiogram: so easy any trauma attending can do it. J Trauma. 2011;71(5):1327–31. discussion 1331-2

Part III
Techniques in the Neck and Chest

Chapter 8
Trachea, Bronchus, and Esophagus Injuries: Techniques

Brandon R. Bruns

Introduction of the Problem

Even in busy urban trauma centers, injuries to the tracheobronchial tree and esophagus remain rare. As the thoracic cage provides protection, these injuries most commonly occur in the neck. Injuries in the neck tend to lend themselves to easier diagnosis, whereas those contained within the thorax may elude diagnosis for prolonged periods of time and lead to adverse outcome.

Tracheobronchial

Because many patients with tracheobronchial injury die before arrival to the hospital, the true incidence of the injury burden remains unknown [1]. Several autopsy studies estimate the incidence to be approximately 3% [2,3]. Compromise of the major airways can restrict oxygenation and ventilation leading to alterations in gas exchange and hemodynamic

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compromise. Any sign of major airway injury should lead the trauma team to rapidly search for the anatomic lesion and initiate treatment.

Blunt injuries to the trachea and bronchus are believed to occur as direct impact to the neck, deceleration injuries suffered at fixed anatomic locations (carina and cricoid), or increased pressure within the tracheobronchial tree from blunt force applied to the thorax or abdomen. Blunt injury to the thoracic trachea most commonly occurs within 1 cm of the carina. Due to the high degree of energy necessary to injure tracheobronchial structures, these injuries are typically only a small part of the larger injury burden.

Penetrating injuries to the trachea in the neck are often the result of stab wounds (Fig. 8.1). Stab wounds are low energy and are frequently associated with vascular and esophageal injuries. Immediate attention to the airway is mandatory in penetrating neck injury, as the situation can rapidly worsen. Penetrating injuries to the trachea and bronchus within the

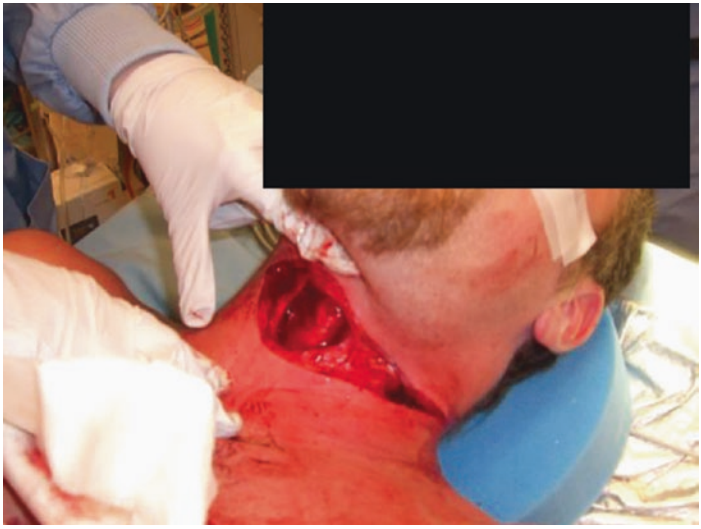

Figure 8.1 External injuries after stab wound to neck

thorax are typically due to gunshot wounds and may be associated with cardiac, pulmonary, major vascular, or esophageal injuries. Iatrogenic injury may also occur during endoscopic evaluations, tracheostomy, and orotracheal intubation.

Esophagus

Penetrating injury is the predominant cause of esophageal trauma. Published in 1985, a series of 26 patients with esophageal trauma were identified over a 15-year time period. Twenty-two of those were penetrating and four blunt, with all blunt injuries occurring in the region of the cervical esophagus [4]. Blunt injuries to the esophagus are often associated with concomitant thoracic injury. Blunt injury occurs as a result of direct force to the cervical esophagus in conjunction with a hyperextended neck or rarely as result of intraluminal distention secondary to blunt force trauma [5].

A 2013 review of the National Trauma Data Bank sought to better describe the current state of penetrating esophageal injury in the United States. Over a 2-year time period, 227 patients with penetrating injuries to the esophagus were treated at either level I or level II trauma centers. The overall mortality was 44%, and the overwhelming majority of deaths occurred within 24 hours. Deaths occurring in the initial 24-hour period were the result of concomitant injury, not the esophageal injury alone. In patients surviving greater than 24 hours, 62% had primary repair, 13% underwent drainage, and 4% underwent resection. The only predictor of death, in those surviving greater than 24 hours, was injury severity score (ISS) [6].

Iatrogenic injury remains the leading cause of penetrating esophageal injury [7]. Nasogastric tube insertion, transesophageal echocardiography, flexible and rigid esophagoscopy, and various other procedures are all potential injurious events. Therapeutic dilation of the esophagus for various strictures and achalasia remains the predominant cause of iatrogenic esophageal perforation [8].

History of Care of Trachea, Bronchus, and Esophagus Injuries

Given the rare nature of tracheobronchial and esophageal injuries, the majority of publications consist of case reports and single-institution series. Thus, the principles of treatment remain the same and have not drastically changed in the past 50 years. Diagnostic adjuncts have continued to improve and assist in earlier identification of tracheobronchial and esophageal injury, which may allow more timely surgical intervention.

Tracheobronchial

Kiser and colleagues performed a meta-analysis of the literature from 1873 to 1996, looking specifically at blunt intrathoracic tracheobronchial injuries. They identified 256 injuries, of which 59% were the result of motor vehicle collisions and 27% were the result of crush injury. The median time to diagnosis was 9 days, with 47% of cases involving the right bronchus and 32% involving the left bronchus (52% of patients with right bronchial injuries and 1% with left-sided bronchial injuries were diagnosed within 24 hours) [9].

Investigators at the Ratchaburi Hospital in Thailand reported on 11 tracheobronchial injuries in 10 years (7 penetrating and 4 blunt). Pneumothorax was present in two patients, subcutaneous emphysema in three patients, and dyspnea in four. The authors concluded that a delay in diagnosis was the leading factor resulting in increased morbidity [10]. Similarly, Cassada and colleagues found that a delay in diagnosis was the single most important factor leading to poor outcome [11].

Esophagus

As diagnostic modalities have advanced, the identification of injury to the esophagus has become easier to accomplish in an expeditious and noninvasive manner. A 1977 report from

the Journal of Trauma describes the case of a young man status post motorcycle collision with multiple injuries, which on the fourth day of his hospitalization manifested with mediastinitis and empyema. Surgical exploration revealed the presence of an esophageal perforation, which was successfully treated with drainage [12]. In the current era of enhanced imaging techniques and the trauma surgeon's increased utilization of endoscopy for diagnosis, clinicians are able to more quickly recognize and intervene for such lesions.

The management of esophageal injury has remained largely unchanged for the past five decades. Some have sought to define the role of endoscopic stenting in esophageal perforation, finding a greater risk of death with stenting than with surgery [13]. This led the authors to conclude that operative therapy is preferred. Thus, the primary goals of treatment remain primary repair, wide drainage, and possible diversion if anatomic repair is impossible or physiology is exceedingly altered [14].

Technique with Personal Tips

Attention to maintenance of the airway, breathing, and circulation is the obvious initial priority in any trauma patient. Exsanguinating injuries must be rapidly identified and hemostasis obtained, by whatever means necessary. Cerebral ischemia from vessel injury should be addressed and a treatment plan devised. The actual tracheobronchial and esophageal injuries should be addressed next.

Tracheobronchial

Clinical examination can lead the astute physician to suspect injury based on physical findings. Stridor and respiratory distress, subcutaneous emphysema, pneumothorax with air leak, and bubbling from epidermal lacerations should increase suspicion for major airway injury.

Obtaining a protected and durable airway is essential. A 1999 series from the University of Tennessee Medical Center

at Knoxville showed 55% of patients with tracheobronchial injury were stable on supplemental oxygen alone and did not require urgent intubation [11]; however, this should not deter the trauma team from intubation in the clinically appropriate setting. Caution must be exercised as partial or complete transection of proximal airways can make intubation difficult, leading to adverse outcomes. Liberal usage of laryngoscopy and fiber-optic bronchoscopy, by providers comfortable with the difficult airway, will increase the chances of successful intubation.

If transection of the trachea occurs in the neck, two lumens may be visible externally. In this instance, intubation of the distal lumen with any available endotracheal tube is wise (Fig. 8.2). After ensuring a temporary airway, definitive airway management can be established when the patient is more

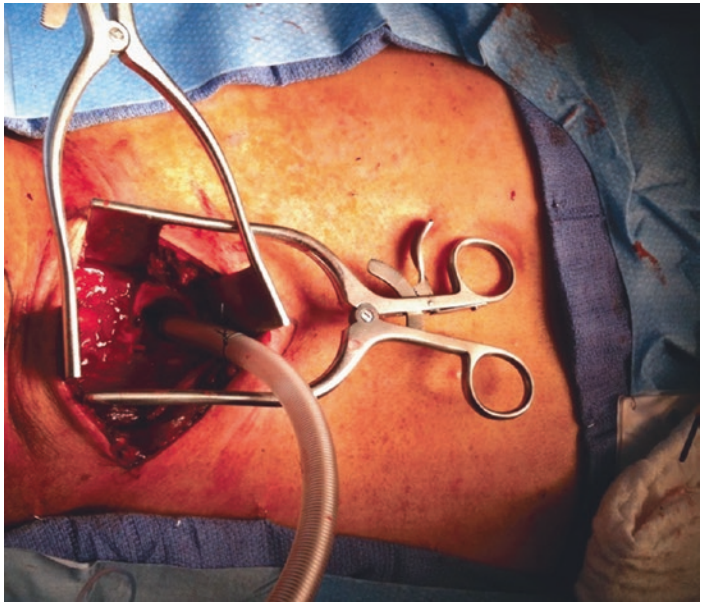

Figure 8.2 Placement of tubing into distal trachea to facilitate ventilation and oxygenation

stable. A surgical airway remains an option in the unstable patient or in patients unable to be successfully intubated.

Bronchoscopy and direct visualization of a mucosal defect remain the optimal study for diagnosis of tracheobronchial injury (Fig. 8.3). If bronchoscopy is performed under general endotracheal anesthesia, the clinician must withdraw the endotracheal tube over the bronchoscope to visualize the entirety of the proximal trachea. The endotracheal tube must be completely free, the balloon deflated, and the tube brought all the way up to the flaring of the upper trachea, analogous to a bronchoscopically assisted percutaneous tracheostomy. Bronchoscopy can be performed in either a trauma

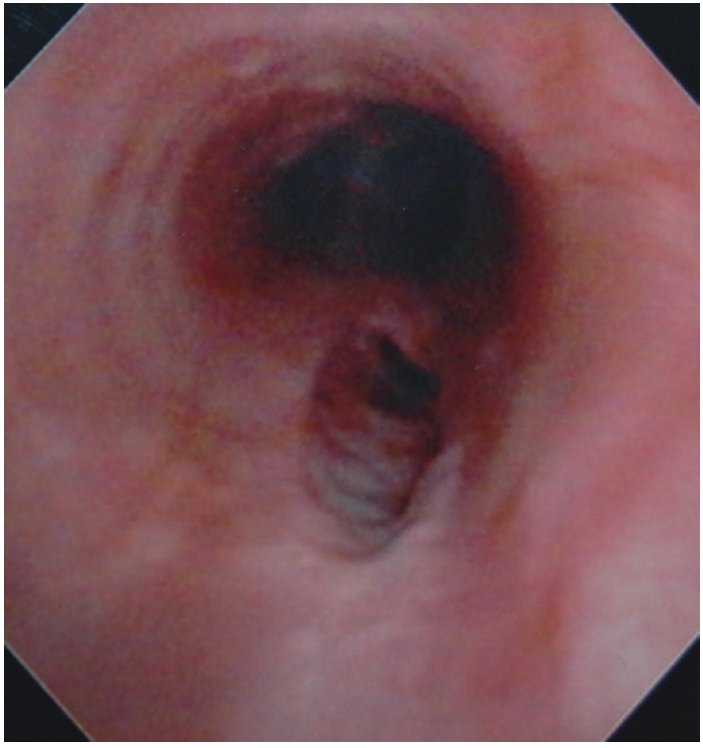

Figure 8.3 Bronchoscopic view of mucosal injury to trachea

resuscitation area or an operating room; however, the operating room provides the safest and most versatile environment for such procedures. If injury is identified, distance from the incisors should be recorded, along with proximity to the carina.

In the patient with major airway injury that requires mechanical ventilation, decreased pressures will lessen the air leak and assist in oxygenation and ventilation. Airway pressure release ventilation (APRV) is a mode of ventilation frequently employed, which allows the patient to comfortably breathe and avoid dis-synchrony with the ventilator; however, the lowest possible pressure should be utilized to minimize ongoing air leak.

CT imaging can be utilized as an adjunct to evaluate for tracheal injury [15] (Fig. 8.4). CT imaging of the neck offers the advantage of evaluating the vasculature and esophagus, in addition to the central airways. In a retrospective review, Chen and colleagues found CT to be 85% sensitive in identification of tracheal injury, and the authors concluded that CT can assist in selecting patients that require bronchoscopy for

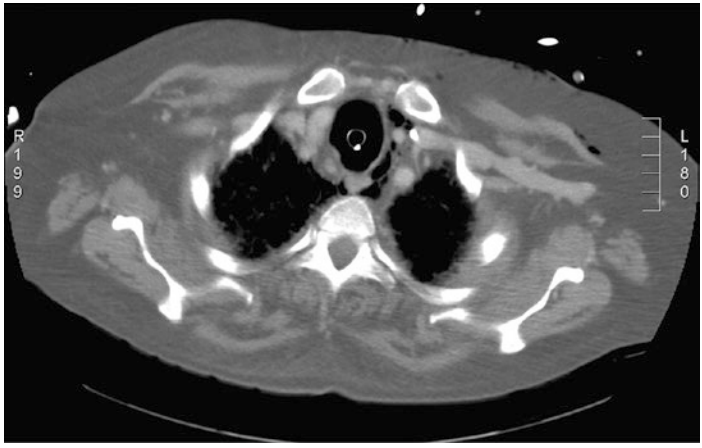

Figure 8.4 CT showing extra-luminal air and tracheal mucosal abnormality suggesting tracheal injury

definitive diagnosis [16]. In penetrating injury, the added advantage of assessing trajectory and determining structures at risk of injury makes axial imaging attractive [17]. In the rare case of suspected tracheal transection after blunt injury, axial imaging in a stable patient with CT may be an adjunct to direct visualization with bronchoscopy [18]. In short, CT findings can suggest injury to the trachea, after which the patient is taken to the operating room for definitive bronchoscopic diagnosis and repair.

Nonoperative approaches to tracheobronchial injury have been described [19, 20], but operative therapy remains the preferred method of definitive treatment. Initial operative management of tracheobronchial injuries begins with appropriate setup of the operating room and operating equipment. A sternal saw, vascular instruments, and a variety of endotracheal and tracheostomy tubes should be available. Fiber-optic bronchoscopy and appropriately trained anesthesia providers are essential. The surgeon should be prepared to perform intraoperative bronchoscopy to assist in identification of the injury. A double-lumen endotracheal tube may be required if single-lung ventilation is desired for a thoracic approach to a bronchial injury.

Cervical injuries involving the trachea are most easily approached via a collar incision performed approximately 2 fingerbreadths above the sternal notch. This incision is carried down through the platysma and sub-platysmal flaps are created. The strap muscles are divided in the midline. The thyroid isthmus can then be retracted cephalad to further increase exposure of the trachea as it enters the mediastinum. Alternatively, the thyroid isthmus can be divided which may increase exposure. The anterior trachea can be mobilized anteriorly using blunt dissection down into the anterior mediastinum. The trachea can then be retracted up into the neck using a tracheal hook. This gives exposure to an additional one to two rings. If the injury extends into the mediastinum, a partial sternal split can be performed without performing a full sternotomy (Fig. 8.5). In the face of a concomitant vascular or esophageal injury, the collar incision can be extended

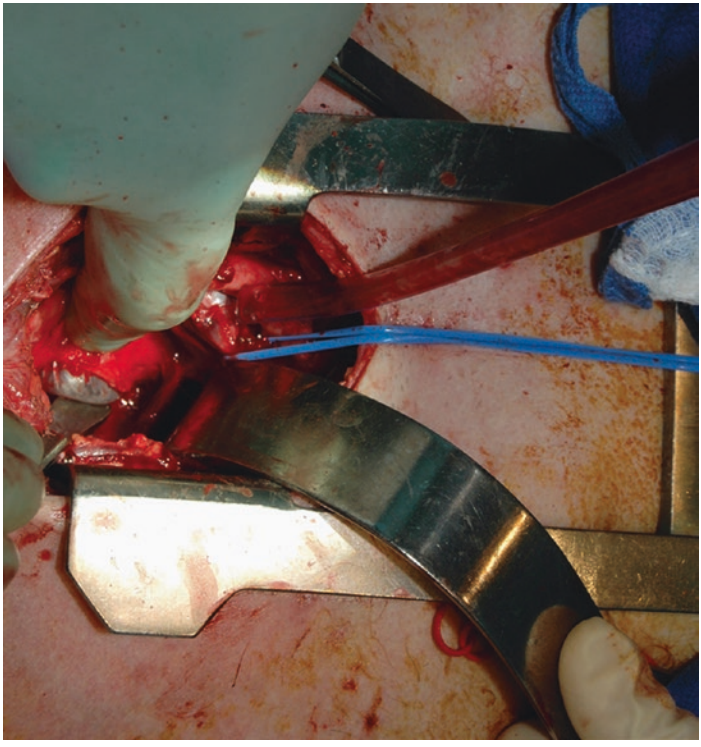

FIGURE 8.5 Partial sternal split for evaluation of tracheal injury. The endotracheal tube balloon is visualized emanating from the injury

superiorly or inferiorly in the plane anterior to the sternocleidomastoid muscle.

The thoracic trachea, right mainstem bronchus, and proximal left mainstem bronchus are best approached via a right posterolateral thoracotomy performed through the fourth intercostal space (Fig. 8.6). Distal left mainstem bronchus injuries are best approached via a left posterolateral thoracotomy. Though lateral decubitus positioning provides optimal exposure of the intrathoracic tracheobronchial structures, the surgeon must recognize that access to other body cavities is exceedingly limited; thus, meticulous identification of other

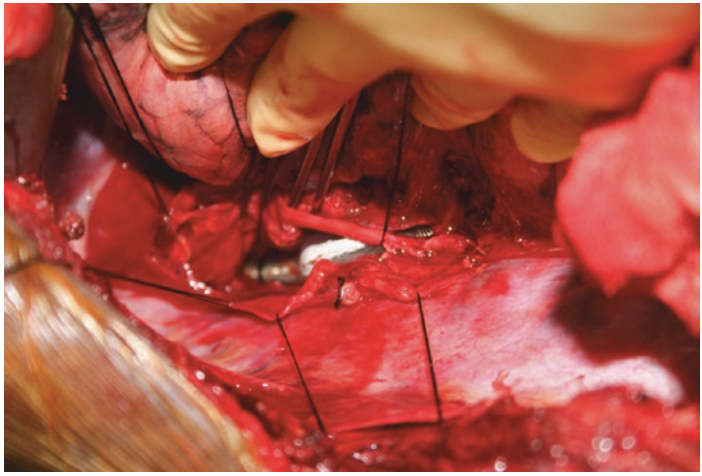

Figure 8.6 Right posterolateral thoracotomy with mediastinal pleura opened and retracted with silk sutures. The endotracheal tube is seen within the lumen of the injured trachea. The lung is retracted upward

injuries prior to proceeding with lateral positioning is prudent.

Primary repair of tracheal injury is desirable. A single layer of absorbable, monofilament suture is utilized to primarily repair the injury. Running or interrupted sutures can be utilized with equivalent results. Smaller diameter structures are best repaired in an interrupted fashion to avoid narrowing of the airway. Stellate areas should be sharply debrided back to healthy-appearing tracheal tissues. Given the mobile nature of the trachea, resection of up to 2–4 cm of trachea with primary anastomosis is feasible with mobilization (Fig. 8.7). Multiple techniques have been described for tracheal mobilization, including division of the inferior pulmonary ligament, cervical flexion, and mobilization of the hilum of the lung. If tension is present on the anastomosis after mobilization, the chin can be sutured to the patient's chest. Mobilization of the trachea should proceed in the anterior and posterior planes as the blood supply comes in laterally. Suture lines are prefer-

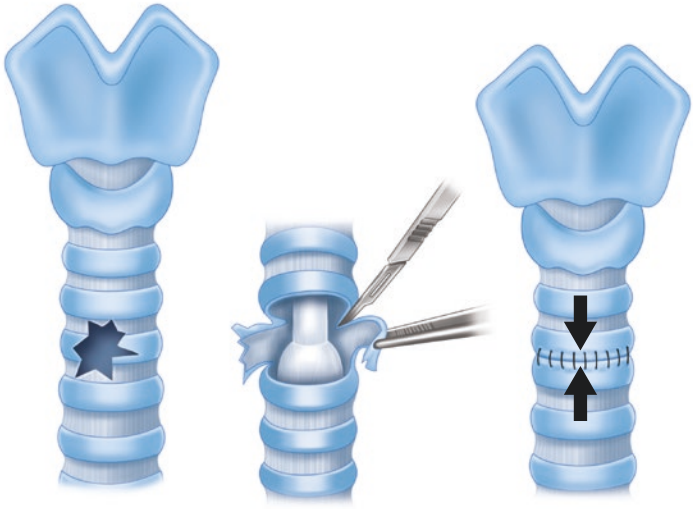

Figure 8.7 Resection of trachea with mobilization and primary anastomosis

ably buttressed with autologous and well-vascularized tissue present in the region of repair (intercostal muscle, pleura, pericardial, strap muscle (Fig. 8.8), or omental flaps). Occasionally, the tracheal injury is located only in the posterior membranous portion, where some can be managed nonoperatively. If repair is necessary, posterior injuries are best approached by opening the trachea anteriorly and repairing the injured trachea from the inside. In these situations, it is necessary to precisely define the level of injury before opening the trachea, which is best done with intraoperative bronchoscopy. With the scope visualizing the posterior injury, the light from the bronchoscope can be seen through the anterior trachea, ensuring the anterior tracheal incision is correctly positioned. As previously mentioned, the endotracheal tube must be withdrawn to allow for adequate visualization of the injury. The maneuver may need to be repeated to be sure adequate oxygenation and ventilation is maintained. The anterior trachea is then closed as described above with placement of a viable muscle flap over the repair.

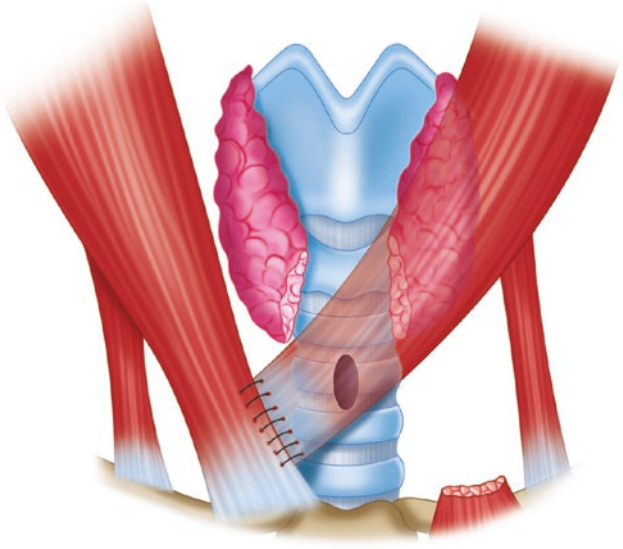

FIGURE 8.8 Autologous flap of strap muscle to buttress tracheal injury/repair

Damage control procedures have also been applied to devastating tracheal injuries with loss of significant length. Investigators at Parkland Hospital described a technique of silicone T-tube placement to bridge a tracheal gap of 6 cm caused by an oblique gunshot wound trajectory. Through the T-tube, an endotracheal tube was placed distally. Eight weeks after the injury, the tracheal injury had healed, without evidence of stenosis [21].

Esophagus

Timely diagnosis of esophageal injury is vital as missed esophageal injuries can lead to life-threatening sepsis. Given the relatively superficial course of the esophagus in the neck, diagnosis of penetrating esophageal injury is sometimes made on the basis of physical exam alone, as saliva or food

particles emanating from the wound are highly suggestive. Diagnosis of thoracic esophageal injury requires a higher degree of suspicion, and various diagnostic modalities can be employed.

Plain film radiography is insufficient to diagnose esophageal injury (Fig. 8.9) but may heighten suspicion. The presence of pneumothorax, hemothorax, displacement of the nasogastric tube, or mediastinal widening all suggest a mechanism of sufficient blunt force to injure the esophagus. Trajectory determination in penetrating injury can give some clue as to structures at risk of injury.

The pervasive use of computed tomography (CT) in the trauma setting has led many to examine its utility in the diagnosis of esophageal injury. Investigators from the Aga Khan

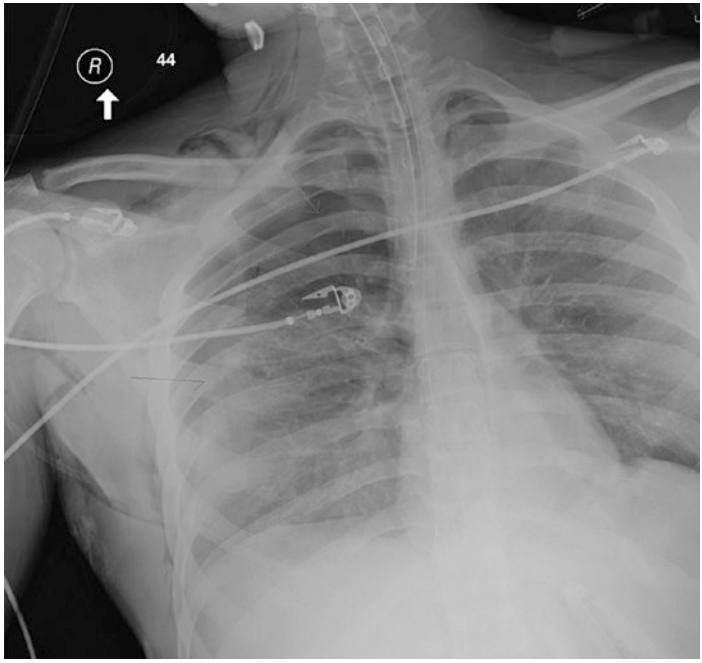

Figure 8.9 Initial chest radiograph in a patient presenting with left-sided chest gunshot wound and esophageal injury

University Hospital showed a sensitivity of 53% when using CT scan for diagnosis of esophageal injury [22]. In a study from 2006, authors concluded that certain CT findings were suggestive of esophageal injury and included fluid and air in the mediastinum, subcutaneous tissues, and pleura; dissection of the esophagus; intramural hematoma formation; esophageal thickening; and left lower lobe atelectasis [23] (Fig. 8.10). However, these findings are far from specific for esophageal injury, and thus endoscopy should be performed.

The gold standard for esophageal injury remains a combination of esophagoscopy and contrast-enhanced esophagography [24]. Because of its readily available nature and minimal resource utilization, surgeon-performed flexible endoscopic evaluation of the esophagus is frequently employed as the initial screening test. However, series have demonstrated a sensitivity of only 38% for flexible endoscopy versus 89% for rigid [24]. If injury is suspected based on trajectory or mechanism, negative endoscopy should be followed by esophagography with gastrografin, to be followed by thickened barium if gastrografin does not show injury.

Selected nonoperative approaches of esophageal injuries may be entertained in a limited number of circumstances. A

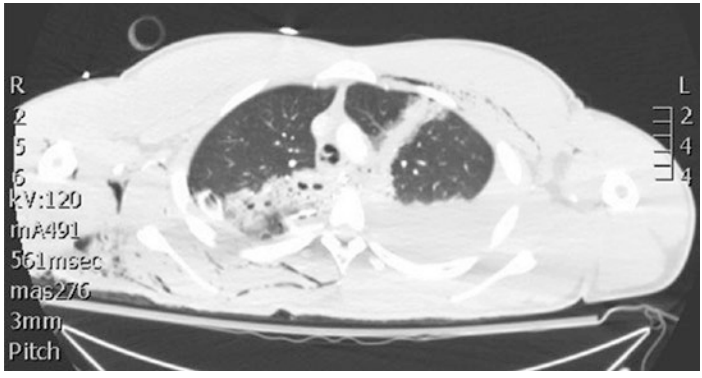

Figure 8.10 Initial chest CT illustrating the trajectory of the missile, left hemothorax, and subcutaneous/mediastinal air; all suggestive of possible esophageal or tracheal injury

2013 meta-analysis of studies evaluating management of esophageal perforation demonstrated a 7.3% pooled mortality for patients managed with esophageal stent versus 13.8% for patients undergoing esophagectomy; however, the authors warn about potential selection bias and limited experience with the technology [25].

Operative repair of esophageal injury remains the mainstay of treatment. Preoperative placement of a nasogastric tube may assist with intraoperative palpation of the esophagus. A shoulder roll may be helpful with neck extension and the patient's head positioned looking to the right to open the operative space. Cervical esophageal injury is best approached via an incision anterior to the sternocleidomastoid muscle on the left. The sternocleidomastoid muscle is retracted laterally, and blunt dissection commences in a medial direction. Circumferential placement of a rubber drain (Penrose) around the esophagus assists in retraction. Care is taken to avoid injury to the recurrent laryngeal nerve along its course in the tracheoesophageal groove.

Thoracic esophageal injury is best approached via a right posterolateral thoracotomy in almost all situations; however, injury in close proximity to the gastroesophageal junction is most easily accessible via a left posterolateral thoracotomy performed through the sixth or seventh interspace, a thoracoabdominal incision, or a laparotomy. If multiple cavities are to be explored, anterolateral thoracotomy with a bump under the thoracic cage can be utilized. Single-lung ventilation, with a dual-lumen endotracheal tube, greatly facilitates visualization of the involved esophagus.

The right posterolateral thoracotomy allows good exposure of the majority of the thoracic esophagus. Ligation of the azygous vein and wide opening of the mediastinal pleura enables the surgeon to bluntly dissect the esophagus and facilitates complete visualization and ease of repair. As in the cervical esophagus, placement of a large Penrose drain can be utilized to encircle the esophagus and assist with retraction (Fig. 8.11).

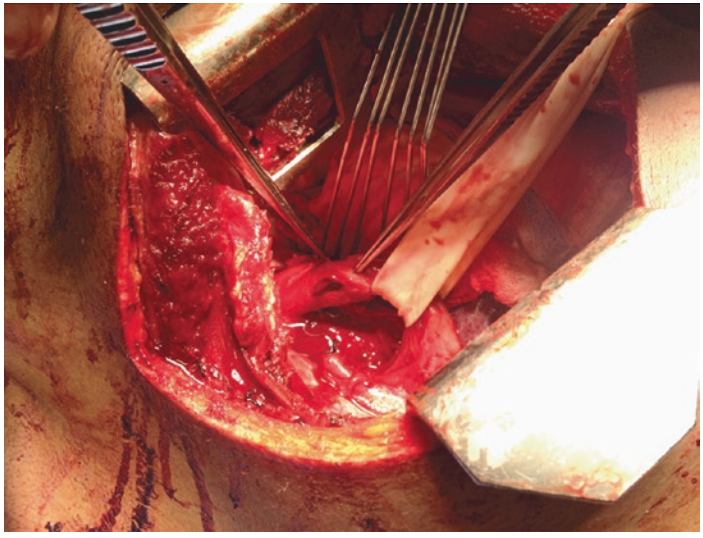

Figure 8.11 Esophageal injury with esophagus retracted by rubber drain (Penrose) and nasogastric tube visible within the lumen of the esophagus. Exposure was obtained via a right posterolateral thoracotomy

Primary repair of esophageal injury remains the goal of treatment. Sharp debridement of nonviable tissue is performed. The surgeon must visualize the entirety of the mucosal injury, realizing that mucosal injury can extend beyond the visualized injury seen through the muscular defect. The muscular layer must be opened to ensure that the whole extent of the underlying injury is visualized and repaired. Esophageal repairs are performed in two layers, with an inner (mucosal) layer of absorbable suture followed by an outer (muscular) layer of absorbable, monofilament suture. The linear course of the outer muscular fibers mandates the surgeon place the sutures through this layer at a slight angle to avoid tearing through the fibers. A "mattress"-type suture can be employed to ensure adequate repair. Placement of a nasogastric tube or bougie, under direct visualization, may help in preventing stenosis at the site of repair (Fig. 8.12).

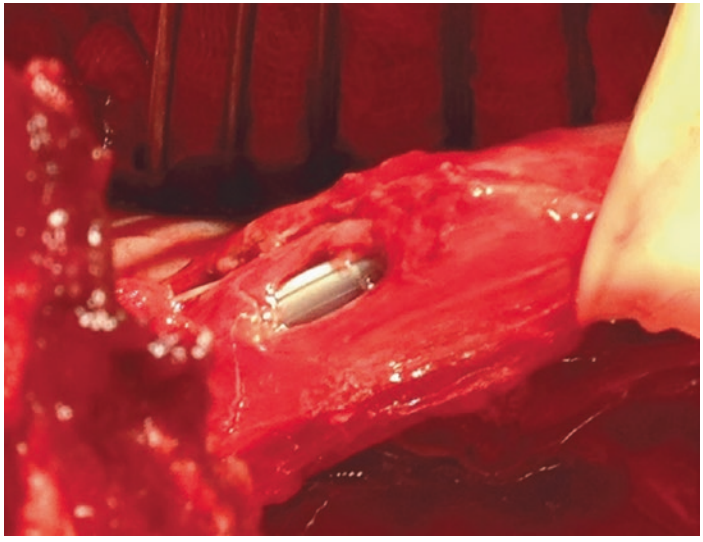

Figure 8.12 Nasogastric tube within the injured esophagus to help prevent repair stenosis

In the case of extensive destruction of esophagus, resection of the nonviable tissue with primary anastomosis is sometimes feasible. In cases of severe hemodynamic compromise, copious contamination and surrounding inflammation, or extensive destruction of the esophagus, damage control principles should be employed. Wide drainage of the area with thoracostomy tubes, in combination with closed suction drains, should be utilized. Additionally, we have successfully managed these destructive lesions with retrograde drainage via trans-gastric tubes which then exit the patient's abdominal wall in combination with nasogastric tube drainage and exterior drainage via thoracostomy tubes and closed suction drains (Fig. 8.13).

As in tracheobronchial injuries, primary repairs and esophageal resection with anastomosis should have the suture line protected with vascularized tissue to prevent anastomotic failure. Intercostal muscle, pleural, pericardial, and omental flaps can all be fashioned in a rather expeditious fashion. We

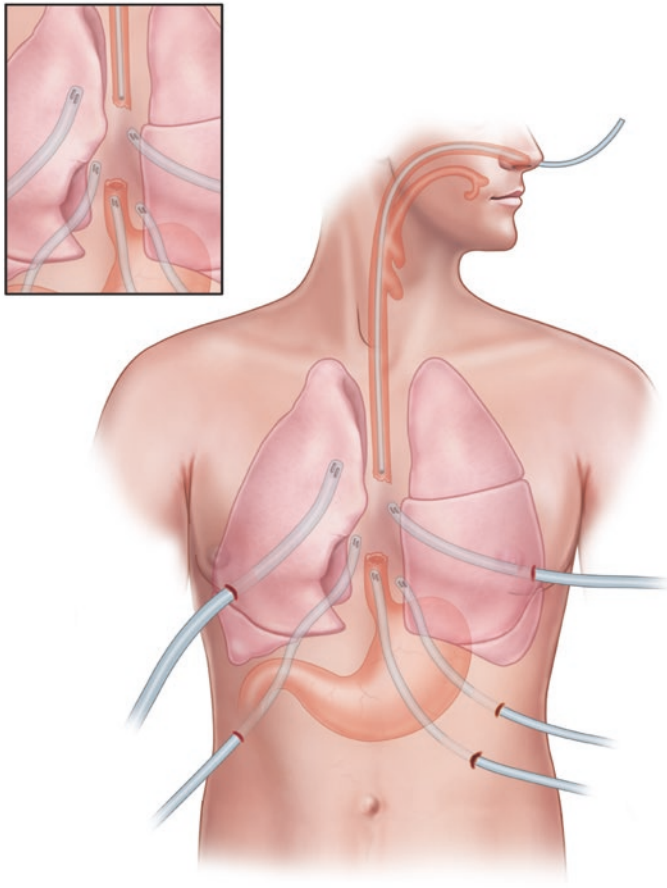

Figure 8.13 Esophageal destructive injury drained via retrograde trans-gastric tubes, thoracostomy tubes, nasogastric tubes, and closed suction drains

employ wide drainage of the repair with thoracostomy tubes and closed suction drainage catheters. At least one drainage tube is left in place until the patient has undergone a contrast-enhanced imaging study that verifies no leak, and they begin tolerating a PO diet without evidence of increasing output from the drainage tube.

Distal enteral access after esophageal repair, resection, or damage control is important, as the patient will remain NPO for a substantial amount of time. To avoid additional surgical access into the enteric tract, a naso-jejunal feeding tube placed under direct visualization at the time of initial operation may be preferred. Additionally, a gastrojejunal (GJ) tube can be placed at the time of initial operation. The GJ tube has the added benefit of gastric decompression while allowing feeding distal to the ligament of Treitz. Another option remains a surgically placed jejunostomy tube.

The Shock Trauma Center practice is to image all repairs at 7–10 days postoperatively with a contrast-enhanced swallow study or via contrast given down a naso-esophageal tube. Initially, gastrografin is administered, which is followed by barium if no leak is evident on gastrografin-enhanced imaging. After the imaging study, the patient is allowed to take PO feeds and the drain output is monitored.

Outcomes

The scarcity of tracheobronchial and esophageal injury limits the data available for true outcome analysis of such injuries. Multi-institutional studies performed over prolonged periods of time occupy the majority of the outcomes literature. The heterogeneity in patient population and varying management strategies makes evaluation of long-term outcomes difficult; however, studies do show the risk of death to be significantly lower in patients cared for at a trauma center versus a non-trauma center [26].

Tracheobronchial Mortality

Morality among patients with tracheobronchial injury has decreased greatly, with published mortality of 36% before 1950 decreasing to 9% in the era after 1970 [9]. In the absence of associated injuries, most patients undergoing major tra-

cheobronchial injury repair or reconstruction will return to their baseline functional status, with minimal morbidity [27, 28].

Esophagus Mortality

A 2013 analysis of trauma patients in Pennsylvania examined 231,964 patients and found 327 (0.14%) with injury to the esophagus. Investigators found that patients with esophageal trauma were significantly more likely to die than those that did not incur esophageal injury. Higher morbidity and mortality were observed in thoracic esophageal injury versus injury occurring in the cervical portion [29].

Tracheobronchial Complications

Described complications of tracheobronchial trauma include postoperative suture granuloma, pneumonia, and surgical site infections [27]. Suture granulomas can be approached and treated using bronchoscopy and laser therapy [28]. Pneumonia is best managed with initiation of antibiotics after culture of bronchial secretions with rapid de-escalation of antibiotic therapy upon identification of the responsible microbe. Surgical site infections are managed with antibiotics and drainage.

Postoperative management of tracheobronchial injuries centers around rapid wean from the ventilator and minimizing intrathoracic pressures. Anastomotic breakdown is the predominant concern in the postoperative period. If tracheal resection was performed or there is any concern of anastomotic tension, a stitch placed from the patient's chin to the soft tissue overlying the manubrium will allow healing and decrease strain on the repair. Repeat bronchoscopy at 7–10 days post-repair will enable visualization of the repair and verification of proper healing.

The identification of a significant postoperative air leak may herald the presence of a bronchopleural fistula.

Management centers on decreasing intra-bronchial pressures and minimizing mean airway pressures through specific ventilator setting adjustments. Nonoperative management is often followed by bronchoscopic techniques, including bronchial blockers, or operative re-exploration.

Esophagus Complications

The most dreaded complication after esophageal injury and repair is anastomotic failure and leak. Leaks in the neck are readily identified by physical examination. The appearance of erythema, induration, pain, and occasionally the extrusion of esophageal contents or purulence onto the skin signal that the anastomosis or repair may have failed. CT imaging can be useful to assist in diagnosis when not clinically evident. Unexplained leukocytosis, fever, or general malaise may indicate a dehiscence of the repair. Cervical leaks, though troubling, are easily accessible surgically and are amenable to wide drainage and irrigation. Rarely, cervical esophagostomy is required, but this remains low on the list of preferred interventions.

Thoracic esophageal leaks pose a much greater threat to the patient, as they are not easily accessible and jeopardize the pleural space and mediastinum. Leaks commonly manifest after an indolent course with fever, respiratory insufficiency or ventilator-dependent respiratory failure, leukocytosis, and a global failure to progress and clinically improve. Chest radiographs can give some suggestion as to the status of the repair, but contrast-enhanced swallow studies or chest CT remains the mainstay of diagnosis. Chest CT is able to evaluate pleural pathology and characterize the presence, or absence, of an empyema.

Treatment of esophageal leak is drainage (for this reason, we leave the drainage tubes until the patient tolerates a PO diet without difficulty), whether surgical or via interventional radiologic techniques. The patient is left NPO and continued on enteral feeds via the distal access obtained at the time of

initial operation. Reoperation with primary repair or muscle buttress of the leak is sometimes necessary but is technically difficult given the adhesions and inflammatory reaction that are invariably present. In the patient who is not deemed an operative candidate, attempts at endoluminal stenting can temporize the situation [30].

References

1. Burke JF. Early diagnosis of traumatic rupture of the bronchus. JAMA. 1962;181:682–6.
2. Lynn RB, Iyengar K. Traumatic rupture of the bronchus. Chest. 1972;61(1):81–3.
3. Roxburgh JC. Rupture of the tracheobronchial tree. Thorax. 1987;42(9):681–8.
4. Glatterer MS Jr, Toon RS, Ellestad C, McFee AS, Rogers W, Mack JW, et al. Management of blunt and penetrating external esophageal trauma. J Trauma. 1985;25(8):784–92.
5. Karmy-Jones R, Jurkovich GJ. Blunt chest trauma. Curr Probl Surg. 2004;41(3):211–380.
6. Patel MS, Malinoski DJ, Zhou L, Neal ML, Hoyt DB. Penetrating oesophageal injury: a contemporary analysis of the national trauma data bank. Injury. 2013;44(1):48–55.
7. Johnson SB. Esophageal trauma. Semin Thorac Cardiovasc Surg. 2008;20(1):46–51.
8. Flynn AE, Verrier ED, Way LW, Thomas AN, Pellegrini CA. Esophageal perforation. Arch Surg. 1989;124(10):1211–4; discussion 1214–5
9. Kiser AC, O'Brien SM, Detterbeck FC. Blunt tracheobronchial injuries: treatment and outcomes. Ann Thorac Surg. 2001;71(6):2059–65.
10. Glinjongol C, Pakdirat B. Management of tracheobrochial injuries: a 10-year experience at Ratchaburi hospital. J Med Assoc Thail. 2005;88(1):32–40.
11. Cassada DC, Munyikwa MP, Moniz MP, Dieter RA Jr, Schuchmann GF, Enderson BL. Acute injuries of the trachea and major bronchi: importance of early diagnosis. Ann Thorac Surg. 2000;69(5):1563–7.
12. Chilimindris CP. Rupture of the thoracic esophagus from blunt trauma. J Trauma. 1977;17(12):968–71.

13. Schweigert M, Beattie R, Solymosi N, Booth K, Dubecz A, Muir A, et al. Endoscopic stent insertion versus primary operative management for spontaneous rupture of the esophagus (boerhaave syndrome): an international study comparing the outcome. Am Surg. 2013;79(6):634–40.
14. Goldstein LA, Thompson WR. Esophageal perforations: a 15 year experience. Am J Surg. 1982;143(4):495–503.
15. Inaba K, Branco BC, Menaker J, Scalea TM, Crane S, DuBose JJ, et al. Evaluation of multidetector computed tomography for penetrating neck injury: a prospective multicenter study. J Trauma Acute Care Surg. 2012;72(3):576–83; discussion 583–4; quiz 803–4
16. Chen JD, Shanmuganathan K, Mirvis SE, Killeen KL, Dutton RP. Using CT to diagnose tracheal rupture. AJR Am J Roentgenol. 2001;176(5):1273–80.
17. Gracias VH, Reilly PM, Philpott J, Klein WP, Lee SY, Singer M, et al. Computed tomography in the evaluation of penetrating neck trauma: a preliminary study. Arch Surg. 2001;136(11):1231–5.
18. Bowley DM, Plani F, Murillo D, Smith M, Degiannis E. Intubated, ventilating patients with complete tracheal transection: a diagnostic challenge. Ann R Coll Surg Engl. 2003;85(4):245–7.
19. Gomez-Caro A, Ausin P, Moradiellos FJ, Díaz-Hellín V, Larrú E, Pérez JA, et al. Role of conservative medical management of tracheobronchial injuries. J Trauma. 2006;61(6):1426–34; discussion 1434–5
20. Carretta A, Melloni G, Bandiera A, Negri G, Voci C, Zannini P. Conservative and surgical treatment of acute posttraumatic tracheobronchial injuries. World J Surg. 2011;35(11):2568–74.
21. Miller BS, Shafi S, Thal ER. Damage control in complex penetrating tracheal injury and silicone T-tube. J Trauma. 2008;64(2):E18–20.
22. Kazi M, Junaid M, Khan MJ, Ali NS, Masoom A. Utility of clinical examination and CT scan in assessment of penetrating neck trauma. J Coll Physicians Surg Pak. 2013;23(4):308–9.
23. de Lutio di Castelguidone E, Merola S, Pinto A, Raissaki M, Gagliardi N, Romano L. Esophageal injuries: spectrum of multidetector row CT findings. Eur J Radiol. 2006;59(3):344–8.
24. Weigelt JA, Thal ER, Snyder WH 3rd, Fry RE, Meier DE, Kilman WJ. Diagnosis of penetrating cervical esophageal injuries. Am J Surg. 1987;154(6):619–22.

25. Biancari F, D'Andrea V, Paone R, Di Marco C, Savino G, Koivukangas V, et al. Current treatment and outcome of esophageal perforations in adults: systematic review and meta-analysis of 75 studies. World J Surg. 2013;37(5):1051–9.
26. MacKenzie EJ, Rivara FP, Jurkovich GJ, Nathens AB, Frey KP, Egleston BL, et al. A national evaluation of the effect of trauma-center care on mortality. N Engl J Med. 2006;354(4):366–78.
27. Rossbach MM, Johnson SB, Gomez MA, Sako EY, Miller OL, Calhoon JH. Management of major tracheobronchial injuries: a 28-year experience. Ann Thorac Surg. 1998;65(1):182–6.
28. Mussi A, Ambrogi MC, Ribechini A, Lucchi M, Menoni F, Angeletti CA. Acute major airway injuries: clinical features and management. Eur J Cardiothorac Surg. 2001;20(1):46–51; discussion 51–2
29. Makhani M, Midani D, Goldberg A, Friedenberg FK. Pathogenesis and outcomes of traumatic injuries of the esophagus. Dis Esophagus. 2014;27:630.
30. Schweigert M, Solymosi N, Dubecz A, Stadlhuber RJ, Muschweck H, Ofner D, et al. Endoscopic stent insertion for anastomotic leakage following oesophagectomy. Ann R Coll Surg Engl. 2013;95(1):43–7.

Chapter 9
Lung Injury: Techniques

Joseph Rabin

Introduction of the Problem

A majority of patients with chest trauma, from either penetrating or blunt injury, can be managed nonoperatively with either observation or a chest tube [1–3]. When surgery is indicated, the purpose of pulmonary resection is to either control lung bleeding or excise injured tissue [4]. Patients who require thoracotomy following blunt trauma often have more severe injuries requiring a more complex operation and are associated with higher mortality [1–5].

Indication for urgent or emergent thoracic surgical exploration includes shock with a penetrating thoracic injury, chest tube output in excess of 1000–1500 cc after chest tube placement, continued chest tube output or ongoing bleeding greater than 200–300 cc/h, massive air leak, and cardiac tamponade [2, 6]. High chest tube output is indicative of continued bleeding that requires surgical control, while large air leak is concerning for a major tracheobronchial injury.

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Bronchoscopy is often necessary to thoroughly evaluate a major airway injury in order to help plan the appropriate operation and incision while patients also require a stable and secure airway to maintain oxygenation and ventilation. Once the patient's airway is established and secured, in the presence of concomitant life-threatening hemorrhage, surgical repair of many airway injuries is deferred until bleeding is controlled. Up to a third of patients who require thoracotomy for traumatic hemorrhage will also require a pulmonary resection [1]. Managing patients with severe chest trauma requires an understanding of thoracic surgical procedures, which can be effectively employed in unstable actively bleeding patients. The first issue is to determine what incision or approach should be utilized, and the second consideration is to determine what procedure should be performed.

Incision

There are various options available for surgical exposure, and it is important to be familiar with the advantages and limitations associated with each approach. The thoracic incision that is ultimately utilized should be versatile enough to address potential injuries in adjacent locations including the neck and abdomen [7]. While covered elsewhere, a few comments may be helpful.

Anterolateral Thoracotomy

This incision is rapid, avoids the time-consuming positioning associated with the traditional posterolateral thoracotomy, and does not require a sternal saw. It provides excellent exposure to the anterior hilum. This incision is the most common approach to the patient in extremis undergoing a salvage resuscitative procedure. It is also utilized in patients undergoing an exploratory laparotomy who decompensate and necessitate an emergent thoracic exploration. Finally, this incision

permits an extension into the contralateral hemithorax providing wide exposure of both hemithoraces and the anterior mediastinum (clamshell thoracotomy). Limitations include limited exposure of the posterior mediastinum especially the esophagus, aorta, and posterior aspect of the lung.

The inframammary crease is the landmark for the incision. The pectoralis muscle is divided followed by the intercostal muscles within the desired interspace, often the fourth or fifth. The internal mammary artery and vein are in close proximity to the sternum, and they should be preserved if possible.

Bilateral Anterior Thoracotomies

Also known as a clamshell thoracotomy, it is performed by starting with an anterolateral incision and extending it across the midline. It provides wide exposure of the anterior mediastinum, bilateral lungs, and pleural cavities. It does require either a Lebsche knife, sternal saw, or Gigli saw to divide the sternum horizontally. Retractors are placed to enhance exposure. This incision requires the identification and ligation of both internal mammary vessels.

Posterolateral Thoracotomy

This is the classic incision utilized for thoracic surgery and provides the best exposure of the thorax, especially the entirety of the lung. It does require more extensive preparation. It should only be utilized if the patient is hemodynamically stable and the injury is confined to a single hemithorax [8]. Correct positioning of the patient is essential and includes lateral positioning with the iliac crest at the level of the table break and rolls or an inflated bean bag to assist in stabilization. An axillary roll should be placed while bending the lower leg to about a 90° angle, keeping the upper leg straight with a pillow in between. The upper arm is placed up toward

the head, flexed at the elbow, and secured to an armrest. Often, one lung isolation is required and achieved with either a double-lumen endotracheal tube or bronchial blocker placed by anesthesia. The bed is flexed to help expand the intercostal spaces.

The incision extends from the level of the mid-scapula in between its edge and the spinous processes, swinging down and anterior through a point about 2–3 cm below the tip of the scapula and then anteriorly to the anterior axillary line and into the inframammary fold as needed. The latissimus dorsi muscle is then divided. The serratus anterior muscle is identified, and the adjacent fascia divided in an attempt to preserve this muscle. A scapular retractor is utilized to elevate the scapula and help identify the desired interspace by counting the ribs. The thorax is entered in the desired interspace by dividing the intercostal muscles with the electrocautery in a posterior to anterior direction along the superior edge of the inferior rib. A Finochietto retractor is then inserted and carefully opened.

All incisions are closed after placing chest tubes, which are taken out through separate stab incisions and secured to the skin. The ribs are reapproximated with interrupted intercostal sutures. The muscles are sutured to the adjacent fascia, followed by a subdermal and skin layer.

Sternotomy

This incision provides excellent exposure to the anterior mediastinum for quick access to the heart, great vessels, pericardium, and thymus. Thus, it is not a primary incision for pulmonary injuries. Injury to the lung may occur with an injury best repaired via a sternotomy. It does provide adequate exposure for many pulmonary procedures except for access to the left lower lobe.

The standard incision is from the jugular notch down to the xiphoid process. The ligamentous tissue just superior to the jugular notch should be divided with electrocautery, and

the retrosternal space bluntly mobilized digitally. The midline of the sternum is identified, scored, and then divided with a sternal saw while respirations are temporarily held. After inspecting the sternal edges and controlling the sternal bleeding, a sternal retractor is placed. After placing chest tubes, the incision is often closed with sternal wires to reapproximate the sternum and Vicryl suture in layers for the soft tissue.

Operative Technique with Personal Tips

Emergent thoracic trauma cases present challenges to achieving the isolated lung ventilation routinely employed in elective thoracic surgery. However, in patients who can be temporarily stabilized, placing a double-lumen tube can be very helpful. Having an experienced anesthesia team helps keep time to a minimum. However, most operations in the trauma setting are performed with a single-lumen endotracheal tube in patients with tenuous respiratory function. Temporary holding of ventilation and manual compression of lung parenchyma are some techniques that may facilitate the surgeon in overcoming this challenge of the lack of isolated lung ventilation [7].

Once the chest is entered, accumulated blood should be cleared and the injury assessed. The inferior pulmonary ligament should be divided to give maximal mobility to the lung (Fig. 9.1). Associated chest wall and/or vascular injuries should be identified before a definitive plan is made. One should assess the adequacy of exposure. If exposure is not adequate, the incision should be widened and/or a counter incision made to facilitate adequate exposure. If a sternotomy has been used, anterolateral thoracotomy should be considered. If an anterolateral thoracotomy has been used, converting to a clamshell should be considered. One should avoid struggling through an inadequate incision.

Formal pulmonary resections for trauma such as lobectomy and pneumonectomy are associated with high mortality rates. Other less morbid “lung-sparing” techniques have

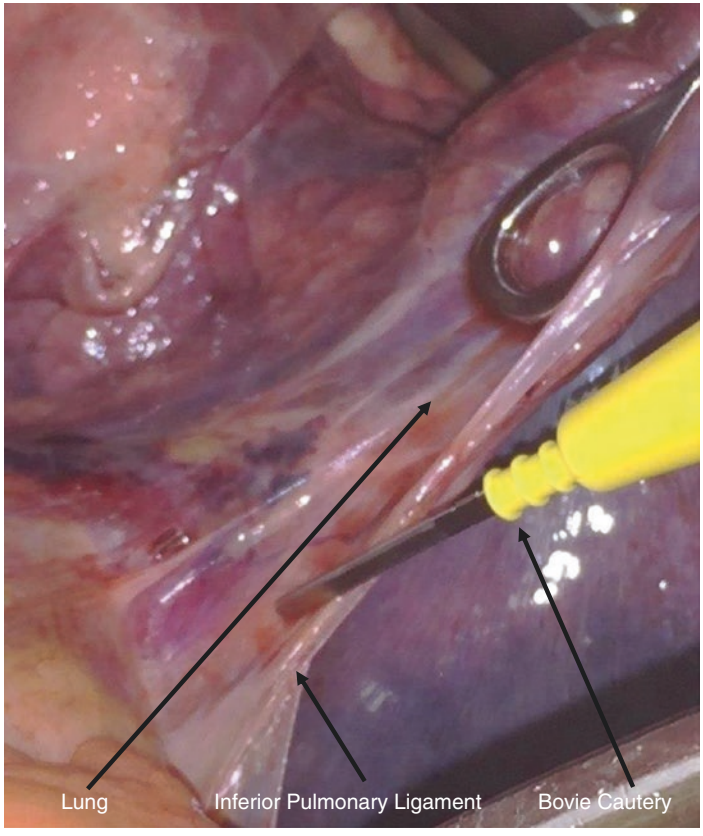

FIGURE 9.1 Dividing the inferior pulmonary ligament

evolved and include pneumonorrhaphy, tractotomy, and nonanatomic pulmonary resections. These less extensive procedures often utilize staplers and have shorter operative times, decreased blood loss, and less parenchymal loss, all of which may contribute to improved outcomes [9, 10]. One should still be familiar with all the possible surgical options but be prepared to perform a more extensive resection if lung-sparing attempts fail [11]. When performing any type of lung procedure, adequate exposure is essential. This is

accomplished by choosing the most appropriate incision as described earlier and by complete mobilization of the lung after entering the thoracic cavity. This includes lysing any pulmonary adhesions [7].

Pneumonorrhaphy

This is a common technique in which hemostasis is achieved and air leak sealed by direct suturing of the actively bleeding pulmonary injury (Fig. 9.2). A running locked suture technique can be employed to help achieve hemostasis [12]. This should only be utilized on peripheral superficial pulmonary injuries. Entry and exit injuries from penetrating wounds should usually not be oversewn since hemostasis may not actually be achieved. The risk is that only visible bleeding may be controlled while active hemorrhage may remain hidden, with continued, uncontrolled bleeding into the underlying pulmonary parenchyma risking the formation of an intrapulmonary shunt, bronchopulmonary fistula, aspiration, pneumonia or infection, and ARDS respiratory failure [2, 9, 13, 14].

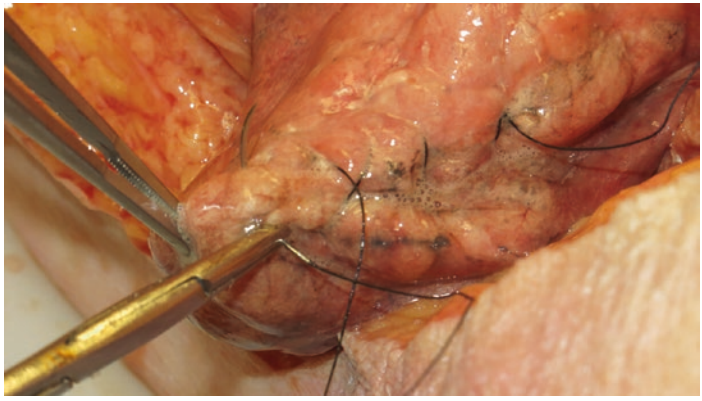

FIGURE 9.2 Pneumonorrhaphy

Tractotomy

This is a technique to rapidly control deep pulmonary parenchymal bleeding that does not involve the hilum or central bronchial vascular structures. It helps avoid a pulmonary resection, which was historically performed for such injuries, thus preserving lung tissue while preventing retention of a parenchymal hematoma [1, 11, 14, 15]. The sites of the entry and exit wounds are identified, and lung clamps are placed along the injury tract (Fig. 9.3). A GIA or TA stapler is placed through these openings and fired, which opens the injury tract. Bleeding vessels and injured airways are identified and ligated with absorbable suture. After controlling bleeding and air leaks, the pulmonary tissue can be closed with a running locked suture, or if feasible, the edges can be stapled [8, 12, 14, 15]. Once these techniques of air leak and bleeding control are completed, the stapled edges of lung tissue may also be sutured together to reapproximate divided parenchyma.

Patients treated with tractotomy often have shorter operative times and lower blood loss relative to formal lung resection. These patients also have less severe hypothermia and coagulopathy. If while performing a tractotomy it becomes evident that bleeding cannot be adequately controlled, often due to a more central injury location, conversion to a more extensive resection should be considered early [13].

Postoperative complications include bleeding and respiratory failure. Bleeding may either be by surgical or secondary to coagulopathy and should be carefully assessed by the surgical team. These patients also need aggressive postoperative pulmonary toilet due to the common occurrence of atelectasis and lobar collapse, which may also require repeated bronchoscopy for secretion clearance to maintain adequate parenchymal aeration [15]. An increased risk of infection has also been reported for those treated with tractotomy [10]; however, these data are limited.

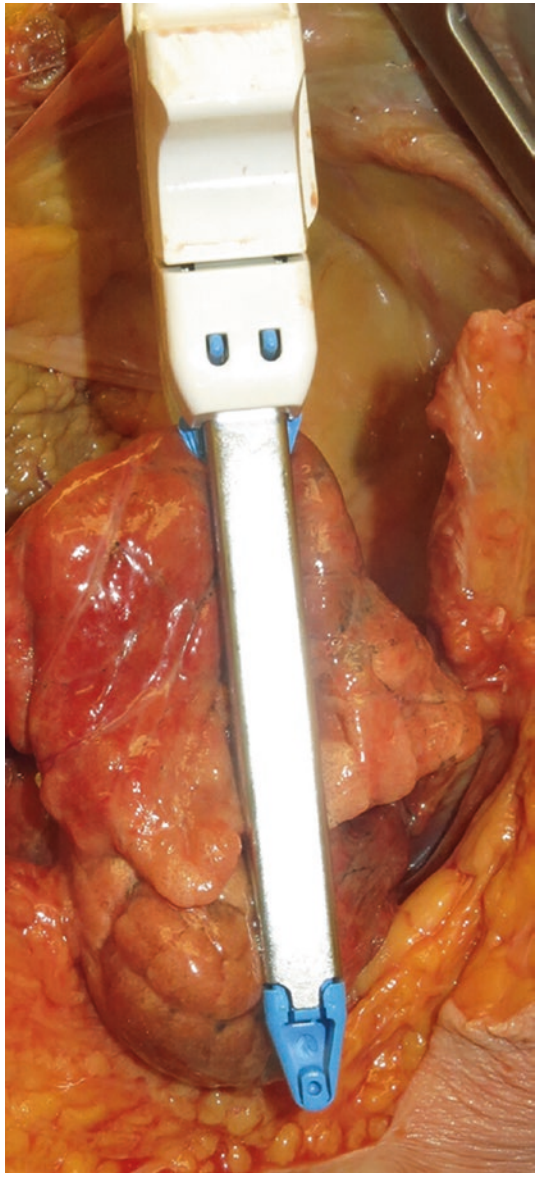

FIGURE 9.3 Tractotomy: a GIA stapler placed through the entry and exit wound sites

Wedge Resection

These are small nonanatomic lung resections of peripheral injuries with surgical staplers [1] with the goal of minimizing the amount of resected normal lung parenchyma. These less extensive nonanatomic resections should be utilized when possible to help avoid the associated morbidities of a formal anatomic lobectomy in a trauma setting [9, 10].

The procedure is often straightforward and is performed by firing a linear cutting stapler across the lung tissue, just under the damaged parenchyma requiring resection which is stabilized with a lung clamp (Fig. 9.4). The staple lines should then be inspected to ensure no air leak and with adequate hemostasis [16]. If necessary, the staple line can be reinforced with an additional firing or be oversewn.

Anatomic Resection: Lobectomy and Pneumonectomy

This procedure is usually reserved for central injuries or extensive lobar involvement involving a complex injury that cannot be managed via limited resection [4, 13]. Most anatomic resections are for hemorrhage control, major bronchial injury, a hilar injury, or significantly damaged lung

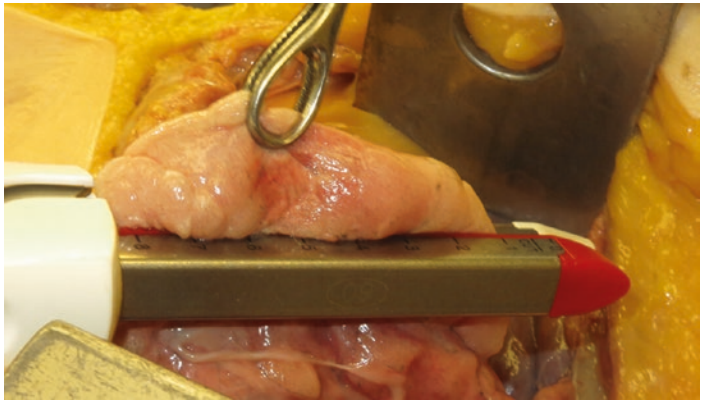

FIGURE 9.4 Right lower lobe wedge resection with GIA stapler

parenchyma in which lung salvage is not feasible. In a case of extensive parenchymal injury isolated to one lobe, it may be managed with a formal lobectomy. This may be performed in a standard anatomic approach or in a more expedited stapled fashion. Anatomic resections have been utilized more often for injuries in proximity to the main pulmonary artery or when stapling was not considered a viable safe option, while stapled lobectomies were often performed in patients who were more unstable and following blunt trauma [10]. A major hilar injury with resulting hemorrhagic shock may require a pneumonectomy. Control of hilar bleeding is challenging, and some techniques that have been utilized include manual compression of the hilum, clamping the hilum en masse, and lung twisting (Figs. 9.5 and 9.6). Proximal hilar

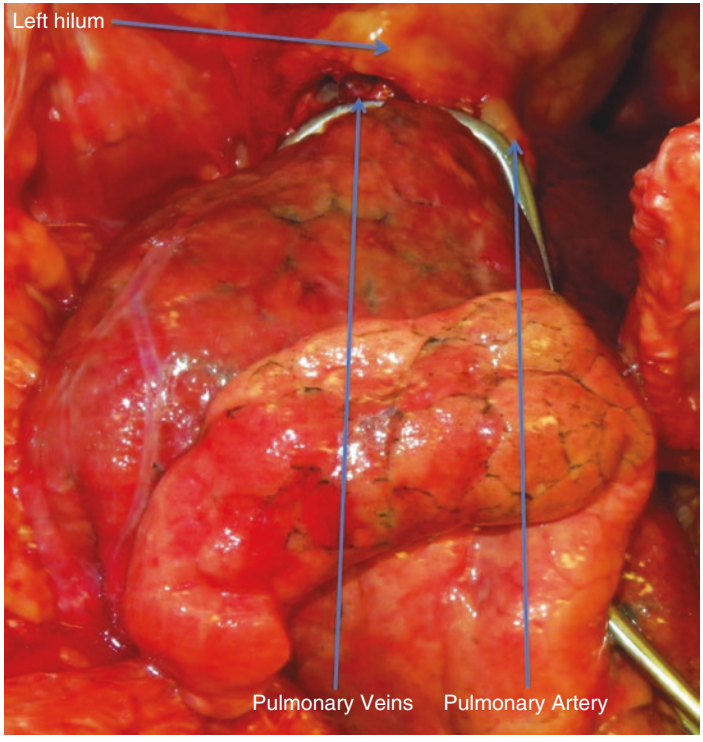

FIGURE 9.5 Clamping the left pulmonary hilum. Left main bronchus not visualized

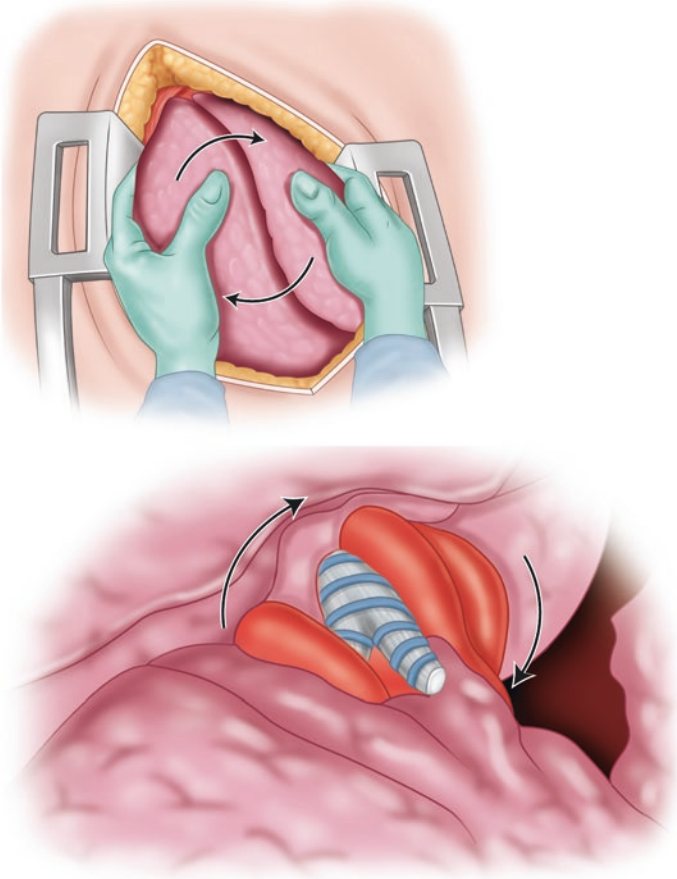

Figure 9.6 Pulmonary hilar twist. The pulmonary hilar twist being performed by rotating the upper and lower lobes clockwise, thereby occluding the proximal pulmonary vasculature

injuries may require opening the pericardium in an attempt to achieve vascular control [7] (Fig. 9.7).

Ideally, the pulmonary artery and vein branches are individually isolated and ligated, often with a vascular load of a stapler. Suture ligation with vascular sutures can also be used. The main stem bronchus is then also stapled, taking care to leave a short stump. If the patient is in extremis, the entire lobe may be resected en masse with a stapler (Fig. 9.8).

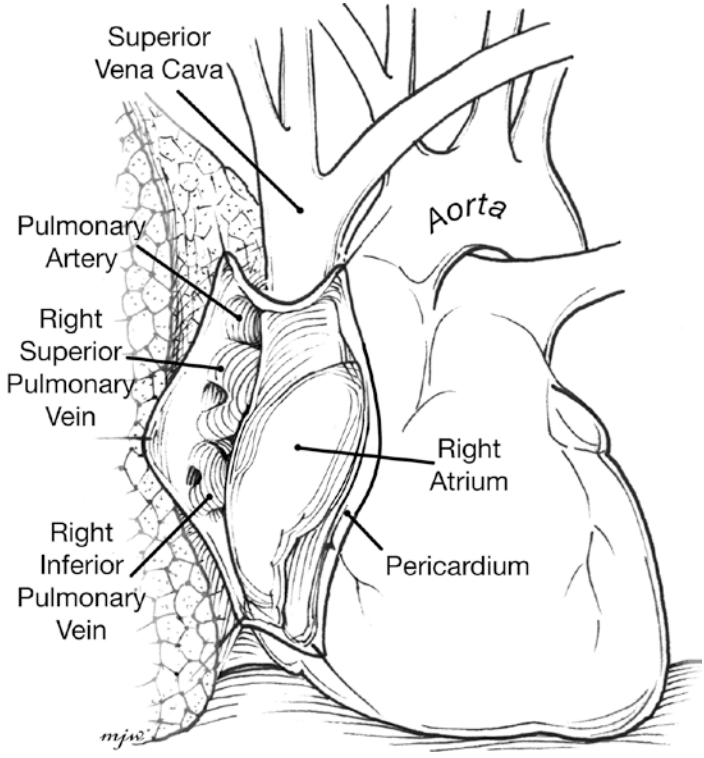

FIGURE 9.7 Intrapericardial exposure of the right hilum. Pericardial incision with exposure of the pulmonary veins lateral to superior vena cava and right atria

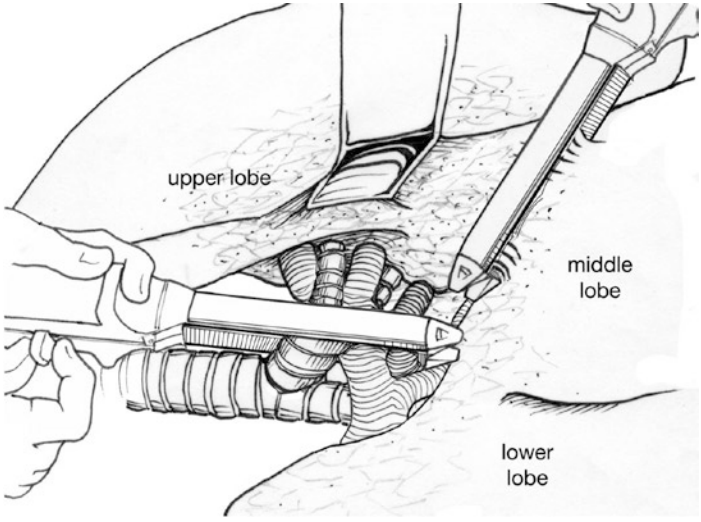

FIGURE 9.8 Stapled lobectomy. An expedited stapled lobectomy performed with a GIA stapler dividing the bronchus and vessels en masse and a GIA stapler dividing the fissure

If possible, we prefer to cover the bronchial stump closure. This is desirable in the event of lobectomy, but we feel more strongly if a pneumonectomy has been performed. Many options exist. Mobilizing an intercostal muscle is the easiest and is usually bulky enough to provide coverage (Fig. 9.9a, b). A tongue of pericardium or diaphragm also can be used. It can be made as large as needed. Finally, either a latissimus dorsi or pectoris flap can be used. These are bulky and provide the best coverage.

Trauma pneumonectomy is associated with significant postoperative morbidity and mortality. This is often associated with right heart failure due to an acute increase in pulmonary vascular resistance, with recent data showing worse outcomes when pneumonectomy is performed after blunt trauma [17]. The most important decision is to decide to do the pneumonectomy early. Acute right heart failure, while common, can be minimized if the procedure is performed

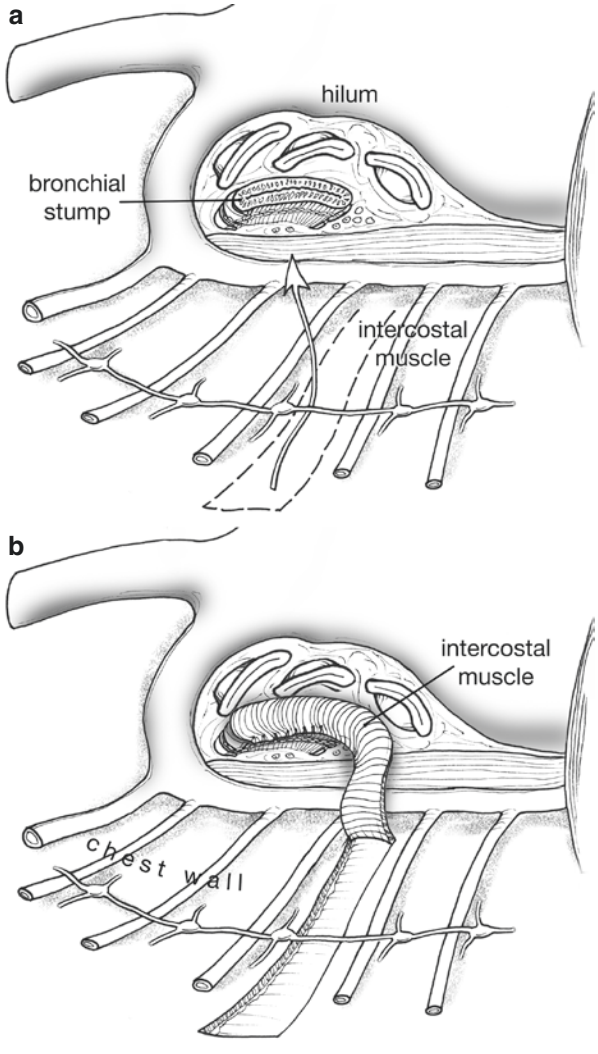

FIGURE 9.9 (**a**, **b**) Intercostal muscle flap. (**a**) Construction of intercostal pedicle flap adjacent to right hilum before application of the pedicle flap over the bronchus. (**b**) Flap applied to bronchus and suture applied to peribronchial tissues

early, before the patient is in refractory shock. We often begin supportive therapy such as pulmonary artery vasodilators and vasodilating inotropic support in the OR. A transesophageal echocardiogram can provide useful information. Finally, we typically leave the pericardium open to allow room for the right side of the heart to swell to avoid tamponade. There are also some limited experiences that suggest early initiation of ECMO support that may also improve outcomes [18]. In contrast to elective pneumonectomy which often does not have continuous post-op drainage of the pleural cavity, trauma pneumonectomy patients will often require at least passive drainage due to concern for postoperative bleeding and coagulopathy. The chest tube should be connected to water seal but not suction.

Damage Control

The principle of damage control involves an abbreviated operative procedure in which life-threatening hemorrhage is controlled, the chest is temporarily closed, and the patient is taken to the intensive care unit. Once stabilized physiologically, the patient is returned to the operating room for definitive management and chest closure [19]. Patients with severe chest trauma and associated physiologic derangements with associated shock that requires emergent thoracic operations should be considered for a damage control approach. The temporary chest closure helps prevent thoracic compartment syndrome while the patient is resuscitated in the intensive care unit [20]. Specific types of pulmonary wounds include those with penetrating thoracic injuries and a systolic blood pressure <90 mmHg and those patients who underwent an emergency department thoracotomy [21].

Maneuvers for thoracic damage control for severe pulmonary injury include cross clamping the pulmonary hilum, stapling across the hilum, and the hilar twist. The hilar twist involves taking down the inferior pulmonary ligament and rotating the lower lobe up, thus twisting the major pulmonary

vasculature and controlling the hemorrhage. Formal lung resections and classic control of the hilar vasculature are deferred for these faster techniques [22–24]. Other thoracic damage control strategies include utilizing large staplers for nonanatomic wedge resections and tractotomy. Suture closure of deep entry and exit wounds to the lung with significant bleeding and air leak should be avoided in order to prevent air embolism and continued intraparenchymal bleeding with subsequent infection [23, 24]. Finally, other considerations to minimize morbidity include minimization of excessive fluid infusion and attempts at blood salvage with cell savers or autotransfusion [25].

Post-op Complications

Postoperative complications are common following thoracic trauma procedures with atelectasis and persistent air leaks as two of the most frequent ones. Treatment of atelectasis involves early mobilization, incentive spirometry, nasotracheal suctioning, and aggressive secretion management that may also necessitate bronchoscopy. Air leaks often resolve, but additional procedures such as bronchoscopy and even reoperation may be necessary for leaks lasting longer than a few weeks. Development of a delayed leak is often associated with infection and requires immediate drainage and antibiotics and if no improvement surgical repair of the bronchial stump with a buttressed closure. In general, mortality and morbidity increase with extensiveness of pulmonary resection. In particular, traumatic pneumonectomy patients have greater than 50% mortality and overall very poor outcomes. Other morbidities include infection, pneumonia, respiratory failure, coagulopathy, and empyema [4, 26]. An important early intervention to try and reduce such postoperative complications is early bronchoscopy. This procedure is frequently performed immediately upon conclusion of the operation in which residual blood and clot is removed from the airways. This helps clear the airways,

which optimizes a patient's oxygenation and ventilation while simultaneously reducing the potential for infection and should be considered a routine procedure.

References

1. Huh J, Wall MW Jr, Estrera AL, Soltero ER, Mattox KL. Surgical management of traumatic pulmonary injury. Am J Surg. 2003;186:620–4.
2. Meredith JW, Hoth JJ. Thoracic trauma: when and how to intervene. Surg Clin North Am. 2007;87:95–118.
3. Stewart KC, Urschel JD, Nakai SS, Gelfand ET, Hamilton SM. Pulmonary resection for lung trauma. Ann Thorac Surg. 1997;63:1587–8.
4. Martin MJ, McDonald JM, Mullenix PS, Steele SR, Demetriades D. Operative management and outcomes of traumatic lung resection. J Am Coll Surg. 2006;203:336–44.
5. Karmy-Jones R, Jurkovich GJ, Nathens AB, Shatz DV, Brundage S, Wall MJ Jr, et al. Timing of urgent thoracotomy for hemorrhage after trauma. Arch Surg. 2001;136:513–8.
6. Demetriades D, Velmahos GC. Penetrating injuries of the chest: indications for operation. Scand J Surg. 2002;91:41–5.
7. DuBose J, O'Connor JV, Scalea TM. Lung, trachea and esophagus. In: Mattox KL, Moore EE, Feliciano DV, editors. Trauma. 7th ed. New York: McGraw Hill; 2013. p. 468–84.
8. Petrone P, Asensio JA. Surgical management of penetrating pulmonary injuries. Scand J Trauma Resusc Emerg Med. 2009;17:8.
9. Cothren C, Moore EE, Biffl WL, Franciose RJ, Offner PJ, Burch JM. Lung-sparing techniques are associated with improved outcome compared with anatomic resection for severe lung injuries. J Trauma. 2002;53:483–7.
10. Karmy-Jones R, Jurkovich GJ, Shatz DV, Brundage S, Wall MJ, Engelhardt S, et al. Management of traumatic lung injury: a western trauma association multicenter review. J Trauma. 2001;51:1049–53.
11. Gasparri M, Karmy-Jones R, Kralovich KA, Patton JH Jr, Arbabi S. Pulmonary tractotomy versus lung resection: viable options in penetrating lung injury. J Trauma. 2001;51:1092–7.
12. Asensio JA, Demetriades D, Berne JD, Velmahos G, Cornwell EE, Murray J, et al. Stapled pulmonary tractotomy: a rapid way to control hemorrhage in penetrating pulmonary injuries. J Am Coll Surg. 1997;185:486–7.

13. Velmahos GC, Baker C, Demetriades D, Goodman J, Murray JA, Asensio JA. Lung-sparing surgery after penetrating trauma using tractotomy, partial lobectomy, and pneumonorrhaphy. Arch Surg. 1999;134:186–9.
14. Livingston DH, Hauser CJ. Chest wall and lung. In: Feliciano DV, Mattox KL, Moore EE, editors. Trauma. 6th ed. New York: McGraw Hill; 2008. p. 525–52.
15. Wall MJ Jr, Villavicencio RT, Miller CC, Aucar JA, Granchi TA, Liscum KR, et al. Pulmonary tractotomy as an abbreviated thoracotomy technique. J Trauma. 1998;45:1015–23.
16. Ferguson MK. Thoracic surgery atlas. Philadelphia, PA: W.B. Saunders; 2007. p. 6–32.
17. Matsushima K, Aiolfi A, Park C, Rosen D, Strumwasser A, Benjamin E, et al. Surgical outcomes after trauma pneumonectomy: revisited. J Trauma Acute Care Surg. 2018;82:927–32.
18. Halonen-Watras J, O'Connor J, Scalea T. Traumatic pneumonectomy: a viable option for patients in extremis. Am Surg. 2011;77:493–7.
19. Vargo DJ, Battistella FD. Abbreviated thoracotomy and temporary chest closure, an application of damage control after thoracic trauma. Arch Surg. 2001;136:21–4.
20. O'Connor JV, Dubose J, Scalea TM. Damage-control thoracic surgery: management and outcomes. J Trauma Acute Care Surg. 2014;77:660–5.
21. Wyrzykowski AD, Feliciano DV. Trauma damage control. In: Feliciano DV, Mattox KL, Moore EE, editors. Trauma. 6th ed. New York: McGraw Hill; 2008. p. 851–69.
22. Wilson A, Wall MJ Jr, Maxson R, Mattox K. The pulmonary hilum twist as a thoracic damage control procedure. Am J Surg. 2003;186:49–52.
23. Phelan HA, Patterson SG, Hassan MO, Gonzalez RP, Rodning CB. Thoracic damage-control operation: principles, techniques, and definitive repair. J Am Coll Surg. 2006;203:933–41.
24. Wall MJ Jr, Soltero E. Damage control for thoracic injuries. Surg Clin North Am. 1997;77:863–78.
25. Reul GJ, Mattox KL, Beall AC Jr, Jordan GL Jr. Recent advances in the operative management of massive chest trauma. Ann Thorac Surg. 1973;16:52–63.
26. Burke SJ, Faber LP. Complications of pulmonary resection. In: Little AG, editor. Complications in cardiothoracic surgery, avoidance and treatment. Elmsford, NY: Blackwell; 2004. p. 67–91.

Chapter 10
Cardiac Injury: Techniques

Ronald Tesoriero

Scope of the Problem

Cardiac wounds are uncommon and seen mainly at urban trauma centers [1]. Despite advancements in surgical care, prehospital treatment, and resuscitation, cardiac injury remains highly lethal. In fact, over several decades survival has not improved, underscoring their lethality [1–6]. One factor that may influence the poor outcome is a mortality bias due to the rapid transit of patients with devastating, non-survivable injuries to the hospital [4, 7].

When considering only the patients who survive to treatment at trauma centers, the incidence of cardiac injury is quite low. Asensio [8] defined the national incidence of penetrating cardiac injuries to be 0.16% in a study utilizing the National Trauma Data Bank (NTDB), and Rhee [4] defined the penetrating cardiac injury rate as 1 in 210 admissions in a single-center retrospective analysis over 7 years. The number

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of patients with blunt cardiac rupture who survive to treatment is even less common, with an overall incidence of 0.041% [9]. However, it is estimated that 8–86% of immediate deaths after significant blunt chest trauma are a result of blunt cardiac rupture [9].

Gunshot wounds (GSWs) account for the majority of penetrating cardiac injuries (58–76%) [1, 4, 8], while stab wounds account for most of the remainder. Unfortunately, the mortality associated with GSWs is significantly higher (32.6–84%) than that of stab wounds (9.7–35%) [1, 4, 10]. Blunt cardiac rupture is the most lethal of cardiac wounds with rates of mortality approaching 90% [9].

History of Care of Cardiac Injury

Claude Beck discussed the history of cardiac injuries in a 1926 review article and described three distinct periods: mysticism (ancient times to the sixteenth century), experimentation and observation (sixteenth to the late nineteenth century), and cardiac repair (early twentieth century to present) [11].

Homer's *Iliad* [12] includes the earliest known description of cardiac injuries, and authors in antiquity including Hippocrates [13, 14], Aristotle [15], and Galen [16] considered these wounds to be universally fatal [14]. In the sixteenth and seventeenth century, Hollerius [14, 17] promoted the idea that not all wounds to the heart were necessarily mortal, and Wolf [18] and Cabriolanus [14] described healed cardiac wounds.

Morgagni reported the first case of traumatic pericardial tamponade. Larrey described successful treatment of traumatic pericardial tamponade by inserting a catheter through a stab wound to the chest [14]. There was strong resistance to surgeons attempting cardiac repairs, which was typified by Billroth's assertion that such an attempt "approaches very closely to that kind of intervention which some surgeons would term a prostitution of the surgical act and other madness" [11, 14].

Despite this resistance, Roberts suggested cardiac injuries could be sutured, and Block demonstrated that successful

repair of cardiac wounds with survival in a rabbit model was possible [11, 14]. This was ultimately demonstrated in humans in the late 1890s when Axel Cappelen successfully repaired a left ventricular laceration, though the patient subsequently succumbed to sepsis [11, 14].

The new era of cardiac repair began when Ludwig Rehn, at the 26th Congress of the German Surgical Society in 1897, described the successful repair of a right ventricular wound with survival. He stated, "The feasibility of cardiorrhaphy no longer remains in doubt … I trust that this case will not remain a curiosity, but rather, that the field of cardiac surgery will be further investigated. Let me speak once more my conviction that by the means of cardiorrhaphy, many lives can be saved that were previously counted as lost" [19].

The ensuing century saw an explosion of innovation and advancement in cardiac surgery, which included the advent of positive pressure ventilation and cardiopulmonary bypass. In parallel were improvements in care of the trauma patient including prehospital care, patient transport, and concepts of volume resuscitation, as well as the development of anesthesia and critical care specialties. These all led to the improved outcome of those sustaining cardiac injuries [20, 21].

Techniques

Diagnosis

Initial Evaluation

Cardiac injury occurs in 6.4% of cases of penetrating thoracic trauma [22]. Wounds involving the "cardiac box" (an imaginary area inferior to the clavicles, superior to the costal margins, and medial to the midclavicular lines) (Fig. 10.1) have been previously described as the most worrisome [23]. However, penetrating thoracic wounds that occur outside "the box" can also result in cardiac injury [24–26]. Thoracic wounds occurring between the anterior and posterior axillary

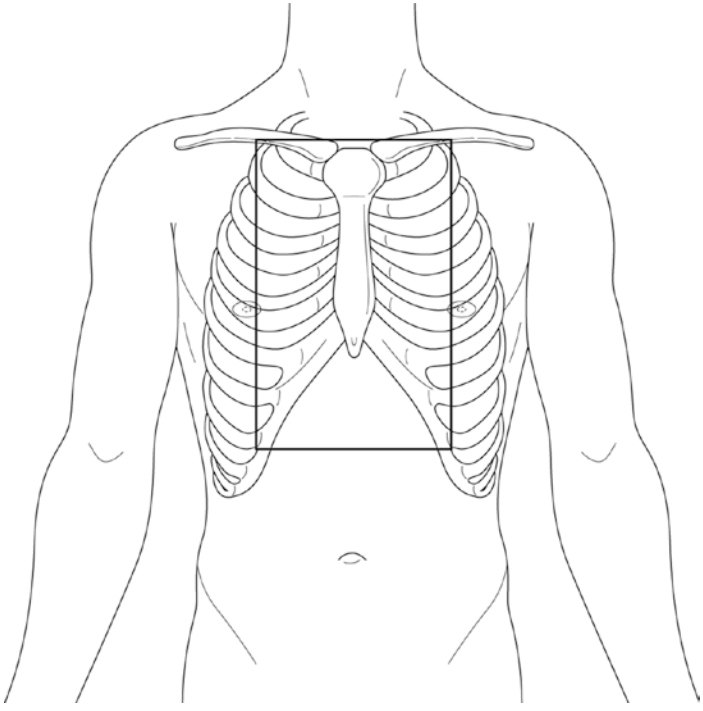

Figure 10.1 The cardiac box. Penetrating injuries within the borders inferior to the clavicles, superior to the costal margin, and between the midclavicular lines should increase the concern for cardiac injury. However, thoracic wounds that occur outside these borders may have an equal likelihood of cardiac injury

line on both the right and left and to the posterior chest medial to the scapulas have as high or a higher risk of cardiac injury compared to those that occur in "the box" [26]. The increased mortality with cardiac injuries that occur with wounds outside "the box" is at least in part related to the clinician having a lower suspicion leading to delays in diagnosis [24].

Patients who arrive at the trauma center after cardiac injury may present anywhere along the range from hemodynamically normal to cardiac arrest. Patients who manifest

hemodynamic compromise may have exsanguinating hemorrhage, cardiac tamponade, or both. The classic findings of Beck's triad (muffled heart sounds, hypotension, and jugular venous distension), caused by the rapid accumulation of a small amount of blood (30–50 ml) in the non-expansible fibrous pericardium, are rarely recognized in the resuscitation suite. Delayed recognition of hemopericardium will result in the development of tamponade physiology, decreased cardiac filling due to high intrapericardial pressures, impaired cardiac output, hypotension, and death.

On presentation, an evaluation that includes a brief history, identification of the location and possible trajectory of wounds, and an evaluation of the cardiopulmonary status of the patient must be completed rapidly. Resuscitative thoracotomy may be lifesaving for patients who present in extremis [1, 10, 20, 23, 27, 28]. As recommended by the Western Trauma Association, its use has expanded to include patients without signs of life with short prehospital times if there is electrical or ultrasound-visualized cardiac activity and in cases of asystole if signs of tamponade are present or cannot be excluded (Box 10.1) [28, 29].

FAST

The focused assessment with sonography for trauma (FAST) examination is extremely sensitive for detecting hemopericardium in precordial and penetrating transthoracic wounds, with sensitivity and specificity that approach 100% [30–32]. It is especially useful in patients who present with maintained hemodynamics to assess the safety of subsequent diagnostic testing [31] and to determine the appropriate management in hemodynamically compromised patients with multiple wounds and potential for multi-cavitary hemorrhage. Occasionally, adequate windows will not be obtainable due to concomitant hemopneumothorax or subcutaneous emphysema. Penetrating precordial or left thoracic wounds with concurrent hemothorax require special consideration as the FAST examination has been shown to produce false-negative

results due to cardiac injury decompressing into the left hemithorax [33]. In these cases, the liberal use of subxiphoid pericardial window is wise, as repeat ultrasonography and echocardiography may fail to diagnose underlying cardiac injury [33].

Extended FAST (EFAST) is extremely useful for the diagnosis of pneumothorax with a high specificity (99%) and sensitivity (>95%) when compared to chest X-ray (CXR) [34, 35]. It may also be utilized to diagnose hemothorax.

CXR

A CXR is important to assist in missile trajectory, presence of concomitant thoracic injury, and operative planning. It can be obtained in most patients even when hemodynamically marginal. For those who need emergent operative intervention, a plate can be placed on the operating room table and a CXR obtained prior to incision [25].

Subxiphoid Pericardial Window

Subxiphoid pericardial window has been validated to be extremely accurate for overt and occult cardiac injuries [6]. It can be utilized to diagnose hemopericardium in patients with equivocal ultrasound results, when ultrasound windows are unobtainable, when ultrasound is unavailable, or when a precordial stab wound is associated with a left hemothorax [33]. It should be performed under general anesthesia in the operating room (OR) with the patient prepared and draped in an adequate fashion to proceed with sternotomy if necessary. When possible, it is wise to have the patient adequately prepared and draped prior to the induction of anesthesia as patients with compensated tamponade may quickly deteriorate to cardiac arrest when preload is decreased from vasodilation and positive pressure ventilation [6]. An incision is made in the midline overlying the xiphoid, which should be excised to facilitate dissection and exposure [6]. The distal sternum is then elevated with a retractor. The diaphragm is

identified and kept inferior, while blunt dissection is utilized to identify the pericardium. Meticulous hemostasis is necessary to prevent difficult-to-interpret results of the pericardial window. Once identified, the pericardium is grasped with Allis or Tonsil clamps and incised. If clot or blood is returned, immediately or after pericardial irrigation, conversion to a sternotomy and direct cardiac inspection should occur.

There have been several studies that suggest the rate of nontherapeutic sternotomy for hemodynamically stable patients with hemopericardium identified on subxiphoid pericardial window is between 38% and 93% [36, 37]. These are identified as tangential superficial partial-thickness endocardial injuries and pericardial lacerations without cardiac injury. Two studies have evaluated the use of subxiphoid pericardial window and drainage alone for hemodynamically stable patients whose hemopericardium resolves after initial lavage [37, 38]. No patients required subsequent conversion to sternotomy or thoracotomy, there were no mortalities, and ICU and hospital length of stay were decreased compared to those undergoing sternotomy. Although interesting, long-term follow-up for these patients is not available, and it is unknown whether delayed complications may occur (ventricular aneurysm) related to unrepaired partial-thickness injuries. At this point, procedures other than direct cardiac inspection after positive subxiphoid pericardial window should only be utilized as part of a research protocol.

Incisions

Median Sternotomy

Median sternotomy is most useful for stable patients who have proven cardiac injury or those that are at least marginally stable [20]. It provides excellent exposure to the mediastinum and can be extended for a laparotomy if indicated. It is our practice to explore most of these wounds via sternotomy even with a concomitant lung injury, as both pleural spaces are accessible. Surgeons with less experience may find the incision limiting and should utilize anterolateral thoracotomy with extension as necessary.

Anterolateral Thoracotomy

Anterolateral thoracotomy is the incision of choice for patients arriving in extremis with severe hemodynamic compromise (Fig. 10.2) and may be extended to a right anterior "clamshell" thoracotomy (Fig. 10.3) when necessary. It is the most useful approach for ED thoracotomy [1, 4, 10, 20, 23, 27]. It is also useful in patients who deteriorate during laparotomy from a suspected cardiac injury and for those who are hemodynamically unstable from injuries that have traversed the mediastinum [20, 23]. Care must be taken to not perform

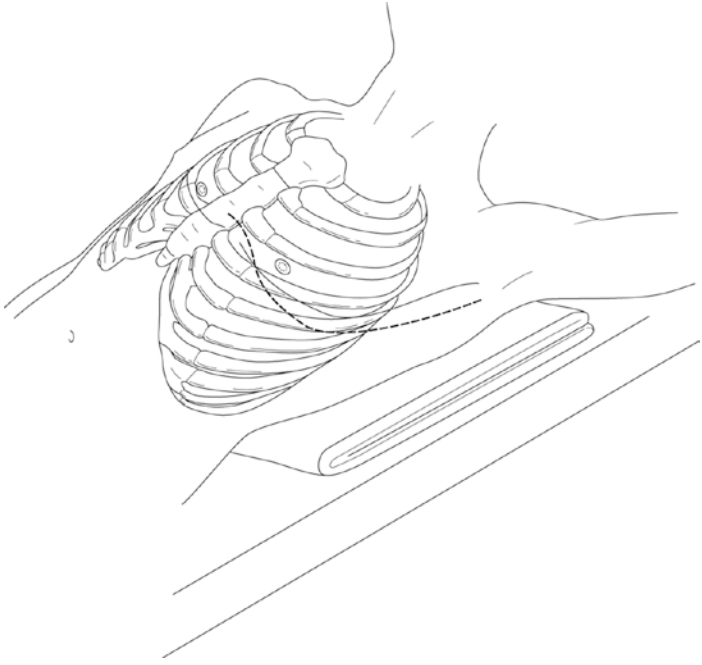

Figure 10.2 Left anterolateral thoracotomy. The incision is made at the inframammary fold, carried into the fourth intercostal space, and curved toward the ipsilateral axilla. Positioning a rolled towel under the left hemithorax allows for additional extension of the incision and improved visualization of the posterior thoracic structures

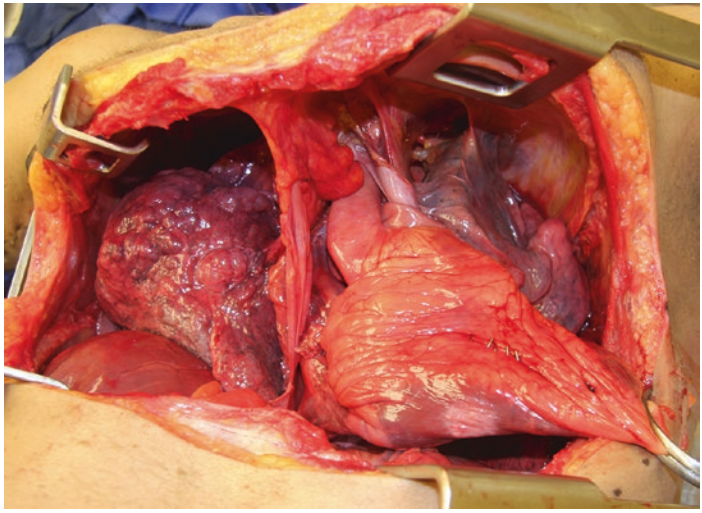

FIGURE 10.3 Bilateral anterolateral "clamshell" thoracotomy. A left anterolateral thoracotomy can be extended across the sternum and into the fourth intercostal space on the right. This affords excellent exposure to the mediastinum and bilateral pleural spaces. Attention must be paid to proper incision placement, as incisions that are made too low may limit access to the upper mediastinum and great thoracic vessels (Facilities: Surgical Laboratory, Anatomical Services Division, School of Medicine, UMB. Cadavers and specimens: Anatomical Donor, Maryland State Anatomy Board)

the incision too inferiorly or optimum exposure may be limited and subsequent thoracic and sternal closure may be compromised [6]. Elevating the left hemithorax 20° by placing a roll under the left chest improves exposure [6] and should be considered in hemodynamically maintained patients and those with penetrating thoracic wounds who are undergoing laparotomy in case rapid entry into the chest is necessary.

When the clamshell thoracotomy is utilized, it is important to ligate the transected internal mammary arteries, after repairing the cardiac injuries [20]. In situations of extremis, the arteries will often be in vasospasm and thus easy to overlook.

The surgeon who omits this step will have the opportunity to return to the operating room to correct their mistake should the patient survive the subsequent hemorrhage.

Exposure and Immediate Control

Pericardiotomy is the first step in evaluation of the injured heart. In the presence of tamponade, the pericardium can be tense and difficult to grasp. Uncontrolled opening can result in injury to the underlying cardiac structures. The use of Allis or Tonsil clamps to grasp and elevate the pericardium helps. Initially, it is wise to make a 1–2 cm incision with a scalpel. The pericardium is further incised with Metzenbaum scissors [20, 23]. When approached from an anterolateral thoracotomy, the phrenic nerve is identified and the pericardium is widely opened anterior and longitudinal to it. The incision can be extended medially at the pericardial base if further exposure is needed. The pericardium is opened in the midline when a sternotomy incision is utilized and may be teed off at its inferior aspect for better exposure, again avoiding the phrenic nerve. In the stable patient, taking the time to suture the pericardium to the skin to create a cardiac sling facilitates visualization and repair [6].

Once the pericardium is opened, the surgeon should rapidly assess for location and degree of cardiac injury, paying special attention to wounding patterns that suggest multiple, posterior, septal, or valvular injuries. Involvement of the thoracic great vessels is also important to identify. The American Association for the Surgery of Trauma (AAST) Organ Injury Scale (OIS) [39] (Table 10.1) can be utilized to classify injury and has been validated to predict mortality [1, 10, 20] but offers little benefit for intraoperative decision-making.

Techniques that can be employed to gain temporary control include direct digital pressure, placement of a Foley catheter, application of a partial occluding clamp, and the use of skin staples. Direct digital control (Fig. 10.4) proves the most

useful for amenable wounds. It allows for tactile feedback, minimizes chances of wound extension, and frees the surgeon's other hand to achieve repair. Foley catheter placement (Fig. 10.5) can be utilized to partially occlude larger wounds, but one must remember to clamp the catheter to prevent continued hemorrhage through it and to inflate the balloon

Table 10.1 American association for the surgery of trauma organ injury scale: heart injury

Grade	Description of injury
I	Blunt cardiac injury with minor ECG abnormality (nonspecific ST or T wave changes, premature atrial or ventricular contraction, or persistent sinus tachycardia)
	Blunt or penetrating pericardial wound without cardiac injury, cardiac tamponade, or cardiac herniation
II	Blunt cardiac injury with heart block (right or left bundle branch, left anterior fascicular, or atrioventricular) or ischemic changes (ST depression or T wave inversion) without cardiac failure
	Penetrating tangential myocardial wound up to, but not extending through, the endocardium, without tamponade
III	Blunt cardiac injury with sustained (≥6 beats/min) or multifocal ventricular contractions
	Blunt or penetrating cardiac injury with septal rupture, pulmonary or tricuspid valvular incompetence, papillary muscle dysfunction, or distal coronary arterial occlusion without cardiac failure
	Blunt pericardial laceration with cardiac herniation
	Blunt cardiac injury with cardiac failure

(continued)

Table 10.1 (continued)

Grade	Description of injury
IV	Penetrating tangential myocardial wound up to, but not extending through, the endocardium, with tamponade
	Blunt or penetrating cardiac injury with septal rupture, pulmonary or tricuspid valvular incompetence, papillary muscle dysfunction, or distal coronary arterial occlusion producing cardiac failure
	Blunt or penetrating cardiac injury with aortic or mitral valve incompetence
	Blunt or penetrating cardiac injury of the right ventricle, right atrium, or left atrium
V	Blunt or penetrating cardiac injury with proximal coronary arterial occlusion
	Blunt or penetrating left ventricular perforation
	Stellate wound with <50% tissue loss of the right ventricle, right atrium, or left atrium
VI	Blunt avulsion of the heart; penetrating wound producing >50% tissue loss of a chamber
Advance one grade for multiple wounds to a single-chamber or multiple-chamber involvement	

Modified with permission of Wolters Kluwer Health Inc from Moore et al. [40]

with saline to prevent air embolism should rupture occur due to inadvertent needle injury. Undue traction on the Foley may easily cause extension and enlargement of the cardiac injury [6]. A partial occluding clamp is an attractive option when the injury is to the atria. Skin staples, though rapid, can

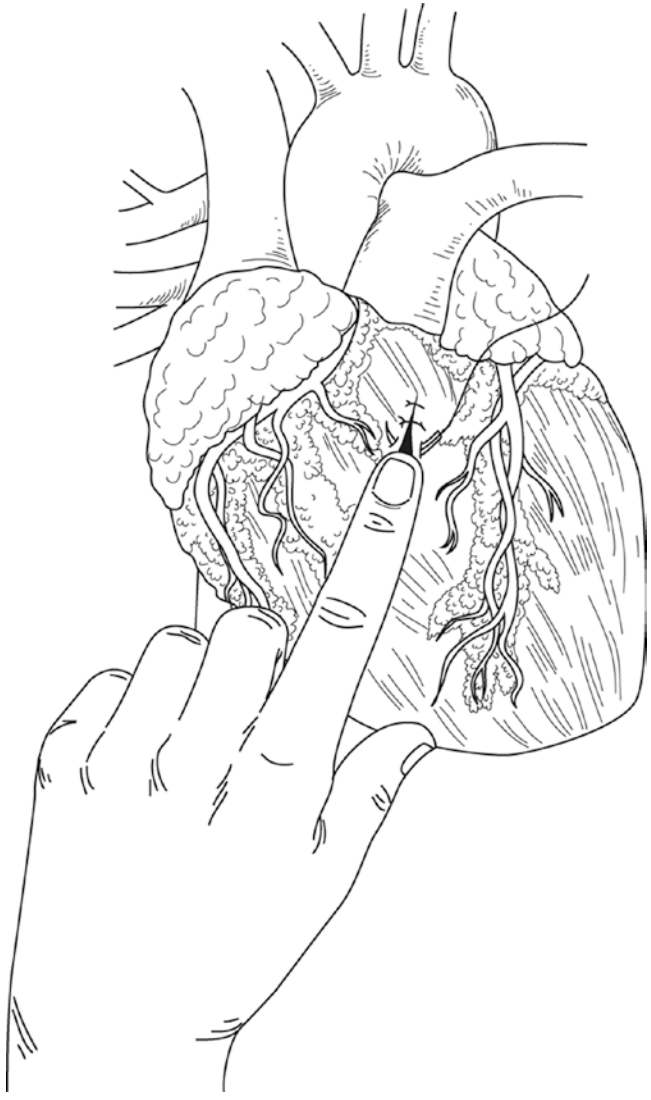

FIGURE 10.4 Direct digital control of cardiac wound. Wounds of amenable size are best controlled with direct digital pressure as there is minimal chance for wound extension. The physician's other hand is freed to achieve repair by placing sutures directly under the finger controlling the wound

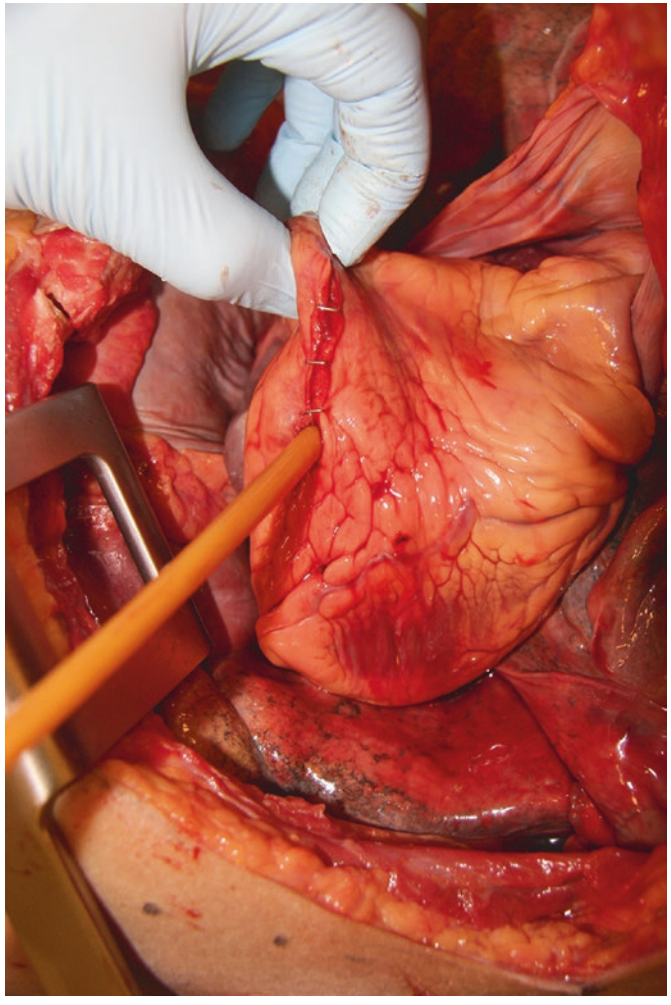

FIGURE 10.5 Foley catheter placement to control cardiac wound. For larger wounds, a Foley catheter may be employed to partially control ongoing hemorrhage while a temporary or permanent repair is achieved. Care must be taken to avoid undue traction on the catheter as wound extension and enlargement may easily occur (Facilities: Surgical Laboratory, Anatomical Services Division, School of Medicine, UMB. Cadavers and specimens: Anatomical Donor, Maryland State Anatomy Board)

be difficult to place to achieve hemostasis, can result in wound extension, should always be followed with definitive repair, and are difficult to remove [1, 10, 20, 23].

Near-total inflow occlusion with Sauerbruch's maneuver can be achieved by placing the third and fourth finger behind the right atrium and compressing it between the second and third finger; vascular clamps may be added to the inferior vena cava and superior vena cava if needed due to injury pattern (Fig. 10.6). It may be necessary for large posterior injuries, left atrial injuries, and injuries that occur in the very lateral aspect of the right atrium and to the superior and inferior atrio-caval junction. Unfortunately, it is poorly tolerated in the acidotic, hypotensive patient and often results in rapid deterioration to cardiac arrest from which the patient may not be resuscitable [20, 23]. However, the brief application of the maneuver to allow identification and positioning of a vascular or series of intestinal allis clamps to control the injury may be life saving.

Patients with central pulmonary injuries may require pulmonary hilar control with cross clamping or a lung twist to arrest hemorrhage and prevent air embolism. These techniques should be used cautiously as they are poorly tolerated, significantly increase the afterload of the right ventricle, and can result in acute right failure or fibrillation in the acidotic and ischemic heart [20, 23]. When employed, rapid control of the injury with stepwise release of the clamp should follow to minimize this risk.

Definitive Repair

Atrial Injuries

Due to their low pressure, atrial injuries are generally easier to repair than ventricular injuries. Control can be achieved with a partial occluding clamp (Fig. 10.7). Once controlled, they should be repaired utilizing 3-0 or 4-0 polypropylene suture in either a running or interrupted horizontal mattress

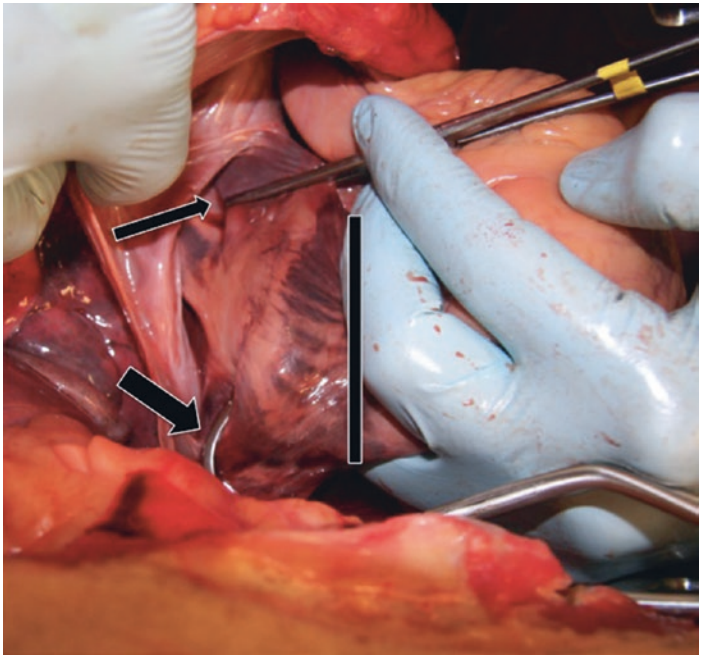

Figure 10.6 Sauerbruch's maneuver. To achieve total inflow occlusion of the heart, the junction of the vena cava and right atrium may be compressed between the third and fourth fingers (solid vertical line). For injuries to the medial aspect of the atrium or the atrio-caval junction, the brief application of vascular clamps to the superior vena cava (narrow arrow) and inferior vena cava (wide arrow) may be necessary to achieve temporary control with a vascular or series of intestinal Allis clamps (Facilities: Surgical Laboratory, Anatomical Services Division, School of Medicine, UMB. Cadavers and specimens: Anatomical Donor, Maryland State Anatomy Board)

fashion. To prevent tearing of their thin walls, we prefer the horizontal technique. Pledgeted repairs are generally unnecessary.

Injuries to the lateral aspect of the right atrium and to the atrio-caval junctions may require total inflow occlusion for

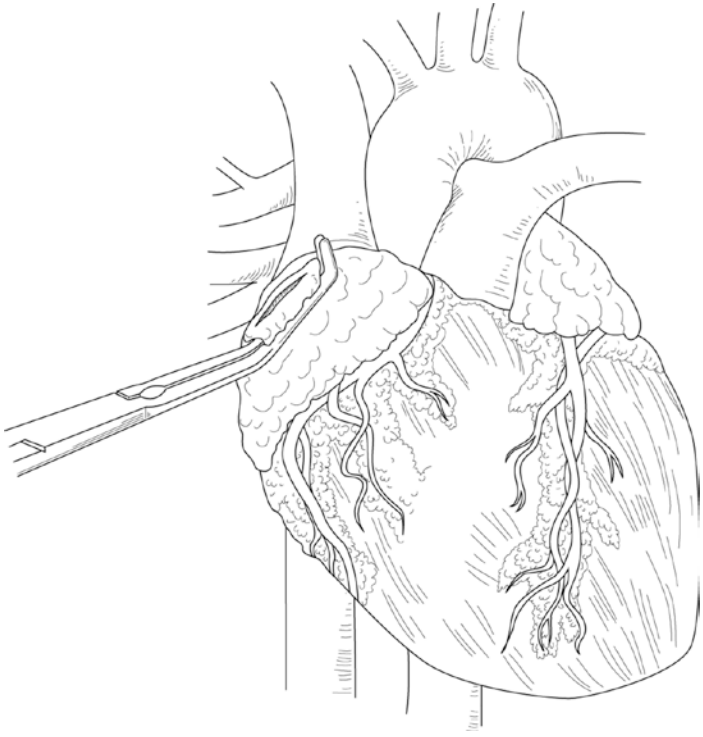

FIGURE 10.7 Atrial control with a Satinsky clamp. The low-pressure nature and anterior location of the right atrium lend itself well to control with an angled vascular clamp. Repair may then proceed with a 4-0 polypropylene suture in a horizontal mattress fashion. Although this also may be employed for left atrial wounds, the posterior location and inability of the heart to tolerate anterior displacement make placement of a clamp impractical

visualization as discussed earlier. This may be followed by the rapid sequential placement of intestinal Allis clamps [41] allowing for release of inflow occlusion and controlled repair of the injury without continued compromise of venous return to the heart.

Ventricular Injuries

The ventricles are the most commonly injured structure in the heart from penetrating trauma [1, 4, 8, 10, 20, 23]. Wounds that are amenable should be controlled with digital pressure as mentioned. Precise needle placement is necessary, and utilizing a needle with appropriate length and curvature, while following the curve of the needle, facilitates adequate tissue bites, thus avoiding myocardial tearing with extension of the injury [6]. We usually use a 3-0 polypropylene suture on an MH needle, but if unavailable, an SH needle will suffice. Timing the suture placement to myocardial contraction will also help to limit the chance of iatrogenic injury [6]. Stab wounds may be closed in a running or horizontal mattress fashion. GSWs tend to create larger injuries with additional contused myocardium due to blast effect. These are best repaired in a horizontal mattress fashion. Though most can be repaired without the use of pledgets, they are useful when the myocardium is thin or especially friable.

Posterior Injuries

Posterior injuries can be exceedingly challenging to repair as their location requires lifting of the heart, causing both inflow and outflow occlusion, with resultant hypotension, bradycardia, and rapid deterioration into cardiac arrest [6]. In patients with a perfusing cardiac rhythm, these repairs are best accomplished in a sequential fashion. The heart should be briefly lifted, the injury assessed, and then the heart placed back in its normal position. After recovery, the heart is again lifted, a suture is placed, the heart is returned to its normal position, and the suture is tagged. This sequence may be repeated multiple times until all sutures are placed and tied. The technique requires surgical patience and close communication with the anesthesia team but offers the best chance for successful completion of repair without patient deterioration [6].

Injuries Adjacent to/and Coronary Artery Injury

Injuries in close proximity to the coronary arteries are especially difficult to repair and require meticulous suture placement to avoid narrowing the artery, or one of its side branches, with resultant distal ischemia [6, 20, 23]. These are best repaired using vertical mattress sutures placed below the bed of the coronary artery (Fig. 10.8). Teflon pledgets should generally be avoided as they make suture placement imprecise and increase the risk of compromising the artery [6]. Administration of 3 mg of adenosine causes brief asystole, has been shown to safely facilitate cardiac repair in penetrating trauma [42], and may be of particular use in these cases.

Direct coronary artery injury greatly increases the complexity of cardiac trauma. If a small side branch or the distal third of the artery is involved, it may be ligated without the resultant ischemia being of severe clinical significance. If the proximal coronary artery is injured, early involvement of a cardiac surgeon to provide coronary bypass, which may need to be done under cardiopulmonary bypass and cardioplegic arrest, is necessary [6, 8, 20, 23].

Intracardiac Injuries

Injury to the internal cardiac structures, including the valves and septum, is often overlooked at the time of initial repair and results in much of the reported postoperative complications in patients sustaining cardiac injuries [7, 43–45]. Rarely, a left-to-right shunt, from a septal injury or aortopulmonary fistula, may be diagnosed intraoperatively due to the clinician noting a dilated right ventricle or pulmonary artery and detecting a palpable thrill [6, 43]. Most post-traumatic valvular lesions present with insufficiency but are generally not recognized at the time of initial cardiac repair. The liberal use of intraoperative transesophageal echocardiography (TEE) can assist in making the diagnosis of intracardiac and proximal great vessel injury [6, 46] and may allow repair by a

Figure 10.8 Repair of cardiac injury in close proximity to coronary artery with mattress suture. A non-pledgeted horizontal mattress suture of 3-0 polypropylene is utilized to repair cardiac wounds that are in close proximity to the coronary arteries to avoid their narrowing or occlusion. Precise suture placement may be facilitated by small doses of adenosine to slow or cause brief episodes of asystole in the heart

cardiac surgeon at the index operation. If initially unavailable, postoperative echocardiography is invaluable in making the diagnosis [7, 43, 44] and should be obtained in all patients who sustain cardiac injury requiring operative repair.

Blunt Cardiac Injury and Rupture

Blunt cardiac rupture is a devastating injury with few survivors. Most patients die at the scene [9, 47], and there is a near 90% mortality for those who survive to be evaluated at a trauma center [9]. Dual-chamber injuries, left ventricular injuries, and injuries with concomitant pericardial laceration have been associated with dismal outcomes [9]. Patients who survive to operative intervention most commonly have tamponade. The most common injuries in these patients are disruptions of the superior vena cava or right atrial junction (36.4%) and right ventricular rupture (36.4%) [47]. Repairs should be pursued as discussed previously but are often more difficult due to the size of the wounds.

Blunt valvular disruption and septal defects, though rare, have been described and are thought to be due to direct trauma to the chest during the isovolumetric phase of the cardiac cycle [8, 45]. This period occurs in late diastole/early systole when the aortic and atrioventricular valves are closed and the heart has maximum volume. Due to the inability of the blood to eject across the valves, sudden increases in intracardiac pressure may lead to valvular and septal injury. Aortic valves are most commonly injured followed by mitral valves.

Symptoms of valvular insufficiency after blunt trauma are often missed in these multiply injured patients. Unexplained hemodynamic compromise, failure to clear lactate, and abnormal conduction patterns or ventricular dysrhythmias on electrocardiogram (ECG) should be aggressively investigated with echocardiography to search for these lesions [45]. When present and symptomatic, they will frequently require repair by a cardiac surgeon under cardiopulmonary bypass and cardioplegic arrest.

Air Embolism

Air embolism may occur much more commonly than recognized in both blunt and penetrating cardiac and thoracic traumas [48]. Mechanism of introduction may be direct entry from atrial or ventricular injury or from traumatic alveolovenous connections due to pulmonary injury. In the latter cases, rapid control of the pulmonary hilum with a vascular clamp or pulmonary twist should be accomplished to prevent continued embolism. Ensuing hemodynamic compromise and cardiac arrest may be due to reduction in cardiac output due to air trapping within the outflow tracts of the heart and/or coronary artery occlusion with resultant cardiac ischemia. The patient with air embolism should be placed in Trendelenburg position to help maintain the air within the apex of the ventricles. Internal cardiac massage may help to dissipate air that is trapped in the coronary circulation and outflow tracts. Needle aspiration of the ventricles, aortic root, and the coronary arteries can be attempted and may be lifesaving. Open cardiac ultrasound may assist in deciding the locations and times when needle aspiration would be appropriate.

Outcomes

The overall survival rate for patients sustaining penetrating cardiac wounds is 19.3% [4], and many die at the scene of injury. The survival rate for patients who are transported and evaluated at a trauma center is 33–43% and is 59% for those with recent signs of life [4, 10, 23, 49]. Those that require emergency resuscitative thoracotomy have a less than 20% [10, 27] chance of survival. Gunshot wounds are associated with a much lower survival (16%) than are victims of stab wounds (65%) [10, 20]. Mortality is substantially higher for those presenting with exsanguination (72%) compared to those presenting with tamponade (16%) [49].

Several studies have attempted to delineate the variables that are predictive of survival. In a large retrospective analysis, Tyburski noted that hemodynamic stability on arrival, stab wounds, and presence of tamponade conferred a survival benefit [2]. Asensio [1] identified mechanism of injury (GSW versus stab), Glasgow Coma Scale (GCS) ≤ 8, revised trauma score (RTS) ≤ 1, cardiovascular-respiratory score (CVRS) ≤ 3, presence of coronary artery injury, need for resuscitative thoracotomy, and absence of sinus rhythm when the pericardium is opened as predictors of mortality. Though the anatomic site of injury did not predict outcomes, the AAST-OIS did predict chance of survival. Mortality worsened as grade increased with grades IV, V, and VI having respective mortality rates of 56%, 76%, and 91%. More recently, Mina identified depressed GCS, initial base deficit <−15, massive hemothorax, need for resuscitative thoracotomy, SBP <75 mm Hg, bradycardia <50, GSW mechanism, scene time >10 minutes, and transit time to trauma center >10 minutes as independent risk factors for mortality [49].

Survival after blunt cardiac rupture is even more dismal. In a 5-year analysis of the NTDB, Teixeira and coauthors [9] defined the survival rate of patients who presented to a trauma center alive after blunt cardiac rupture as 10.8%. For those who survived to the operating room, this was increased to 32.4%. Patients with isolated right atrial injuries may have a reasonable chance at a good outcome, while bi-chamber injuries and blunt cardiac rupture with exsanguinating hemorrhage due to pericardial laceration and loss of containment are nearly uniformly fatal [50].

Complications

Complications common in all injured patients who present in shock—including hospital-/ventilator-acquired pneumonia, prolonged ventilator requirements, and multisystem organ dysfunction—also affect the patient with cardiac injury. Patients presenting in extremis with cardiac arrest have the added risk of sustaining neurologic and cognitive deficit,

which may be profound. The risk approaches 20% in those surviving a resuscitative thoracotomy [28]. Additionally, the risks of surgical site infection—including infections of the mediastinum, pericardium, pleural spaces, and chest wall—are ever present in patients whose thoracic incisions are often made in times of duress and compromised sterile surgical technique. Unfortunately, rates have not been reported in the literature [51].

Cardiac-specific complications are comprised mostly of missed injuries to the septum, valves, and conduction system [7, 43]. Severe conduction system injuries, though life-threatening, can be easily managed with cardiac pacing wires placed at the time of surgery and generally resolve spontaneously [52]. Patients are also at risk for ventricular aneurysm, cardiac pseudoaneurysm, pericarditis, retained intracardiac missiles, and missile embolus. Tang and coauthors [7] reported a 17.4% incidence of cardiac complications when echocardiography was routinely used in postoperative evaluation of patients who underwent repair of cardiac injury. Of those complications, pericardial effusion (47%) was most frequent, followed by abnormal wall motion (42%), ejection fraction less than 45% (42%), intramural thrombus (21%), valvular injury (21%), conduction abnormality (10%), ventricular pseudoaneurysm (5%), and ventricular aneurysm (1%). No patients required operative management. In contrast, Cha and colleagues [39] reported the rate of post-procedural cardiac complication to be 23%, with nearly all patients requiring further operative intervention. The most common complication was ventricular septal defect (45%) followed by aortic valve injury (18%) and conduction abnormalities (18%). Most patients had new-onset murmurs, and many developed dyspnea on exertion, cardiomegaly, and congestive heart failure prior to diagnosis.

As many of these complications are clinically silent or masked by concomitant injury, liberal use of intraoperative TEE is warranted to prevent delays in diagnosis and increased morbidity [6, 46]. When unavailable, the use of postoperative echocardiography in all patients who sustain cardiac injury requiring repair is wise [7, 43, 44].

Conclusion

Cardiac injury remains a devastating event, and most patients succumb before ever reaching the trauma center. For initial survivors, the mortality is exceedingly high, despite shortened transport times and advances in prehospital efforts, surgical care, and resuscitation strategies. Many survivors are plagued by persistent neurologic and cognitive deficits, although there is hope that post-resuscitation therapeutic hypothermia may improve this outcome [6]. Successful outcomes require a high index of suspicion, swift diagnosis, and an aggressive, precise, and coordinated effort on the part of the surgeon, trauma service, and anesthesia team.

Box 10.1 Limits of Resuscitative Thoracotomy

Prehospital CPR >10 min after blunt trauma without response[a]
Prehospital CPR >15 min after penetrating trauma without response[a]
Asystole is presenting rhythm, and there is no pericardial tamponade

Used with permission of Wolters Kluwer Health Inc. from Ernest et al. [28]
[a]Cardiac electrical activity present or cardiac activity detected by focused assessment with sonography for trauma (FAST)

References

1. Asensio JA, Murray J, Demetriades D, Berne J, Cornwell E, Velmahos G, et al. Penetrating cardiac injuries: a prospective study of variables predicting outcomes. J Am Coll Surg. 1998;186:24–34.
2. Tyburski JG, Astra L, Wilson RF, Dente C, Steffes C. Factors affecting prognosis with penetrating wounds of the heart. J Trauma. 2000;48:587–90. discussion 590–1

3. Naughton MJ, Brissie RM, Bessey PQ, McEachern MM, Donald JM Jr, Laws HL. Demography of penetrating cardiac trauma. Ann Surg. 1989;209:676–81. discussion 682–3
4. Rhee PM, Foy H, Kaufmann C, Areola C, Boyle E, Maier RV, et al. Penetrating cardiac injuries: a population-based study. J Trauma. 1998;45:366–70.
5. Kaplan AJ, Norcross ED, Crawford FA. Predictors of mortality in penetrating cardiac injury. Am Surg. 1993;59:338–41.
6. O'Connor J, Ditillo M, Scalea T. Penetrating cardiac injury. J R Army Med Corps. 2009;155:185–90.
7. Tang AL, Inaba K, Branco BC, Oliver M, Bukur M, Salim A, et al. Postdischarge complications after penetrating cardiac injury: a survivable injury with a high postdischarge complication rate. Arch Surg. 2011;146:1061–6.
8. Asensio JA, Garcia-Nunez LM, Petrone P. Trauma to the heart. In: Feliciano DV, Mattox KL, Moore EE, editors. Trauma. 6th ed. New York, NY: McGraw-Hill Medical; 2008. p. 569–86.
9. Teixeira PG, Inaba K, Oncel D, DuBose J, Chan L, Rhee P, et al. Blunt cardiac rupture: a 5-year NTDB analysis. J Trauma. 2009;67:788–91.
10. Asensio JA, Berne JD, Demetriades D, Chan L, Murray J, Falabella A, et al. One hundred five penetrating cardiac injuries: a 2-year prospective evaluation. J Trauma. 1998;44:1073–82.
11. Beck C. Wounds of the heart. Arch Surg. 1926;13:205–27.
12. Homer. The Iliad. New York, NY: Farrar, Straus and Giroux; 2004.
13. Hippocrates. The genuine works of Hippocrates. New York: William Wood and Co; 1886:252. Vol 2, sec 6, aphorism 18.
14. Juan A, Patrizio P, Bruno P, Diego P, Supparerk P, Taichiro T, et al. Penetrating cardiac injuries: a historic perspective and fascinating trip through time. J Am Coll Surg. 2009;208:462–72.
15. Aristotle (384–322 BC). The Partibus Animalium. Lib III, chap 4. Opera Edidit Academia Regia Borrusca, vol 3, 328. As quoted by Beck CS. Wounds of the heart. Arch Surg. 1926;13:205–27.
16. Galen (130–200AD). Medicorum Graecorum Opera. Vol VIII, Lipsiae Prostat in officina Libraria Car. Cuoblochii 1824, Le Locis Affectis. Edited by Kuhn DC: Tome VIII, lib V, chap 2, 304. As quoted by Beck CS. Wounds of the Heart. Arch Surg. 1926;13:205–27.
17. Hollerius J. Communis aphorismi allegati. Quoted from Fisher G: Die Wuden des Herzeus und des Herzbeutels. Arch Klin Chir.

1868;9:571. As quoted by Beck CS. Wounds of the heart. Arch Surg. 1926;13:205–27.
18. Wolf I. Cited by Fischer G. D Die Wunden des Herzeus und des Herzbeutels. Arch Klin Chir. 1868;9:571.
19. Saplacan V, Cuttone F, Massetti M. The ignored birth of cardiac surgery: the history of the surgical treatment of heart wounds and pericardial effusions. In: Piciche M, editor. Dawn and evolution of cardiac procedures: research avenues in cardiac surgery and interventional cardiology. Italy: Springer; 2013. p. 24.
20. Asensio JA, Soto SN, Forno W, Roldan G, Petrone P, Salim A, et al. Penetrating cardiac injuries: a complex challenge. Injury. 2001;32:533–43.
21. Westaby S, Bosher C. Landmarks in cardiac surgery. Oxford: Isis Medical Media Ltd.; 1997.
22. Mandal AKS, Sanusi M. Penetrating chest wounds: 24 years experience. World J Surg. 2001;25:1145–9.
23. Asensio JA, Stewart BM, Murray J, Fox AH, Falabella A, Gomez H, et al. Penetrating cardiac injuries. Surg Clin North Am. 1996;76:685–724.
24. Elias D, Peter L, Dietrich D, Fabrizio B, Douglas B, Martin S. Penetrating cardiac injuries: recent experience in South Africa. World J Surg. 2006;30:1258–64.
25. Claassen CW, O'connor JV, Gens D, Sikorski R, Scalea TM. Penetrating cardiac injury: think outside the box. J Trauma. 2010;68:E71–3.
26. Jhunjhunwala R, Mina MJ, El R, Dente CJ, Carr JS, Dougherty SD, et al. Reassessing the cardiac box: a comprehensive evaluation of the relationship between thoracic gunshot wounds and cardiac injury. J Trauma Acute Care Surg. 2017;83(3):349–55.
27. Rhee PM, Acosta J, Bridgeman A, Wang D, Jordan M, Rich N. Survival after emergency department thoracotomy: review of published data from the past 25 years. J Am Coll Surg. 2000;190:288–98.
28. Ernest M, Knudson MM, Clay B, Kenji I, Rochelle D, Walter B, et al. Defining the limits of resuscitative emergency department thoracotomy: a contemporary Western Trauma Association perspective. J Trauma. 2011;70:334–9.
29. Burlew CC. Resuscitative thoracotomy. Available at: http://westerntrauma.org/algorithms/WTAAlgorithms_files/gif_8.htm.
30. Rozycki GS, Ballard RB, Feliciano DV, Schmidt JA, Pennington SD. Surgeon-performed ultrasound for the assessment of trun-

cal injuries: lessons learned from 1540 patients. Ann Surg. 1998;228:557–67.
31. Rozycki GS, Feliciano DV, Ochsner MG, Knudson MM, Hoyt DB, Davis F, et al. The role of ultrasound in patients with possible penetrating cardiac wounds: a prospective multicenter study. J Trauma. 1999;46:543–51.
32. Vivek T, Michael B, John M, Christian T, Michael T. FAST (focused assessment with sonography in trauma) accurate for cardiac and intraperitoneal injury in penetrating anterior chest trauma. J Ultrasound Med. 2004;23:467–72.
33. Chad B, Brian W, Amy W, Jeffrey N, Grace R, David F. A caveat to the performance of pericardial ultrasound in patients with penetrating cardiac wounds. J Trauma. 2009;67:1123–4.
34. Kirkpatrick AW, Sirois M, Laupland KB, Liu D, Rowan K, Ball CG, et al. Hand-held thoracic sonography for detecting post-traumatic pneumothoraces: the extended focused assessment with sonography for trauma (EFAST). J Trauma. 2004;57:288–95.
35. Olga B, Nira B-R, Subhi A, Janna F, Anat I, Diana L, et al. Sonographic detection of pneumothorax by radiology residents as part of extended focused assessment with sonography for trauma. J Ultrasound Med. 2009;28:749–55.
36. Thorson CM, Namias N, Van Haren RM, Guarch GA, Ginzburg E, Salerno TA, et al. Does hemopericardium after chest trauma mandate sternotomy? J Trauma Acute Care Surg. 2012;72(6):1518–24.
37. Nicol AJ, Navsaria PH, Hommes M, Ball CG, Edu S, Kahn D. Sternotomy or drainage for a hemopericardium after penetrating trauma: a randomized controlled trial. Ann Surg. 2014;259(3):438–42.
38. Chestovich PJ, McNicoll CF, Fraser DR, Patel PP, Kuhls DA, Clark E, et al. Selective use of pericardial window and drainage as sole treatment for hemopericardium from penetrating chest trauma. Trauma Surg Acute Care Open. 2018;3(1):e000187.
39. Moore EE, Cogbill TH, Malangoni M, Jurkovich GJ, Champion HR. Scaling system for organ specific injuries. Available at: http://www.aast.org/Library/TraumaTools/InjuryScoringScales.aspx.
40. Moore EE, Malangoni MA, Cogbill TH, Shackford SR, Champion HR, Jurkovich GJ, et al. Organ injury scaling IV: thoracic vascular, lung, cardiac and diaphragm. J Trauma. 1994;36(3):299–300.

41. Henry SM, Duncan AO, Scalea TM. Intestinal Allis clamps as temporary vascular control for major retroperitoneal venous injury. J Trauma. 2001;51:170–2.
42. Kokotsakis J, Hountis P, Antonopoulos N, Skouteli E, Athanasiou T, Lioulias A. Intravenous adenosine for surgical management of penetrating heart wounds. Tex Heart Inst J. 2007;34:80–1.
43. Cha EK, Mittal V, Allaben RD. Delayed sequelae of penetrating cardiac injury. Arch Surg. 1993;128:836–9.
44. Loizos A, Petros P, Christos E, Evagoras N. A penetrating heart injury resulting in ventricular septal defect. Hell J Cardiol. 2011;52:71–4.
45. Menaker J, Tesoriero RB, Hyder M, Sikorski R, Scalea TM. Traumatic atrial septal defect and papillary muscle rupture requiring mitral valve replacement after blunt injury. J Trauma. 2009;67:1126.
46. Diana Y, Nir H, Wendy R, Fardad E, Paul S, Aman M. Intraoperative transesophageal echocardiography in chest trauma. J Trauma. 2008;65:924–6.
47. Nan Yu-Yun L, Ming-Shian LK-S, Yao-Kuang H, Feng-Chun T, Jaw-Ji C, Pyng L. Blunt traumatic cardiac rupture: therapeutic options and outcomes. Injury. 2009;40:938–45.
48. Cothren CC, Moore EE. Emergency department thoracotomy for the critically injured patient: objectives, indications, and outcomes. World J Emerg Surg. 2006;1:4.
49. Mina MJ, Jhunjhunwala R, Gelbard RB, Dougherty SD, Carr JS, Dente CJ, et al. Factors affecting mortality after penetrating cardiac injuries: 10-year experience at urban level I trauma center. Am J Surg. 2017;213(6):1109–15.
50. May AK, Patterson MA, Rue LW 3rd, Schiller HJ, Rotondo MF, Schwab CW. Combined blunt cardiac and pericardial rupture: review of the literature and report of a new diagnostic algorithm. Am Surg. 1999;65:568–74.
51. Gilman K, Ipaktchi K, Moore EE, Barnett C, Gurunluoglu R. Reconstruction of an emergency thoracotomy wound with free rectus abdominis flap: anatomic and radiologic basis for the surgical technique. World J Emerg Surg. 2010;5:12.
52. Jhunjhunwala R, Dente CJ, Keeling WB, Prest PJ, Dougherty SD, Gelbard RB, et al. Injury to the conduction system: management of life-threatening arrhythmias after penetrating cardiac trauma. Am J Surg. 2016;212(2):352–3.

Part IV
Techniques in the Abdomen

Chapter 11
Liver Injuries: Techniques

Benjamin J. Moran and Deborah M. Stein

Introduction of the Problem

The liver is the largest organ in the abdomen and is frequently injured. Liver injuries make up approximately 5% of all trauma admissions [1]. Although minor or moderate injuries may be managed with simple observation, the extensive vascular supply of the liver makes severe liver injuries extremely challenging. Frequently, patients who require liver-specific operations have exsanguinating hemorrhage. Despite

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improvements in resuscitation, mortality of operative liver injuries remains approximately 50% [2, 3], with mortality of up to 80% for patients with juxtahepatic venous injuries [4–7]. Morbidity rates following operation for high-grade liver injuries are as high as 30–80% depending on the grade of injury and types of complications considered [8–11].

History of Care of Liver Injuries

Management of hepatic trauma has undergone a revolution, from the first description of liver injuries in Greek and Roman mythology to Hogarth Pringle's description of the "Pringle maneuver" in 1908 [12]. Since then, the management of hepatic injuries has transformed from early laparotomy with definitive repair and resection to damage control techniques in unstable patients and nonoperative management (NOM) in hemodynamically stable patients [13–15]. Successful operative management of liver injury was first reported in the early seventeenth century [16]. Military and civilian surgeons documented successful operative management of hepatic injury, but morbidity and mortality rates remained extremely high, as was true for all major hemorrhage prior to the era of modern medicine [17–21]. With Root's description of the first diagnostic peritoneal lavage (DPL) in 1965, the incidence of liver injury and experience with operative management dramatically increased as the standard of care dictated that patients with abdominal trauma and hemoperitoneum on DPL required operative management [22]. In the early 1980s, introduction of computed tomography (CT) allowed selective management of liver injuries to emerge [23]. In 1989, Moore and colleagues introduced the American Association for the Surgery of Trauma Organ Injury Scale (AAST-OIS) for liver injury, which was updated in 1995 and allows for a stratified qualification of liver injuries that is used to both guide intervention and allow for scientific comparisons of hepatic trauma [24, 25]. Grades from I to VI are described. Grades I and II are

Table 11.1 AAST-OIS liver injury scale

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

Used with permission of Wolters Kluwer from Moore et al. [25]

minor, grade III is moderate, and grades IV and V injuries are major. Grade VI injuries are uniformly fatal hepatic avulsion. See Table 11.1.

It was in 1908 when Pringle published the landmark study first described the "Pringle maneuver," and he was also likely the first surgeon to advocate for NOM of low-grade injuries and stratify treatment options by severity of injury:

> It is very probable that slight ruptures will occasionally heal without surgical interference in consequence of this increased tension of the abdominal wall leading to the arrest of hemorrhage, but in the cases of severe injury to the liver this will not happen [12].

Although for low-grade injuries, NOM has been advocated for decades, there was considerable controversy about the management of high-grade injuries. Studies by Croce and Pachter in the mid-1990s convincingly demonstrated that high-grade injuries could be managed safely in stable patients [26, 27]. Both found that NOM is safe for hemodynamically stable patients regardless of injury severity and/or amount of hemoperitoneum. The current paradigm is that hemodynamics alone should dictate which patients require immediate operative intervention for hepatic trauma [28].

Technique with Personal Tips

Knowledge of hepatic vascular anatomy—the hepatic arteries, the portal venous system, and the hepatic veins—is essential to manage hemorrhage while preserving hepatic function in complex hepatic injury. Familiarity with the anatomic relationships and preservation of main conduits of the biliary tree is also essential (Fig. 11.1a–c). The classic description of the liver anatomy is based on the gross external appearance in which the falciform ligament extends from the diaphragmatic surface to the abdominal wall and divides the liver into the right and left anatomic lobes. The liver is most commonly described by the eight Couinaud's segments, which are dictated by the vascular anatomy rather than external landmarks [29] (Figs. 11.2a, b and 11.3a–d). This classification is more useful for operative liver management, since it divides the liver into units based on the key structures essential for safe operative technique. The center of each Couinaud's segments contains the supplying branch of the portal vein and hepatic artery running with the bile duct while hepatic veins lie in the periphery of each segment. A functional left and right liver is divided by a main portal fissure known as Cantlie's line, which runs from the middle of the gallbladder fossa to the inferior vena cava (IVC) and contains the middle hepatic vein.

The portal vein divides the liver into upper and lower segments by the left and right portal veins, which then branch

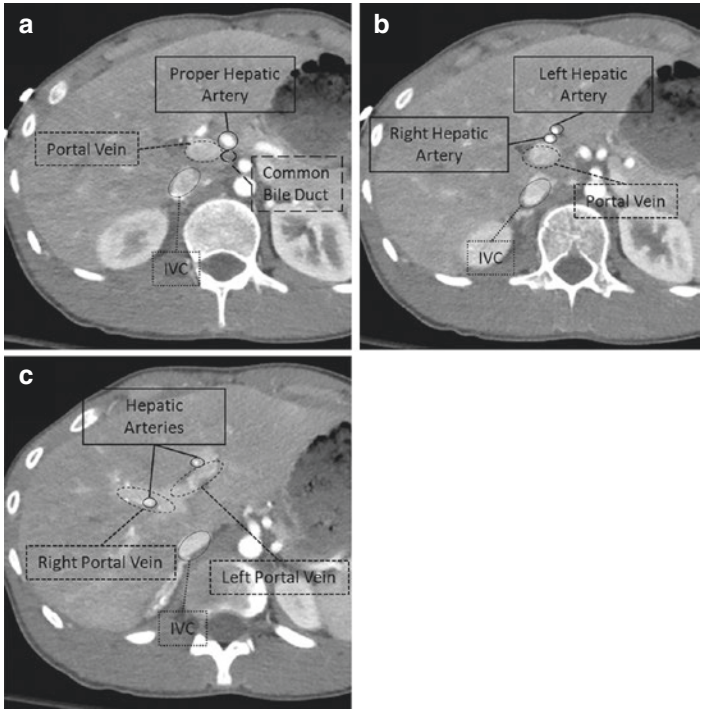


FIGURE 11.1 (**a**–**c**) Hepatic anatomy. (**a**) The relationship of the portal vein (dashed line), proper hepatic artery (solid line), and common bile duct (large dashed line) in the porta hepatis. (**b**) The relationship of the portal vein (dashed line) and right and left hepatic arteries (solid lines). (**c**) Bifurcation of the portal vein (dashed line) separating segments 8 (superiorly) and 5 (inferiorly) on the right from segment 4 on the left. The main branches of the hepatic arteries (solid line) can be seen running with the portal vein

superiorly and inferiorly. Additionally, the hepatic arteries and main biliary ducts run with the portal veins. The hepatic venous drainage includes the right hepatic vein, which divides the right lobe into anterior and posterior segments; the middle hepatic vein, which divides the liver into right and left lobes in a plane along Cantlie's line; and the left hepatic vein, which divides the left lobe into a medial and lateral part.

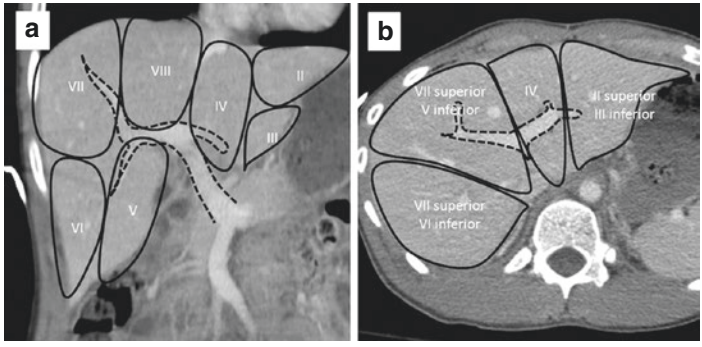


FIGURE 11.2 (**a**, **b**) Portal venous anatomy (dashed line) dividing the liver into Couinaud's segments. Segment I (not shown) is the caudate lobe. (**a**) Coronal view. (**b**) Axial view

The caudate lobe, segment I, is drained by one or more short hepatic veins that drain directly into the IVC. Another key consideration is understanding the external hepatic artery anatomy, which can be aberrant in 25% of patients [30]. The most common of these is the "replaced right hepatic artery" in which the right hepatic artery originates from the superior mesenteric artery rather than the proper hepatic artery, which lies posterior to the portal triad. The second most common is a replaced left hepatic artery (10–15%), where the left hepatic artery originates from the left gastric artery and travels in the gastrohepatic ligament. These are two key considerations when dissecting out the porta hepatis and/or applying a "Pringle maneuver."

Operative management of liver injuries typically occurs in one of two settings:

1. The patient is being explored for both diagnosis and therapy—most typically in the situation of hemodynamic instability, peritonitis, or a transperitoneal penetrating trajectory.
2. The patient is being explored for another indication such as bowel or bladder injury, and a liver injury is encountered.

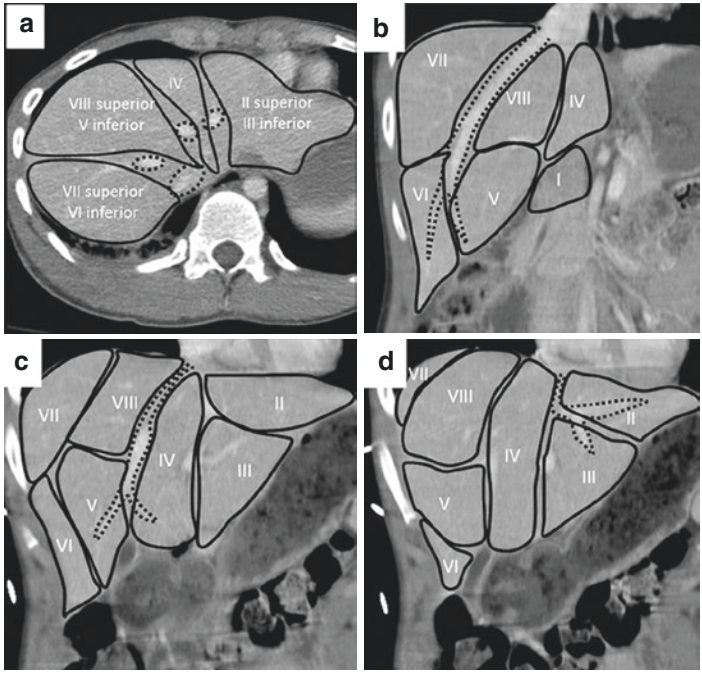


FIGURE 11.3 (**a**–**d**) Hepatic venous anatomy (dotted line) dividing the liver into Couinaud's segments. Segment I is the caudate lobe. (**a**) Axial view. Dotted circles indicate IVC, right, middle, and left hepatic veins (clockwise from bottom). Coronal views showing the right hepatic vein (**b**), middle hepatic vein (**c**), and left hepatic vein (**d**) and relationships to the functional hepatic segments

In the first situation, a standard midline trauma exploratory laparotomy should always be performed with initial packing (Fig. 11.4). Exsanguinating hemorrhage should be anticipated, and activation of a massive transfusion protocol, availability of rapid transfusion systems, blood salvage devices, and careful coordination with the anesthesia providers are essential. The anterior portion of the falciform ligament is taken down between hemostats and tied to minimize risk of bleeding from a patent remnant umbilical vein. At this point in the operation, the falciform ligament should not be taken

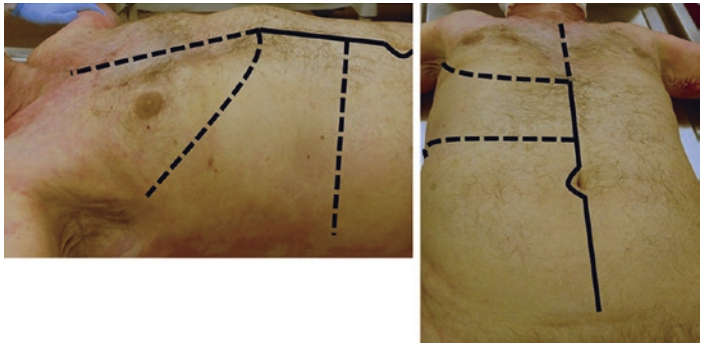


Figure 11.4 Exposure for operative management of liver injuries. Standard midline laparotomy (solid line). Additional incisions for exposure when needed (dashed line). (Facilities: Surgical Laboratory, Anatomical Services Division, School of Medicine, UMB; Cadavers and Specimens, Anatomical Donors, Maryland State Anatomy Board; Ronn Wade, State Anatomy Board, Department of Health and Mental Hygiene)

down any farther posteriorly than required to gain adequate access to the peritoneal cavity to minimize the risk of release of retrohepatic tamponade. Packing around the liver typically includes three laparotomy pads ("lap pads") above and below the liver to compress the liver up against the diaphragm (Fig. 11.5). Even at this early stage in the operation, significant liver injuries should be readily apparent. There are two crucial questions that need to be answered:

1. Is the patient bleeding through packs?
2. Does the patient's physiology dictate that a damage control (DC) approach should be used?

If the answer to question #1 is "no," then time is given to allow the anesthesia providers to administer blood products and stabilize the patient's hemodynamics. Definitive repair and hemostasis are then dictated by the answer to question #2. If the patient is physiologically normal, then definitive hemostasis and repair can be performed as discussed below. If a DC approach is advisable and the patient is not actively

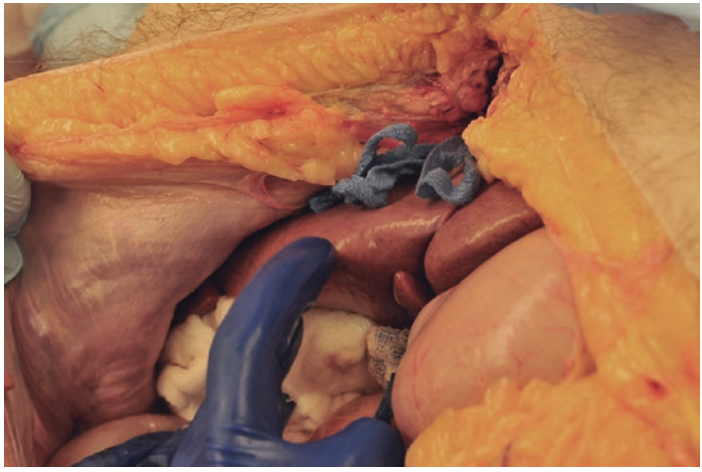


FIGURE 11.5 Simple "lap pad" packing of the liver. Typically, three lap pads are packed above and three lap pads below the liver for temporary hemostasis. (Facilities: Surgical Laboratory, Anatomical Services Division, School of Medicine, UMB; Cadavers and Specimens, Anatomical Donors, Maryland State Anatomy Board; Ronn Wade, State Anatomy Board, Department of Health and Mental Hygiene)

bleeding with the lap pads in place, no further liver-specific intervention is indicated at that time and other active issues can be addressed such as control of enteric contamination.

If the answer to question #1 is "yes," then a series of maneuvers should be performed in an orderly and sequential fashion (Fig. 11.6). The right upper quadrant should be unpacked and inspected. *Manual compression* is the first-line therapy for all hemorrhage from the liver and is surprisingly effective. While the assistant is compressing the liver parenchyma, the liver must be fully mobilized. The exception to this is in patients with retrohepatic hematoma that appears to be stable. In this situation, full mobilization of the liver may result in release of tamponade from bleeding from a major hepatic venous injury or retrohepatic caval injury. All other injuries are best approached with *full mobilization of the liver*

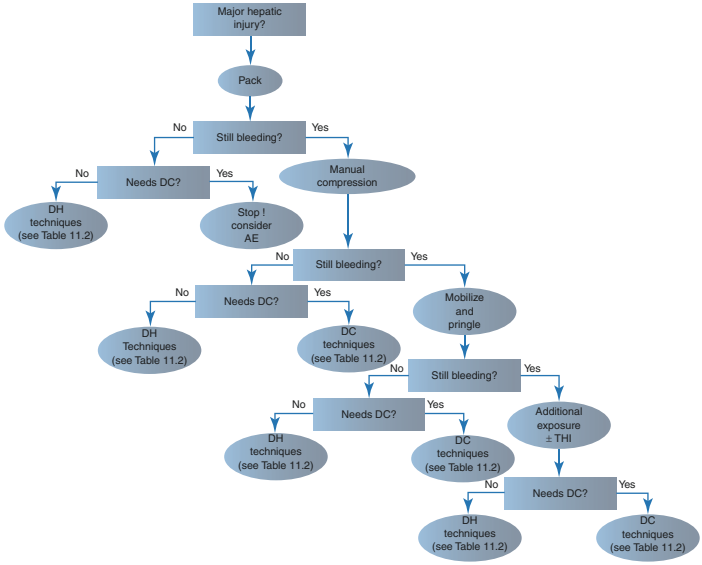


FIGURE 11.6 Algorithm for initial operative management of major hepatic injury. DC = damage control, DH = definitive hemostasis, THI = total hepatic isolation

(Fig. 11.7a–d). The liver is mobilized by taking down the falciform ligament all the way to the suprahepatic IVC using either electrocautery or Metzenbaum scissors. The hepatic veins are not encountered until the leaflets of the falciform ligament diverge so the ligament can be dissected all the way back to the IVC with impunity. The right and left triangular ligaments are similarly divided until the liver can be mobilized out of the right upper quadrant and inspected. This is done extremely rapidly and should only take 1 or 2 minutes to completely mobilize the liver. At this point, the liver can be fully inspected, and the major source of hemorrhage can be identified.

If active hemorrhage is still encountered that cannot be controlled with simple manual pressure, a "Pringle maneuver" is next applied (Fig. 11.8a–c). The gastrohepatic ligament is opened, taking care to avoid injury to the left gastric artery

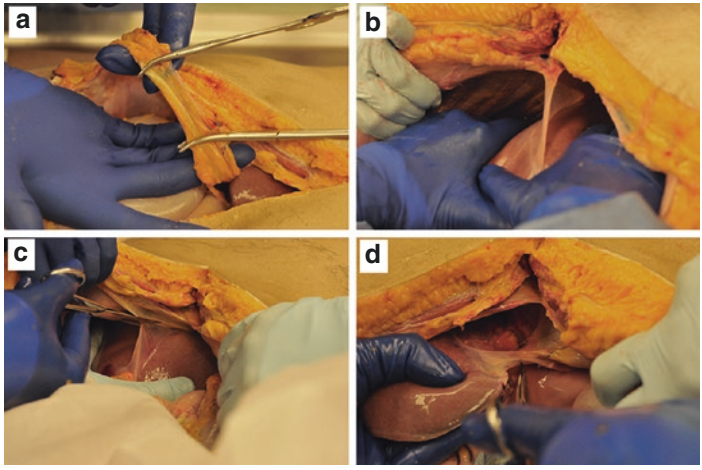


FIGURE 11.7 (**a**–**d**) Mobilization of the liver. (**a**) The anterior portion of the falciform ligament is clamped and tied. (**b**, **c**) The liver is retracted downward, and the falciform ligament is sharply transected back to the IVC. (**d**) The right (not shown) and left triangular ligaments are similarly transected. (Facilities: Anatomical Services Division, School of Medicine, UMB; Cadavers and Specimens: Anatomical Donors, Maryland State Anatomy Board; Ronn Wade, State Anatomy Board, Department of Health and Mental Hygiene)

and a potentially replaced left hepatic artery, and the porta hepatis is encircled through the foramen of Winslow. A vascular clamp or Penrose drain can then be used to compress the portal vein and proper hepatic artery at the porta hepatis. This maneuver controls all but hepatic venous flow, so if the hemorrhage from the liver is stemmed, a major hepatic venous injury is unlikely. Decisions about definitive hemostasis for a hepatic arterial or portal venous injury can then be made. If massive exsanguination continues, then the diagnosis of hepatic venous injury is likely, and the techniques described next will need to be rapidly employed. If the majority of the bleeding appears to be from the posterior aspect of the liver, a major hepatic vein or retrohepatic caval injury is likely.

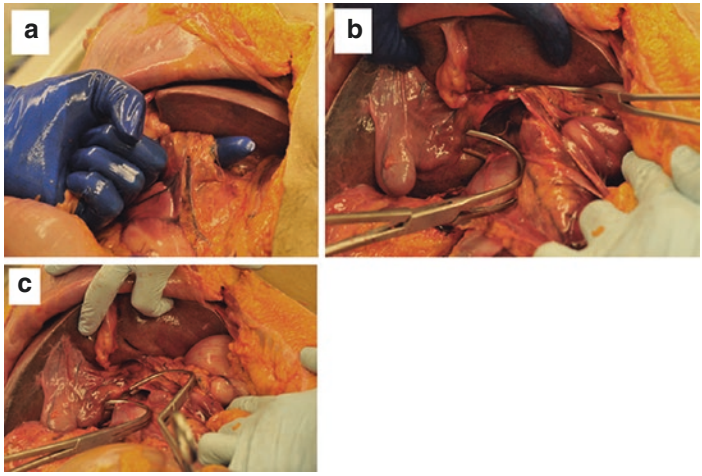


Figure 11.8 (**a**) Dissecting out the porta hepatis for a "Pringle maneuver." (**b**) Exposure to the infrahepatic IVC. (**c**) Infrahepatic IVC occlusion with the portal triad occluded. (Facilities: Surgical Laboratory, Anatomical Services Division, School of Medicine, UMB; Cadavers and Specimens, Anatomical Donors, Maryland State Anatomy Board; Ronn Wade, State Anatomy Board, Department of Health and Mental Hygiene)

If a major hepatic vein or retrohepatic caval injury is suspected, *additional exposure* is advisable. There are several strategies for this. Access to the hepatic veins and IVC requires a more extensive mobilization and, in our opinion, is best accomplished by opening the thorax. The best exposure will be obtained by extending the midline laparotomy incision across the right chest at about the eighth intercostal space and transecting the costal cartilages (Fig. 11.4). The diaphragm is then taken down from the anterior midline radially all the way back to the IVC, remembering to leave enough diaphragm on the chest wall side of the incision for later repair (Fig. 11.9a, b). At this point, a chest retractor can be placed and exposure to the entire retrohepatic area is achieved (Fig. 11.10). The other strategy that can be used to gain additional exposure to the liver, particularly the poste-

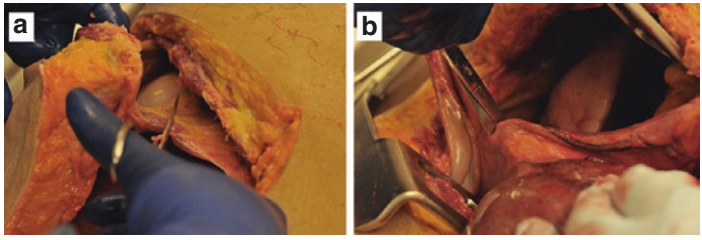


FIGURE 11.9 (**a**, **b**) Extension of the midline laparotomy to the right chest at the eighth intercostal space and transecting the costal cartilages. The diaphragm is then taken down from the anterior midline (**a**) radially all the way back to the IVC (**b**), remembering to leave enough diaphragm on the chest wall side of the incision for later repair. (Facilities: Surgical Laboratory, Anatomical Services Division, School of Medicine, UMB; Cadavers and Specimens, Anatomical Donors, Maryland State Anatomy Board; Ronn Wade, State Anatomy Board, Department of Health and Mental Hygiene)

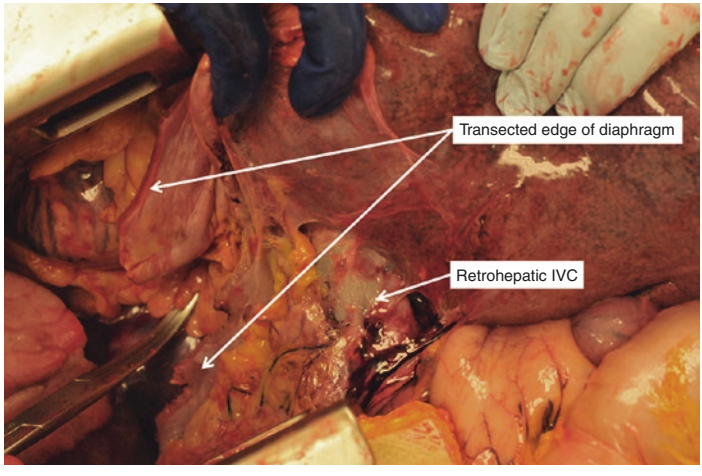


FIGURE 11.10 Retrohepatic IVC exposure after extension of the midline laparotomy into the right thorax. (Facilities: Surgical Laboratory, Anatomical Services Division, School of Medicine, UMB; Cadavers and Specimens, Anatomical Donors, Maryland State Anatomy Board; Ronn Wade, State Anatomy Board, Department of Health and Mental Hygiene)

rior right lobe and retrohepatic area, is to "t-off" the laparotomy incision through a right lateral transverse incision in which the rectus muscles are transected (Fig. 11.4). This allows for improved access to these structures and facilitates mobilization and the ability to apply direct circumferential pressure to the liver parenchyma.

Although classically taught, the technique of *total hepatic isolation* is, in our opinion, not as effective or as straightforward as typically described. Additionally, the profound reduction in preload that occurs with clamping of the suprahepatic IVC in a patient who already is in shock often leads to cardiac arrest. If total hepatic isolation is desired, however, once the chest is open, the suprahepatic IVC can be easily clamped by simple incision of the right lateral pericardium, taking care to avoid injury to the phrenic nerve (Fig. 11.11). Alternative access to the suprahepatic IVC without thoracic exposure can be

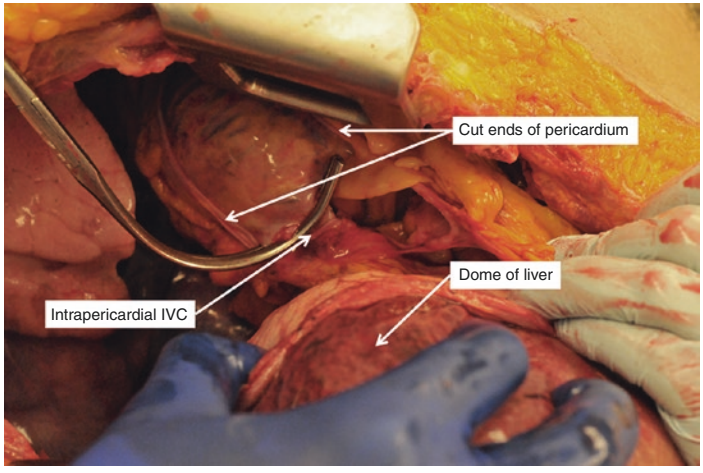


Figure 11.11 Suprahepatic IVC clamped after exposure through the right thorax with incision in the pericardium. (Facilities: Surgical Laboratory, Anatomical Services Division, School of Medicine, UMB; Cadavers and Specimens, Anatomical Donors, Maryland State Anatomy Board; Ronn Wade, State Anatomy Board, Department of Health and Mental Hygiene)

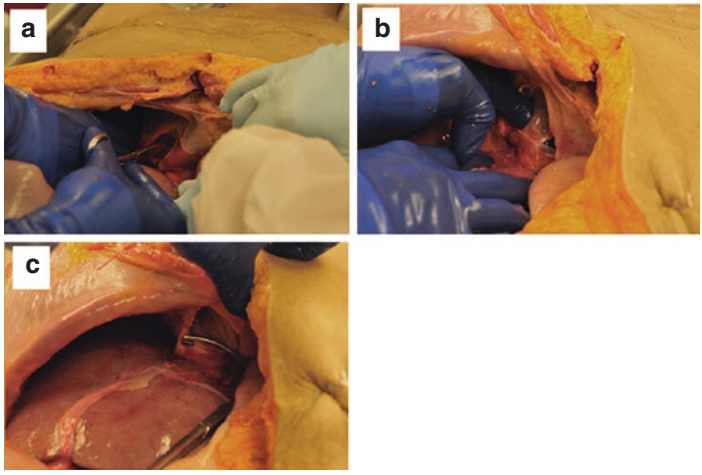


FIGURE 11.12 (**a**–**c**) Accessing the suprahepatic IVC. The pericardium is incised through the diaphragm (**a**), and the suprahepatic IVC is encircled (**b**) and clamped (**c**). (Facilities: Surgical Laboratory, Anatomical Services Division, School of Medicine, UMB; Cadavers and Specimens, Anatomical Donors, Maryland State Anatomy Board; Ronn Wade, State Anatomy Board, Department of Health and Mental Hygiene)

obtained via gentle traction down on the dome of the liver with upward traction on the diaphragm and intraperitoneal dissection and exposure. An incision through the peritoneal side of the diaphragm into the pericardium and clamping of the suprahepatic IVC in the pericardium is another approach to suprahepatic IVC control (Fig. 11.12a–c). A median sternotomy also provides immediate access to the suprahepatic IVC. The infrahepatic IVC is accessed either through the gastrohepatic ligament or, more commonly, via a complete right medial visceral rotation with a Kocher maneuver. The IVC is carefully encircled above the renal veins and clamped with a vascular clamp (Fig. 11.8a–c). With the porta hepatis, the suprahepatic IVC, and the intrahepatic IVC now occluded, total hepatic vascular isolation is achieved. As stated previously, however, this is typically poorly tolerated by patients in extremis.

Endovascular approaches for infrahepatic and suprahepatic IVC control are becoming more available with innovations in catheter therapy. REBOA balloon catheters have been used with some success in animal models to help stem bleeding from retrohepatic IVC injuries by occluding the suprahepatic IVC. This is useful tool to keep in the surgeon's armamentarium as many patients may already have established femoral venous access prior to the operating room [31]. Additionally, there are venous occlusion devices designed for temporization of inadvertent superior vena cava injuries during cardiac pacemaker or implantable defibrillator lead placement or removal that may be helpful in providing tamponade for hemorrhage from the IVC.

The use of veno-venous bypass can allow for preservation of preload and venous return to the heart with total hepatic isolation [32, 33]. Venous cannulae can be placed percutaneously in the right internal jugular and right femoral veins allowing for preservation of flow to the heart. Additionally, this technique can be employed without the use of total hepatic isolation as a way to markedly decrease bleeding from the hepatic veins and the IVC to allow for better visualization and a reduction in blood loss during repair or ligation of these structures. Veno-venous bypass is exceptionally helpful when major resection is needed at planned "second-look" laparotomy or for management of major complications.

The atriocaval shunt was first described in 1968 by Schrock and colleagues as a strategy of controlling hemorrhage from retrohepatic IVC and hepatic venous injury [34]. The technique includes placement of a large-bore chest tube into the right atrium after a purse-string suture is placed in the atrial appendage. The side hole of the chest tube must be within the atrium to allow for maintenance of venous inflow. The tip of the tube is clamped, and the tube is then passed down into the IVC and secured at the intrapericardial and infrahepatic IVC with simple ties, Rummel tourniquets, or umbilical tape. When used with a Pringle maneuver, it should provide almost total hepatic vascular occlusion. Case series have reported survival in these highly lethal injuries [35, 36]. In 1986, Pachter reported six consecutive patients with juxta-

hepatic venous injuries managed without a shunt with a remarkable survival of 83% [37]. While we are not big proponents of the use of a Schrock shunt, the key to its successful employment is clearly the decision to utilize the technique early in the operation before the patient is moribund [35].

Specific techniques for hemostasis are dictated by three main factors: the location of the injury, the anatomy of the injury, and the need for a DC approach (Table 11.2). Each of the techniques that will be described in this chapter is divided into "definitive hemostasis" and "damage control" or "temporary hemostasis." Any of the definitive hemostasis

Table 11.2 Preferred techniques for hemostasis

Type of injury	Definitive hemostasis	Damage control
Capsular tear/ subcapsular hematoma or any "raw surface"	Electrocautery	Lap pad packing
	Argon beam coagulation	QuikClot® or other nonabsorbable hemostatic agent
	Topical hemostatic agents	
	Absorbable mesh with hemostatic agent (EVICEL®, Crosseal™, etc.)	
Minor parenchymal injury	Electrocautery	Lap pad packing
	Argon beam coagulation	QuikClot® or other nonabsorbable hemostatic agent
	Topical hemostatic agents	
	Absorbable mesh with hemostatic agent (EVICEL®, Crosseal™, etc.)	
	Direct vessel ligation	
	Suture hepatorrhaphy (horizontal mattress 0 chromic liver suture with omental patch)	

(continued)

Table 11.2 (continued)

Type of injury	Definitive hemostasis	Damage control
Major parenchymal injury		
Peripheral	Direct vessel ligation	Lap pad packing
	Suture hepatorrhaphy (horizontal mattress 0 chromic liver suture with omental patch)	QuikClot® or other nonabsorbable hemostatic agent
	Hepatotomy with direct vessel ligation	
	Resectional debridement/ nonanatomic resection	
	Anatomic resection	
	AE	
Central	Direct vessel ligation	Lap pad packing
	Hepatotomy with direct vessel ligation Resectional debridement/ nonanatomic resection	QuikClot® or other nonabsorbable hemostatic agent
	Anatomic resection Selective hepatic artery ligation AE	Foley balloon tamponade
		Veno-venous bypass
		Total hepatectomy

Table 11.2 (continued)

Type of injury	Definitive hemostasis	Damage control
Transhepatic GSW	Direct vessel ligation	Lap pad packing
	Hepatotomy/"tractotomy" with direct vessel ligation Resectional debridement/ nonanatomic resection	QuikClot® or other nonabsorbable hemostatic agent
	Selective hepatic artery ligation AE	Foley balloon tamponade
		Saline-filled Penrose drain tamponade
		Veno-venous bypass
		Total hepatectomy
Major hepatic vein injury	Direct vessel ligation or repair	Lap pad packing
Retrohepatic IVC injury	Vessel repair	Lap pad packing
	Interposition graft	Total hepatic vascular isolation Atriocaval shunt with veno-venous bypass Total hepatectomy if not repairable

Any of the techniques for definitive hemostasis can also be used for DC if it will be the most rapid way of controlling hemorrhage and are appropriate for use at both the index operation or during the take-back second-look laparotomy after DC

DH definitive hemostasis, *DC* damage control, *GSW* gunshot wound, *IVC* inferior vena cava, *AE* angiographic embolization

EVICEL®, Ethicon, Inc., Somerville, NJ, USA

Crosseal™, OMRIX Biopharmaceuticals, Ltd, New Brunswick, NJ, USA

QuikClot® Z-Medica, Wallingford, CT, USA

techniques can also be used for DC if it will be the most rapid way of controlling hemorrhage, and any of these techniques are appropriate for use at both the index operation or during the take-back second-look laparotomy after DC and in the event of re-exploration for recurrent hemorrhage.

Capsular tears of the liver are typically minor and easily controlled. In the hemodynamically stable and physiologically normal patient, definitive treatment of these injuries ranges from no intervention to simple hepatorrhaphy with electrocautery, argon beam coagulation, or topical hemostatic agents, such as EVICEL® Fibrin Sealant (Ethicon, Inc., Somerville, NJ, USA) or Crosseal™ Fibrin Sealant (OMRIX Biopharmaceuticals, Ltd, New Brunswick, NJ, USA). A very helpful technique useful for minor bleeding is placement of a piece of SURGICEL® (Ethicon, Medline Industries, Inc., Somerville, NJ, USA) or other absorbable hemostatic agent, which can then be cauterized on the surface of the liver by "arcing" the electrocautery or using the argon beam. Large capsular tears can be additionally treated with a technique of the use of an absorbable material such as Vicryl™ Knitted Mesh or SURGICEL® NU-KNIT® Absorbable Hemostat (Ethicon, Medline Industries, Inc., Somerville, NJ, USA) sprayed with a topical hemostatic agent. These are also applicable to treat "ooze" from the raw liver surface that is left after hepatotomy or hepatectomy. In the physiologically deranged patient, DC approach includes simple laparotomy pad placement or the use of a temporary topical hemostatic gauze pack or pad such as QuikClot® Trauma Pad™ (Z-Medica, Wallingford, CT, USA). This product is kaolin impregnated, which activates factors in the blood, thus triggering the coagulation cascade. This product is only US Food and Drug Administration (FDA) approved for external use but is X-ray detectable. The majority of subcapsular hematomas require no specific treatment in the stable patient, but in a DC setting, they can expand significantly due to underlying coagulopathy and may require any of the above approaches.

Minor parenchymal injuries can typically be managed as described previously for capsular tears, particularly in the

setting of definitive hemostasis. An additional technique that is used for either definitive hemostasis or DC includes suture hepatorrhaphy. If bleeding vessels are visualized, they should be directly ligated, typically with a 2-0 Vicryl suture. Another technique useful for more significant parenchymal injury is placement of "liver sutures" whereby a 0 or #1 chromic with a blunt-tipped needle is placed on either side of the defect in the parenchyma in a horizontal mattress fashion, creating a tamponade effect on the injured liver (Fig. 11.13a–d). A few tricks can be very helpful. The defect

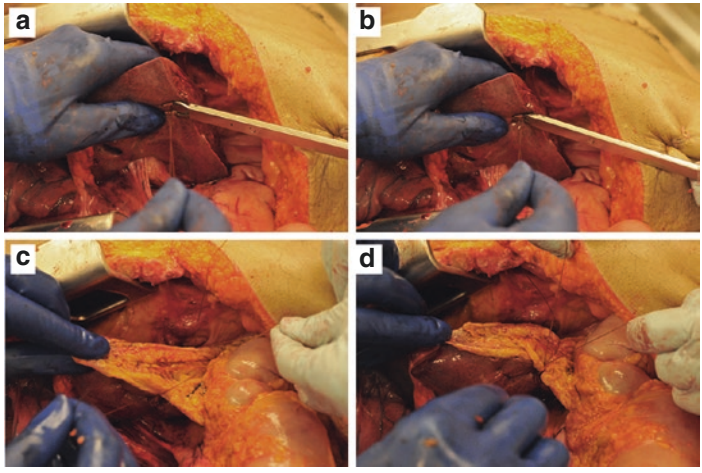


FIGURE 11.13 (**a**–**d**) Suture hepatorrhaphy. Placement of "liver sutures" whereby a 0 chromic with a blunt-tipped needle is placed on either side of the defect in the parenchyma in a horizontal mattress fashion creating a tamponade effect on the injured liver. (**a**, **b**) To help prevent tearing of the capsule, a hemoclip can be used to "set the tension" and then tie down the suture. (**c**, **d**) If the sutures are placed opposite each other and left long, the omentum can be placed on the defect and the sutures tied over it to minimize the need to place additional sutures. (Facilities: Surgical Laboratory, Anatomical Services Division, School of Medicine, UMB; Cadavers and Specimens, Anatomical Donors, Maryland State Anatomy Board; Ronn Wade, State Anatomy Board, Department of Health and Mental Hygiene)

can be packed with hemostatic material such as SURGICEL® (Ethicon, Medline Industries, Inc., Somerville, NJ, USA) or omentum prior to tying down the sutures. By placing sutures opposite one another and leaving the tails long, the omentum or other hemostatic agent can be placed on the defect, and the sutures tied over it to eliminate the need for additional sutures. Additionally, one technique that helps prevent tearing of the capsule is to "set the tension" on the suture using a hemoclip to press into the liver capsule along the suture, fire at the designated tension, and then tie down the suture. We typically do not use pledgets, but if we do, we avoid the use of nonabsorbable materials as these can become a nidus for infection. Instead, a piece of rolled or folded SURGICEL®, Vicryl™ Knitted Mesh, or NU-KNIT® can be used.

Major parenchymal disruptions require the use of more advanced surgical techniques whether as a damage control procedure or in the setting of definitive repair. Techniques include suture and other hepatorrhaphy techniques as described previously, hepatotomy with direct vessel ligation, resectional debridement/nonanatomic resection, or anatomic resection. Often, a combination of these techniques is required. Peripheral and central injuries often require different approaches.

Major peripheral injuries are best served by resectional debridement or nonanatomic resection. The liver tends to bleed from both "halves" of the injured liver, and once the lateral parenchyma is resected, the bleeding is stemmed by "half" and is easily controlled with manual compression and direct vessel ligation. This tends to be the most rapid way to control hemorrhage. Techniques for transecting liver parenchyma are identical for resectional debridement, hepatotomy, nonanatomic resection, and anatomic resection and include a number of techniques such as finger fracture or crush clamping and devices such as the ultrasonic desiccator (Cavitron Ultrasonic Surgical Aspirator—CUSA), water-jet dissectors (HELIX HYDRO-JET®, Stormoff, Duesseldorf, Germany), harmonic scalpel (Ethicon Endo-Surgery, Inc.,

Cincinnati, OH, USA), or dissecting/radio-frequency sealers (TissueLink, Medtronic, Inc, Minneapolis, MN, USA). These specialized devices are too slow for use in acute trauma due to the need for prolonged tissue contact time for efficacy. These devices can be very helpful, however, upon reoperation for definitive hemostasis after DC or for management of liver-related complications. We typically favor a simple finger fracture technique or the use of gastrointestinal anastomosis (GIA) staplers. The finger fracture technique uses gentle fracturing of the liver parenchyma between the thumb and index finger or uses the blunt tip of an instrument in a back-and-forth motion after the liver capsule is "scored." The liver parenchyma is dissected, leaving the blood vessels and biliary ducts exposed in the tract for ligation with sutures or clips. The use of short-handle endo GIA staplers using a "vascular" load, typically 2.5 mm staples, accomplishes the same thing in less time. The anvil of the stapler is inserted gently into the liver parenchyma, and with small adjustments, the stapler will find its "sweet spot" where it slides easily into the liver parenchyma. The stapler is fired, dividing the parenchyma and sealing the vessels. If this is unable to be done safely, a tract should be created first by the gentle insertion of a hemostat (Fig. 11.14). We

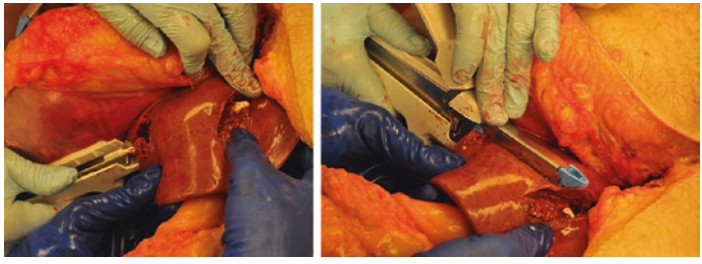


Figure 11.14 Hepatotomy/tractotomy of liver parenchyma using GIA stapler. (Facilities: Surgical Laboratory, Anatomical Services Division, School of Medicine, UMB; Cadavers and Specimens, Anatomical Donors, Maryland State Anatomy Board; Ronn Wade, State Anatomy Board, Department of Health and Mental Hygiene)

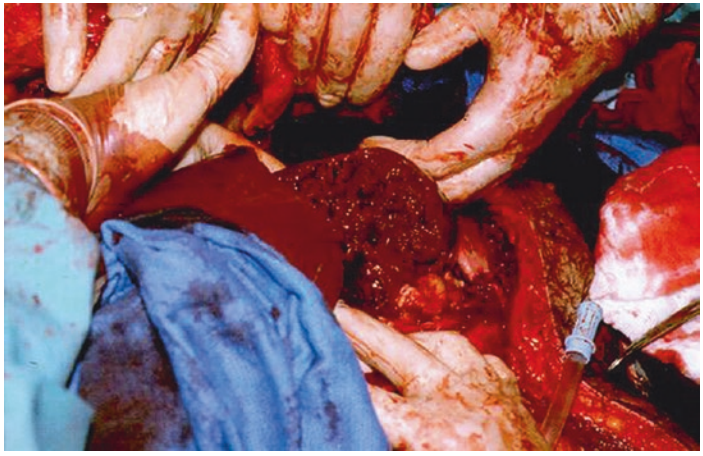


Figure 11.15 Example of major central parenchymal injury

always have our operating room (OR) personnel have two open devices on the operative field so that a loaded device is always available to allow rapid parenchymal division. The raw edge of the liver can then be treated with any of the techniques described previously for hepatorrhaphy. For all major parenchymal injuries or if a significant "raw" edge of the liver is left, we place closed suction drains in anticipation of biliary leaks that occur frequently [12].

Major central injuries to the liver can be exceptionally difficult to manage (Fig. 11.15). These patients are almost universally unstable and therefore DC is wise. If the injury can be largely controlled with simple packing, we use temporary abdominal closure and emergent diagnostic angiography and angiographic embolization (AE). If bleeding cannot be controlled, then more advanced techniques are indicated. Suture hepatorrhaphy with large liver sutures to "close down the parenchymal defect" should not be used for these injuries. Although approximation of tissue may create a local tamponade effect, it risks rebleeding, intrahepatic hematoma, abscess, and biloma formation [38]. Additionally, the blind placement of large central liver

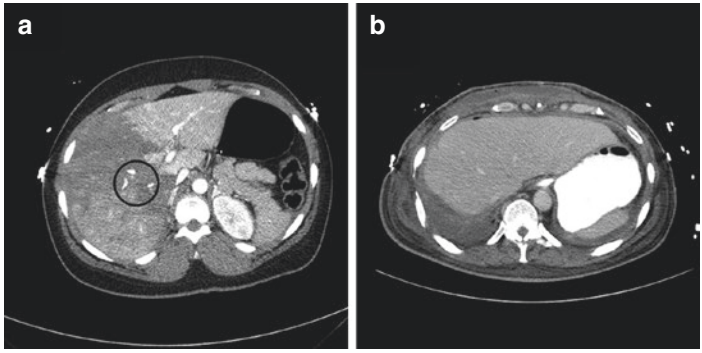


Figure 11.16 (**a**) CT scan of a 48-year-old female after a motor vehicle crash with a grade V liver injury with active areas of extravasation (circle). The patient was initially hemodynamically stable but then decompensated and was taken to the OR where she had percutaneous cannulation for veno-venous bypass and a nonanatomic resection of the right lobe of her liver. Forty-eight hours later, she was taken back for completion of anatomic resection and decannulation. (**b**) CT scan done 4 weeks postoperatively

sutures causes a risk of injury to underlying major vascular and biliary structures. These injuries are best treated with hepatotomy with direct vessel ligation, often with portal occlusion in place. These injuries particularly require a keen appreciation of hepatic anatomy to avoid injury or transection of noninjured essential vascular and biliary structures. If anatomically amenable, without causing disruption of the vascular supply to the uninjured liver, nonanatomic resection or resectional debridement can be performed (Fig. 11.16a, b). Anatomic resection with direct vascular ligation of major vascular and biliary structures can be performed. However, it is rare that the patient is stable enough to allow for anatomic resection at the index operation, and this technique is usually reserved for the "take-back" operation or for management of complications such as major hepatic necrosis [39]. Another option for major central hepatic parenchymal injuries is selective hepatic artery ligation. This technique should be reserved

for use in patients with exsanguinating hemorrhage in which there is not time for more selective AE to be done. There are a few additional DC techniques that can be applied to the patient with central parenchymal injuries, particularly transhepatic gunshot wounds. Tamponade with a Foley balloon can be used to provide intraparenchymal pressure on vascular structures for temporary hemostasis, and the technique of a saline-filled Penrose drain passed through a central injury tract for temporary hemostasis can be lifesaving [40–42] (Fig. 11.17a–e).

The use of angiographic embolization (AE) is an essential adjunct to any major hepatic parenchymal injury, whether peripheral or central. AE is typically used as a part of NOM, for management of subsequent complications, and as an adjunctive measure for operative management, typically in the setting of DC. Numerous studies have advocated the use of adjunctive AE either pre- or postoperatively [3, 5, 10, 11, 43–50]. The key to successful use of AE is anticipating its need. In most institutions, mobilizing the interventional radiology (IR) team takes some time, especially off-hour on nights and weekends. We recommend notifying the IR team as soon as we identify that a patient has severe hepatic trauma. This allows for the team to be ready so the patient can be transported directly to the IR suite from the OR. Ongoing resuscitation can be carried out in the IR suite, and the patient brought directly back to the OR as needed. If personnel can be spared, it is useful to have a team member assist with resuscitation in the IR suite. Additionally, if ongoing substantial bleeding has occurred since the index operation and with successful AE, we advocate returning to the OR for a quick "unpack and repack" as the lap pads soaked with blood no longer provide effective tamponade. A temporary abdominal closure is then performed, and the patient is brought to the ICU for ongoing resuscitation. At our institution, we have Acute Care Vascular Surgeons who perform these procedures in our hybrid operating room (OR) which precludes the need to take the patient out of the OR to the IR suite. As hybrid

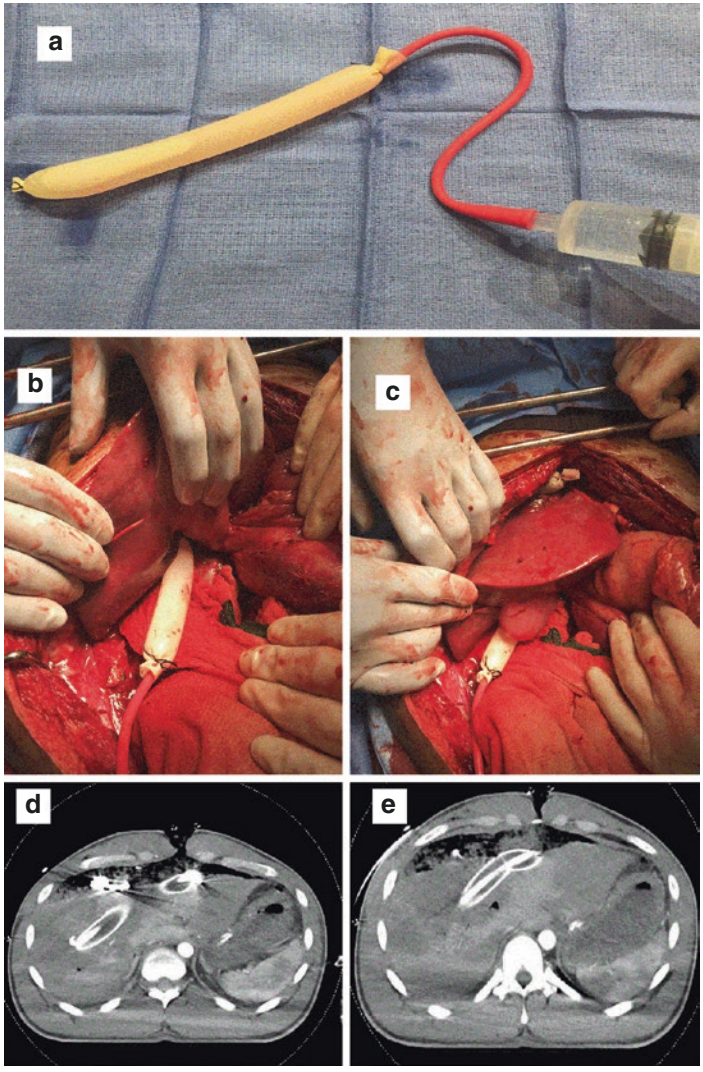


FIGURE 11.17 (**a**) Foley balloon tamponade – constructed with Penrose drain over red rubber. (**b**, **c**) Intraoperative placement of a Foley balloon for tamponade in a central hepatic injury. (**d**, **e**) CT imaging of Foley balloon tamponade

operating suites are becoming more widely available, the patient can ideally undergo an AE procedure either by an Acute Care Vascular Surgeon or IR personnel while in the OR, allowing for ongoing resuscitation without extensive movement through the hospital.

Retrohepatic caval and hepatic venous injuries are both lethal and extremely challenging. Access to visualize and ligate or repair these structures is the key to successful management. Direct pressure should be applied during hepatic mobilization and attainment of additional exposure. Typically, these injuries are associated with massive hemorrhage, and even when exposure is optimal, it is exceptionally difficult to maintain a dry enough field to identify bleeding sites. Once the injury is visualized, the hepatic veins can typically be ligated without consequence. The retrohepatic IVC, however, should be repaired whenever possible. One effective technique that can be applied to all major venous injuries is the use of "intestinal Allis" clamps, which can be used to approximate the edges of the vein and provide temporary hemostasis [51]. The clamps are "stacked" along the injury, and a suture can then be run under the clamps for repair of the vessel wall.

In the patient with a massively destructive liver injury, total hepatectomy is a viable, albeit last resort, option [8, 38, 52, 53]. Outcomes following total hepatectomy and transplantation are acceptable enough to make this an option in patients with liver injury not amenable to other hemostatic techniques. If total hepatectomy is to be done, there are a few key steps that should be undertaken. First, the IVC should be left as intact as possible or reconstructed with a prosthetic graft to maintain venous drainage and preload to the heart. Second, portal decompression is essential. This can be accomplished with either extracorporeal support using veno-venous bypass or with the creation of a portal-systemic shunt/bypass. Failure to provide portal decompression will almost invariably result in mesenteric ischemia and death from sepsis. Use of veno-venous bypass in these patients with or without a portal decompression cannula is also extremely helpful in

stabilizing the patient enough to allow them to survive to transplantation. Third, involvement of a transplant service or referral to a transplant center should be done immediately so that the patient can be listed as a "Status 1" candidate for emergent hepatic transplantation.

Outcomes

Outcomes following liver injury are highly variable depending on the type and severity of injury. For patients who are candidates for NOM, liver-related mortality is well below 1% [27]. For patients that require operative intervention, mortality rates have been reported to be as low as 2% for highly selected patients [9] but >80% for patients with retrohepatic IVC or major hepatic venous injuries [35, 36]. In most large studies of patients with operative hepatic trauma, mortality rates are typically approximately 50% [2, 3, 54, 55]. In more selected patient populations in which early deaths from acute hemorrhage were excluded, liver-specific mortality rates are 11–30% [10, 12]. Morbidity rates for patients who require operative intervention are as high as 80%, with the most frequent complications being recurrent hemorrhage, sepsis and organ system dysfunction, abdominal compartment syndrome (ACS), bile leak, hepatic necrosis, and liver abscess [6, 10–12, 43]. After adjunctive AE, complication rates as high as almost 90% have been reported due to the severity of the primary injury combined with the hepatic ischemia from embolization [11].

Complications with Treatment

Patients with severe liver injury can develop devastating sepsis and/or organ dysfunction that can require intensive supportive therapy. The complications discussed here are the ones that specifically require additional operative therapy or intervention.

Rebleeding or recurrent hemorrhage occurs both after NOM and operative management. Strategies for management are dependent on the original approach used, degree of hemodynamic stability, presence of coagulopathy, and availability of resources. If major vascular bleeding is suspected, the patient can be managed with either AE or laparotomy. Angiography is the procedure of choice if immediately available in a patient who underwent a diagnostic angiogram initially without AE performed or in a patient who has already had DC laparotomy without postoperative angiography. If angiography is not immediately available and the patient is hemodynamically unstable, then additional operative intervention is warranted. Diffuse "ooze" and coagulopathy are best managed with emergent factor and platelet administration. Returning these patients to the operating room to repack may be beneficial, but the risks of additional operative insult and physiological derangements must be weighed against the potential benefits.

Hepatic abscesses occur following major liver injury, particularly in the setting of concomitant bowel injury. Most can be managed with percutaneous drainage and antibiotics. Operative intervention is typically reserved for patients in whom this approach fails to resolve the abscess or with signs and symptoms of persistent sepsis.

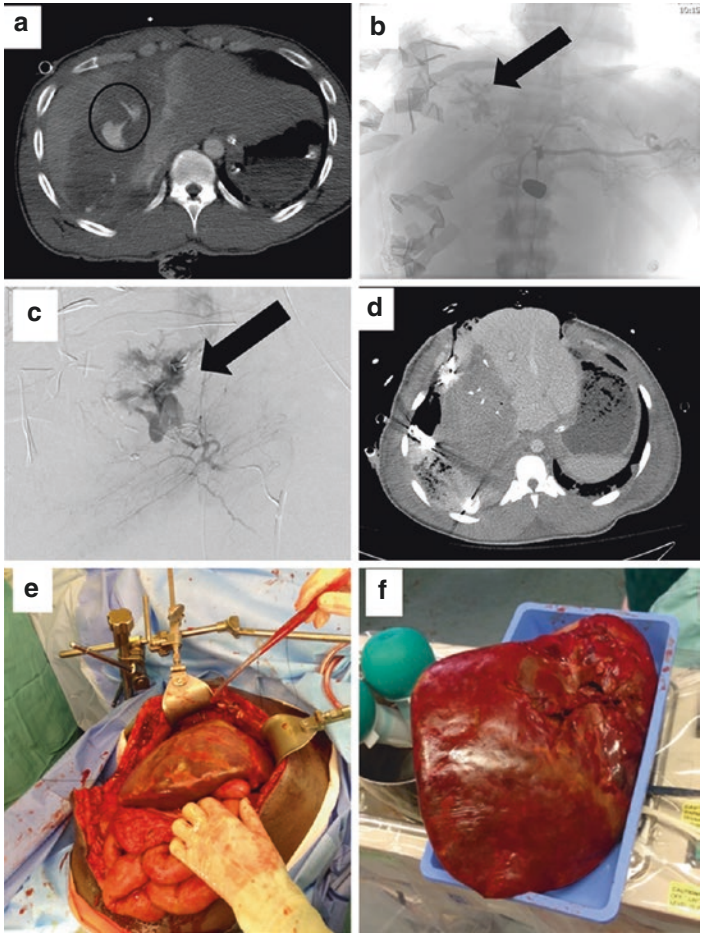
Biliary complications are some of the most frequently encountered after major hepatic injury [43, 56]. Bile leaks, biloma formation, and hemobilia can all occur. Bile leaks are typically diagnosed when persistent bilious drainage is noted from operatively placed drains or with the development of bile peritonitis. Hepatobiliary iminodiacetic acid (HIDA) scan is diagnostic [56]. Small contained leaks can be effectively managed with drainage alone. Persistent or high-volume leaks are best treated with drainage, endoscopic retrograde cholangiopancreatography (ERCP), and common duct stent placement, which is highly effective [57]. Diffuse leaks with bile peritonitis are best managed with operative "washout" [56]. Bilomas are typically intraparenchymal contained bile leaks. Small bilomas often will

resolve, but larger ones should be treated with percutaneous drainage +/– ERCP and stenting if persistent. Hemobilia, typically a late complication of severe liver injury, is caused by an arterial fistula to some part of the biliary tree. These patients present most typically with upper gastrointestinal bleeding. Treatment is AE of the involved artery and fistulous connection. Gallbladder necrosis is also seen following AE for high-grade liver injuries and requires cholecystectomy [10].

Major hepatic necrosis is now a well-recognized complication following severe liver injury, particularly when AE is used to control hemorrhage and is reported to occur in 15–40% of AE cases [10, 11, 58] (Fig. 11.18a–f). It typically presents with fever, leukocytosis, hyperlactemia, and persistent elevation of transaminases [10]. Contrast-enhanced CT is diagnostic. These patients are often critically ill, and areas of necrosis must be distinguished from simple abscess. We, and others, have favored an aggressive surgical approach to these complications with anatomic or nonanatomic resection of the necrotic liver [11, 39]. For large areas of necrosis, we favor placing the patient on veno-venous bypass for these procedures as it markedly reduces intraoperative blood loss and facilitates resection. Alternatives to resection include a combination of operative debridement and percutaneous drainage. Often, multiple procedures are required, and the complication rate is higher with this approach [39].

Hepatic failure may occur as a result of severe liver trauma. The mainstay of treatment has been supportive care, but this is not enough in some cases. Extracorporeal hepatic replacement by Molecular Adsorbent Recirculating System (MARS, Baxter International, Deerfield, IL, USA) may be used to support patients until recovery of liver function or as a bridge to transplantation. Although experience with use of MARS in severe hepatic trauma is limited, it has shown some improvements in survival. Indications for its use are elevated ammonia, impaired neurological function, and rising creatinine and lactate levels. More experience is needed with

MARS, but the major benefit is the ability to support the patient through the initial insult and allow support and temporization of multisystem organ dysfunction caused by failure of clearance of toxins in hepatic failure. Additionally, this approach may give a transplant team further time on decisions on suitability for liver transplantation in cases in which hepatic failure does not resolve [59].



References

1. Taghavi S, Askari R. Liver Trauma. StatPearls. Treasure Island: StatPearls Publishing; 2018. Available from: http://www.ncbi.nlm.nih.gov/books/NBK513236.
2. 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.
3. Lin B-C, Fang J-F, Chen R-J, Wong Y-P. Surgical management and outcome of blunt major liver injuries: experience of damage control laparotomy with perihepatic packing in one trauma centre. Injury. 2013;45:122.
4. Fabian TC, Croce MA, Stanford GG, Payne LW, Mangiante EC, Voeller GR, et al. Factors affecting morbidity following hepatic trauma: a prospective analysis of 482 injuries. Ann Surg. 1991;213:540–8.
5. Asensio JA, Roldan G, Petrone P, Rojo E, Tillou A, Kuncir E, et al. Operative management and outcomes in 103 AAST-OIS grades IV and V complex hepatic injuries; trauma surgeons still need to operate, but angioembolization helps. J Trauma. 2003;54:647–54.

Figure 11.18 (**a**) CT scan of a 17-year-old male after a gunshot wound with a grade V liver injury with large area of extravasation (circle). The patient became hemodynamically unstable and was taken to the OR where hepatotomy and direct vessel ligation was performed with packing. (**b**, **c**) The patient was brought emergently to IR, which demonstrated active bleeding from the right hepatic artery (arrows). Coil embolization was performed. Three days later, patient developed fever, leukocytosis, and increasing transaminases. (**d**) CT scan demonstrated hepatic necrosis of the entire right lobe of the liver. (**e**) Intraoperative photograph of the right hepatic lobe with extensive necrosis. A formal right hepatic lobectomy was performed with veno-venous bypass. (**f**) Resected right lobe of the liver demonstrating the previous hepatotomy site and parenchymal necrosis

6. Denton JR, Moore EE, Coldwell DM. Multimodality treatment for grade V hepatic injuries: perihepatic packing, arterial embolization, and venous stent. J Trauma. 1997;42:964–8.
7. Cogbill TH, Moore EE, Jurkovich GJ, Feliciano DV, Morris JA, Mucha P. Severe hepatic trauma: a multicenter experience with 1335 liver injuries. J Trauma. 1988;28:1433–8.
8. 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;65:1264–70.
9. Petri SL, 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;78:20–5.
10. Dabbs DN, Stein DM, Scalea TM. Major hepatic necrosis: a common complication after angioembolization for treatment of high-grade liver injuries. J Trauma. 2009;66:621–9.
11. 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.
12. Pringle JH. V. Notes on the arrest of hepatic hemorrhage due to trauma. Ann Surg. 1908;48(4):541–9.
13. Schwab CW. Selection of nonoperative management candidates. World J Surg. 2001;25:1389–92.
14. Hawkins ML, Wynn JJ, Schmacht DC, Medeiros RS, Gadacz TR. Nonoperative management of liver and/or splenic injuries: effect on resident surgical experience. Am Surg. 1998;64:552–7.
15. Rutledge R, Hunt JP, Lentz CW, Fakhry SM, Meyer AA, Baker CC, et al. A Statewide, population-based time-series analysis of the increasing frequency of non-operative management of abdominal solid organ injury. Ann Surg. 1995;225:311–26.
16. Shedden K. Cut Gurlt. Geschichte de Chir. 1898;3722.
17. Tilton BJ. Considerations regarding wounds of the liver. Ann Surg. 1905;61:20–30.
18. Krieg EG. Hepatic trauma: analysis of sixty cases. Arch Surg. 1936;32:907–14.
19. Lamb CA. Rupture of the liver. N Engl J Med. 1939;221:855–9.
20. Beck C. Surgery of the liver. JAMA. 1902;38:1063–8.
21. Trunkey DD. Hepatic trauma: contemporary management. Surg Clin N Am. 2004;84:437–50.
22. Root HD, Howser CW, McKinley CR, Lafave JW, Mendiola RP Jr. Diagnostic peritoneal lavage. Surgery. 1965;57:633–7.

23. Federle MP, Goldberg HI, Kaiser KA, Moss AA, Jeffrey RB Jr, Mall JC. Evaluation of abdominal trauma by computed tomography. Radiology. 1981;138:a637–44.
24. Moore EE, Shackford SR, Pachter HL, McAninch JW, Browner BD, Champion HR, et al. Organ injury scaling: spleen, liver and kidney. J Trauma. 1989;29:1664–5.
25. Moore EE, Shackford SR, Pachter HL, Shackford SR, Malangoni MA, Champion HR. Organ injury scaling: spleen, liver and kidney. J Trauma. 1995;38(3):323–4.
26. Croce MA, Fabian TC, Menke PG, Waddle-Smith L, Minard G, Kudsk KS, 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;221:744–55.
27. 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. 1995;40:31–8.
28. 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.
29. Couinaud C. Le foie: études anatomiques et chirurgicales [The liver: anatomical and surgical studies] (in French). Paris: Masson; 1957.
30. Hiatt JR, Gabbay J, Busuttil RW. Surgical anatomy of the hepatic arteries in 1000 cases. Ann Surg. 1994;220(1):50–2.
31. Reynolds CL, Celio AC, Bridges LC, Mosquera C, O'Connell B, Bard MR, et al. REBOA for the IVC? Resuscitative balloon occlusion of the inferior vena cava (REBOVC) to abate massive hemorrhage in retrohepatic vena cava injuries. J Trauma Acute Care Surg. 2017;83(6):1041–6.
32. Baumgartner F, Scudamore C, Nair C, Karusseit O, Hemming A. Venovenous bypass for hepatic and caval trauma. J Trauma. 1995;39:671–3.
33. Horowitz JR, Black T, Lally KP, Andrassy RJ. Venovenous bypass as an adjunct for the management of a retrohepatic venous injury in child. J Trauma. 1995;39:584–5.
34. Schrock T, Blaisdell FW, Mathewson C. Management of blunt trauma to the liver and hepatic veins. Arch Surg. 1968;96:698–704.
35. Burch JM, Feliciano DV, Mattox KL. The atriocaval shunt. Facts and fiction. Ann Surg. 1988;207(5):555–68.

36. Kudsk KA, Sheldon GF, Lim RC. Atrial-caval shunting (ACS) after trauma. J Trauma. 1982;22(2):81–5.
37. Pachter HL, Spencer FC, Hofstetter SL, Liang HC, Coppa GF. The management of juxtahepatic venous injuries without an atriocaval shunt. Surgery. 1986;99:569–75.
38. Peitzman AB, Marsh JW. Advanced operative techniques in the management of complex liver injury. J Trauma Acute Care Surg. 2012;73(3):765.
39. Dabbs DN, Stein DM, Philosophe B, Scalea TM. Treatment of major hepatic necrosis: lobectomy versus serial debridement. J Trauma. 2010;69(3):562.
40. Morimoto R, Bironlini D, Junqueria A, Poqqetti R, Horita L. Balloon tamponade for transfixing lesion of the liver. Surg Gynecol Obstet. 1987;164:87.
41. Poggetti RS, Moore EE, Moore FA, Mitchell MB, Read RA. Balloon tamponade for bilobar transfixing hepatic gunshot wounds. J Trauma. 1992;33(5):694.
42. Seligman JY, Egan M. Balloon tamponade: an alternative in the treatment of liver trauma. Am Surg. 1997;63(11):1022–3.
43. Kozar RA, McNutt MK. Management of adult blunt hepatic trauma. Curr Opin Crit Care. 2010;16:596–601.
44. Misselbeck TS, Teicher E, Cipolle M, Pasquale MD, Shah KT, Dangleben DA, et al. Hepatic angioembolization in trauma patients: indications and complications. J Trauma. 2009;67:769–73.
45. Johnson JW, Gracias VH, Gupta R, Guillamondegui O, Reilly PM, Shapiro MB, et al. Hepatic angiography in patients undergoing damage control laparotomy. J Trauma. 2002;52:1102–6.
46. Denton JR, Moore EE, Coldwell DM. Multimodality treatment of grade v hepatic injuries: perihepatic packing, arterial embolization, and venous stenting. J Trauma. 1997;42:965–8.
47. Asensio JA, Petrone P, Garcia-Nunez L, Kimbrell B, Kuncir E. Multidisciplinary approach for the management of complex hepatic injuries AAST-OIS grades IV-V: a prospective study. Scand J Surg. 2007;96:214–20.
48. Wahl WL, Ahrns KS, Brandt MM, Franklin GA, Taheri PA. The need for early angiographic embolization in blunt liver injuries. J Trauma. 2002;52:1097–101.
49. 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;55:1077–81.
50. Matsumoto S, Cantrell E, Jung K, Smith A, Coimbra R. Influence of postoperative hepatic angiography on mortality after laparot-

omy in Grade IV/V hepatic injuries. J Trauma Acute Care Surg. 2018;85(2):290–7.
51. Henry SM, Duncan AO, Scalea TM. Intestinal Allis clamps as temporary vascular control for major retroperitoneal venous injury. J Trauma. 2001;51(1):170–2.
52. Heuer M, Kaiser GM, Lendemans S, Vernadakis S, Treckmann JW, Paul A. Transplantation after blunt trauma to the liver: a valuable option or just a "waste of organs"? Eur J Med Res. 2010;15(4):169–73.
53. Tucker ON, Marriott P, Rela M, Heaton N. Emergency liver transplantation following severe liver trauma. Liver Transpl. 2008;14:1204–10.
54. Liu PP, Chen CL, Cheng YF, Hsieh PM, Tan BL, Jawan B, et al. Use of a refined operative strategy in combination with the multidisciplinary approach to manage blunt juxtahepatic venous injuries. J Trauma. 2005;59:940–5.
55. Doklestić K, Stefanović B, Gregorić P, Ivančević N, Lončar Z, Jovanović B, et al. Surgical management of AAST grades III-V hepatic trauma by damage control surgery with perihepatic packing and definitive hepatic repair-single centre experience. World J Emerg Surg WJES. 2015;10:34.
56. Wahl WL, Brandt MM, Hemmila MR, Arbabi S. Diagnosis and management of bile leaks after blunt liver injury. Surgery. 2005;138(4):742–8.
57. 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.
58. Green CS, Bulger EM, Kwan SW. Outcomes and complications of angioembolization for hepatic trauma: a systematic review of the literature. J Trauma Acute Care Surg. 2016;80(3):529–37.
59. Hanish SI, Stein DM, Scalea JR, Essien E-O, Thurman P, Hutson WR, et al. Molecular adsorbent recirculating system effectively replaces hepatic function in severe acute liver failure. Ann Surg. 2017;266(4):677–84.

Chapter 12
Spleen Injuries: Techniques

Amanda M. Chipman, Matthew Lissauer, and Rosemary Kozar

Introduction of the Problem

The spleen is one of the most commonly injured organs following blunt trauma. The injured spleen is a major source of morbidity and mortality and can be rapidly fatal if hemostasis is not promptly achieved. Up to one-third of patients with splenic injuries proceed directly to the operating room for surgical exploration [1]. Patients who have had splenectomy are at risk for the usual morbidities of laparotomy such as

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incisional hernia and small bowel obstruction as well are at increased risk for overwhelming sepsis syndromes due to asplenia. Nonoperative management also has risks including failure of nonoperative management, delayed hemorrhage, splenic infarction and abscess, and complications of angiography, if used. Given the common nature of the injury and the risks with all forms of management, the trauma surgeon must be well versed in the techniques required to care for this injury.

History of Care of Spleen Injuries

In the past, the spleen seemed a mystery and was considered nonessential for survival. The Talmud describes the spleen as a laughter-generating organ. Babylonian, Greek, and Roman texts incriminate the spleen as a cause of poor athletic performance [2]. Galen, Vesalius, and Malpighi all debated whether or not the spleen was necessary for survival [3]. There were accounts of asplenic patients, however, with no documented obvious adverse sequela suggesting lack of function of the organ.

During the early history of surgery for the injured spleen, splenectomy, though usually fatal, was considered as the only treatment for injury since its true function was unknown and death seemed more certain without operation. The spleen was considered frail and unable to heal. Early attempts at splenectomy for trauma described in the 1800s were quite often failures. Still, no consideration at that time was given to splenic salvage. It was incorrectly thought that the bone marrow and thyroid could substitute for the spleen's perceived "blood producing quality."

By the early twentieth century, however, splenectomy became more safe and hundreds of total splenectomies for trauma had been reported [4], many of them successful. Over the course of the twentieth century, the immunologic function of the spleen became better known and characterized. As early as 1952, data emerged that asplenic patients were at

higher risk for overwhelming sepsis [5]. These data were generated in children with hematologic diseases, but the principles were used to manage all patients with splenic injury. As this risk became better delineated, splenic salvage and conservative management of the spleen emerged as management options [6].

Prior to the era of computed tomography (CT) scan, conservative management of splenic injury was splenorrhaphy or repair since diagnosis was usually made at laparotomy. With the advent and widespread use of CT scans, preoperative diagnosis became common. Concurrently, advances in catheter-based technologies allowed development of angiography as another diagnostic tool and embolization as therapy. These technologies permitted true nonoperative, conservative management of the injured spleen. Splenectomy for trauma became significantly more uncommon.

The incidence of overwhelming postsplenectomy sepsis now appears lower than once perceived, perhaps due to advances in vaccine and prophylactic therapy. Further, compared to nonoperative management, the morbidity of laparotomy is high. One must weigh the benefits of definitive bleeding control and the risks of laparotomy against the potential benefits and risks of nonoperative management, including a defined failure rate. While splenectomy for trauma can be a relatively straightforward procedure, it can be associated with operative complications such as pancreatic tail injury and postoperative complications such as overwhelming postsplenectomy sepsis. This chapter will focus on the diagnosis and treatment options for the injured spleen.

Techniques and Tips

Techniques for Diagnosis

Approximately 30% of patients with splenic injury are treated with primary operative exploration. Typically, these patients present with hemodynamic instability and hemoperitoneum.

While hemoperitoneum may be readily diagnosed in the trauma bay by focused assessment with sonography for trauma (FAST) , splenic injury as the source of the hemoperitoneum is typically diagnosed in the operating room (OR). Standard techniques of trauma laparotomy described earlier in this book should be followed, with packing of all four quadrants to obtain immediate hemostasis followed by controlled removal with inspection for injury and plans for prompt hemorrhage control as injuries are identified.

Many patients with splenic injury, however, are hemodynamically stable and can underdo additional diagnostic workup to evaluate the source of hemoperitoneum. Additionally, up to 25% of patients with splenic injury may have injury within the parenchyma and will not have hemoperitoneum [7]. A negative FAST in a hemodynamically stable patient who has significant mechanism concerning for solid organ injury should undergo further evaluation.

The diagnostic modality of choice is now multidetector CT imaging. CT provides very high sensitivity and specificity for diagnosing injury to the spleen. Dual-phase imaging with both arterial phase and portal venous phase demonstrates the best diagnostic performance and can help guide management [8]. However, some patients may have discrete contraindications to intravenous (IV) contrast. While image quality will not be as good and detection of vascular lesions not possible, determination of significant injuries can sometimes be made with a non-contrast-enhanced CT scan [9]. It is important to keep in mind that using suboptimal or no contrast will limit the accurate diagnosis of splenic trauma and that a "normal"-appearing spleen can indeed be injured. CT scanning remains the most useful modality for directing initial care of the hemodynamically stable injured patient [10].

Once a splenic injury is diagnosed, there are several important radiologic features which should be evaluated as they impact treatment and outcomes. These features include grade of injury, the presence of a vascular injury, the extent of hemoperitoneum, and the presence of associated intra-abdominal injuries. The American Association for the Surgery

of Trauma (AAST) organ injury scale for spleens has recently been revised [11]. The most significant change in the 2018 revision is the incorporation of CT-diagnosed vascular injuries into the grading scale. In this context, a vascular injury is defined as either as a pseudoaneurysm or arteriovenous fistula. The presence of a vascular injury is now at least a grade 4 injury, regardless of the extent of parenchymal injury.

Techniques for Nonoperative Management

Conservative management includes admission to the hospital, serial abdominal exams, serial hemoglobin measurements, close observation and in some cases, angioembolization and/or repeat imaging. It is appropriate for most splenic injuries in the stable patient. However, there may be other issues such as brain injury, comorbidities, or other concurrent injuries that change this decision-making.

Nonoperative management is best guided by protocols. For instance, if predetermined triggers are reached, nonoperative management should be considered a failure, and the patient should proceed to the operating room. In adult patients with isolated splenic injury, the need for any blood transfusion should prompt serious consideration for operative exploration. Similarly, changes in hemodynamics or clinical exam should be indications for laparotomy. Institutional protocols have proven useful to optimize outcomes and guide management [12]. National evidenced-based guidelines may assist in the creation of institutional protocols [13], and consensus strategies have been evaluated that may also assist with creation of local institutional protocols [14].

Numerous risk factors have been identified that may predispose patients to failure of conservative treatment. High AAST injury grade, quantity of hemoperitoneum, evidence of active hemorrhage, and vascular lesions predict failure of conservative therapy [1, 11]. Age is a controversial risk factor for failure. While advancing age may be a risk [15–17], some analysis suggests age should not be considered when deciding to pursue

nonoperative therapy [18]. A recent review of the National Trauma Data Bank showed that while geriatric patients (age >65) failed nonoperative management more often than younger patients, there was no difference in mortality in geriatric patients who had successful versus failed nonoperative management [19]. Thus, they concluded that a trial of nonoperative management of blunt splenic injury is safe in the geriatric population. Another review identified clinical features including age over 40 years, splenic injury grade of 3 or higher, Injury Severity Score (ISS) of 25 or higher, abdominal Abbreviated Injury Score of 3 or greater, TRISS of less than 0.80, the presence of an intraparenchymal contrast blush, and an increased transfusion need as predictors for failure of nonoperative management [20]. All of these factors should be considered when deciding upon the appropriate therapeutic algorithm.

Angioembolization has a major role in conservative therapy for high-grade lesions. Several recent studies have shown that the use of angioembolization is associated with decreased rate of splenectomy [21, 22]. At the R Adams Cowley Shock Trauma Center, stable patients with evidence of vascular injury and those with grade 4 and 5 injuries undergo urgent angiography and embolization. Stable patients with grade 3 splenic injuries also undergo angiography, with angioembolization if any vascular injury is identified. These patients also undergo repeat imaging with CT scan within 48–72 hours. Angiogram is not indicated in patients with grade 1 and 2 injuries if there is no vascular injury seen on good-quality CT. Several studies also support the use of mandatory angioembolization of grade 4/5 injuries and selective angioembolization in lower-grade injuries [23, 24].

Proximal coil embolization is the technique of choice as it minimizes the risk of ischemia of the spleen, as it maintains adequate blood supply through the short gastric vessels. Distal coil embolization, although effective, is associated with higher rates of both failure of nonoperative management and splenic infarction. Proximal coil embolization involves first performing a diagnostic angiogram. The catheter is advanced into the proximal splenic artery, and the size of the artery is

determined. A coil, slightly larger than the artery, is then introduced into the artery just beyond the first pancreatic branch. Repeat angiography should demonstrate both occlusion of the splenic artery and distal filling of the splenic artery and spleen via collaterals such as the short gastric vessels, the gastroepiploic vessels, and the pancreatic branches.

Early analysis of angioembolization demonstrated splenic salvage rates as high as 92%, even in patients with vascular lesions [25, 26]. A splenic salvage rate of 80–95% can be achieved for grade 4 and 5 injuries, and embolization significantly improves salvage rates in patients with high-grade injuries and contrast blush on CT scan [26–29]. Even if no extravasation or pseudoaneurysm is noted on CT scan, a patient with a grade 4 or 5 splenic injury should undergo embolization to maximize the chance for success of nonoperative therapy [30]. Patients managed nonoperatively with grade 3 or higher injury should be observed in a monitored setting. Abdominal exams and serial hemoglobin measurements should be performed. If repeat imaging demonstrates delayed or persistent pseudoaneurysms, repeat embolization is indicated and salvage rates remain greater than 90% [31].

Delayed splenectomy after 24 hours of successful nonoperative management is rare [32]. However, if a patient fails conservative therapy, splenectomy is generally indicated. There are reports, however, of angioembolization for salvage therapy. In one series, over 80% of patients with delayed splenic rupture were successfully treated nonoperatively either with observation or angioembolization [33].

Figure 12.1 represents the R Adams Cowley Shock Trauma management algorithm for splenic trauma as of 2017. Follow-up CT scan is not necessary for low-grade injuries as less than 2% of patients require delayed intervention, and those who did were noted to have decreasing hemoglobin levels early in their hospital course [34]. When managing patients nonoperatively, thromboembolism prophylaxis with low-molecular-weight heparin should be provided as it does not increase the rate of transfusion or rate of failure of therapy [35]. All patients who either undergo splenectomy or

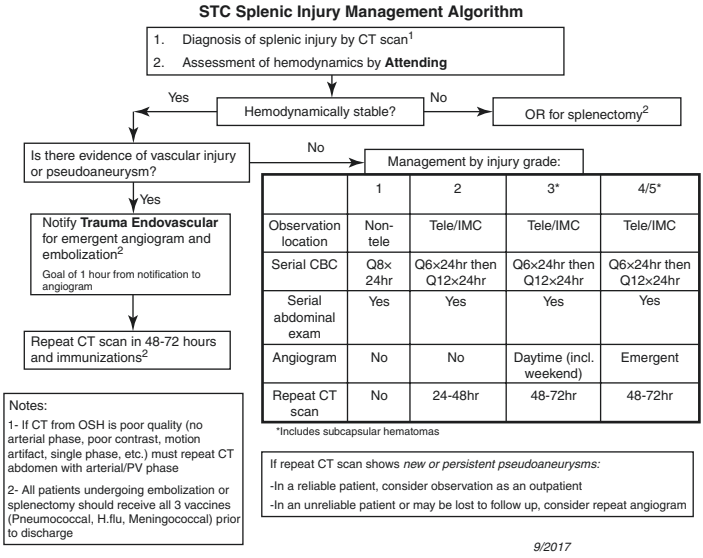


FIGURE 12.1 R Adams Cowley Shock Trauma Center (University of Maryland) blunt splenic injury protocol

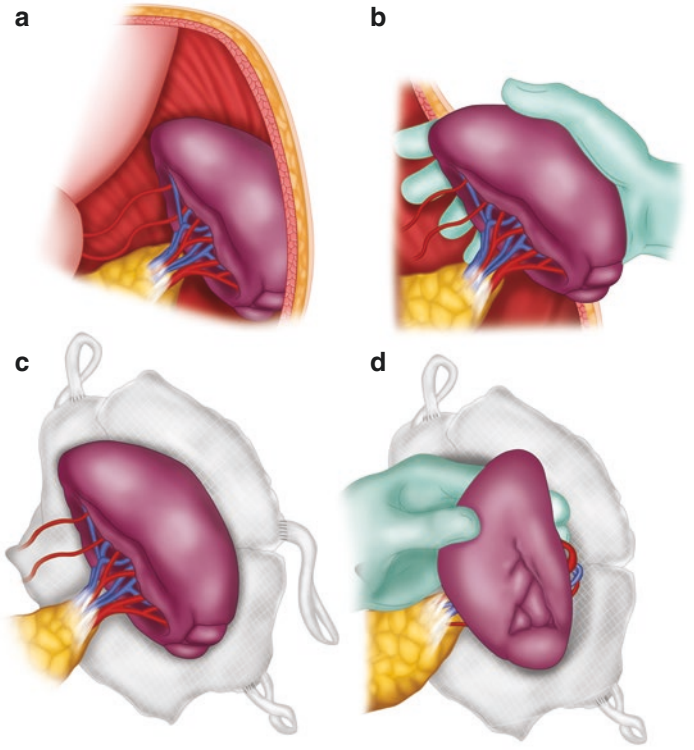
receive coil embolization of the spleen receive vaccines to reduce the incidence of postsplenectomy sepsis prior to discharge. In our opinion, the risk of losing patients to follow-up after discharge is not worth the benefit that may be obtained by delayed vaccination. There is some evidence as well that embolization alone may allow preserved splenic immune function [36–38]. With further study, it may prove unnecessary to vaccinate patients who undergo angiographic embolization of the spleen, but currently, sufficient data are not available to preclude vaccinations.

Techniques for Operative Treatment of Splenic Trauma

For the patient who is taken immediately to the operating room due to hemodynamic instability, hemorrhage control must be rapid. If at laparotomy the spleen is found to be the

cause of hemorrhage, splenectomy is usually indicated. Our technique for emergent splenectomy is described:

While standing on the right side of the patient, the surgeon uses his/her right hand to rapidly mobilize the spleen by bluntly dividing the splenorenal and splenophrenic ligaments (Fig. 12.2a, b). The spleen is mobilized medially into the abdominal incision. It is an error to operate on the spleen in the left upper quadrant, as exposure is inadequate. The spleen can be elevated on some lap pads (Fig. 12.2c). If needed, a left



FIGURERE 12.2 (**a**) The spleen in situ. (**b**) Manual mobilization of the spleen from its attachments. (**c**) The spleen mobilized into the operative field resting on laparotomy pads. (**d**) Medial rotation of the spleen to control the hilum from its posterior aspect

medial visceral rotation can be rapidly completed almost entirely with blunt dissection if other organs are injured in proximity to the spleen or retroperitoneal hemorrhage is identified in addition to splenic hemorrhage. We generally prefer to approach the spleen from its posterior aspect as it allows the best visualization of the pancreatic tail (Fig. 12.2d). The splenic vascular anatomy is variable. Sometimes, the splenic artery and vein extend onto the hilum. If so, they can be ligated flush with the spleen. Other times, they branch proximal to the hilum. If so, each branch should be ligated separately. Regardless of how the hilar vessels are handled, we reinforce the ties with suture ligatures. The short gastric vessels are then ligated and the spleen is removed. The stomach should be carefully inspected to be sure every short gastric has been ligated. Even if all short gastric vessels are ligated, some oin our group bury them with silk Lembert stitches or re-liagte them, also with sild sutures to ensure they do not bleed post operatively.

If the patient is in extremis, a one clamp splenectomy can be performed. An aortic vascular clamp is placed across the hilum and short gastric vessels. Care should be taken with this technique to avoid clamping the stomach. The clamp should be placed distal enough to avoid the tail of the pancreas as well (Fig. 12.3). Scissors are then used to remove the organ. The hilum and short gastric vessels can be controlled separately at this point by either ties or suture ligation. We use a combination of ties on all major vessels followed by additional suture ligature to reinforce the splenic artery and vein. If there is any concern for pancreatic injury from the rapid splenectomy, a closed suction drain can be left as energy transmitted to the injured spleen could also cause occult injury to the tail of the pancreas.

If the hemodynamically stable patient is found to have a splenic injury in the OR, splenorrhaphy can be an option. In general, a stable patient with minimal blood loss and low-grade injury (grade 1 or 2) is a candidate for splenorrhaphy. However, if a patient has a severe traumatic brain injury such that recurrent hemorrhage may cause secondary brain injury or if the patient is coagulopathic or has comorbidities that may predispose to bleeding (such as chronic liver disease), splenectomy may be a better choice. We usually prefer sple-

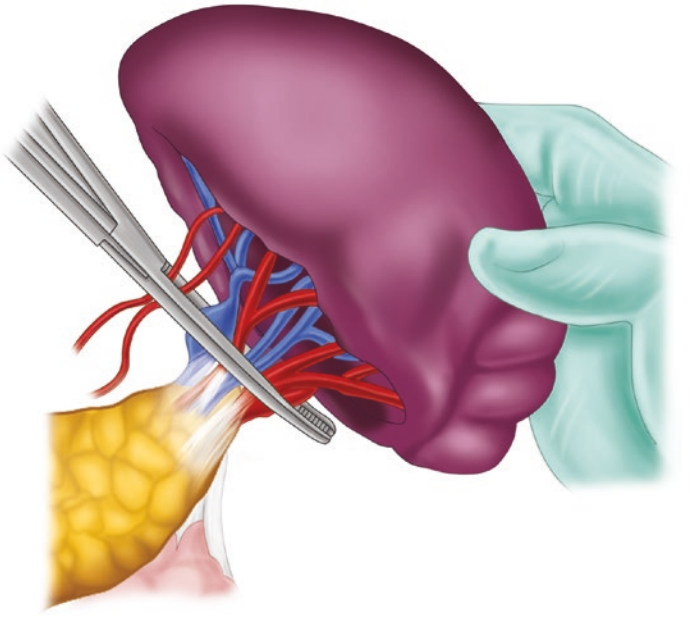


FIGURE 12.3 Single clamp splenectomy with the clamp obtaining total vascular control of the hilum and short gastric vessels

nectomy for higher-grade injuries to avoid repeat laparotomy as failure rates are higher.

If splenorrhaphy is chosen, there are many options for surgical technique. Partial splenectomy can be done if the injury is limited to one pole. After mobilizing the spleen, temporary hemostasis may be obtained with finger pressure (Fig. 12.4). The corresponding vascular supply to that pole is ligated and divided. The splenic parenchyma is then cut with a scalpel (Fig. 12.5). The raw edge of the parenchyma is compressed with pledgeted horizontal mattress sutures. 0-Vicryl or 2-0 Vicryl suture works well, and some of the new bioabsorbable pledgets may reduce infectious risk (Fig. 12.6). Argon beam coagulation or placement of fibrin hemostatic sealant can be added to augment hemostasis. For smaller injuries, direct pledgeted suturing can be used to control

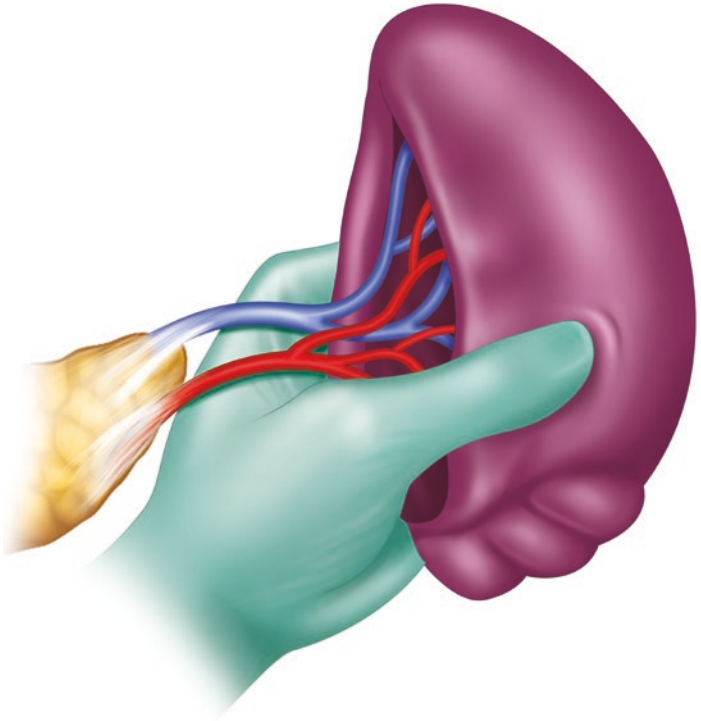


Figure 12.4 Direct manual control of splenic bleeding in preparation for splenorrhaphy

bleeding from the spleen (Fig. 12.7). If the capsule of the spleen is torn and peeling off the spleen, but parenchymal damage is limited, either an omental patch or mesh wrap can be used (Fig. 12.8). For mesh, we prefer knitted Vicryl mesh soaked in a fibrin gel sealant. The mesh is wrapped around the area of injury (Fig. 12.9a). The sealant allows the mesh to adhere to the parenchyma and provides some additional hemostatic effect. Vicryl sutures can be used to tighten the mesh to compress the spleen and provide further hemostasis (Fig. 12.9b). The argon beam can be useful to weld the mesh

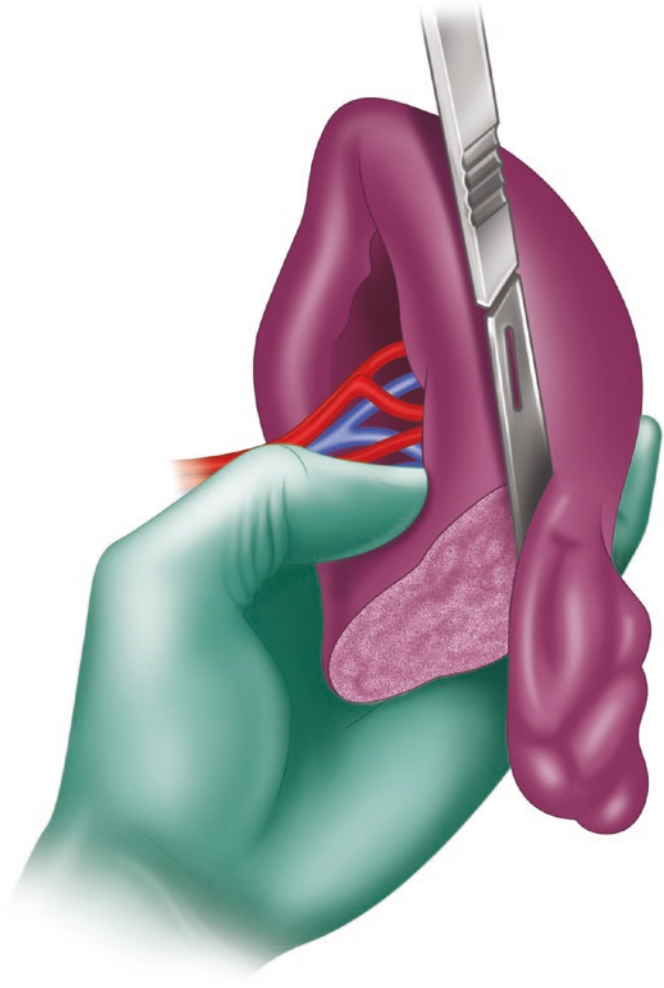


FIGURE 12.5 Partial splenectomy for splenic salvage. Manual pressure is used to maintain control until the cut end is hemostatic

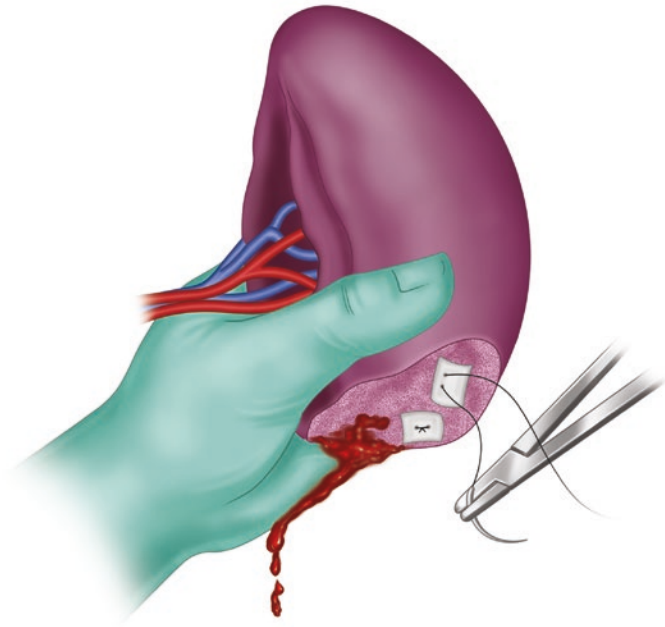


Figure 12.6 Mattress repair of the cut end of the spleen

to the spleen in some cases (Fig. 12.10). For very minor injuries, direct suture repair may work (Fig. 12.11).

Outcomes

Attributable mortality to splenic injury is not well reported, but with current management protocols, death from splenic injury should be exceedingly rare. Most patients that die succumb to hemorrhage from multiple injuries. In general, bleeding from the spleen should rarely be fatal unless the patient arrives in extremis from the field.

Overall, the success rate of nonoperative management is greater than 96% when all grade injuries are included. The salvage rate is significantly higher in trauma centers that perform a high volume of angioembolization as well, with 99%

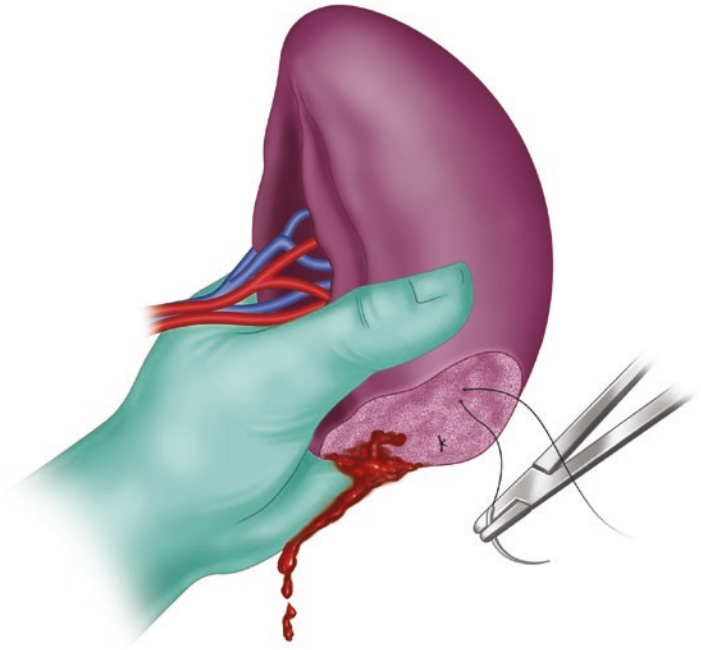


FIGURE 12.7 Direct repair of vessels of cut end of spleen

salvage rates reported [16]. This difference is more marked in higher-grade injury with high-volume angiography centers reporting salvage rates of 90% compared to salvage rates of 79% at low-volume angiography centers. Nonoperative management can be utilized for splenic salvage even in high-grade injuries as long as patients remain hemodynamically stable.

Complications

Complications of splenectomy include the usual complications of laparotomy such as incisional hernia and small bowel obstruction. Specific to splenectomy, one major risk is post-splenectomy sepsis. A recent analysis demonstrated that splenectomy as compared to splenic salvage was the most significant factor predicting infectious complications in the

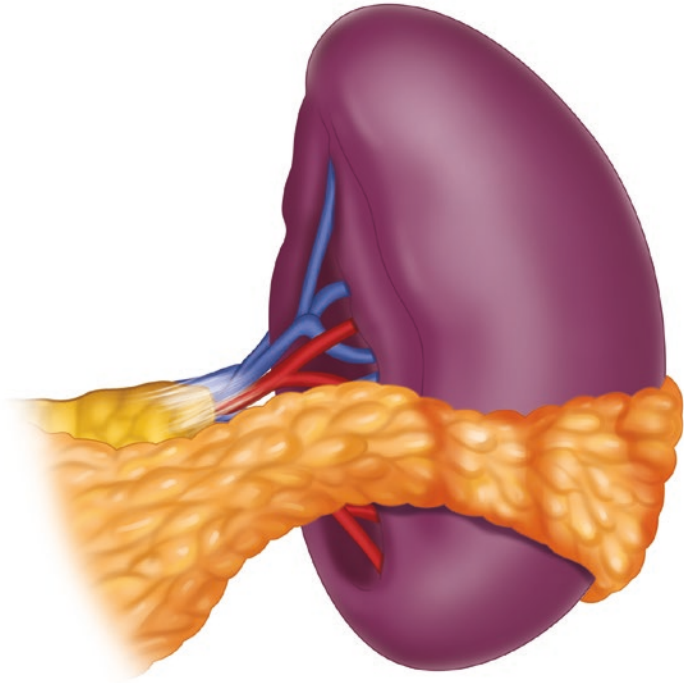


FIGURE 12.8 Omentum wrapped around small splenic injury

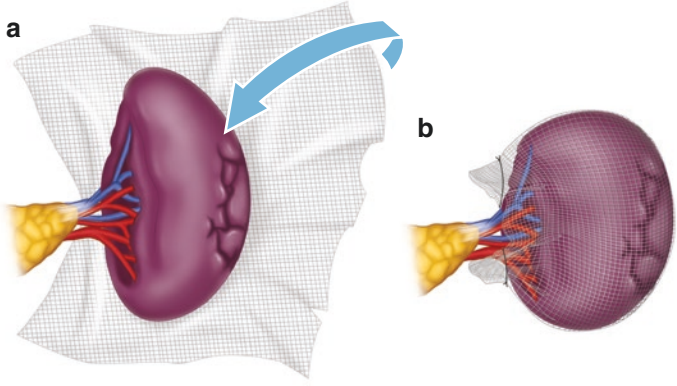


FIGURE 12.9 (**a**, **b**) Vicryl mesh wrap of the injured spleen

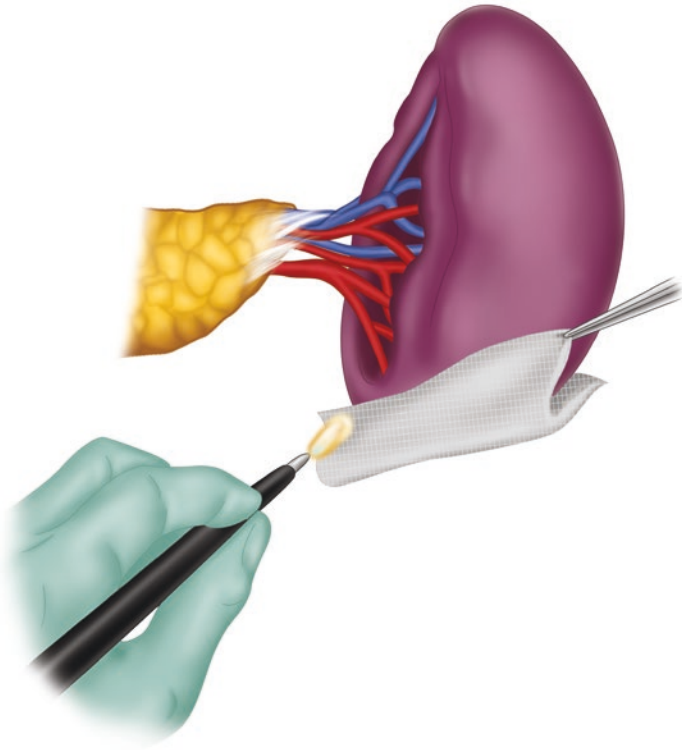


FIGURE 12.10 Argon beam coagulation of the spleen

hospital, even when controlling for injury severity and other confounders [39]. After discharge, patients must remain vigilant for signs of postsplenectomy overwhelming sepsis, as rates up to 3.2% have been described in patients after splenectomy. The mortality of sepsis is higher after splenectomy than the general population as well [40].

Though conservative management with angioembolization is associated with improved outcomes and fewer infectious complications, there are still many significant associated complications associated with this procedure. Patients can have procedural-associated bleeding, failure of therapy, pseudoaneurysms or hematomas of the access vessel, contrast-

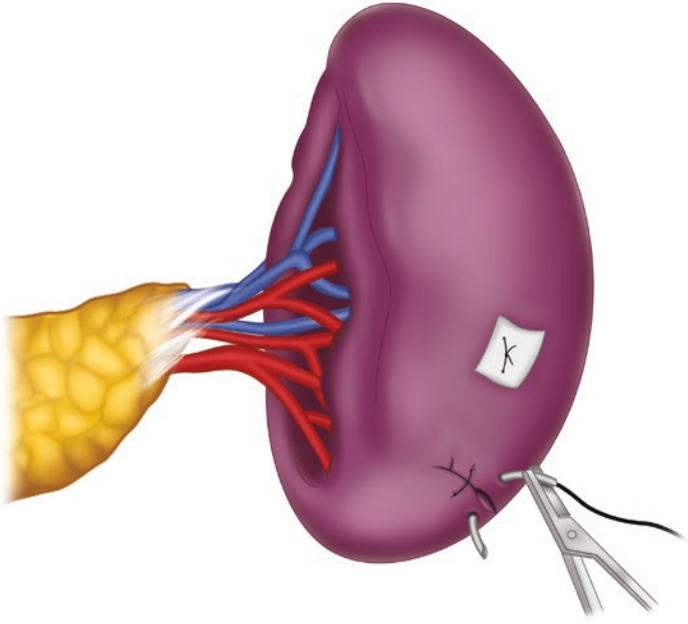


Figure 12.11 Suture repair with pledget of grade 1 splenic injuries

induced nephropathy, and complications of anesthesia. Up to 20% of patients may suffer major complications. One common complication is infection due to splenic infarction that may occur in up to 4% of patients who undergo angioembolization [41–43]. Splenic infarction may be managed conservatively unless the patient has severe pain or systemic signs of sepsis in which case splenectomy should be considered. Percutaneously placed drains may also be appropriate for localized infarction or abscess. The elderly, who may benefit the most from avoidance of laparotomy, may suffer from an even higher complication rate [44]. There may be differences in complications depending on the method of angioembolization used. Proximal coil embolization of the main splenic artery is associated with similar initial failure rate to selective distal embolization but may lead to fewer splenic infarcts [45] and fewer major complications [43].

In summary, while simple in anatomy, the spleen is difficult in concept. In appropriately selected patients, the benefits of nonoperative therapy usually outweigh the risks as complications of splenic salvage techniques tend to be less morbid than those of laparotomy. Conservative management remains the best option in stable patients, but care has to be taken to ensure that the care provider acknowledges when nonoperative management has failed to avoid serious morbidity or mortality from hemorrhage. Early splenectomy and hemorrhage control in the unstable patient is equally important for maximizing outcomes.

References

1. Peitzman AB, Heil B, Rivera L, Federle MB, Harbrecht BG, Clancy KD, et al. Blunt splenic injury in adults: multi-institutional study of the eastern association for the surgery of trauma. J Trauma. 2000;49:177–87.
2. Wilkens B. Historical review: the spleen. Br J Haematol. 2002;117:265.
3. McClusky DA, Skandalakis LJ, Colborn GJ, Skandalakis JE. Tribute to a triad: history of splenic anatomy, physiology, and surgery—part 1. World J Surg. 1999;23:311–25.
4. McClusky DA, Skandalakis LJ, Colborn GJ, Skandalakis JE. Tribute to a triad: history of splenic anatomy, physiology, and surgery—part 2. World J Surg. 1999;23:514–26.
5. King H, Shumacker HB. Splenic studies. I. Susceptibility to infection after splenectomy performed in infancy. Ann Surg. 1952;136:239–42.
6. Gerritsen GP, Madern GC. Conservative management of splenic trauma. Neth J Surg. 1980;32:62–5.
7. Boscak A, Shanmuganathan K. Splenic trauma: what is new? Radiol Clin N Am. 2012;50:105–22.
8. Boscak AR, Shanmuganathan K, Mirvis SE, Fleiter TR, Miller LA, Sliker CW, et al. Optimizing trauma multidetector CT protocol for blunt splenic injury: need for arterial and portal venous phase scans. Radiology. 2013;268:79–88.
9. Murken DR, Weis JJ, Hill GC, Alarcon LH, Rosengart MR, Forsythe RM, et al. Radiographic assessment of splenic injury without contrast: is contrast truly needed? Surgery. 2012;152:676–82.

10. Marmery H, Shanmuganathan K, Mirvis SE, Richard H 3rd, Sliker C, Miller LA, et al. Correlation of multidetector CT findings with splenic arteriography and surgery: prospective study in 392 patients. J Am Coll Surg. 2008;206:685–93.
11. Kozar RA, Crandall M, Shanmuganathan K, Zarzaur B, Coburn M, Cribari C, et al. Organ injury scaling 2018 update: spleen, liver, and kidney. J Trauma Acute Care Surg. 2018;85(6):1119–22.
12. Haan J, Ilahi ON, Kramer M, Scalea TM, Myers J. Protocol-driven nonoperative management in patients with blunt splenic trauma and minimal associated injury decreases length of stay. J Trauma. 2003;55:317–21.
13. Stassen NA, Bhullar I, Cheng JD, Crandall ML, Friese RS, Guillamondegui OD, et al. Selective nonoperative management of blunt splenic injury: an eastern association for the surgery of trauma practice management guideline. J Trauma Acute Care Surg. 2012;73:S294–300.
14. Olthof DC, van der Vlies CH, Joosse P, van Delden OM, Jurkovich GJ, Goslings JC, et al. Consensus strategies for the nonoperative management of patients with blunt splenic injury: a Delphi study. J Trauma Acute Care Surg. 2013;74:1567–74.
15. Bhangu A, Nepogodiev D, Lal N, Bowley DM. Meta-analysis of predictive factors and outcomes for failure of non-operative management of blunt splenic trauma. Injury. 2012;43:1337–46.
16. Banerjee A, Duane TM, Wilson SP, Haney S, O'Neill PJ, Evans HL, et al. Trauma center variation in splenic artery embolization and spleen salvage: a multicenter analysis. J Trauma Acute Care Surg. 2013;75:69–74.
17. Brault-Noble G, Charbit J, Chardon P, Barral L, Guillon F, Taourel P, et al. Age should be considered in the decision making of prophylactic splenic angioembolization in nonoperative management of blunt splenic trauma: a study of 208 consecutive civilian trauma patients. J Trauma Acute Care Surg. 2012;73:1213–20.
18. Bhullar IS, Frykberg ER, Siragusa D, Chesire D, Paul J, Tepas JJ 3rd, et al. Age does not affect outcomes of nonoperative management of blunt splenic trauma. J Am Coll Surg. 2012;214:958–64.
19. Trust MD, Teixeira PG, Brown LH, Ali S, Coopwood B, Aydelotte JD, et al. Is it safe? Nonoperative management of blunt splenic injuries in geriatric trauma patients. J Trauma Acute Care Surg. 2018;84:123–7.
20. Olthof DC, Joosse P, van der Vlies CH, de Haan RJ, Goslings JC. Prognostic factors for failure of nonoperative management

in adults with blunt splenic injury: a systematic review. J Trauma Acute Care Surg. 2013;74:546–57.
21. Capecci LM, Jeremitsky E, Smith RS, Philp F. Trauma centers with higher rates of angiography have a lesser incidence of splenectomy in the management of blunt splenic injury. Surgery. 2015;158:1020–4.
22. Rosenberg GM, Weiser TG, Maggio PM, Browder TD, Tennakoon L, Spain DA, et al. The association between angioembolization and splenic salvage for isolated splenic injuries. J Surg Res. 2018;229:150–5.
23. Bhular IS, Tepas JJ 3rd, Siragusa D, Loper T, Kerwin A, Frykberg ER. To nearly come full circle: nonoperative management of high-grade IV-V blunt splenic trauma is safe using a protocol with routine angioembolization. J Trauma Acute Care Surg. 2017;82(4):657–64.
24. Crichton JCI, Naidoo K, Yet B, Brundage SI, Perkins Z. The role of splenic angioembolization as an adjunct to nonoperative management of blunt splenic injuries: a systematic review and meta-analysis. J Trauma Acute Care Surg. 2017;83(5):934–43.
25. Haan J, Scott J, Boyd-Kranis R, Ho S, Kramer M, Scalea TM. Admission angiography for blunt splenic injury: advantages and pitfalls. J Trauma. 2001;51:1161–5.
26. Skattum J, Naess PA, Eken T, Gaarder C. Refining the role of splenic angiographic embolization in high-grade splenic injuries. J Trauma Acute Care Surg. 2013;74:100–3.
27. Haan JM, Bochicchio GV, Kramer N, Scalea TM. Nonoperative management of blunt splenic injury: a 5-year experience. J Trauma. 2005;58:492–8.
28. Bhullar IS, Frykberg ER, Siragusa D, Chesire D, Paul J, Tepas JJ 3rd, et al. Selective angiographic embolization of blunt splenic traumatic injuries in adults decreases failure rate of nonoperative management. J Trauma Acute Care Surg. 2012;72:1127–34.
29. Jeremitsky E, Kao A, Carlton C, Rodriguez A, Ong A. Does splenic embolization and grade of splenic injury impact nonoperative management in patients sustaining blunt splenic trauma? Am Surg. 2011;77:215–20.
30. Bhullar IS, Frykberg ER, Tepas JJ III, Siragusa D, Loper T, Kerwin AJ. At first blush: absence of computed tomography contrast extravasation in grade IV or V adult blunt splenic trauma should not preclude angioembolization. J Trauma Acute Care Surg. 2013;74:105–11.

31. Haan JM, Marmery H, Shanmuganathan K, Mirvis SE, Scalea TM. Experience with splenic main coil embolization and significance of new or persistent pseudoaneurysm: reembolize, operate, or observe. J Trauma. 2007;63:615–9.
32. Zarzaur BL, Kozar R, Myers JG, Claridge JA, Scalea TM, Neideen TA, et al. The splenic injury outcomes trial: an American Association for the Surgery of Trauma multi-institutional study. J Trauma Acute Care Surg. 2015;79(3):335–42.
33. Liu P, Liu H, Hsieh T, Huang CY, Ko SF. Nonsurgical management of delayed splenic rupture after blunt trauma. J Trauma Acute Care Surg. 2012;72:1019–23.
34. Haan JM, Boswell S, Stein D, Scalea TM. Follow-up abdominal CT is not necessary in low-grade splenic injury. Am Surg. 2007;73:13–8.
35. Eberle BM, Schnüriger B, Inaba K, Cestero R, Kobayashi L, Barmparas G, et al. Thromboembolic prophylaxis with low-molecular-weight heparin in patients with blunt solid abdominal organ injuries undergoing nonoperative management: current practice and outcomes. J Trauma. 2011;70:141–6.
36. Malhotra AK, Carter RF, Lebman DA, Carter DS, Riaz OJ, Aboutanos MB, et al. Preservation of splenic immunocompetence after splenic artery angioembolization for blunt splenic injury. J Trauma. 2010;69:1126–30.
37. Skattum J, Naess PA, Gaarder C. Non-operative management and immune function after splenic injury. Br J Surg. 2012;99(Suppl 1):59–65.
38. Skattum J, Titze TL, Dormagen JB, Aaberge IS, Bechensteen AG, Gaarder PI, et al. Preserved splenic function after angioembolisation of high grade injury. Injury. 2012;43:62–6.
39. Demetriades D, Scalea TM, Degiannis E, Barmparas G, Konstantinidis A, Massahis J, et al. Blunt splenic trauma: splenectomy increases early infectious complications: a prospective multicenter study. J Trauma Acute Care Surg. 2012;72:229–34.
40. Bisharat N, Omari H, Lavi I, Raz R. Risk of infection and death among post-splenectomy patients. J Infect. 2001;43:182–6.
41. Haan J, Bochicchio G, Kramer M. Scalea TAir following splenic embolization: infection or incidental finding? Am Surg. 2003;69:1036–9.

42. Haan JM, Biffl W, Knudson MM, Davis KA, Oka T, Majercik S, et al. Splenic embolization revisited: a multicenter review. J Trauma. 2004;56:542–7.
43. Ekeh AP, Khalaf S, Ilyas S, Kauffman S, Walusimbi M, McCarthy MC. Complications arising from splenic artery embolization: a review of an 11-year experience. Am J Surg. 2013;205:250–4.
44. Wu S, Fu C, Chen R, Chen YF, Wang YC, Chung PK, et al. Higher incidence of major complications after splenic embolization for blunt splenic injuries in elderly patients. Am J Emerg Med. 2011;29:135–40.
45. Schnüriger B, Inaba K, Konstantinidis A, Lustenberger T, Chan LS, Demetriades D. Outcomes of proximal versus distal splenic artery embolization after trauma: a systematic review and meta-analysis. J Trauma. 2011;70:252–60.

Chapter 13
Pancreas and Duodenum Injuries: Techniques

David V. Feliciano

Introduction of the Problem

Injuries to the pancreas or duodenum are noted at only 5–6% and 1.5–11% of all laparotomies for trauma, respectively [1]. With their retroperitoneal location in the upper abdomen, associated injuries to the gastrointestinal tract and to upper abdominal vessels are common. Operative repairs are complicated by these factors in addition to the shared blood supply of the head of the pancreas and C-loop of the duodenum.

Once associated vascular and gastrointestinal repairs are completed, management of the injured pancreas is accomplished by drainage only or resection in almost all patients. Injuries to the duodenum are managed with closure, Roux-en-Y duodenojejunostomy, or resection. In patients with combined pancreatoduodenal or complex duodenal injuries, a proximal diversion procedure such as pyloric exclusion with gastrojejunostomy should still be considered.

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Pancreas

Surgical Anatomy

The retroperitoneal pancreas has a thin mesothelial capsule and is 15–20 cm in length, 1.0–1.5 cm in thickness, and approximately 3.0 cm in width. It crosses the abdomen at the level of the first and second lumbar vertebrae.

The main pancreatic duct of Wirsung passes through the entire length of the pancreas just above a line halfway between the superior and inferior edges. It enters the left side of the common bile duct in the head of the pancreas to form the ampulla of Vater.

The vascular anatomy of the upper abdomen complicates management of pancreatic injuries. There is a shared blood supply of the head of the pancreas and the C-loop of the duodenum through the anterior and posterior branches of the superior and inferior pancreaticoduodenal arteries. Therefore, a major injury to either organ to the right of the spine usually mandates a combined resection.

Penetrating wounds to the head of the pancreas may involve the underlying inferior vena cava, the right renal vessels, and the left renal vein as it enters the cava. In similar fashion, injuries to the neck and uncinate process are in proximity to the inferior pancreaticoduodenal artery, superior mesenteric vein, and superior mesenteric artery. Finally, the body of the pancreas lies over the visceral portion of the abdominal aorta, while the body and tail encircle or are intimately adherent to the splenic artery and vein.

Clinical Presentation and Diagnosis

With blunt trauma, a direct blow, the lower rim of the steering wheel, or a misplaced lap seat belt can cause compression of upper abdominal viscera and vessels against the spine and is the most common mechanism of injury to the pancreas. Even with transection over the spine at the level of the neck of the pancreas, the patient may have only modest epigastric pain and tenderness

because of its retroperitoneal location. The initial serum amylase is neither sensitive nor specific enough to confirm or rule out an injury to the pancreas. For example, salivary amylase elevations related to acute alcohol intoxication account for a significant percentage of the hyperamylasemia seen on admission to trauma centers. An elevated serum amylase level, particularly one drawn 3 hours after trauma, does warrant further investigation. In the modern era, a 128-slice multidetector abdominal CT scan with intravenous contrast is the diagnostic modality of choice. CT findings suggestive or diagnostic of an injury to the pancreas are listed in Box 13.1 [2]. An injury noted on multidetector CT or at the time of laparotomy is described by using the Pancreatic Organ Injury Scale (OIS) of the American Association for the Surgery of Trauma (Table 13.1) [3].

Box 13.1 CT Findings Suggestive or Diagnostic of a Pancreatic Injury

Suggestive of injury
Fluid in the lesser sac
Fluid between pancreas and splenic vein
Hematoma of transverse mesocolon
Thickening of left anterior renal fascia
Duodenal hematoma or laceration
Injury to spleen, left kidney, and left adrenal gland
Chance (transverse) facture of lumbar spine, especially in a child
Diagnostic of injury
Parenchymal hematoma or laceration
Obvious transection of the parenchyma/duct with fluid in the lesser sac
Disruption of the head of pancreas
Diffuse swelling characteristic of posttraumatic pancreatitis

Used with permission of Wolters Kluwer from Feliciano [2]

Table 13.1 Pancreatic organ injury scale of the American Association for the Surgery of Trauma

Grade[a]	Type of injury	Description of injury	ICD-9[b]	AIS 90
I	Hematoma	Minor contusion without duct injury	863.81–863.74	2
	Laceration	Superficial laceration without duct injury		2
II	Hematoma	Major contusion without duct injury or tissue loss	863.81–863.74	2
	Laceration	Major laceration without duct or injury or tissue loss		3
III	Laceration	Distal transection or parenchymal injury with duct injury	863.92–863.94	3
IV	Laceration	Proximal[c] transection of parenchymal injury involving ampulla	863.91	4
V	Laceration	Massive disruption of pancreatic head	863.91	5

Used with permission of Wolters Kluwer from Moore et al. [3]
[a]Advance one grade for multiple injuries up to Grade III
[b]863.51, 863.91, head; 863.99, 862.92, body; 863.83, 863.93, tail
[c]Proximal pancreas is to the patient's right of the superior mesenteric vein

If a CT is not available or the result is equivocal (unclear if ductal injury is present, with or without associated hyperamylasemia), an endoscopic retrograde cholangiopancreatogram (ERCP) or magnetic resonance cholangiopancreatogram (MRCP) is appropriate [4].

Nonoperative Management

In the absence of associated intra-abdominal injuries mandating an emergent or urgent laparotomy, a blunt OIS Grade I or II pancreatic injury on CT is managed nonop-

eratively. Such contusions or lacerations would be expected to heal, though an occasional patient may develop a peripancreatic fluid collection or pseudocyst. A follow-up abdominal CT scan is appropriate if the patient develops new-onset epigastric pain or hyperamylasemia while being observed.

Operative Exposure

Once areas of hemorrhage are repaired or ligated and gastrointestinal perforations or ruptures are repaired or resected, the entire pancreas should be exposed. Medial mobilization of the distal ascending colon and hepatic flexure is performed first. This is followed by an extensive Kocher maneuver (to the superior mesenteric vein) which will allow for visualization of the anterior and posterior aspects of the head and the anterior aspect of the neck of the pancreas. The anterior aspect of the body is visualized after division of the gastrocolic omentum. Division of the retroperitoneal attachments to the inferior border of the pancreas while carefully avoiding the inferior mesenteric vein is then performed. Bimanual gentle elevation of the inferior border of the pancreas will allow for visualization of the posterior body. Finally, division of the lienorenal and splenocolic ligaments will allow for elevation and medial mobilization of the spleen. In the absence of a history of pancreatitis, the tail of the pancreas can be elevated, as well, by sweeping the surgeon's right hand under the pancreas toward the midline.

When the thin mesothelial capsule of the pancreas has a "hematoma" underneath it, the capsule should always be opened. A complete transection of the pancreas may be under the hematoma if the retroperitoneum has not been disrupted by the blunt trauma. A penetrating wound of the pancreas should be exposed in the same fashion to see what its relationship is to the likely area of the duct of Wirsung.

A transection or injury to the duct of Wirsung is suggested by all the operative findings listed in Box 13.2. On occasion,

overt signs are lacking, but the surgeon is still concerned that an injury to the main duct is present. Any clot or loose pancreatic tissue is first debrided away. Then, the surgeon looks for leakage of clear pancreatic fluid from the area of injury using magnifying loupes for 3–5 minutes. Some textbooks recommended injecting the patient with 1 mg/kg of secretin to stimulate pancreatic secretion during the period of observation.

Box 13.2 Operative Findings Suggestive of Injury to the Duct of Wirsung

Extensive fat necrosis in the lesser sac
Leakage of clear fluid from area of injury
Central perforation from gunshot wound
Blunt laceration involving half the width of the gland
Significant disruption of the parenchyma at any location

A routine intraoperative cholangiopancreatogram or one using methylene blue dye is another option to assess the integrity of the duct of Wirsung [5]. A #5 pediatric feeding tube is inserted into the cystic duct. After the anesthesiologist injects fentanyl to cause spasm of the sphincter of Oddi, standard cholangiogram contrast or 1 ampule of methylene blue solution in 200 ml normal saline is injected through the feeding tube. The spasm should cause reflux of the dye into the pancreatic duct and, when methylene blue is injected, staining and leakage at the site of a ductal injury. The obvious disadvantage of the approach is the need for a cholecystectomy after the cystic duct has been opened to perform the cholangiopancreatogram. Performing an intraoperative pancreatogram after a distal pancreatectomy of normal pancreas or after a duodenotomy and cannulation of the pancreatic duct is rarely performed in the modern era as they add further injury.

Operative Management: Factors in Repair

Hypotension and physiologic exhaustion secondary to associated injuries are the most important factors in operative management. Once peripancreatic hemorrhage is controlled, no other operative procedure on the pancreas is necessary at a "damage control" operation.

A delay in diagnosis is another factor in choice of repair as autodigestion, pancreatic fistulas, and suture line leaks are more likely to occur in the postoperative period. After a delayed repair or resection, consideration should be given to inserting a feeding jejunostomy tube and extra peripancreatic drains as well as initiating empiric subcutaneous injections of octreotide. The value of octreotide is only that it may decrease the daily volume of any pancreatic fistula that might occur.

In hemodynamically stable patients with an early diagnosis and operation, the most important factor in choosing drainage only, resection, Roux-en-Y reconstruction, and/or diversion is whether the duct of Wirsung has been injured or not [1, 2]. Ductal transection mandates resection in most patients in the modern era (see below).

Finally, a combined pancreatoduodenal injury increases the complexity of management and, possibly, increases the fistula rate from either repair.

Operative Management: OIS Grade I–II Injury

An OIS Grade I injury noted at laparotomy does not need to be drained. With an OIS Grade II "major laceration," one option is to fill the laceration with a viable omental plug held in place with an absorbable suture. While there are no data proving the value of this adjunct, theoretic benefits (based on its use in hepatic trauma) include the following: (1) tamponade of venous bleeding, (2) lower incidence of postoperative pancreatic fistula, and (3) bring mobile macrophages to the area of injury. Again, there are no clear-cut data regarding the need to drain OIS Grade II pancreatic injuries.

Operative Management: OIS Grade III Injury

Selected patients with isolated pancreatic ductal transection have been treated with endoscopic insertion of a temporary stent for over 30 years. Otherwise, transection of the duct of Wirsung over or to the left of the superior mesenteric vein is almost always treated with a distal pancreatectomy with splenectomy in adults (Fig. 13.1). The splenic artery and vein are suture ligated with 4-0 polypropylene 2 cm proximal to where the duct has been injured or where the pancreas is to be divided. Along with coverage of the stumps of the vessels with a viable omental pedicle, separate sites of division and ligation isolate the stumps from a distal pancreatic fistula occurring in the postoperative period.

A variety of surgical techniques have been used over the years by elective and trauma pancreatic surgeons to lower rates of postoperative pancreatic fistulas after distal resection. These have included the following: (1) fishmouth central beveled resection and posterior to anterior suture closure with or without ligation of the pancreatic duct, (2) use of TIA or

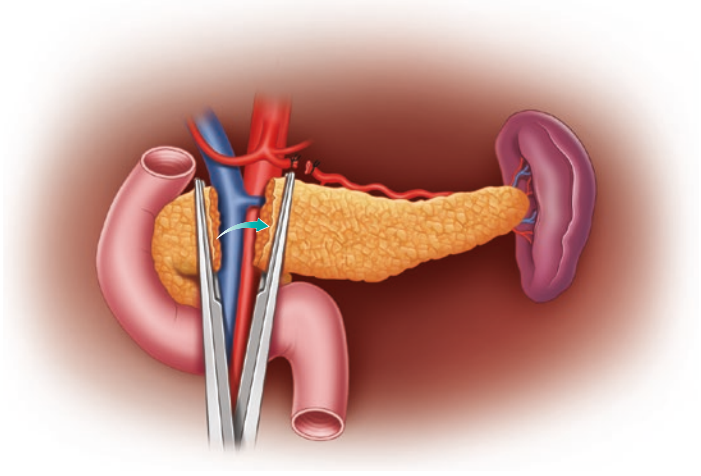

Figure 13.1 Distal pancreatectomy with splenectomy is performed for most Grade III or IV pancreatic injuries

endovascular stapler, (3) suture closure buttressed with fibrin glue, omental patch, or falciform mesothelial membrane [6, 7].

Large retrospective and prospective studies on elective and trauma pancreatectomies, some using the 2005 International Study Group on Pancreatic Fistula Definition, have now been performed [8]. A 28–32% rate of fistulas has been consistent no matter which type of closure is performed [6, 7, 9]. A Jackson-Pratt or Blake drain is always inserted after a distal pancreatectomy. Because of the importance of splenic immunity in children, a distal pancreatectomy is performed without splenectomy. This is accomplished by using 4-0 ties on the splenic vessel side and small clips on the pancreatic side of the multiple small vessels connecting these structures.

One of the issues with a distal resection after an OIS Grade III injury is that it is actually a 70–80% pancreatectomy (Fig. 13.2). This is related to the location of the neck of the pancreas, the most common site of blunt transection, over the spine. It is important to inform patients after this operation how much of the exocrine and islet cell masses

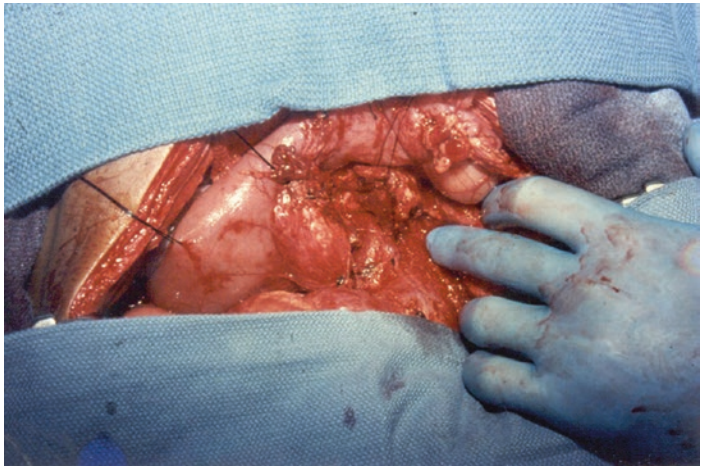

Figure 13.2 If this Grade IV pancreatic injury is treated with distal resection, it will be a 90% pancreatectomy and splenectomy. (Used with permission of McGraw-Hill Education from Feliciano [10])

has been resected and the risks of alcoholism and obesity as they age.

Operative Management: OIS Grade IV Injury

There are two options to manage the rare transection of the duct of Wirsung to the right of the superior mesenteric vein. A subtotal or 80–90% pancreatectomy and splenectomy in an adult is complicated by the need to close over the bulky proximal stump in the head-neck junction or head of the pancreas. Closure of this stump is usually with sutures as standard staplers may be too narrow to fit around it.

In highly selected patients who are hemodynamically stable and have no or few associated injuries, a Letton-Wilson procedure can be performed for an OIS Grade III or IV injury [11] (Fig. 13.3). Originally performed in 1957 at Grady

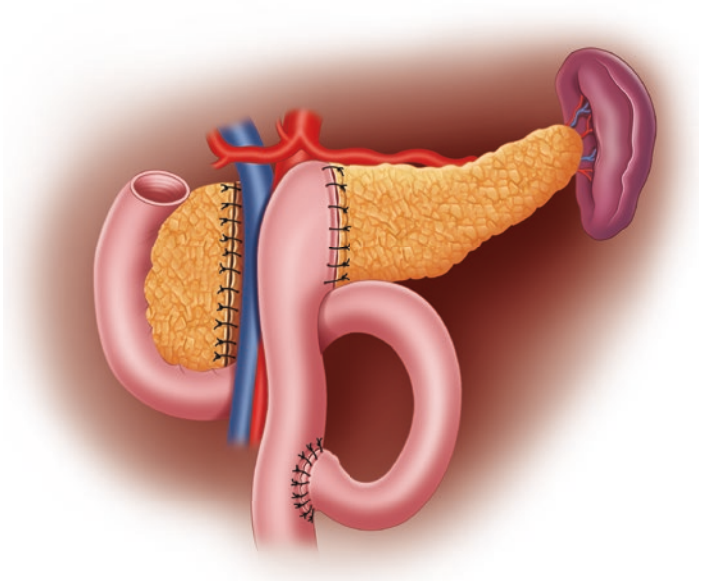

Figure 13.3 Letton-Wilson procedure for highly selected patients with proximal Grade III or Grade IV pancreatic injuries

Memorial Hospital in Atlanta, Georgia, the first step in the procedure is ligation of the exposed duct of Wirsung in the open transected proximal pancreatic remnant (head or neck). This opened end is then oversewn or stapled in the surgeon's usual fashion. The next step is mobilization/elevation of 2 cm of the opened end of the distal fragment (body and tail) off the splenic vessels. A 40-cm jejunal Roux limb is then created, passed through the right side of the transverse mesocolon, a 2-layer end-to-end distal pancreatojejunostomy is performed, and the Roux limb is fixated in the mesocolon with circumferential sutures. Finally, the standard end-to-side jejunojejunostomy is performed, and closed suction drains are inserted.

Operative Management: OIS Grade V Injury

"Massive disruption" of the head of the pancreas, devascularization of the C-loop of the duodenum, or destruction of the ampulla of Vater mandates a pancreatoduodenectomy. The timing will depend on the patient's hemodynamic status, physiologic state, and the magnitude of associated injuries. In certain patients with blunt compression injuries, the trauma itself has "performed the Whipple," i.e., the head of the pancreas and C-loop of the duodenum have been devascularized and are already partially separated from surrounding structures. A patient with any of these injuries in the presence of physiologic exhaustion can have a delayed resection, followed by reconstruction at a first reoperation. The most significant disadvantage to delaying the Whipple resection itself or the reconstruction is the marked edema and swelling of the midgut over the next 3–5 days. As for techniques of reconstruction, a pylorus-preserving Whipple procedure can be performed depending on the extent of pancreatoduodenal trauma.

Summary of Operative Management (Table 13.2)

In large reviews in the past, drainage was performed 73% of the time, resection 17%, and exclusion procedures or a Whipple procedure 3.5% [12].

Table 13.2 Modern operative management of pancreatic trauma

Injury	Technique
AAST OIS Grade I–II injury	Closed suction drainage, consider viable omental plug to defect
AAST OIS Grade III injury	Distal pancreatectomy with closure using 4.8 mm staples or sutures
AAST OIS Grade IV injury (hemodynamically stable)	Oversew proximal stump, Roux-en-Y distal end-to-end pancreatojejunostomy
AAST OIS Grade V injury	Whipple procedure with delayed reconstruction if necessary
Combined head of pancreas and duodenal injuries (Whipple not justified)	Closed suction drainage of pancreas, duodenal repair, consider pyloric exclusion with gastrojejunostomy

Used with permission of Southeastern Surgical Congress from Feliciano [1]

Complications and Results

In older large reviews, the incidence of postoperative pancreatic fistulas with all grades of injury has ranged from 3% to 17% with a mean of 6%; however, trauma centers have used a variety of definitions of this complication over time. In one recent series of distal pancreatectomies performed for trauma, pancreatic fistulas occurred in 29% of patients [9]. This is the exact same figure for the incidence of fistulas after elective distal pancreatectomies. The second most common complication is a postoperative intra-abdominal abscess, with a range of 5–18% and a mean of 5%.

Mortality after pancreatic trauma is obviously affected by the mechanism of injury, magnitude of the pancreatic injury, number and magnitude of associated injuries, and the patient's hemodynamic status on admission. Overall mortality is approximately 20% with the following based on mechanism of injury: stab wound 2.8–5%, gunshot wound 15.4–22%, and blunt trauma 16.9–19% [2].

Duodenum

Anatomy

The duodenum is a retroperitoneal organ 30 cm ("12 finger-breadths" according to Herophilus in ancient Greece) in length and has four anatomical sections. The short superior duodenum (D_1) starts beyond the pylorus of the stomach and extends to where it crosses over the gastroduodenal artery and common bile duct in the hepatoduodenal ligament. This is the most common area for anterior perforations and posterior bleeding from acid-peptic-Helicobacter ulcers. The descending duodenum (D_2) extends from the hepatoduodenal ligament to the ampulla of Vater, while the transverse duodenum (D_3) continues over to the superior mesenteric vein and artery. The ascending duodenum (D_4) then continues over to the duodenojejunal junction at the ligament of Treitz.

The main ventral pancreatic duct of Wirsung enters the common bile duct to form the ampulla of Vater as previously noted. This then empties through the major papilla in the medial duodenal wall at the junction of D_2–D_3 and is readily palpable through a duodenotomy. The accessory dorsal pancreatic duct of Santorini enters D_2 at the minor papilla in the proximal medial duodenal wall in 70% of individuals.

The vascular anatomy of the duodenum is similar to that of the head of the pancreas. The arterial supply of the upper half of the duodenum is from the superior pancreaticoduodenal artery, a branch of the gastroduodenal artery. The arterial supply of the lower half of the duodenum is from the inferior pancreaticoduodenal artery, a branch of the superior mesenteric artery.

Penetrating wounds to D_2–D_3 may involve the underlying inferior vena cava, the right renal vessels, and the left renal vein as it enters the cava. Wounds to D_3–D_4 may involve the underlying visceral abdominal aorta and the left renal vessels.

Clinical Presentation and Diagnosis

While the duodenum is held in place by its anatomic attachments and the retroperitoneum, its two main points of fixation are at the pyloroduodenal and duodenojejunal junctions. Therefore, it has long been hypothesized that angling or closure of these in a deceleration-type motor vehicle crash might explain the "blowout" injuries seen in D_2 or D_3. And, similar to the pancreas, the duodenum can be crushed by a direct blow, the lower rim of the steering wheel, or a misplaced lap seat belt against the spine.

A submucosal or subserosal hematoma from a direct blow, such as a handlebar injury in a child, may cause complete duodenal obstruction. The patient may have moderate epigastric pain without peritonitis but then develops persistent vomiting within 6–12 hours. A 128-multidetector CT of the abdomen will document the presence of the hematoma in D_2 or D_3. If gastrointestinal contrast is used, a "coiled spring" sign or complete obstruction will be visualized.

Blunt ruptures of the duodenum as described above may not be obvious on the initial physical examination if the retroperitoneum remains intact. The bacterial count of the duodenum is low, and pancreatic bicarbonate tends to neutralize gastric acid, so "retroperitonitis" tends to be masked in the first hours after injury. In the days before multidetector CT, an abdominal X-ray was performed on patients without peritonitis after blunt trauma. One of the goals was to see if there was a "sliver" of retroperitoneal air or other accumulation of gas outlining the C-loop of the duodenum – a radiologic sign pathognomonic of a blunt duodenal rupture. In the modern era, this abnormal collection of air would be noted on the initial abdominal CT. Confirmation of the rupture, if felt to be necessary, would be by adding gastrointestinal contrast.

All other patients undergoing emergent or urgent laparotomies after trauma will have the duodenum exposed and explored at that time. An injury noted on multidetector CT or at the time of laparotomy is described by using the Duodenum OIS of the American Association for the Surgery of Trauma (Table 13.3) [3].

Table 13.3 Duodenum organ injury scale of the American Association for the Surgery of Trauma

Grade[a]		Description of injury[b]	ICD-9	AIS 85	AiS-90
I	Hematoma	Involving single portion of duodenum	863.21	2	2
	Laceration	Partial thickness, no perforation	863.21	2	3
II	Hematoma	Involving more than one portion	863.21	2	2
	Laceration	Disruption <50% of circumference	863.31	3	4
III	Laceration	Disruption 50–75% circumference of D2	863.31	4	4
		Disruption 50–75% circumference of D1, D3, D4		4	4
IV	Laceration	Disruption >75% circumference of D2	863.31	4	5
		Involving ampulla or distal common bile duct		4	5
V	Laceration	Massive disruption of duodenopancreatic complex	863.31	5	5
	Vascular	Devascularization of duodenum	863.31	5	5

Used with permission of Wolters Kluwer from Moore et al. [3]
D1 1st portion duodenum, *D2* 2nd portion duodenum, *D3* 3rd portion duodenum, *D4* 4th portion duodenum
[a]Advance one grade for multiple injuries to the same organ
[b]Based on most accurate assessment at autopsy, laparotomy, or radiologic study

Nonoperative Management

In the absence of associated intra-abdominal injuries mandating an emergent or urgent laparotomy, a blunt OIS Grade I duodenal hematoma diagnosed on CT is managed nonoperatively. A nasogastric tube and central intravenous line are inserted, and the patient is maintained on total parenteral nutrition. While textbooks describe waiting for up to 3 weeks for the obstructive symptoms to resolve, this is a long and expensive period for a patient, especially a child, to remain NPO in the hospital. Therefore, a repeat CT scan with gastrointestinal contrast is performed after 10–14 days in many centers to see if there has been any resolution of the hematoma. If the hematoma has not started to resolve at that point, a discussion should be held with the patient or parents. The options to be discussed would include continued observation, percutaneous drainage, or open or laparoscopic evacuation [13, 14].

Operative Exposure

Once areas of hemorrhage are repaired or ligated and other gastrointestinal perforations or ruptures are repaired or resected, the entire duodenum should be exposed. As with exposure of the head of the pancreas, medial mobilization of the distal ascending colon and hepatic flexure is performed first. To simplify performing an extensive Kocher maneuver in obese patients, 2–3 Babcock clamps are applied to D_2 and D_3 through the retroperitoneum. Elevation of these clamps by an assistant will allow for better visualization of the right lateral aspect of the C-loop of the duodenum as scissors are used to initiate the Kocher maneuver. Continued mobilization of the hepatic flexure inferiorly and elevation of D_2–D_3 superiorly will allow for a complete Kocher maneuver. In obese individuals, it is helpful to remember that the middle colic vein can be used as a marker to where the superior mesenteric vein lies inferior to the pancreas. The Kocher maneu-

ver allows for visualization of anterior D_1 and anterior and posterior D_2 and a portion of D_3.

The next step is division of the ligament of Treitz medially to mobilize the duodenojejunal junction until the fingers enter the space to the right where the Kocher maneuver was performed. This will allow for complete visualization of D_4 and reasonable visualization of D_3 (anterior wall can be visualized, posterior wall palpated) [15]. On rare occasions, to improve visualization for a repair of D_3, the Cattell-Braasch maneuver described in 1960 is used [16]. The cecum, ascending colon, and hepatic flexure are mobilized to the midline. Then, the retroperitoneal attachments of the mesentery of the small bowel are sharply divided from the right lower quadrant to the duodenojejunal junction. Evisceration of the small bowel to the left allows for complete visualization of D_3.

Operative Management: Factors in Repair

Much as with injuries to the pancreas, hypotension and physiologic exhaustion secondary to associated injuries are the most important factors in operative management. The difference with duodenal injuries is that an effort should be made to temporarily or permanently close the hole in the duodenum at the first "damage control" procedure. This is appropriate even if the repair results in significant narrowing. This decreases contamination and inflammation in an immunocompromised patient who will need more laparotomies.

A delay in diagnosis of a duodenal rupture or perforation for 24 hours, a rare event in the modern era, led to a mortality of 40% in the past [17]. This was due to autodigestion of the retroperitoneum and difficult duodenal repairs due to edema and eversion of exposed mucosa at the time of diagnosis.

In hemodynamically stable patients, the most important factor in choosing suture repair, Roux-en-Y reconstruction, or resection is whether or not there has been a significant loss of tissue from the duodenal wall. As surgeons who performed open biliary/pancreatic sphincteroplasty in the past learned,

it is surprisingly easy to narrow D_2–D_3 with a longitudinal suture line.

And, as previously noted, a combined pancreatoduodenal injury is obviously a more complex injury than a duodenal injury alone and has a greater potential for postoperative complications.

Operative Management: OIS Grade II and III and Selected Grade IV Injuries (Table 13.4) [1]

The duodenum has reasonable mobility except medially and an excellent blood supply. Therefore, repairs of isolated blunt or penetrating injuries without loss of a portion of the wall will have a leak rate of only 1–3%. A routine antimesenteric blunt rupture in D_2 or D_3 is closed in a transverse or oblique fashion with an inner full-thickness row of 3-0 absorbable suture and an outer seromuscular row of 3-0 silks. Even with a near transection, an end-to-end repair can be performed as long as the major papilla on the medial wall is not involved.

Table 13.4 Modern operative management of duodenal trauma

Injury	Technique
AAST OIS Grade II–III injuries	Primary transverse or oblique 2-layer repair
AAST OIS Grade II–III injuries with loss of tissue	Retrocolic Roux-en-Y side-to-end duodenojejunostomy
AAST OIS Grade IV (with ampulla)–V injuries	Whipple procedure with delayed reconstruction if necessary
Narrowed or discolored duodenal repair or combined head of pancreas and duodenal injury (Whipple not justified)	Duodenal repair, closed suction of pancreas, consider pyloric exclusion with gastrojejunostomy[a]

Used with permission of Southeastern Surgical Congress Feliciano [1]

[a]Exclusion performed with #1 polypropylene suture. Patient screened for *Helicobacter pylori* and undergoes upper gastrointestinal X-ray series before discharge

A routine antimesenteric bullet hole is closed in the same fashion, while adjacent holes are connected and closed in a transverse or oblique direction. When either the entrance or exit wound involves the medial wall, the antimesenteric perforation is extended in a transverse direction. This will allow for visualization and palpation of the major papilla to verify that it is not injured. If the papilla/ampulla complex is not injured, but the pancreas is visible through the medial hole, an attempt should be made to close this with a single layer of interrupted 3-0 absorbable sutures. Even if a postoperative pancreatic fistula develops through this duodenal repair, it should drain internally into the duodenal lumen. Some surgeons then cover a long primary repair with a viable omental pedicle as a buttress. Data on the value of this adjunct are lacking, but it can be easily performed in less than 5 minutes.

A more complex repair is needed in a patient with loss of a portion of the anterior, antimesenteric, or posterior wall. The same would be true in a patient with significant narrowing or an early leak after a rapid repair at a prior "damage control" laparotomy. When there is not an associated injury to the pancreas or papilla/ampulla complex, any previous sutures are removed and the injured edges of the duodenal defect are debrided back to bleeding tissue. A 40-cm jejunal Roux limb is created, passed through the right side of the transverse mesocolon, and a 2-layer side-to-end duodenojejunostomy is performed. Once again, the Roux limb is fixated in the mesocolon with circumferential sutures. Finally, the jejunojejunostomy is performed. This mucosa-to-mucosa repair is much preferred to the jejunal serosal patch described in older publications [18].

On occasion, the surgeon is concerned that the repair of the duodenum is compromised but a major resection is not indicated. Examples in D_2 or D_3 would include the following: (1) narrowed repair, (2) discolored or bruised repair, and (3) delayed repair in the presence of retroperitoneal autodigestion and mucosal eversion. The addition of a proximal diversion procedure such as pyloric exclusion with gastrojejunostomy should be considered (Fig. 13.4a, b). This procedure was first

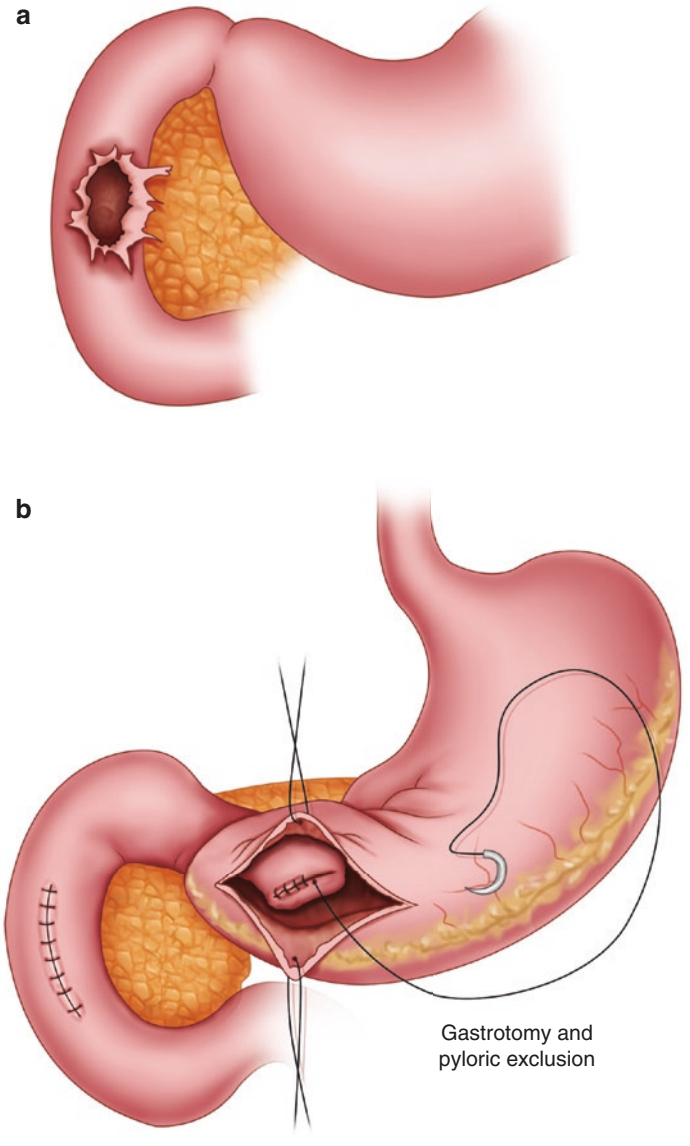

Figure 13.4 (**a**, **b**) Pyloric exclusion performed through dependent gastrotomy with #1 polypropylene suture

described by Albert A. Berg at Mt. Sinai Hospital in New York City in 1907 for patients with duodenal fistulas [19]. It was later revivified at Ben Taub General Hospital in Houston in a 1977 publication [20]. The first step is to skeletonize approximately 10 cm of the dependent greater curve starting within 6–10 cm of the pylorus. A dependent gastrotomy is made through the skeletonized area, and the pyloric muscle ring (not the prepyloric antrum) is grabbed at 12 and 6 o'clock with Babcock clamps. Two rows of #1 Prolene suture are placed deep into the pyloric muscle ring to complete the exclusion. An antecolic gastrojejunostomy is then sutured or stapled at the site of the gastrotomy. Prior to discharge from the hospital, the patient is screened for the presence of *Helicobacter pylori* and treated as needed. Also, an upper gastrointestinal contrast X-ray is performed to verify continued closure of the pylorus. The exclusion opens between 14 and 21 days in 94% of patients, so it is most helpful in minimizing output from duodenal fistulas in the first two postoperative weeks [21]. Several studies have been critical of pyloric exclusion with gastrojejunostomy over the past 12 years, but the fistula rate will always be higher as it is only used in high-risk duodenal repairs.

The use of a closed suction drain near a routine primary repair is a matter of personal preference for the trauma surgeon. Delayed, compromised, or complex repairs are almost always drained. The addition of a nasojejunal feeding tube or feeding jejunostomy tube after duodenal repair should be considered in patients with complex repairs, multiple associated injuries, or need for a prior "damage control" operation.

Operative Management: OIS Grade IV Injury Involving the Ampulla or Distal Common Bile Duct and Grade V Injury (Table 13.4) [1]

The indications for performing a pancreatoduodenectomy for the injuries listed are the same as those previously described for OIS Grade V injuries of the pancreas. There have been,

however, isolated reports of reimplantation of the transected ampulla back into the medial wall of the duodenum instead of a pancreatoduodenectomy over the years. Such a rare operation should only be performed with the assistance of an experienced hepatopancreatobiliary surgeon.

Complications and Results

In older large reviews, the incidence of postoperative duodenal fistulas with all grades of injury ranged from 4% to 16.6%, with a mean of 6.6% [22]. A review of 44 patients with "complex penetrating wounds" in 2014 noted a fistula rate of 33% [23]. A 19-year experience with 125 patients who survived longer than 24 hours after penetrating duodenal wounds in 2016 described a fistula rate of 8% [24].

Mortality after duodenal trauma in older series has ranged from 5.5% to 30%, with a mean of 17% [22]. In the two more recent series, mortality was 11.8% ("complex penetrating wounds") and 10.4%, respectively [23, 24]. Mortalities in a series of 147 patients in the National Trauma Data Bank comparing primary repair alone (#119) versus repair plus pyloric exclusion (#28) were 15.1% and 10.7%, respectively [25].

Combined Pancreatoduodenal Injuries

Even in the busiest urban centers with penetrating wounds accounting for 25–30% of all trauma admissions, only seven to ten patients with combined injuries will be treated each year. And, there is a wide spectrum of injuries.

Operative Management

The first principle of operative management is to treat geographically separate, but combined, injuries individually. An example would be a patient with multiple gunshot wounds and Grade III injuries to the mid-pancreas and to D_2. This patient should have a distal pancreatectomy and splenec-

tomy, a transverse repair of the duodenum, and insertion of a closed suction chain adjacent to the stump of the pancreas.

The second principle of operative management is to treat adjacent destructive injuries such as the aforementioned disruption of the head of the pancreas, devascularization of the C-loop of the duodenum, or destruction of the ampulla of Vater with a pancreatoduodenectomy.

The third principle of operative management is to consider adding a proximal diversion procedure such as a pyloric exclusion with gastrojejunostomy to treat adjacent nondestructive injuries.

Complication and Mortality

There were 108 patients surviving longer than 48 hours in the largest series in the American literature published in 1987 [26]. Postoperative complications included pancreatic fistulas (25.9%), intra-abdominal abscesses (16.6%), and duodenal fistulas (6.5%).

Overall mortality in the aforementioned series (129 patients) was 29.4%, while the mortality in the 13 patients undergoing an emergent pancreatoduodenectomy was 46.2% [26]. In two more recent reports on the outcome of pancreatoduodenectomies after trauma, mortality rates were 33% and 13% at Los Angeles County Hospital and Harborview Medical Center, respectively [27, 28].

References

1. Feliciano DV. Abdominal trauma revisited. Am Surg. 2017;83:1193–202.
2. Feliciano DV. Operative management of pancreatic trauma. In: Fischer JE, editor. Fischer's mastery of surgery. 7th ed. Philadelphia: Wolters & Kluwer; 2019. p. 1573–8.
3. Moore EE, Cogbill TH, Malangoni MA, Jurkovich GJ, Champion HR, Gennarelli TA, et al. Organ injury scaling II: pancreas, duodenum, small bowel, colon, and rectum. J Trauma. 1990;30:1427–9.

4. Subramanian A, Feliciano DV. Pancreatic trauma revisited. Eur J Trauma Emerg Surg. 2008;1:3–10.
5. Cortes V. Methylene blue pancreatography. In: Jacobs LM, editor. Advanced trauma operative management. Woodbury, CT: Cine-Med Publishing; 2010. p. 180–1.
6. Ferrone CR, Warshaw AL, Rattner DW, Berger D, Zheng H, Rawal B, et al. Pancreatic fistula rates after 462 distal pancreatectomies: staplers do not decrease fistulas rates. J Gastrointest Surg. 2008;12:1691–8.
7. Diener MK, Seiler CM, Rossion I, Kleeff J, Glanemann M, Butturini G, et al. Efficacy of stapler versus hand-sewn closure after distal pancreatectomy (DISPACT): a randomized, controlled multicenter trial. Lancet. 2011;377:1514–22.
8. Bassi C, Dervenis C, Buttirini G, Fingerhut A, Yeo C, Izbicki J, et al. Postoperative pancreatic fistula: an international study group (ISGPF) definition. Surgery. 2005;138:8–13.
9. Peck GL, Blitzer DN, Bulauitan G, Huntress LA, Truche P, Feliciano DV, et al. Outcomes after distal pancreatectomy for trauma in the modern era. Am Surg. 2016;82:526–32.
10. Feliciano DV. Abdominal trauma. In: Schwartz SI, Ellis H, editors. Maingot's abdominal operations. 9th ed. Norwalk, MA: Appleton & Lange; 1989. p. 457–512.
11. Letton AH, Wilson JP. Traumatic severance of pancreas treated by Roux-Y anastomosis. Surg Gynecol Obstet. 1959;109:473–8.
12. Cushman JG, Feliciano DV. Contemporary management of pancreatic trauma. In: Maull KI, Cleveland HC, Feliciano DV, et al., editors. Advances in trauma and critical care, vol. 10. Chicago: Mosby-Year Book; 1995. p. 309–36.
13. Gullotto C, Paulson EK. CT-guided percutaneous drainage of a duodenal hematoma. AJR. 2005;184:231–3.
14. Nolan GJ, Bendinelli C, Gani J. Laparoscopic drainage of an intramural duodenal hematoma: a novel technique and review of the literature. World J Emerg Surg. 2011;6:42–6.
15. Asensio JA, Demetriades D, Berne JD, Falabella A, Gomez H, Murray J, et al. A unified approach to the surgical exposure of pancreatic and duodenal injuries. Am J Surg. 1997;174:54–60.
16. Cattell RB, Braasch JW. A technique for exposure of the duodenum. Surg Gynecol Obstet. 1960;133:379–80.
17. Lucas CE, Ledgerwood AM. Factors influencing outcome after blunt duodenal injury. J Trauma. 1975;15:839–46.
18. Ivatury RR, Gaudino J, Ascer E, Nallathambi M, Ramirez-Schon G, Stahl WM. Treatment of penetrating duodenal injuries: pri-

mary repair vs. repair with decompressive enterostomy/serosal patch. J Trauma. 1985;25:337–41.
19. Berg AA. Duodenal fistula: its treatment by gastrojejunostomy and pyloric occlusion. Ann Surg. 1907;45:721–9.
20. Vaughan GD III, Frazier OH, Graham DY, Mattox KL, Petmecky FF, Jordan GL Jr. The use of pyloric exclusion in the management of severe duodenal injuries. Am J Surg. 1977;134:785–90.
21. Martin TD, Feliciano DV, Mattox KL, Jordan GL Jr. Severe duodenal injuries. Treatment with pyloric exclusion and gastrojejunostomy. Arch Surg. 1983;118:631–5.
22. Asensio JA, Feliciano DV, Britt LD, Kerstein MD. Management of duodenal injuries. Curr Probl Surg. 1993;30:1021–100.
23. Ordonez C, Garcia A, Parra MW, Scavo D, Pino LF, Millán M, et al. Complex penetrating duodenal injuries: less is better. J Trauma Acute Care Surg. 2014;76:1177–83.
24. Schroppel TJ, Saleem K, Sharpe JP, Magnotti LJ, Weinberg JA, Fischer PE, et al. Penetrating duodenal trauma: a 19-year experience. J Trauma Acute Care Surg. 2016;80:461–5.
25. DuBose JJ, Inaba K, Teixeira PGR, Shiflett A, Putty B, Green DJ, et al. Pyloric exclusion in the treatment of severe duodenal injuries: results from the National Trauma Data Bank. Am Surg. 2008;74:925–9.
26. Feliciano DV, Martin TD, Cruse PA, Graham JM, Burch JM, Mattox KL, et al. Management of combined pancreatoduodenal injuries. Ann Surg. 1987;205:673–80.
27. Asensio JA, Petrone P, Roldan G, Kuncir E, Demetriades D. Pancreaticoduodenectomy: a rare procedure for the management of complex pancreaticoduodenal injuries. J Am Coll Surg. 2003;197:937–42.
28. Thompson CM, Shalhub S, DeBoard ZM, Maier RV. Revisiting the pancreaticoduodenectomy for trauma: a single institution's experience. J Trauma Acute Care Surg. 2013;75:225–8.

Chapter 14
Stomach, Small Bowel, and Colon Injuries: Techniques

Carlos J. Rodriguez

Introduction of the Problem

The intra-abdominal gastrointestinal (GI) tract includes the stomach, small bowel, and colon. Injury to these organs can result from either blunt or penetrating trauma, although they are much more common in penetrating injury. Injuries include contusions, hematomas, and partial- or full-thickness injuries and can often involve the mesentery and underlying vasculature. Unrecognized injuries to the gastrointestinal tract carry the risk of intra-abdominal contamination, infectious complications, and morbidity and mortality. A study from the Eastern Association for the Surgery of Trauma (EAST) revealed an incidence of only 0.3% injury in more than 275,000 blunt trauma victims; however, in patients with any blunt abdominal trauma, 4–7% will have hollow viscus injury. The presence of solid organ injury increases the likelihood of concomitant hollow viscus injury [1].

Gunshot wounds (GSWs) violating the peritoneal cavity have a higher incidence (70% versus 30%) of hollow viscus injury than stab wounds (SWs). This is not surprising due to

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the greater force and pressure wave transmitted from higher-velocity penetrating injury. However, not all penetrating abdominal wounds require operative intervention. Citing similar outcomes, some centers have implemented structured processes that allow selective nonoperative management, vice mandatory operative exploration, of abdominal GSWs. These processes are predicated on the patient's hemodynamic normality, absence of peritonitis, adequate projectile path imaging, and neurologic status allowing surgeons to perform serial abdominal examinations [2–5]. Given the low energy and lack of blast wave associated with SW, nonoperative management of these wounds has enjoyed a wider, faster adoption rate. However, they are still predicated on the same criteria as nonoperative management of GSWs [6, 7]. Penetrating flank wounds can also cause gastrointestinal injuries and, due to the nature of injury patterns, may not cause immediate peritonitis. Triple-contrast CT has been advocated in these cases [8–12].

Given its compliance characteristics, gastric rupture from blunt trauma is relatively uncommon but can lead to large amounts of intraperitoneal contamination. This is most likely to occur when large amounts of energy are rapidly transferred to a patient, as in cases of pedestrians being struck by motor vehicles. Oftentimes, the force required to cause blunt gastric rupture is transmitted to other parts of the victim's body causing additional injuries. As such, the presence of blunt gastric rupture should prompt a thorough exploration of the entire abdomen [13].

Motor vehicle collisions (MVC) remain the most common cause of blunt small bowel rupture. While seat belts have clearly been shown to save lives, they are also more likely to lead to small bowel injury. In fact, studies have shown that the risk of small bowel injury is increased 4.38 times with the use of three-point restraints and up to 10 times with the use of lap belts. The classic "seat belt" sign (SBS) involves an ecchymosed abdominal wall following the shape of a seat belt with or without abdominal wall embarrassment. Historically, this physical exam finding has been found to be associated with a

significantly greater chance of abdominal and small bowel injury. A multi-institutional study found a 4.7% increase in risk of small bowel perforation after MVC if a seat belt sign was present [1, 14, 15].

Recently, in the air bag era, this dictum has been challenged where one retrospective study showed no association of a SBS and intra-abdominal injury. Interestingly, the results of this study showed that SBS was more associated with cervicothoracic injury. Other investigators have looked at clinically visible SBS, its depth on multi-detector computed tomography (MDCT) scan as a function of abdominal wall thickness, and the SBS relationship to the ASIS. These investigators found the presence of deeper SBS seen on MDCT and SBS cephalad to the ASIS was more predictive of surgically significant hollow viscus injury. SBS and its location on the abdominal wall should certainly heighten awareness of additional injury [16, 17].

Lumbar spine fractures, occurring with or without a seat belt sign, can result as the force of energy is transmitted posteriorly to the spine. These Chance fractures are often associated with hollow viscus injury, involving either the small bowel or colon.

Colonic injury is commonly seen as a result of penetrating abdominal trauma but only involved in less than 5% of blunt abdominal trauma [18]. During the Civil War, colon and rectal injuries carried a mortality rate of >90% and were typically managed expectantly. This strategy was challenged during the latter stages of World War I when surgeons began the practice of primary repair or exteriorization. These maneuvers decreased mortality rates to 60–75%.

As World War II progressed, the Office of the Surgeon General mandated colostomy for all colonic injuries secondary to unacceptably high morbidity and mortality rates. These maneuvers dropped the mortality rates to 22–35%. Antibiotics and other therapeutic advancements allowed primary repair to be considered an option in the early 1950s, and it was only in the 1970s that mandatory colostomy was challenged. A prospective randomized study by Chaping in 1999 concluded

there were no increases in complications with primary colonic repair after penetrating trauma. A meta-analysis by Nelson and colleagues in 2003, which included five prospective studies, also showed no differences in mortality between primary repair and colostomy for colonic injury [18, 19]. More recently, both the Eastern Association for the Surgery of Trauma (EAST) [20] and Western Trauma Association (WTA) [21] are suggesting primary repair or resection and anastomosis are acceptable strategies in almost all cases. Additionally, with the widespread adoption of damage control laparotomy and damage control resuscitation techniques, guidelines from both EAST and WTA advocate delayed anastomoses in patients who originally underwent abbreviated laparotomy secondary to physiologic derangement [22, 23].

History of Care of Stomach, Small Bowel, and Colon Injuries

Much of the early written work on GI injuries came as a result of military conflicts. Up until the late nineteenth century, the lack of adequate anesthesia, improper technique, and poor antiseptic measures often led to early patient demise.

The 25th president of the United States, William McKinley, was shot twice in the abdomen on the grounds of the Pan-American Exposition in Buffalo, New York, in late 1901. McKinley was rushed to a local hospital where he was given an injection of morphine and strychnine to ease his pain and ether for sedation. He underwent surgical exploration and was found to have an anterior and posterior gunshot wound to the stomach, which was primarily repaired. Unfortunately, he died several days later of gangrene and septic shock [24].

Improvements in mortality from GI injuries were first seen in World War II and the Korean and Vietnam wars. Triage, rapid transport, antiseptic technique, and management of specific intra-abdominal injuries all improved as a result of wartime experience and were subsequently adopted by civilian surgeons.

The application of skills learned in the military arena continues today as a result of the wars in Afghanistan and Iraq [25–27].

General Management

The initial approach to a patient with suspected gastrointestinal tract injury should begin with the primary survey and the expedient control of catastrophic hemorrhage. Measures to control catastrophic hemorrhage include direct pressure or tourniquets in the setting of extremity trauma to more advanced techniques of resuscitative endovascular balloon occlusion of the aorta (REBOA) in cases of noncompressible junctional or abdominopelvic hemorrhage. In an era of increasing dependence on radiographic imaging, the history and physical exam are often overlooked but can be helpful in guiding diagnostic and therapeutic decisions. Wounds should be noted and the abdomen inspected for signs of peritonitis. It is important to identify the anatomic location of entrance wounds and delineate between the abdomen, the flank, or the back. Visualizing the trajectory of penetrating injuries can help delineate injury pattern.

Standard lab values in the early stages of gastric, small bowel, and colonic injuries are of little diagnostic value. Markers of resuscitation, such as lactic acid and base deficit, correlate well with the adequacy of resuscitation or ongoing hemorrhage [28]. Lab values are more likely to help in the diagnosis of delayed hollow viscus injury presentation that can present after the first 24–72 h of admission. Peritonitis and hemodynamic lability are absolute indications for urgent exploratory laparotomy. If the patient's abdomen is tender but there is no peritonitis, serial examinations and radiographic studies can be considered.

The focused assessment with sonography for trauma (FAST) is now a standard diagnostic tool. It is helpful in identifying free fluid but is not very sensitive in diagnosing hollow viscus injury [29]. FAST has supplanted diagnostic peritoneal lavage or aspirate (DPL/DPA) as an initial means to diagnose

free fluid in the peritoneal cavity. However, DPL/DPA should remain in the toolbox of the acute care surgeon, especially in the setting of an equivocal FAST exam. Neither the FAST nor DPL/DPA will identify blood in the retroperitoneum. DPLs should be performed caudal to the umbilicus, and findings of succus entericus, stool, or organic matter are all signs of hollow viscus injury [30]. An alkaline phosphatase level in the DPL fluid of greater than 10 international units has a specificity of 99.8% and sensitivity of 94.7% for hollow viscus injury [31].

Computed tomography (CT) scan of the abdomen is used very often in patients with blunt trauma who are hemodynamically stable. Findings such as bowel wall thickening, lack of enhancement of the bowel wall on a contrast scan, mesenteric stranding or hematoma, free intraperitoneal fluid or contrast extravasation, pneumatosis, and pneumoperitoneum are all signs of possible hollow viscus injury [32, 33]. The overall sensitivity and specificity of CT for bowel injury have been shown to be as high as 88.3% and 99.4%, respectively [34]. Free intra-abdominal fluid can be misleading as it does not always indicate bowel injury and can occur as a result of large-volume resuscitation. Fahkry and colleagues found that only 29% of patients with free fluid in the abdomen had full-thickness bowel injury. Yet, another study found that 12% of patients with normal abdominal CT were subsequently found to have bowel injury [35].

In the absence of initial physical or radiologic findings necessitating immediate operative intervention, one particularly difficult injury to identify is the so-called "bucket-handle" injury. This occurs in blunt abdominal trauma when a shearing force applied to the abdominal wall causes the mesentery or mesocolon to separate from a short segment of the bowel wall serosa (Fig. 14.1). The small amount of bleeding that occurs with this shear can lead to free fluid in the abdomen without concomitant solid organ injury. Patients with these types of injuries may initially have a benign abdominal examination; however, local bowel ischemia may occur, causing necrosis and delayed perforation. Serial abdominal and laboratory exams in patients with free fluid without solid organ injury are mandatory [36].

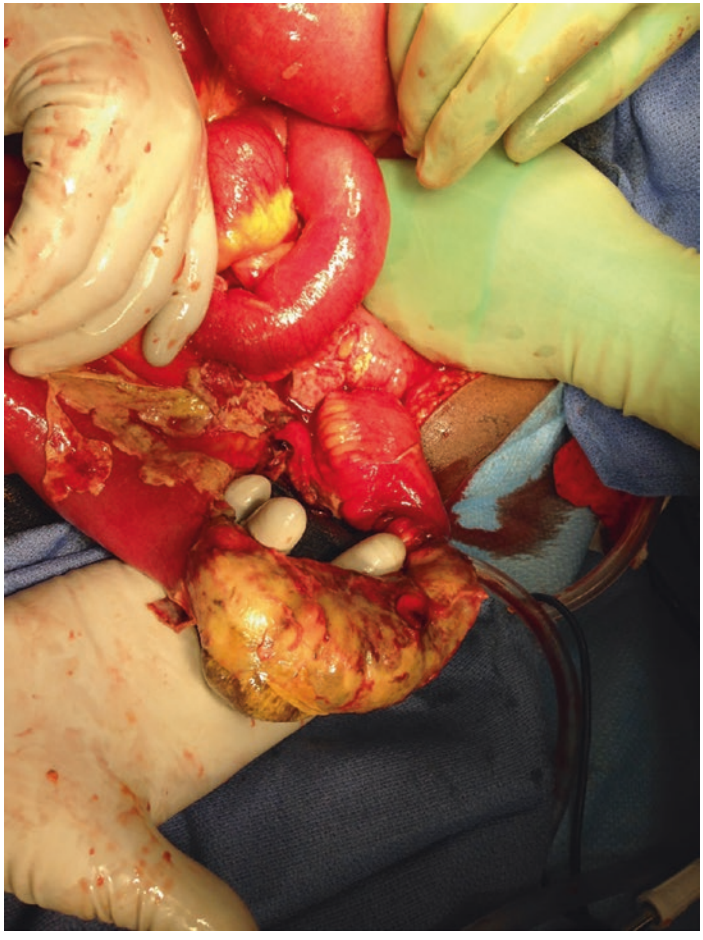

FIGURE 14.1 Necrotic segment of the bowel shown is the sequelae of "bucket-handle" injury at 24 h

Although the utility of CT is established in blunt abdominal injury, its role in penetrating injury is less well defined. Velmahos and colleagues have proposed the utility of CT scan in the selective nonoperative management of abdominal gunshot wounds [37]. In our institution, hemodynamically

stable patients with penetrating abdominal wounds who do not exhibit absolute indications for surgery are scanned and observed. Missile trajectory is also used to help guide operative versus nonoperative decision-making.

Whether deciding to take the patient for operative exploration of the abdomen secondary to blunt or penetrating trauma, the goals of the procedure are the same: hemorrhage control, contamination control, injury identification, and, if patient physiology allows, injury repair. The single key to successful operative intervention is visualization. This is accomplished by good, systematic hemostatic packing; small bowel evisceration allowing full exposure of the mesentery and the retroperitoneum; and opening the lesser sac to fully evaluate the posterior stomach and pancreas.

Minimally invasive techniques have become ubiquitous in surgery, and some authors are advocating its use in trauma. Many of the same advantages seen in elective surgery are being reported in trauma: shorter hospitalization, fewer postoperative wound infections, less postoperative pain, and shorter hospital lengths of stay. Several studies have shown that laparoscopy is safe in selected patients with blunt and penetrating abdominal trauma, minimizes nontherapeutic laparotomies, and allows for the minimal invasive management of selected intra-abdominal injuries. Should laparoscopic evaluation be chosen, it is just as imperative to visualize the bowel from GE junction to the rectum as it is in open exploration. Surgeon experience and supporting hospital infrastructure are important variables when considering its use [38–43].

Organ-Specific Injury: Management and Personal Tips

Stomach

The stomach is very well vascularized due to its redundant blood supply. The distended stomach is at higher risk for rupture because of direct compression or acute increase in intra-

luminal pressure. During initial exploration, the entire gastrointestinal tract from the gastroesophageal (GE) junction to the rectum should be visualized and injuries identified. Blunt gastric injury usually occurs as a result of a blowout-type injury and is usually found on the anterior surface of the stomach. Taking down the gastrohepatic and gastrocolic ligaments increases exposure. When taking down these ligaments, care must be taken to avoid injury to the right and left gastric arteries, the gastroepiploic arteries, and the middle colic artery.

Dividing the left triangular ligament of the left lobe of the liver and placing the patient in reverse Trendelenburg position can also improve visualization of the gastroesophageal junction and the diaphragm. Visualization of the diaphragm is important to rule out injury and to prevent potential contamination of the pleural cavity.

To explore the posterior gastric wall, one must open the lesser sac. This is done most easily by gently grasping the stomach in one hand and the transverse colon in the other and lifting it up. The lesser sac is then entered by dividing the gastrocolic omentum. Completely dividing the gastrocolic omentum and taking the short gastric vessels up to the GE junction provide ultimate exposure to the stomach (Fig. 14.2). Should the suspected gastric injury not be found by visual inspection alone, the surgeon may ask the anesthesia team to fill the stomach with saline and a small amount of methylene blue dye. Gentle occlusion of the GE junction and pylorus while simultaneously applying manual pressure to the stomach can help identify the site of gastric injury. Alternatively, an endoscope can be placed into the stomach for direct inspection of the gastric mucosa.

Gastric injuries are graded according to the American Association for the Surgery of Trauma (AAST) Organ Injury Scale (Tables 14.1, 14.2, 14.3, and 14.4). Grade I stomach injuries are contusions or hematomas or a partial-thickness laceration. A grade II stomach injury is defined as a laceration less than 2 cm at the GE junction or pylorus, less than 5 cm in the proximal one-third of the stomach, or less than 10 cm in

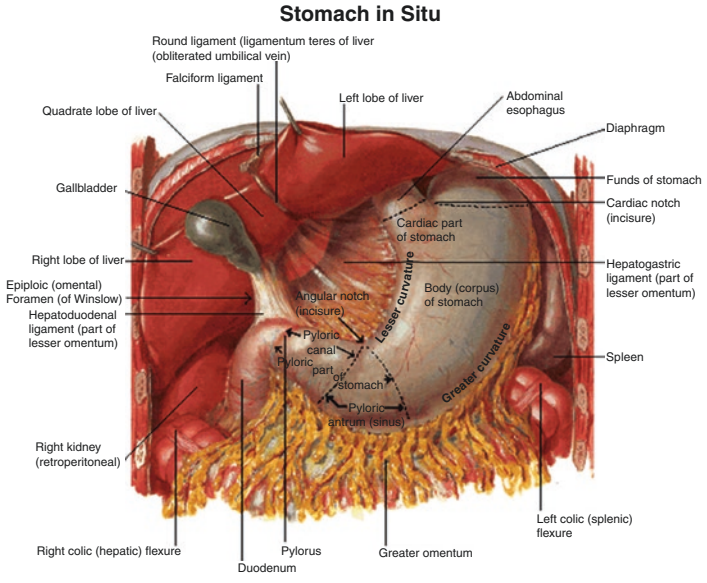

FIGURE 14.2 General anatomy and exposure of the stomach

TABLE 14.1 American Association for the Surgery of Trauma (AAST)—stomach injury scale

Grade	Description of injury	AIS-90
I	Contusion/hematoma	2
	Partial-thickness laceration	2
II	Laceration <2 cm in the GE junction or pylorus	3
	<5 cm in the proximal 1/3 of the stomach	3
	<10 cm in the distal 2/3 of the stomach	3
III	Laceration >2 cm in the GE junction or pylorus	3
	>5 cm in the proximal 1/3 of the stomach	3
	>10 cm in the distal 2/3 of the stomach	3
IV	Tissue loss or devascularization <2/3 of the stomach	4
V	Tissue loss or devascularization >2/3 of the stomach	4

Used with permission of Wolters Kluwer from Moore et al. [68]

Table 14.2 American Association for the Surgery of Trauma (AAST)—small bowel injury scale

Grade	Type of injury	Description of injury	AIS-90
I	Hematoma	Contusion or hematoma without devascularization	2
	Laceration	Partial thickness, no perforation	2
II	Laceration	Laceration <50% of circumference	3
III	Laceration	Laceration >50% of circumference without transection	3
IV	Laceration	Transection of the small bowel	4
V	Laceration	Transection of the small bowel with segmental tissue loss	4
	Vascular	Devascularized segment	4

Used with permission of Wolters Kluwer from Moore et al. [69]

Table 14.3 American Association for the Surgery of Trauma (AAST)—colon injury scale

Grade	Type of injury	Description of injury	AIS-90
I	Hematoma	Contusion or hematoma without devascularization	2
	Laceration	Partial thickness, no perforation	2
II	Laceration	Laceration <50% of circumference	3
III	Laceration	Laceration ≥50% of circumference without transection	3
IV	Laceration	Transection of the colon	4
V	Laceration	Transection of the colon with segmental tissue loss	4
	Vascular	Devascularized segment	4

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Table 14.4 American Association for the Surgery of Trauma (AAST)—rectum injury scale

Grade	Type of injury	Description of injury	AIS-90
I	Hematoma	Contusion or hematoma without devascularization	2
	Laceration	Partial-thickness laceration	2
II	Laceration	Laceration <50% of circumference	3
III	Laceration	Laceration ≥50% of circumference	4
IV	Laceration	Full-thickness laceration with extension into the perineum	5
V	Vascular	Devascularized segment	5

Used with permission of Wolters Kluwer from Moore et al. [69]

the distal two-thirds of the stomach. A grade III stomach injury is defined as a laceration greater than 2 cm at the GE junction or pylorus, greater than 5 cm in the proximal one-third of the stomach, or greater than 10 cm in the distal two-thirds of the stomach. A grade IV stomach injury is when there is significant tissue loss or devascularization.

Once identified, surgical repair of the gastric injury is straightforward. Smaller injuries can be repaired primarily after debridement of devitalized tissue. Most grade I injuries and small perforations can be managed with interrupted silk Lembert sutures in either single- or two-layered closure. Larger perforations, > 2 cm, should be repaired in 2 layers. The inner layer is closed with a continuous running absorbable suture, and the outer layer is closed with interrupted silk Lembert sutures, taking imbricating seromuscular bites. Grade III injuries with major involvement of the greater curvature can be dealt with by performong a sleeve gastrectomy using the gastrointestinal anastomotic (GIA) stapler (Fig. 14.3). Careful attention should be paid to avoid narrowing of the stomach lumen. Injuries causing damage to the pylorus may also necessitate pyloroplasty. Grade IV stomach injuries, with total loss or devascularization of greater than 2/3 of the stomach, may require major resection

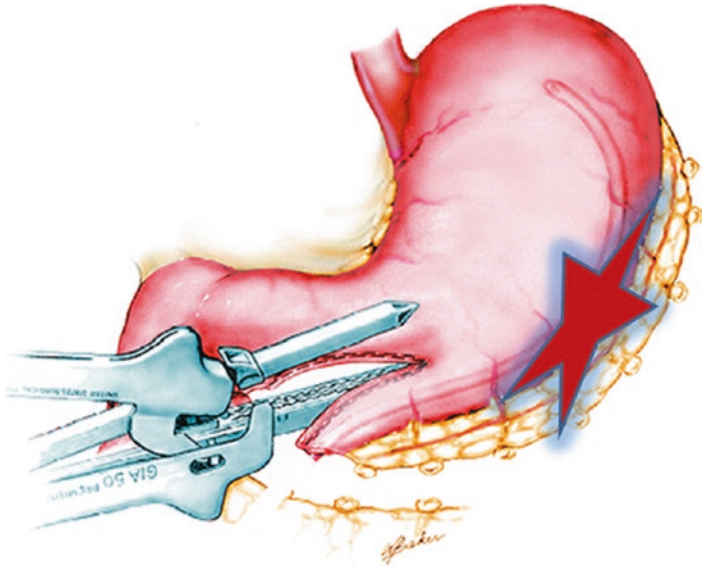

FIGURE 14.3 Grade III injuries with major involvement of the greater curvature can be repaired using the gastrointestinal anastomotic (GIA) stapler

and either gastroduodenostomy (Billroth I) or gastrojejunostomy (Billroth 2) anastomoses (Fig. 14.4). Grade V injuries are rare and may require total gastrectomy followed by Roux-en-Y esophagojejunostomy [7, 44–47].

Complications after gastric injury include rebleeding, gastric fistula formation, gastroparesis, anastomotic leak, and sepsis. Additionally, in patients with resection and with reconstruction, the development of postgastrectomy syndromes is possible. Symptoms include early satiety, delayed gastric emptying, Roux-en-Y stasis syndrome, early and late dumping syndrome, alkaline reflux gastritis, postvagotomy diarrhea, afferent and efferent loop syndromes, internal hernias leading to obstruction, and marginal ulcers. Management may require alterations in diet, prokinetic agents, or reconstruction of the initial anastomosis [44, 47, 48].

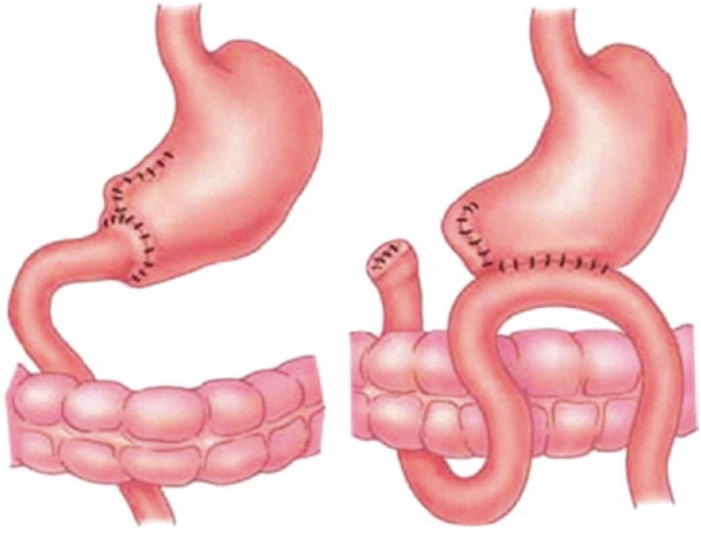

Figure 14.4 Billroth reconstructions

Small Bowel

The small intestine is well vascularized and is less prone to ischemic injury compared to the colon. Distal to the ligament of Treitz, it is suspended on its mesentery with the majority of its blood supply originating from the superior mesenteric artery (SMA) along with multiple arcades. Upon entering the peritoneal cavity, the first priority of management should be hemorrhage control. Mesenteric lacerations can be a source of major hemorrhage and should be identified and controlled rapidly by systematic inspection of the small bowel starting from the ligament of Treitz to the ileocecal valve. Once hemorrhage has been controlled, controlling contamination should be the second step (Fig. 14.5). This can be done with either nonpenetrating clamps, simple suture repair, or stapled division.

Particular attention should be paid to the technique of running the bowel in patients who have penetrating trauma. It is our practice to run the bowel using the index finger and

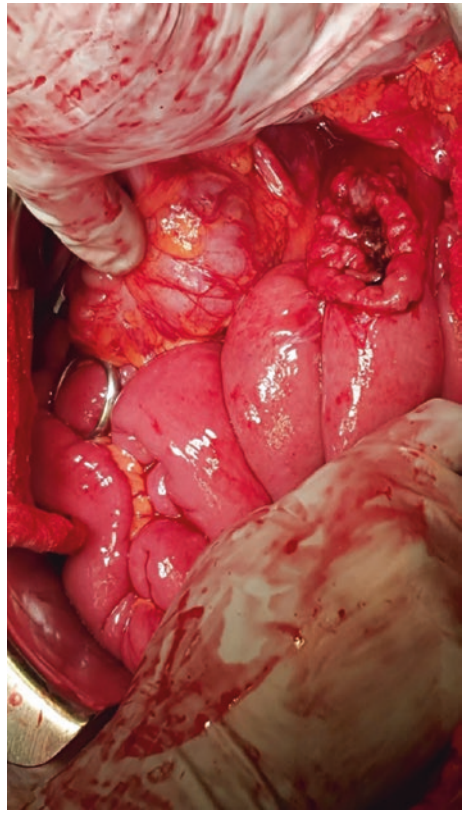

Figure 14.5 When found during exploration, small bowel destructive lesions, such as these, often require techniques aimed at contamination control to minimize spillage of bowel contents

middle finger of each hand. From the left-hand side of the table, the surgeon places the right-hand middle and index fingers at the ligament of Treitz and gently slides along the bowel with the index finger and middle finger of the left-hand control traveling a short distance. This technique helps avoid missing foreign bodies retained within the wall of the bowel, particularly helpful after a shotgun injury with multiple pellets entering the abdominal cavity (Fig. 14.6).

Careful inspection of the mesentery should be performed. Hematomas of the bowel wall should raise suspicion of underlying bowel wall injury. Grade I small bowel injuries can usually be treated by simple inversion with seromuscular

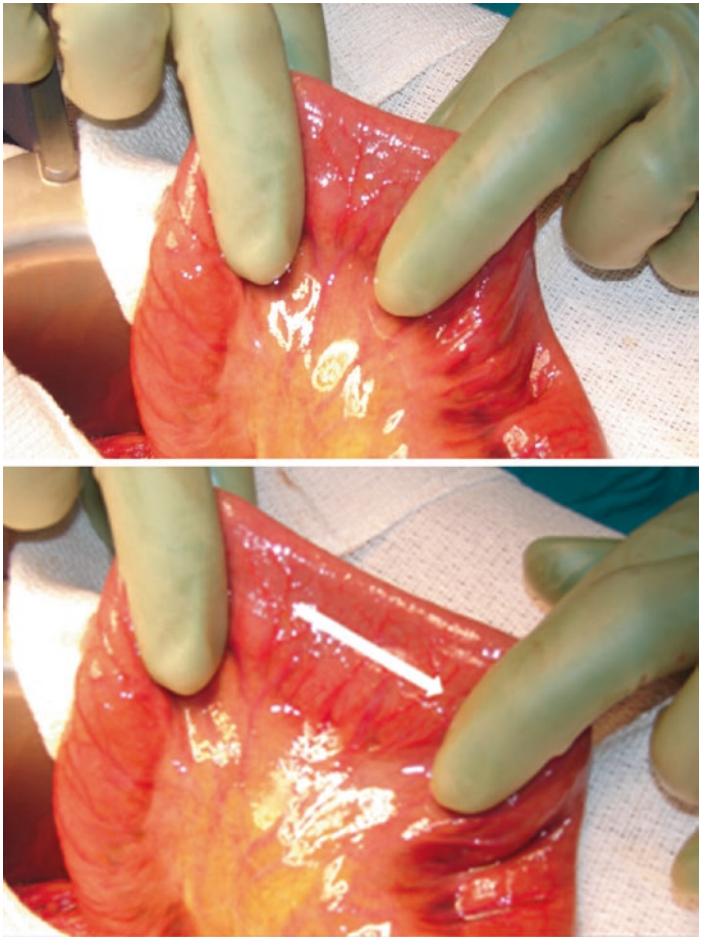

Figure 14.6 Manual inspection of the small bowel

sutures. Grade II bowel injuries should be debrided and managed with single- or double-layered closure with either absorbable or nonabsorbable suture. Grade III and IV injuries will more likely require bowel resection. Careful attention must be paid to avoid narrowing the small bowel lumen. Injuries should be closed transversely, and those involving a

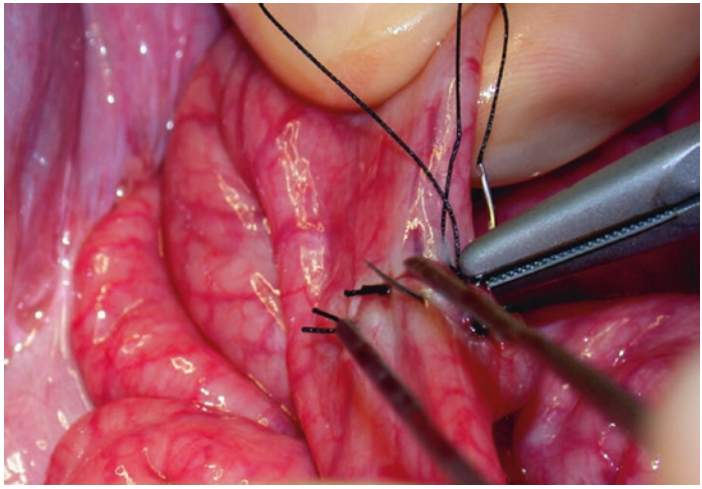

FIGURE 14.7 Primary repair of small bowel injury

>50% circumference should be managed with segmental resection and, if the patients' hemodynamic and acid-base status allows, primary anastomoses (Fig. 14.7). The small bowel mesentery should be closed with either interrupted or running absorbable or nonabsorbable suture after anastomoses to prevent the potential for internal hernia. Patients left in discontinuity should return to the operating room for definitive management as soon as possible. Adjunctive techniques for assessing bowel viability can be used if there is any question of viability. Doppler ultrasound, fluorescence with Wood's lamp, or the newer SPY Elite® Fluorescent Imaging System from Stryker (Kalamazoo, MI, USA) can all be used.

As a recent, large multicenter study failed to demonstrate superiority, the ideal technique for small bowel anastomosis remains unknown [49]. Some prefer stapled anastomoses, while others prefer either single- or two-layered hand-sewn. There may be higher leak rates with stapled versus hand-sewn anastomoses in trauma patients, particularly if damage control has been used. Our practice has been to perform stapled anastomoses for routine resections in non-edematous

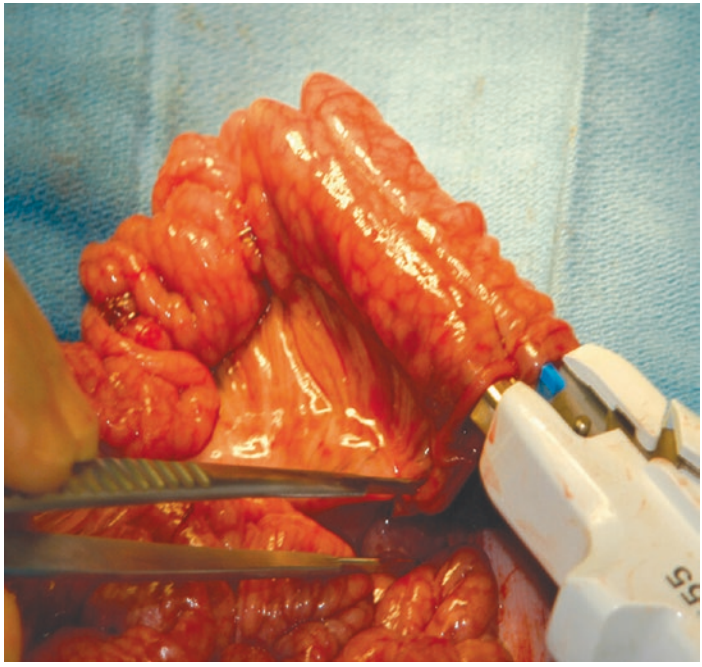

Figure 14.8 Stapled anastomosis using GIA stapler

bowel and hand-sewn double-layered anastomoses, particularly in the setting of bowel edema (Fig. 14.8).

While it is rare for the isolated small bowel injury to require abbreviated, damage control surgery, grade V injuries typically involve significant tissue loss or a devascularized segment warranting resection. If there is a concern for blood flow compromise, a delayed second-look laparotomy within 24 h to assess bowel viability prior to definitive closure may be wise. The open abdomen can be managed by a temporary wound vacuum-assisted closure (VAC) using either a large, fenestrated, sterile plastic barrier, Kerlix gauze, nasogastric tubes, or an Ioban® dressing, with a traditional black sponge negative-pressure therapy KCI VAC® or the KCI ABThera™ VAC (San Antonio, TX, USA) [50, 51].

Open abdomen management, while not a focus of this chapter, has changed significantly over the years. Ratio-driven, blood product-focused resuscitation strategies have led to less bowel wall and retroperitoneal edema leading to improved success rates at closing rectus fascia on subsequent operating room trips. It is imperative to close the abdomen as soon as possible, during the first take back if at all possible, to minimize anastomotic leak, fistula formation, and intra-abdominal infections [52, 53].

Complications After Small Bowel Injury

Postoperative complications can develop in the immediate perioperative or can be delayed by several days to weeks. Bleeding, wound infection, dehiscence or evisceration, and the development of abdominal compartment syndrome can all occur within 72 h of surgery. Less common complications can include stomach or bowel ischemia, enterocutaneous fistula, and empyema.

Any surgical wound is at risk for the development of a surgical site infection. Necrotizing abdominal soft tissue infection can also develop rapidly and be fatal. Prompt aggressive surgical debridement can help lessen the degree of morbidity and mortality associated with this condition.

Intra-abdominal abscesses can develop and usually form as a result of residual contamination from the original injury, new spillage as a result of an anastomotic breakdown, or the development of a fistula. Patients exhibiting signs of fever with leukocytosis approximately a week after operation should undergo CT imaging of the abdomen and pelvis to rule out abscess formation. If found, percutaneous drainage can be performed. In a small number of cases, reoperative exploration will be required.

Diaphragmatic injury in the setting of gastric injury can occasionally result in empyema. The early use of video-assisted thoracoscopic surgery (VATS) for empyema can be helpful. A small subset of these patients may eventually require thoracotomy for debridement.

Any postoperative trauma patient with intra-abdominal injuries should be monitored for abdominal compartment syndrome in the early postoperative phase. This is especially true in cases of damage control surgery or when high-volume resuscitation was used. Particular attention should be paid to the overall hemodynamic status, abdominal distention, urine output per hour, and ventilatory difficulties. Bladder pressure can serve as a marker of intra-abdominal pressure and should be measured if there is clinical concern for intra-abdominal hypertension or abdominal compartment syndrome. Some authors have recommended the early and routine use of bladder pressure monitoring in the postoperative setting [54].

A rare but morbid complication is the short gut syndrome, which can occur after resections of large amounts of the small bowel. Approximately 100 cm of the small bowel is required to allow for oral nutrition without the colon. If the colon remains intact, a minimum of 50–60 cm of the small bowel is needed. The ileocecal valve should be preserved if possible [55].

Colorectal

Colon and rectal injuries can occur as a result of either blunt or penetrating trauma, although they are much more commonly caused by penetrating trauma. In blunt trauma, the colon can be injured by creation of a closed loop and blowout rupture due to increased intraluminal pressure, by shear injury due to tearing at the junction of the mobile and immobile colon, and by deceleration mechanisms causing mesenteric avulsion injury.

Diagnosis of colorectal injuries should begin with physical examination. Close attention should be given to examination of the perineum, the anus, the rectal vault, and the prostate in men. Obvious tears of the anus, the presence of gross blood, disruption of the bony pelvis, a high-riding prostate, or exquisite pain in the perineal region can all provide clues to a possible rectal injury. CT scan of the abdomen and pelvis looking

for the presence of extra-luminal air, extravasation of contrast, colonic wall thickening, or mesenteric stranding can also be helpful. Triple-contrast studies can be helpful, particularly with injuries to the flank. Patients with confirmed or suspected colorectal injury should be placed in stirrups for initial sigmoidoscope evaluation followed by exploratory laparotomy [5]. This facilitates access to the rectal vault and concomitant sigmoidoscopic evaluation should the injury be difficult to find intra-abdominally. In the abdomen, mandatory exploration of hematomas of the colonic wall or mesentery should be performed to avoid missing underlying injury [56].

Both the Eastern Association for the Surgery of Trauma (EAST) [20] and the Western Trauma Association (WTA) [21] have recently published management guidelines/algorithms aimed at treating colorectal injuries. Both organizations have concluded that direct repair, or resection and primary anastomosis on either index operation or first take back if a damage control strategy is employed, is acceptable in virtually all colonic injuries. WTA recommends diversion (with ostomy creation) be considered in the following conditions:

1. Ongoing acidosis or hemorrhagic shock
2. Chronic immunosuppression
3. Inability to close fascia at second laparotomy
4. Concomitant pancreas injuries

While loop ileostomy is associated with the easiest, most straightforward reversal options, they can be associated with dehydration and electrolyte abnormalities—which may be especially troublesome in the elderly, frail population. The type and location of ostomy should depend on the patient's body habitus, the baseline (i.e., pre-injury) physiologic condition, the injury location, and the patient's ability to care for themselves [57–59].

Patients diagnosed with colonic injury warranting exploration should be given preoperative broad-spectrum antibiotics continued for 24 h post-op. The principles of management

remain the same: hemorrhage control followed by control of contamination [60]. Colonic mobilization is performed by dissecting along the white line of Toldt. The surgeon should exercise judgment in determining which patients will be best suited for primary versus colonic diversion [61]. Our practice has been to consider primary repair in patients who have no evidence of hypotension/shock based on physiologic and acid-base parameters, those with minimal stool contamination [62].

Similar to the management for small bowel injuries, colonic injuries can be managed with either stapled or hand-sewn anastomoses. Intraperitoneal rectal injuries are treated similarly to colonic injuries. Extraperitoneal colonic injuries located near the anus can be managed trans-anally and repaired if <25% of the colonic wall is involved. In destructive cases, proximal diversion is advised (WTA) [21]. Presacral drainage or rectal "washouts" are no longer advocated.

Complications of Colorectal Injury

The most common cause of death in patients with colorectal injury is exsanguination from the concomitant mesenteric injury. Sepsis leading to multisystem organ failure, especially in cases where an injury is missed, is the second most common cause of death. Infection complications are common in patients who sustain colorectal injury. Repeat imaging should be performed to evaluate for intra-abdominal abscesses in patients who develop signs of infection. Should they be present, percutaneous drainage is the preferred approach. Wound infections can be avoided by leaving the skin open to heal by secondary intention or the use of a wound VAC. Delayed primary closure can be considered once the initial postoperative period has passed. Delayed complications can occur as a result of suture line failure or development of fistulas. Fistula management is dependent on ensuring there is no distal obstruction, foreign body, or active inflammatory process. Localization should be performed by either CT scanning or fistulogram. Non-septic patients can usually be managed nonoperatively. In addition,

these distal GI fistulas are generally low-output fistulas (<500 cc/day) and can be managed by continuing patients on an oral diet. Multiple, proximal small bowel or high-output fistulas will generally require total parenteral nutrition [63–67].

References

1. Watts D, Fakhry S, Scalea T, Cooper C, Wahl W, Ahrms K, et al. Blunt hollow viscus injury (HVI) and small bowel injury (SBI): prevalence, mortality, and morbidity results from a large multi-institutional study. J Trauma. 2001;51:1233.
2. Lamb G. Selective non-operative management of civilian gunshot wounds to the abdomen: a systematic review of the evidence. Injury. 2014;5(4):659–66.
3. Peponis K, Kasotakis G, Yu J, Alouidor R, Burkott B, Maung AA, et al. Selective nonoperative management of abdominal gunshot Wounds from Heresy to adoption: a multicenter study of the Research Consortium of New England Centers for Trauma (ReCoNECT). JACS. 2017;224(6):1036–45.
4. Al Rawahi AN, Al Hinai FA, Boyd JM, Doig CH, Ball CG, Velmahos GC, et al. Outcomes of selective nonoperative management of civilian abdominal gunshot wounds: a systematic review and meta-analysis. World J Emerg Surg. 2018;13:55.
5. Saksobhavivat N, Shanmuganathan K, Boscak AR, Silker CW, Stein DM, Bodanapally UK, et al. Diagnostic accuracy of triple-contrast multi-detector computed tomography for detection of penetrating gastrointestinal injury: a prospective study. Eur Radiol. 2016;26(11):4107–20.
6. Clarke DL, Allorto NL, Thomson SR. An audit of failed non-operative management of abdominal stab wounds. Injury. 2010;41(5):488–91.
7. Como JJ, Bokhari F, Chiu WC, Duane TM, Holevar MR, Tandoh MA, et al. Practice management guidelines for selective nonoperative management of penetrating abdominal trauma. J Trauma. 2010;68(3):721–33.
8. Biffl WL, Kaups KL, Cothren CC, Brasel KJ, Dicker RA, Bullard MK, et al. Management of patients with anterior stab wounds: a Western Trauma Association Multicenter trial. J Trauma. 2009;66:1294–301.

9. Velmahos GC, Demetriades D, Toutouzas KG. Selective nonoperative management in 1,856 patients with abdominal gunshot wounds should laparotomy still be the standard of care? Ann Surg. 2001;234:395–403.
10. Inaba K, Branco BC, Moe D, Barmparas G, Okoye O, Lam L, et al. Prospective evaluation of selective nonoperative management of torso gunshot wounds: when is it safe to discharge? J Trauma Acute Care Surg. 2012;72(4):884–91.
11. Fikry K, Velmahos GC, Bramos A, Janjua S, de Moya M, King DR, et al. Successful selective nonoperative management of abdominal gunshot wounds despite low penetrating trauma volumes. Arch Surg. 2011;146(5):528–32.
12. Inaba K, Demetriades D. The nonoperative management of penetrating abdominal trauma. Adv Surg. 2007;41:51–62.
13. Zeppa R. Stomach and duodenum. In: Nora PF, editor. Operative surgery: principles and techniques. 2nd ed. Philadelphia: Lea & Febiger; 1980. p. 371–80.
14. Kahane CJ. Injury vulnerability and effectiveness of occupant protection technologies for older occupants and women. Washington, D.C.: National Highway Traffic Safety Administration. Report No. DOT HS 811 766.; 2013.
15. Anderson PA, Rivara FP, Maier RV, Drake C. The epidemiology of seatbelt associated injuries. J Trauma. 1991;31:60.
16. Glover JM, Waychoff MF, Casmaer M, April MD, Hunter CJ, Trexler ST, et al. Association between seatbelt sign and internal injuries in the contemporary airbag era: a retrospective cohort study. Am J Emerg Med. 2018;36(4):545–50.
17. Johnson MC, Eastridge BJ. Redefining the abdominal seatbelt sign: enhanced CT imaging metrics improve injury prediction. Am J Surg. 2017. Dec;214(6):1175–9.
18. Chappuis CW, Fry DJ, Dietzen CD, Panetta TP, Buechter KJ, Cohn IJR. Management of penetrating colonic injuries. A prospective randomized trial. Ann Surg. 1999;213:492–7.
19. Nelson R, Singer M. Primary repair for penetrating colon injuries. Cochrane Database Syst Rev. 2003;3:CD002247.
20. Cullinane DC, Jawa RS, Como JJ, Moore A, Morris DS, Cheriyan J, et al. Management of penetrating intraperitoneal colon injuries: a meta-analysis and practice management guideline from the Eastern Association for the Surgery of Trauma. J Trauma Acute Care Surg. 2019;86(3):505–15.
21. Biffl WL, Moore EE, Feliciano DV, Albrecht RM, Croce MA, Karmy-Jones R, et al. Management of colorectal injuries: a

Western Trauma Association critical decisions algorithm. J Trauma Acute Care Surg. 2018;85(5):1016–20.
22. Sharpe JP, Magnotti LJ, Fabian TC, Croce MA. Evolution of the operative management of colon trauma. Trauma Surg Acute Care Open. 2017;2(1):e000092.
23. Lasinski AM, Gil L, Kothari AN, Anstadt MJ, Gonzalez RP. Defining outcomes after colon resection in blunt trauma: is diversion or primary anastomosis more favorable? Am Surg. 2018;84(8):1288–93.
24. Miller S. The president and the assassin. New York: Random House; 2011.
25. Weiner SL, Barrett J. Abdominal injuries. In: Weiner SL, Barrett J, editors. Trauma management for civilian and military physicians. 1st ed. Philadelphia: WB Saunders Co; 1986. p. 212–50.
26. Palm K, Apodaca A, Spencer D, Costanzo G, Bailey J, Blackbourne LH, et al. Evaluation of military trauma system practices related to damage-control resuscitation. J Trauma Acute Care Surg. 2012;73(6 Suppl 5):S459–64.
27. Prat N, Pidcoke HF, Sailliol A, Cap AP. Evolution of US military transfusion support for resuscitation of trauma and hemorrhagic shock. Transfus Clin Biol. 2013;20(2):225–30.
28. Chiara O, Pelosi P, Segala M, Turconi MG, Brazzi L, Bottino N, et al. Mesenteric and renal oxygen transport during hemorrhage and reperfusion: evaluation of optimal goals for resuscitation. J Trauma. 2001;51(2):356–62.
29. Udobi KF, Rodriguez A, Chiu WC, Scalea TM. Role of ultrasonography in penetrating abdominal trauma: a prospective clinical study. J Trauma. 2001;50(3):475–9.
30. Rhodes CM, Smith HL, Sidwell RA. Utility and relevance of diagnostic peritoneal lavage in trauma education. J Surg Educ. 2011;68(4):313–7.
31. Jaffin JH, Ochsner G, Cole FL, Rozycki GS, Kass M, Champion HR. Alkaline phosphatase levels in diagnostic peritoneal lavage fluid as a predictor of hollow visceral injury. J Trauma. 1993;34:829.
32. Donahue JH, Federle MP, Griffiths BG, Trunkey DD. Computed tomography in the diagnosis of blunt intestinal and mesenteric injuries. J Trauma. 1987;27:11–7.
33. Phillips T, Sclafani SJ, Goldstein A, Scalea T, Panetta T, Shaftan G. Use of the contrast-enhanced CT enema in the management of penetrating trauma to the flank and back. J Trauma. 1986;26(7):593–601.

34. Malhotra AK, Fabian TC, Katsis SB, Gavant ML, Croce MA. Blunt bowel and mesenteric injuries: the role of screening computed tomography. J Trauma. 2000;48(6):991–8; discussion 998–1000
35. Fahkry SM, Watts DD, Luchette FA, EAST Multi-Institutional Hollow Viscus Injury Research Group. Current diagnostic approaches lack sensitivity in the diagnosis of perforated blunt small bowel injury: analysis from 275,557 trauma admissions from the EAST Multi-Institutional HVI trial. J Trauma. 2003;54:295–306.
36. Mahmood I, Tawfek Z, Abdelrahman Y, Siddiuqqi T, Abdelrahman H, El-Menyar A, et al. Significance of computed tomography finding of intra-abdominal free fluid without solid organ injury after blunt abdominal trauma: time for laparotomy on demand. World J Surg. 2014;38(6):1411–5.
37. Velmahos GC, Constantinou C, Tillou A, Brown CV, Salim A, Demetriades D. Abdominal computed tomographic scanning for patients with gunshot wounds of the abdomen selected for non-operative management. J Trauma. 2005;59:1155–61.
38. Ivatury RR, Simon RJ, Stahl WM. A critical evaluation for laparoscopy in penetrating abdominal trauma. J Trauma. 1993;34:822.
39. Kaban GK, Novitsky YW, Perugini RA, Haveran L, Czerniach D, Kelly JJ, et al. Use of laparoscopy in evaluation and treatment of penetrating and blunt abdominal injuries. Surg Innov. 2008;15(1):26–31.
40. Johnson JJ, Garwe T, Raines AR, Thurman JB, Carter S, Bender JS, et al. The use of laparoscopy in the diagnosis and treatment of blunt and penetrating abdominal injuries: 10-year experience at a level 1 trauma center. Am J Surg. 2013;205(3):317–20; discussion 321.
41. Chestovich PJ, Browder TD, Morrissey SL, Fraser DR, Ingalls NK, Fildes JJ. Minimally invasive is maximally effective: diagnostic and therapeutic laparoscopy for penetrating abdominal injuries. J Trauma Acute Care Surg. 2015;78(6):1076–85.
42. Sumislawski JJ, Zarzaur BL, Paulus EM, Sharpe JP, Savage SA, Nawaf CB, et al. Diagnostic laparoscopy after anterior abdominal stab wounds: worth another look? J Trauma Acute Care Surg. 2013;75(6):1013–8.
43. Matsevych OY, Koto MZ, Motilall SR, Kumar N. The role of laparoscopy in management of stable with PAT and organ evisceration. J Trauma Acute Care Surg. 2016;81(2):307–11.
44. Jordan PH Jr. Stomach and duodenum. In: Hardy JD, editor. Hardy's textbook of surgery. 1st ed. Philadelphia: JB Lippincott; 1983. p. 497–521.

45. Rodkey GV, Welch CE. Injuries to the stomach and duodenum. In: Schwartz SI, Ellis H, editors. Maingot's abdominal operations. 8th ed. Norwalk: Appleton-Century-Crofts; 1985. p. 605–99.
46. Moore EE, Jurkovich GJ, Knudson MM, Cogbill TH, Malangoni MA, Champion HR, et al. Organ injury scaling VI: extrahepatic biliary, esophagus, stomach, vulva, vagina, uterus, fallopian tube ovary. J Trauma. 1995;39:1069–70.
47. Carvajal SH, Mulvihill SJ. Postgastrectomy syndromes: dumping and diarrhea. Gastroenterol Clin N Am. 1994;23(2):261–79.
48. Karamanolis G, Tack J. Nutrition and motility disorders. Best Pract Res Clin Gastroenterol. 2006;20(3):485–505.
49. Bruns BR, Morris DS, Zielinski M, Mowery NT, Miller PR, Arnold K, et al. Stapled versus hand-sewn: a prospective emergency surgery study. An American Association for the Surgery of Trauma multi-institutional study. J Trauma Acute Care Surg. 2017;82(3):435–43.
50. Fahkry SM, Brownstein M, Watts DD, Baker CC. Relatively short diagnostic delays (<8 hours) produce morbidity and mortality in blunt small bowel injury. J Trauma. 2000;48:409–15.
51. Burch JM, Franciose RJ, Moore EE. Single-layer continuous versus two-layer interrupted intestinal anastomosis: a prospective randomized trial. Ann Surg. 2000;231:832–7.
52. Joseph B, Zangbar B, Pandit V, Vercruysse G, Aziz H, Kulvatunyou N, et al. The conjoint effect of reduced crystalloid administration and decreased damage-control laparotomy use in the development of abdominal compartment syndrome. J Trauma Acute Care Surg. 2014;76(2):457–61.
53. Hatch QM, Osterhout LM, Podbielski J, Kozar RA, Wade CE, Holcomb JB, et al. Impact of closure at the first take back: complication burden and potential overutilization of damage control laparotomy. J Trauma. 2011;71(6):1503–11.
54. Luckianow GM, Matthew Ellis M, Governale D, Kaplan LJ. Abdominal compartment syndrome: risk factors, diagnosis, and current therapy. Crit Care Res Pract. 2012;2012:8.
55. Dabney A, Thompson J, DiBaise J, Sudan D, McBride C. Short bowel syndrome after trauma. Am J Surg. 2004;188(6):792–5.
56. Stone H, Fabian TC. Management of perforating colon trauma: randomization between primary closure and exteriorization. Ann Surg. 1979;1990:430–6.
57. Lavenson GS, Cohen A. Management of rectal injuries. Am J Surg. 1971;122(2):226–30.

58. Murray JA, Demetriades D, Colson M, Song Z, Velmahos GC, Cornwell EE 3rd, et al. Colonic resection in trauma: colostomy versus anastomosis. J Trauma. 1999;46:250.
59. Gonzalez RP, Faliminsky ME, Holevar MR. The role of presacral drainage in the management of penetrating rectal injuries. J Trauma. 1998;45:656–61.
60. Woodhall JP, Ochsner A. The management of perforating injuries of the colon and rectum in civilian practice. Surgery. 1951;29:305–20.
61. Demitriades DD, Rabinowitz B, Sofianos C, Prümm E. The management of colonic injuries by primary repair or colostomy. Br J Surg. 1985;72:881–3.
62. Cayten CG, Fabian TC, Garcia VF, Ivatury RR, Morris JA Jr. Patient management guidelines for penetrating colon injuries. Trauma Practice Guidelines. Eastern Association for the Surgery of Trauma; 1998. http://www.east.org.
63. Demetriades D, Murray JA, Chan LC, Ordoñez C, Bowley D, Nagy KK, et al. Penetrating colon injuries requiring resection: diversion or primary anastomosis? An AAST Prospective Multicenter Study. J Trauma. 2005;59:1155–61.
64. Zmora D, Manhajna A, Bar-Zakai B, Rosin D, Hershko D, Shabtai M, et al. Colon and rectal surgery without mechanical bowel preparation: a randomized prospective trial. Ann Surg. 2003;237:363–7.
65. Velmahos GG, Degiannis E, Wells M, Souter I, Saadia R. Early closure of colostomy in trauma patients: a prospective randomized trial. Ann Surg. 1995;118:815–20.
66. Jeffrey RB Jr, Federle MP, Stein SM, Crass RA. Intramural hematoma of the cecum following blunt trauma. J Comput Assist Tomogr. 1982;6:404–5.
67. Moore EE, Cogbill TH, Malongoni MA, Jurkovich GJ, Champion HR, Gennarelli TA, et al. Organ injury scaling. II. Pancreas, duodenum, small bowel, colon, and rectum. J Trauma. 1990;30:1427–9.
68. Moore EE, Jurkovich GJ, Knudson MM, Cogbill TH, Malangoni MA, Champion HR, Shackford SR. Organ injury scaling. VI: Extrahepatic biliary, esophagus, stomach, vulva, vagina, uterus (nonpregnant), uterus (pregnant), fallopian tube, and ovary. J Trauma. 1995;39(6):1069–70.
69. 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. 1990;30(11):1427–9.

Chapter 15
Trauma of the Kidney, Ureter, and Bladder: Techniques

Margaret Lauerman and Stacy Shackelford

Introduction of the Problem

GU trauma is infrequently seen by trauma surgeons [1–4]. Despite the infrequent occurrence of GU trauma, patients with GU trauma can be critically ill and difficult to manage. For example, patients with renal injuries can present hemodynamically unstable and require emergent intervention [5]. In stable patients, GU trauma can still cause significant morbidity through leak, renal failure, and stricture.

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Renal Trauma

Renal trauma is the most common GU trauma [4] (Fig. 15.1). Renal trauma occurs in 1.2% of trauma overall. Most renal injuries occur after blunt trauma, specifically after motor vehicle crashes [2]. A majority of renal injuries are contusions or hematomas, with lacerations and vascular injuries less common [2]. Renal trauma is graded on the American Association for the Surgery of Trauma (AAST) scale from 1 to 5. Lower-grade renal injuries are more commonly seen than higher-grade injuries [4, 6].

A majority of patients with renal injuries can be managed with nonoperative management (NOM) successfully, with only 11–13% of patients with renal injuries requiring operative intervention [2, 5]. Nephrectomy is the most commonly used surgical technique, with 61–64% of patients with renal injuries who require operative intervention undergoing nephrectomy [2, 5]. Unsurprisingly nephrectomy is more frequently required in higher AAST grade injuries [5].

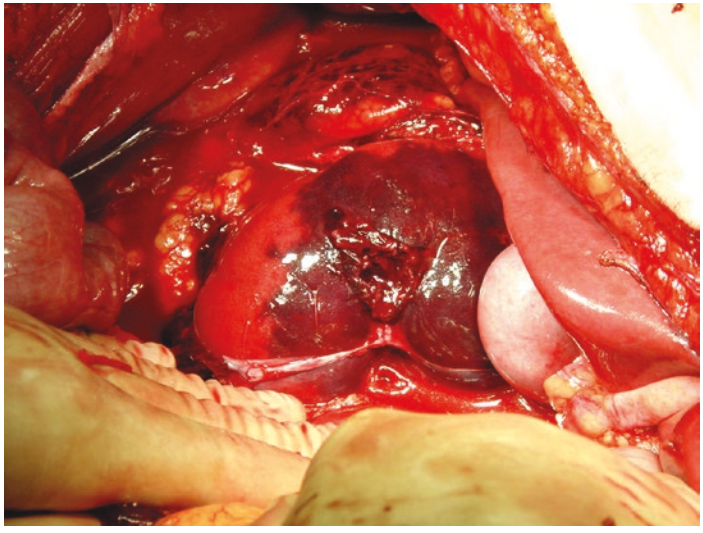

Figure 15.1 Renal injury to the hilum with devascularization

Diagnosis of renal injury depends on the hemodynamic stability of the patient, as patients with renal injuries can present with hypotension [5]. At our institution, hemodynamically unstable patients are explored without a preoperative diagnosis of renal injuries. These unstable patients are seen at laparotomy to have a zone 2 retroperitoneal hematoma, and the diagnosis of a renal injury is made in the operating room when this hematoma is explored. Other patients who undergo exploration of a zone 2 hematoma are patients with an expanding zone 2 hematoma, active bleeding from a zone 2 hematoma, or penetrating injury.

Hemodynamically stable patients undergo CT imaging to diagnose renal injuries. CT is the optimal diagnostic test for renal trauma, as renal injuries have multiple, often concurrent characteristics. CT quantifies amount of parenchymal injury, presence of vascular injury (such as active extravasation or pseudoaneurysm), presence of collecting system injury, and amount of perinephric hematoma.

With renal trauma, further delayed images (beyond the venous phase) allow time for intravenous contrast to be excreted by the kidney into the collecting system. These delayed images are examined for contrast pooling in the retroperitoneum, which indicates collecting system injury. Investigation for collecting system injury is important as 29% of high-grade kidney injuries have urinary extravasation [7]. Repeat imaging should be considered with grade 5 injuries, collecting system injuries, and if directed by the patients' clinical courses, such as with sepsis, worsening anemia, or hemodynamic changes [8].

There are no hard guidelines for which renal injuries require operative intervention beyond those in patients with hypotension and renal pedicle avulsion [9]. Hilar injuries are unlikely to be successfully managed with NOM (Fig. 15.2), although many patients with high-grade renal injuries can be managed with NOM as long as they remain hemodynamically normal. This NOM often requires a multidisciplinary approach including endovascular specialists and urologists.

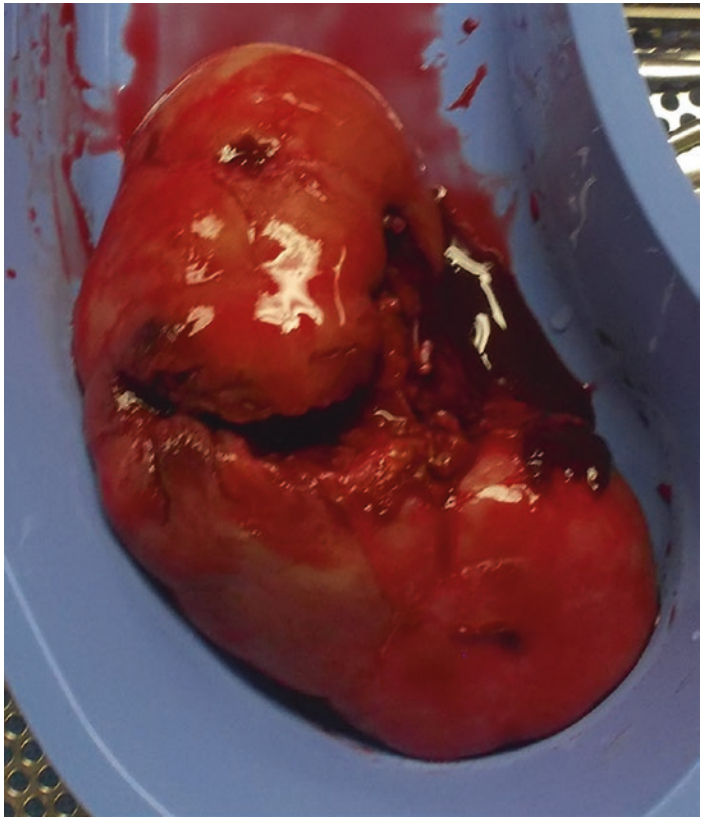

Figure 15.2 Renal hilar injury requiring nephrectomy

Collecting system injury on CT does not mandate operative repair, particularly if the patient does not have another indication for laparotomy. Treatment with Foley catheter drainage, stenting, or percutaneous nephrostomy may then be successful (Fig. 15.3). In addition, collecting system injuries can resolve on repeat CT without intervention [10]. Using ureteral stents for collecting system injuries will exclude the area of injury, with stenting used in 29% of patients with contrast extravasation from the GU tract [7].

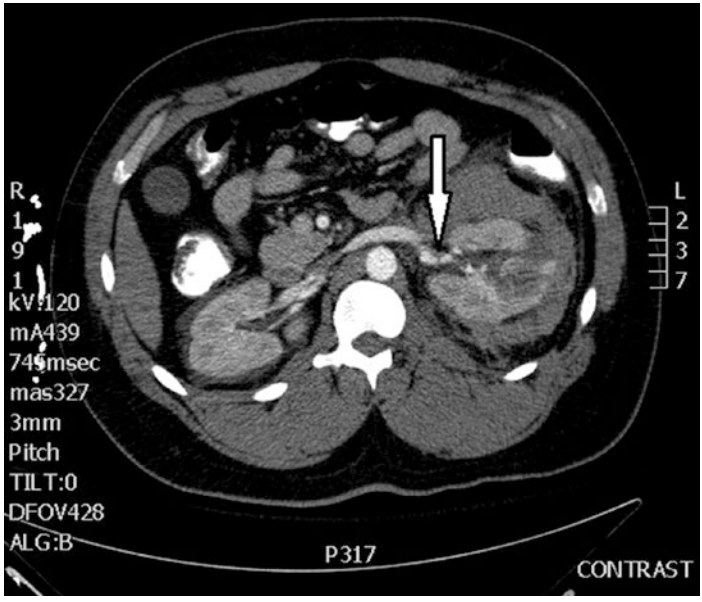

Figure 15.3 Urine extravasation on CT treated with nephrostomy and stenting

Similarly active vascular contrast extravasation, pseudoaneurysm, and arteriovenous fistula do not mandate operative intervention (Fig. 15.4a, b). There are no standardized indications for angiography in renal trauma; however, active contrast extravasation and perirenal hematoma size are associated with use of angioembolization [11–13]. In hemodynamically stable patients, we often attempt endovascular embolization for these vascular injuries. Repeat embolization is sometimes required for vascular injuries [14, 15]. Unlike other solid visceral injuries, embolization for renal injuries is not highly successful, with approximately 27% of patients failing NOM after embolization with ongoing bleeding, abdominal compartment syndrome, pain, or increased free fluid seen on CT [16].

In patients with renal injuries requiring operative management, we use a midline laparotomy. We believe that a laparotomy is the ideal incision as it allows rapid access to

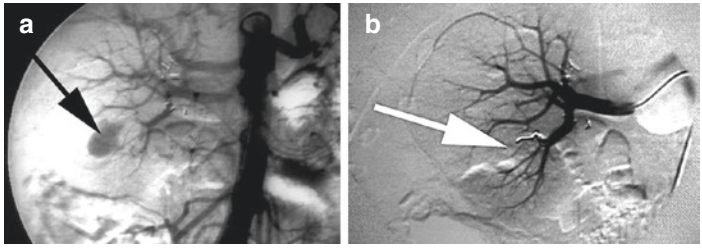

FIGURE 15.4 (**a**, **b**) Active vascular contrast extravasation on angiogram (**a**) and subsequent embolization (**b**)

proximal vascular control if needed. The right kidney is explored by performing a right to left medial visceral rotation. The left kidney is explored in a similar fashion starting with a left to right medial visceral rotation.

These maneuvers expose Gerota's fascia. Gerota's fascia is entered by making a liberal vertical incision. The renal injury is explored. The fatty perirenal tissue is dissected free with care taken not to deserosalize the kidney, which can cause bleeding or iatrogenic injury, especially with blunt dissection at the area of injury. This allows the renal injury to be defined. The kidney should be mobilized out of the retroperitoneum toward the midline to improve visualization by rotating the kidney on its vascular pedicle. This is especially helpful for visualizing the posterior surface of the kidney. Further operative interventions will depend on the components of the injury. If the kidney is not felt to be salvageable, then a nephrectomy is performed. Nephrectomy will mostly be required with destructive injuries that cannot be reconstructed or with bleeding that is unable to be controlled locally.

Some surgeons advocate for obtaining proximal control of the renal artery and vein prior to entering Gerota's fascia. However, vascular control prior to opening Gerota's fascia does not decrease the nephrectomy rate [17]. In our practice, we have not found obtaining proximal control to be commonly necessary. Trying to attempt to obtain proximal vascular control often results in entering the hematoma and

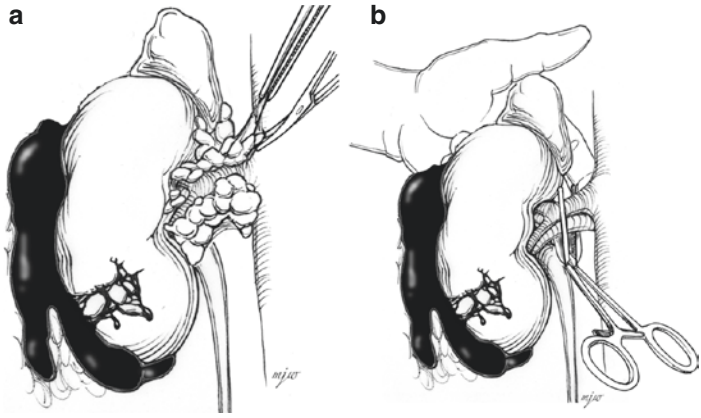

FIGURE 15.5 (**a**, **b**) Method for obtaining en bloc control of the renal vascular pedicle

decompressing the vascular injury. We most often simply mobilize the kidney and control bleeding directly with suture ligatures or manual compression after Gerota's fascia is entered. Another strategy described is to obtain renal pedicle control en bloc next to the kidney, which avoids a lengthy time to dissect the renal pedicle (Fig. 15.5a, b).

In renal injuries that are salvageable, the simplest method of repair is with suture renorrhaphy. This is used when the edges of the laceration can be approximated. Definitive bleeding control is first obtained with suture ligatures. We then look for an underlying collecting system injury, which if present is primarily repaired (Fig. 15.6). The parenchyma is then reapproximated using pledgets (either with Teflon or with Surgicel® Nu-Knit® [Ethicon, Somerville, NJ, USA)] as the renal parenchyma is quite soft and the sutures can pull through, which worsens the injury. Drains are placed after the repair (we use one posterior to the repair and one anterior to the repair). Topical hemostatic agents are also helpful on the kidney to obtain hemorrhage control and maximize renal salvage.

Another option for renal salvage is partial nephrectomy. After vascular control is obtained either at the level of the hilum or locally with pressure, the non-salvageable portion of

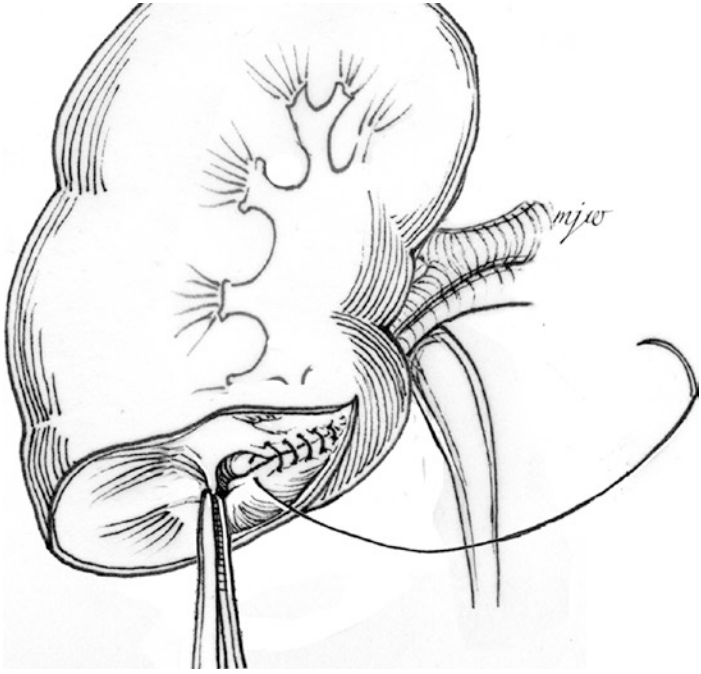

FIGURE 15.6 Closure of injuries to the collecting system prior to management of the parenchymal injury

the kidney is debrided. Hemostasis is achieved and the collecting system repaired if a collecting system injury is present. This debridement, however, can leave a cut end of the kidney that may not be able to be closed. We typically try to close the cut surface down with some mattress 0 chromic liver sutures, setting the tension with a hemoclip. When possible, we cover the free surface of the kidney with a tissue flap, such as an omental flap, and suture this flap onto the cut edge. We also place drains after partial nephrectomy.

We will evaluate all stable patients for renorrhaphy or partial nephrectomy to maximize renal salvage. However, nephrectomy is sometimes required. To perform a nephrectomy, the renal artery and vein are suture ligated. The ureter is divided distally and ligated. We recommend feeling for a

contralateral kidney prior to nephrectomy but do not routinely perform intravenous pyelograms in patients without preoperative imaging.

Mortality is substantial in patients undergoing operative management of their renal injury, at 23% [18]. Other complications include urinoma in approximately 10% of renal injuries. Persistent hematuria was seen more frequently in patients undergoing observation at 16%. Abscess was seen in less than 2% of patients with renal injuries. Renal failure is present in 4–5% of patients with renal injury [18], which underscores the importance of renal salvage when possible. Delayed bleeding can be seen [19] and can be managed with surgical exploration or endovascular therapy depending on hemodynamic stability and injury anatomy. Hypertension can be seen after renal trauma with causes including renal artery occlusion or renal parenchymal compression, with subsequent increase in renin release [9]. Infected urinoma or abscess can occur from neighboring bowel or pancreatic injuries [19].

Ureter Trauma

Blunt and penetrating ureter trauma are rare. Blunt ureter trauma occurs in 0.03% of overall trauma patients [3]. While an uncommon injury, blunt ureter trauma should always be kept in mind as a possibility. Penetrating mechanism is present in 62% of patients with ureteral injury, with gunshot wounds as the majority [3]. Care must be taken in evaluation of injury trajectory (either on CT or at operative exploration) that the ureter is uninjured.

There are no physical exam findings that specifically define ureteral injury. Ureter injury should be suspected with a retroperitoneal trajectory. Flank hematoma is a nonspecific sign of ureter injury. Ureteral injury similarly does not always occur with hematuria, and this cannot be relied upon to screen patients.

In hemodynamically stable patients, multiple radiographic modalities can be used to diagnose ureter injury: retrograde pyelogram and CT. CT for diagnosis of a ureter injury is with

an anterograde study. In general, we will first use CT to evaluate for ureteral injury in stable patients where injury trajectory potentially contacts with the ureter. Delayed CT images can be obtained after intravenous contrast administration to look for contrast filling of the ureter and contrast pooling within the retroperitoneum. Caution must be used with CT for definitive diagnosis of ureter injuries if the trajectory is concerning given the possibility for missed ureteral injuries, with one study noting 50% of ureteral injuries without contrast extravasation seen on the initial CT [20].

In unstable patients, operative exploration is used for diagnosis of ureteral injuries. The ureter can be difficult to find in destructive injuries or with a large retroperitoneal hematoma. A reproducible way to locate the ureter is to identify it at the level of the iliac bifurcation [21]. This is an almost universally reliable anatomic relationship. The ureter should be explored for the distance required to definitely exclude an injury within the entire zone of potential injury, with 360 degree exposure required to fully explore the entirety of ureter wall. The blood supply to the ureter is from multiple vessels, including the renal artery, gonadal artery, aorta, common iliac artery, and hypogastric artery, so extensive circumferential dissection should be avoided so as to not devascularize the ureter [21].

Operative repair of ureteral injuries is most simply done with a ureteroureterostomy with an end-to-end anastomosis to the ipsilateral ureter. Ureteroureterostomy is the most common procedure to repair ureteral injuries [22] and is ideal for mid-ureteral injuries. Care should be taken to assure that the ureteroureterostomy is tension-free, which can be difficult with destructive injuries. The ureter should be mobilized if needed to assure a tension-free anastomosis. The ureter is debrided back to healthy tissue. Both ends of the ureter are spatulated. Interrupted monofilament sutures are placed for the back wall of the anastomosis. A double-J stent is placed and the anterior wall is closed over the top of the stent (Fig. 15.7). The stent remains in place for at least 6 weeks after ureter repair. We will decompress the GU tract with a Foley catheter as well for at least 1 week.

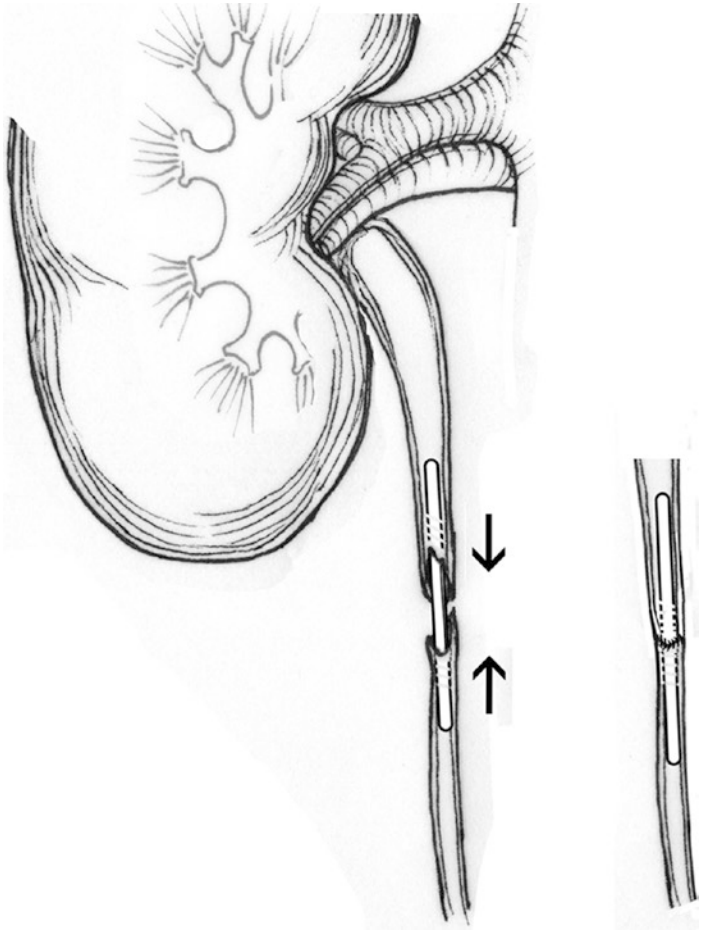

FIGURE 15.7 Repair of ureteral injury over a stent

Another option for mid-ureteral injuries with destruction where primary anastomosis to the ipsilateral ureter is not possible is with ureteroureterostomy to the contralateral ureter or transureteroureterostomy (Fig. 15.8). We do not recommend using ureteroureterostomy to the contralateral ureter as anastomosis to the contralateral ureter exposes the unin-

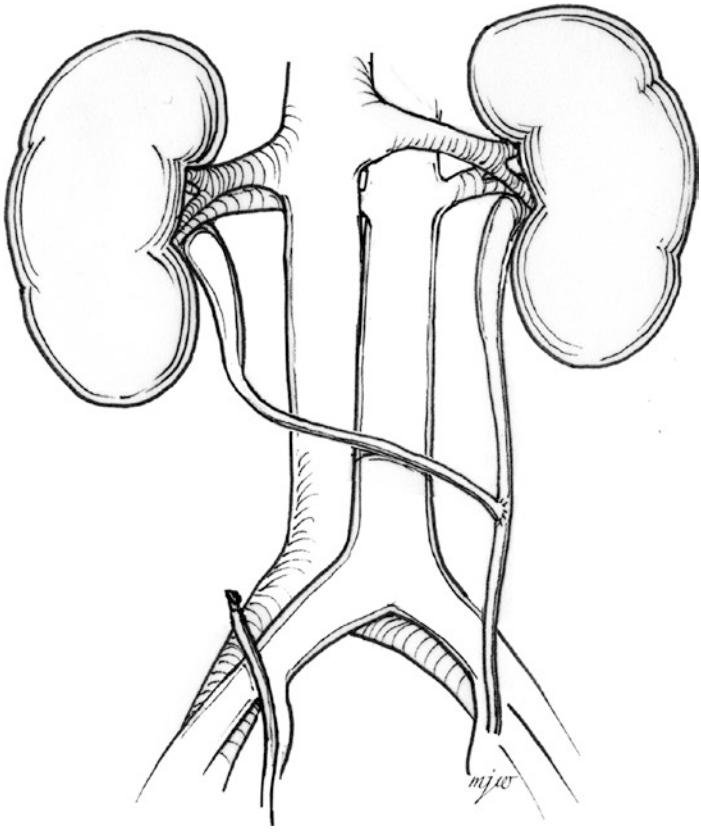

FIGURE 15.8 Ureteroureterostomy to the contralateral ureter

jured ureter to stricture and leak. This takes one ureter injury and potentially creates injury to the other ureter. We have not found the technique of ureteroureterostomy to the contralateral ureter to be required in our practice.

Distal ureteral injuries should be repaired with ureteral reimplantation into the bladder or ureterocystostomy. In ureter injuries without substantial tissue loss, ureterocystostomy can be done without further manipulation. With destructive injuries, reaching the distal ureter to the bladder without tension can be difficult. One technique to allow ureterocystostomy is

the psoas hitch. In this technique, the bladder is dissected free from the surrounding tissue and sutured superiorly (to the psoas). This allows a ureterocystostomy to be created with less ureteral length. Another technique to allow ureterocystostomy is with the Boari flap. In the Boari flap, a tube of the bladder is created and the ureter anastomosed to the tubularized bladder. The defect in the bladder is then closed primarily.

In patients undergoing damage control surgery, resuscitation takes priority over definitive ureteral reconstruction. Our preferred technique is to place a pediatric feeding tube through the cut end of the proximal ureter and secure the feeding tube in the ureter with a suture. The pediatric feeding tube is externalized through the abdominal wall (Fig. 15.9). Urine can then drain through the pediatric feeding tube and be recorded while resuscitation is ongoing without spending time repairing in the ureter.

Missed ureteral injuries are a possibility, even after operative exploration, and a high index of suspicion for ureter injury should be maintained [23]. A missed ureter injury will not present in a reproducible fashion, nor will it necessarily present with symptoms specific to the GU tract. For example, ileus, urine leaking from the wound, sepsis, and uremia could all be caused by missed ureteral injury. Typically, patients with large volume urine leak develop hyperchloremic, metabolic acidosis as the urine is reabsorbed visa the peritoneum. Other complications include ureteral fistulae which are usually treated with stenting and drainage [24]. Stricture is a known complication with ureter injury, likely caused by ischemia from dissection or local blast injury. Strictures can often be managed endoscopically with dilation or stenting, although open repair is sometimes required [24].

Bladder Trauma

Bladder injuries are a similarly infrequent occurrence, occurring in 0.7% of trauma patients [1]. Bladder injuries can occur in both penetrating and blunt trauma, with 35%–85% occurring in blunt trauma [1, 25]. Bladder injuries do not frequently

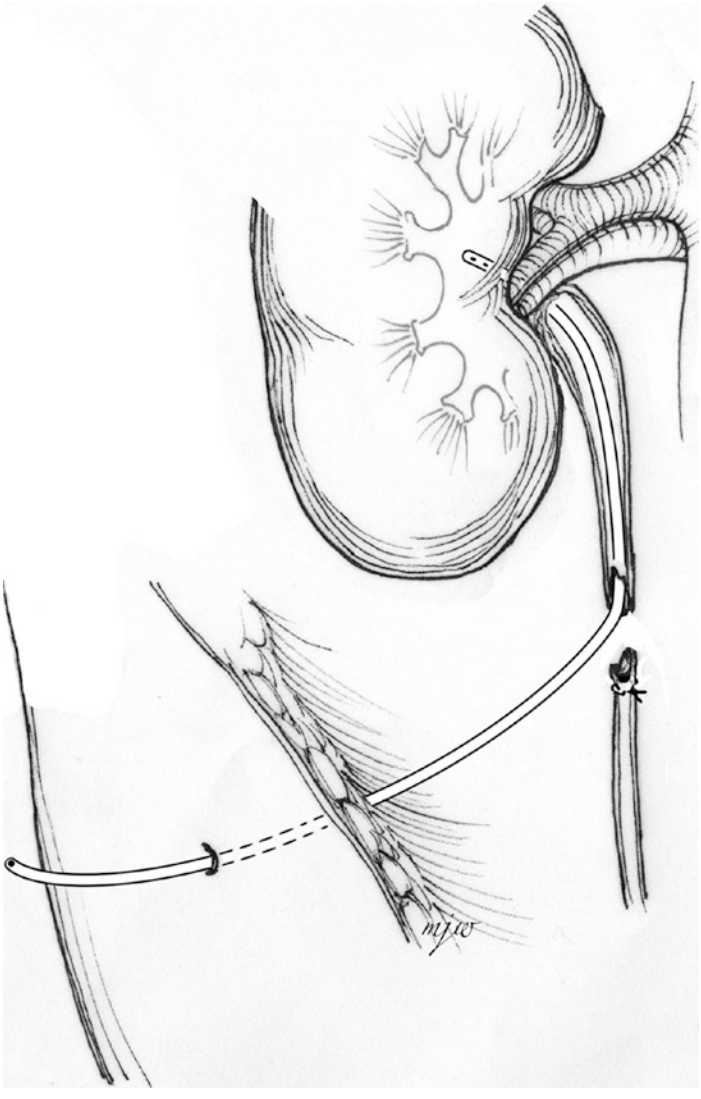

FIGURE 15.9 Damage control exteriorization of an injured ureter using a pediatric feeding tube

occur in isolation, with 44% occurring with another intra-abdominal injury and 32%–46% with concurrent pelvic fractures [1, 25]. Simultaneous injuries to the colon, small intestine, rectum, other solid organs, and urethra are also seen [1]. Bladder injuries are not usually associated with hemodynamic instability [26]. Hematuria is commonly seen with bladder injuries [26].

Bladder injuries are commonly managed with NOM, with only 54% undergoing repair [25], particularly after blunt trauma. The need for operative intervention depends on the type of bladder injury. Intraperitoneal bladder injuries almost always undergo operative repair, as do penetrating bladder injuries [1]. Conversely, most extraperitoneal bladder ruptures after blunt trauma do not require operative repair [1].

CT cystogram is our most common radiographic technique to diagnose bladder injuries. Concern for urethral injury should prompt a retrograde urethrogram prior to Foley placement for the cystogram in stable patients (Fig. 15.10). The cystogram may be performed using regular X ray imaging or CT scanning. Contrast is injected through the Foley catheter retrograde into the bladder, and the Foley catheter clamped to distend the bladder. Usually, several hundred cc's are needed to distend the bladder. Extravasation can then be seen on CT, if present (Fig. 15.11). Passive bladder filling on CT is not adequate to evaluate for bladder injury [27]. Patients with bladder injuries often undergo CT imaging as part of their initial trauma evaluation, as concurrent injuries are common [1, 25]. Thought should be given to the timing of performing cystograms in patients with pelvic fractures to avoid contrast obscuring angiographic imaging if needed for pelvic bleeding.

Another commonly used imaging study for bladder injuries is a flat plate cystogram. This involves three images. The first image is an x-ray prior to contrast injection. The second image is an x-ray after contrast is instilled through the Foley catheter and the Foley catheter clamped, with this image showing the distended bladder. The third image is an x-ray

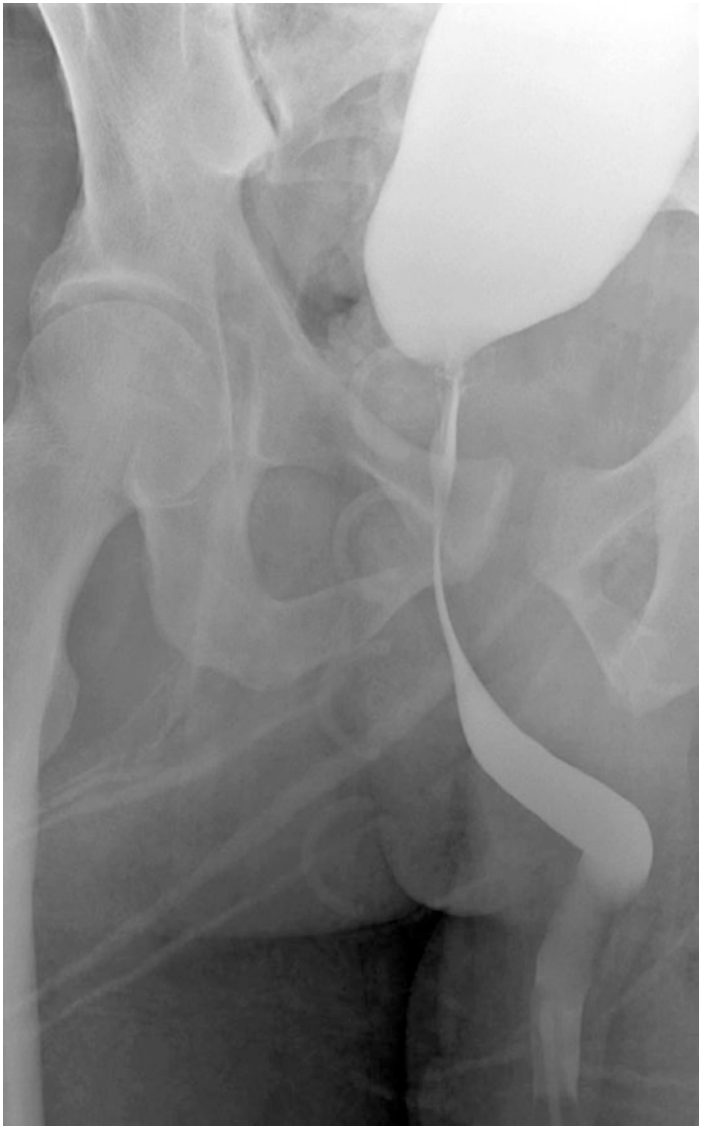

Figure 15.10 Normal retrograde urethrogram prior to Foley catheter placement for cystogram

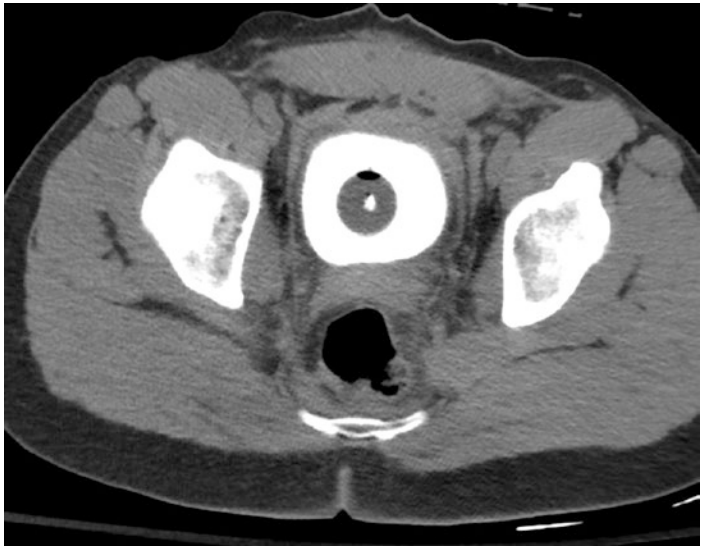

Figure 15.11 Normal CT cystogram in evaluation for bladder injury

after the Foley catheter is unclamped and the contrast allowed to drain and should show the bladder empty to rule out posterior bladder injury.

Blunt intraperitoneal bladder injuries are most often at the dome of the bladder and can be large, occurring due to overpressurization and subsequent rupture. Intraperitoneal bladder injuries are repaired at laparotomy. Prior to repair, the surgeon should look though the bladder defect and visualize the trigone. This assures that the bladder repair does not ligate a ureteral orifice. The bladder repair is usually undertaken in two layers (Fig. 15.12a, b). We favor an inner running layer of 2-0 Vicryl and outer Lembert layer of 2-0 Vicryl. We will place an intraperitoneal drain next to the repair and decompress the bladder with a Foley catheter.

Blunt extraperitoneal bladder injuries are mostly associated with pelvic fractures. These injuries can occur with direct (penetrating) injury from pelvic bone fragments or due to pelvic disruption. These extraperitoneal bladder injuries are managed with NOM with Foley catheter drainage [27].

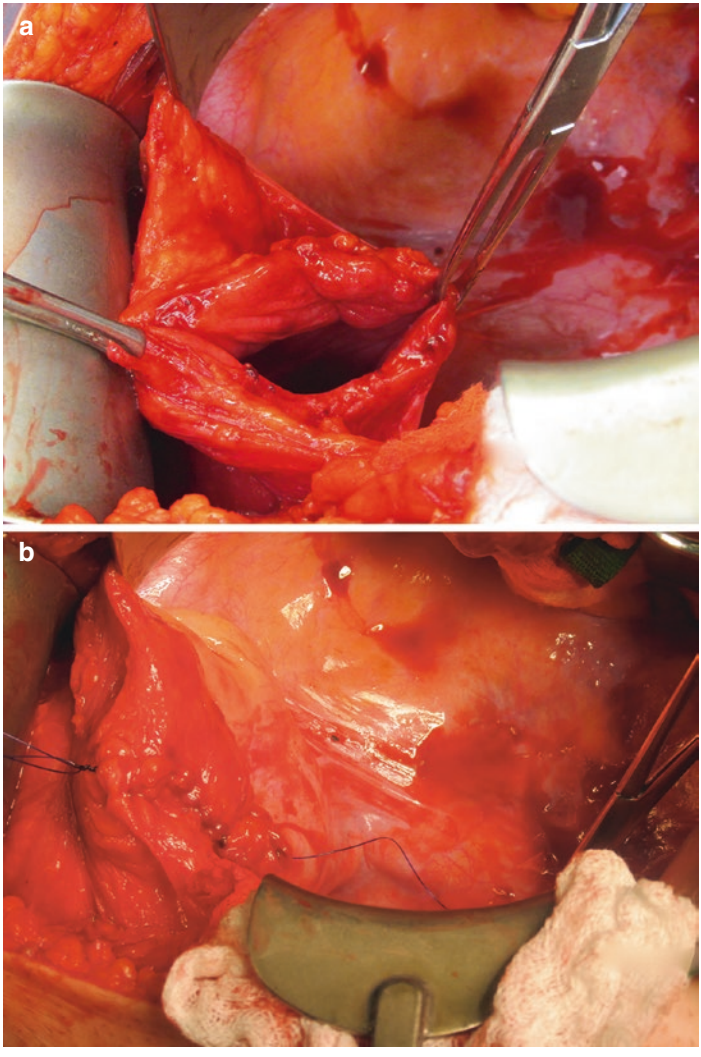

FIGURE 15.12 (**a**, **b**) Repair of an intraperitoneal bladder injury

Repair of extraperitoneal bladder ruptures can be considered when the injury is large, the patient is undergoing fixation of their pelvic fracture and the operative exposure will unroof the bladder injury, with concurrent rectal or vaginal injury, with injuries to the bladder neck, with inadequate Foley drainage, or when undergoing laparotomy for other (non-bladder) injuries [28]. In general, we will repair these extraperitoneal bladder injuries in two layers as well.

In penetrating bladder injuries, control of pelvic bleeding will take priority over the bladder repair. Penetrating bladder injuries do not occur in reproducible patterns and are dependent on the trajectory of the penetrating object. Basic concepts include identifying the trigone and evaluating multiple bladder injuries. If one penetrating bladder injury is found, the surgeon must thoroughly explore for a second, as 90–100% of gunshot wounds to the bladder traverse through the bladder [29]. This exploration can be from the outside of the bladder, either by direct exploration of the portion of the bladder at risk or by examining the inside of the bladder through a cystotomy, almost always made in the dome of the bladder. Repair is in a similar multilayer fashion as in blunt injuries.

We do not routinely obtain a post-repair cystogram in either simple blunt or penetrating bladder injuries, as leaks are not commonly demonstrated [26]. Similarly we do not routinely place a suprapubic tube and instead drain with a Foley catheter [30].

Complications in bladder injuries are infrequent. Leak is the most common postoperative complication, occurring in approximately 3% of bladder injuries [1]. This leak rate is seen without routine placement of suprapubic tubes and when managed mostly by trauma surgeons [1]. Missed injury can similarly present with urinary leakage and a variety of GU and non-GU symptoms including urinoma, sepsis, abscess, or electrolyte abnormalities [31].

References

1. Urry RJ, Clarke DL, Bruce JL, Laing GL. The incidence, spectrum and outcomes of traumatic bladder injuries within the pietermaritzburg metropolitan trauma service. Injury. 2016;47(5):1057–63.
2. Wessells H, Suh D, Porter JR, Rivara F, MacKenzie EJ, Jurkovich GJ, et al. Renal injury and operative management in the United States: results of a population-based study. J Trauma. 2003;54(3):423–30.
3. Siram SM, Gerald SZ, Greene WR, Hughes K, Oyetunji TA, Chrouser K, et al. Ureteral trauma: patterns and mechanisms of injury of an uncommon condition. Am J Surg. 2010;199(4):566–70.
4. Bjurlin MA, Goble SM, Fantus RJ, Hollowell CM. Outcomes in geriatric genitourinary trauma. J Am Coll Surg. 2011;213(3):415–21.
5. McClung CD, Hotaling JM, Wang J, Wessells H, Voelzke BB. Contemporary trends in the immediate surgical management of renal trauma using a national database. J Trauma Acute Care Surg. 2013;75(4):602–6.
6. Hotaling JM, Wang J, Sorensen MD, Rivara FP, Gore JL, Jurkovich J, et al. A national study of trauma level designation and renal trauma outcomes. J Urol. 2012;187(2):536–41.
7. Keihani S, Anderson RE, Fiander M, McFarland MM, Stoddard GJ, Hotaling JM, et al. Incidence of urinary extravasation and rate of ureteral stenting after high-grade renal trauma in adults: a meta-analysis. Transl Androl Urol. 2018;7(Suppl 2):S169–78.
8. Davis P, Bultitude MF, Koukounaras J, Royce PL, Corcoran NM. Assessing the usefulness of delayed imaging in routine followup for renal trauma. J Urol. 2010;184(3):973–7.
9. Santucci RA, Wessells H, Bartsch G, Descotes J, Heyns CF, McAninch JW, et al. Evaluation and management of renal injuries: consensus statement of the renal trauma subcommittee. BJU Int. 2004;93(7):937–54.
10. Matthews LA, Smith EM, Spirnak JP. Nonoperative treatment of major blunt renal lacerations with urinary extravasation. J Urol. 1997;157(6):2056–8.
11. Nuss GR, Morey AF, Jenkins AC, Pruitt JH, Dugi DD 3rd, Morse B, et al. Radiographic predictors of need for angiographic embolization after traumatic renal injury. J Trauma. 2009;67(3):578–82; discussion 582.

12. Charbit J, Manzanera J, Millet I, Roustan JP, Chardon P, Taourel P, et al. What are the specific computed tomography scan criteria that can predict or exclude the need for renal angioembolization after high-grade renal trauma in a conservative management strategy? J Trauma. 2011;70(5):1219–27; discussion 1227–8.
13. Lin WC, Lin CH, Chen JH, Chen YF, Chang CH, Wu SC, et al. Computed tomographic imaging in determining the need of embolization for high-grade blunt renal injury. J Trauma Acute Care Surg. 2013;74(1):230–5.
14. Hotaling JM, Sorensen MD, Smith TG 3rd, Rivara FP, Wessells H, Voelzke BB. Analysis of diagnostic angiography and angioembolization in the acute management of renal trauma using a national data set. J Urol. 2011;185(4):1316–20.
15. Prakash SV, Mohan CG, Reddy VB, Reddy VK, Kumar A, Reddy UM. Salvageability of kidney in grade IV renal trauma by minimally invasive treatment methods. J Emerg Trauma Shock. 2015;8(1):16–20.
16. Menaker J, Joseph B, Stein DM, Scalea TM. Angiointervention: high rates of failure following blunt renal injuries. World J Surg. 2011;35(3):520–7.
17. Gonzalez RP, Falimirski M, Holevar MR, Evankovich C. Surgical management of renal trauma: is vascular control necessary? J Trauma. 1999;47(6):1039–42; discussion 1042–4.
18. van der Wilden GM, Velmahos GC, Joseph DK, Jacobs L, Debusk MG, Adams CA, et al. Successful nonoperative management of the most severe blunt renal injuries: a multicenter study of the research consortium of New England centers for trauma. JAMA Surg. 2013;148(10):924–31.
19. Husmann DA, Gilling PJ, Perry MO, Morris JS, Boone TB. Major renal lacerations with a devitalized fragment following blunt abdominal trauma: a comparison between nonoperative (expectant) versus surgical management. J Urol. 1993;150(6):1774–7.
20. Baghdanian AH, Baghdanian AA, Armetta A, Babayan RK, LeBedis CA, Soto JA, et al. Utility of MDCT findings in predicting patient management outcomes in renal trauma. Emerg Radiol. 2017;24(3):263–72.
21. Frober R. Surgical anatomy of the ureter. BJU Int. 2007;100(4):949–65.
22. Pereira BM, Ogilvie MP, Gomez-Rodriguez JC, Ryan ML, Peña D, Marttos AC, et al. A review of ureteral injuries after external trauma. Scand J Trauma Resusc Emerg Med. 2010;18:6. https://doi.org/10.1186/1757-7241-18-6.

23. Ghali AM, El Malik EM, Ibrahim AI, Ismail G, Rashid M. Ureteric injuries: diagnosis, management, and outcome. J Trauma. 1999;46(1):150–8.
24. Brandes S, Coburn M, Armenakas N, McAninch J. Diagnosis and management of ureteric injury: an evidence-based analysis. BJU Int. 2004;94(3):277–89.
25. Deibert CM, Spencer BA. The association between operative repair of bladder injury and improved survival: results from the national trauma data bank. J Urol. 2011;186(1):151–5.
26. Inaba K, Okoye OT, Browder T, Best C, Branco BC, Teixeira PG, et al. Prospective evaluation of the utility of routine postoperative cystogram after traumatic bladder injury. J Trauma Acute Care Surg. 2013;75(6):1019–23.
27. Gomez RG, Ceballos L, Coburn M, Corriere JN Jr, Dixon CM, Lobel B, et al. Consensus statement on bladder injuries. BJU Int. 2004;94(1):27–32.
28. Elliott SP, McAninch JW. Extraperitoneal bladder trauma: delayed surgical management can lead to prolonged convalescence. J Trauma. 2009;66(1):274–5.
29. Cinman NM, McAninch JW, Porten SP, Myers JB, Blaschko SD, Bagga HS, et al. Gunshot wounds to the lower urinary tract: a single-institution experience. J Trauma Acute Care Surg. 2013;74(3):725–30; discussion 730–1.
30. Parry NG, Rozycki GS, Feliciano DV, Tremblay LN, Cava RA, Voeltz Z, et al. Traumatic rupture of the urinary bladder: is the suprapubic tube necessary? J Trauma. 2003;54(3):431–6.
31. Kong JP, Bultitude MF, Royce P, Gruen RL, Cato A, Corcoran NM. Lower urinary tract injuries following blunt trauma: a review of contemporary management. Rev Urol. 2011;13(3):119–30.

Part V
Techniques in Vascular Trauma

Chapter 16
Cervical Vascular Injuries: Techniques

Joseph J. DuBose

Introduction of the Problem

The complex anatomical relationships within a small area make the diagnosis and management of both penetrating (PNVI) and blunt neck vascular injuries (BNVI) challenging. Radiographic evaluation continues to evolve, with a shift from invasive to noninvasive diagnostics. Despite advances in both diagnosis and therapeutics, the optimal management of neck injuries remains a matter of active investigation.

The epidemiology of penetrating and blunt vascular injuries to the neck is distinctly different. Among penetrating injuries, firearms are responsible for about 43%, stab wounds for about 40%, shotguns for about 4%, and other weapons for about 12% [1]. Overall, about 35% of all gunshot wounds (GSWs) and 20% of stab wounds (SWs) to the neck cause significant injuries, but only 16% of GSWs and 10% SWs require surgical therapy. Even though transcervical GSWs cause significant injuries in 73% of victims, only 21% require surgery [2].

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Blunt vascular injury to the neck follows a distinctly different epidemiology. Advancements in imaging capabilities and repeated reconsideration of optimal screening criteria [3] have promoted the improved detection of these injuries. Although these changes have revealed that BVI is more common than once believed, when cervical spine injuries are excluded, injuries to the remaining structures are rare. Though uncommon, blunt cerebrovascular injuries to the vertebral and carotid arteries can be associated with significant lethality. With increased appreciation and availability of noninvasive diagnostics, the rates of these injuries are now between 1.0 and 2.0% [4–11].

History of Care of Cervical Vascular Injuries

Historically, open surgical techniques were utilized for both diagnosis and treatment of cervical vascular injuries. In this context, the division of the cervical region into three anatomical zones facilitated algorithms for evaluation and operative planning (Fig. 16.1). Zone I comprises the area between the clavicles and the cricoid cartilage. Critical structures include

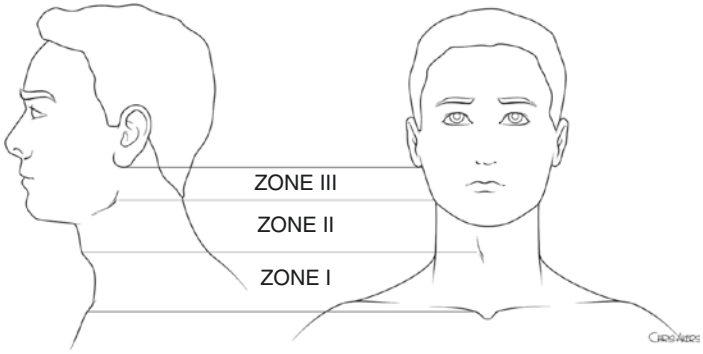

Figure 16.1 Surgical zones of the neck: Zone I is between the clavicle and the cricoid, zone II is between the cricoid and the angle of the mandible, and zone III is between the angle of the mandible and the base of the skull

the innominate vessels, the origin of the common carotid artery, the subclavian vessels and the vertebral artery, the brachial plexus, the trachea, the esophagus, the apex of the lung, and the thoracic duct. Surgical exposure in zone I can be difficult because of the presence of the clavicle and bony structures of the thoracic inlet. Zone II comprises the area between the cricoid cartilage and the angle of the mandible and contains the carotid and vertebral arteries, internal jugular veins, trachea, and esophagus. This zone is more accessible to clinical exam and surgical exploration using standard incisions than the other zones. Zone III extends between the angle of the mandible and the base of the skull and includes the distal carotid and vertebral arteries and the pharynx. The proximity to the skull base makes zone III structures less amenable to physical exam and difficult to explore. Overall, zone II is the most commonly injured area (47%) after PNI, followed by zones III (19%) and I (18%) [1]. In 16%, injuries will involve more than one zone [1].

Using these zone categorizations to guide operative planning, for many years, mandatory operation for all patients with penetrating injuries of the neck that violated the platysma was standard. The rationale was that clinical examination was not reliable. In addition, it has been suggested that routine operation avoided expensive investigations and does not prolong hospital stay [12]. Routine surgical exploration is associated with an unacceptably high incidence of unnecessary operations, however, ranging from 30 to 89% [12, 13]. Improved appreciation of the reliability of physical exam and "hard" and "soft" (Table 16.1) signs of vascular injury, combined with noninvasive diagnostic capabilities that can be utilized to investigate patients with "soft" signs of injury, has resulted in the use of selective nonoperative management at most centers [1, 14, 15].

GSWs are associated with a higher incidence of significant injuries requiring operation than SWs. However, more than 80% of GSWs to the neck do not require an operation, and there is strong evidence that these patients can be identified and spared an unnecessary operation [1, 14–17].

Table 16.1 Hard and soft signs of injury after penetrating neck trauma

Hard signs of injury	Soft signs of injury
Active arterial hemorrhage	Stable hematoma
Absent peripheral pulse on affected side	Trajectory
Expanding hematoma	Dysphagia
Air or saliva from wound	Pulse abnormality on affected side
Bruit	Nerve deficit
Hemoptysis	

Transcervical GSWs are associated with a much higher incidence of significant injuries than GSWs that have not crossed the midline (73% vs. 31%) [18]. It has been suggested that all such patients undergo exploration, irrespective of clinical exam [19]. However, many of these injuries, such as spinal cord or nerve injuries, do not require operation. In one prospective study of transcervical GSWs, 73% of patients had injuries to vital structures, but only 21% required operation [15, 18]. Several studies have demonstrated that CT angiography with thin cuts can reliably identify those patients who do not need further investigation or those who might benefit from specific studies [19–24].

Endovascular management of cerebrovascular trauma continues to evolve and has demonstrated promise in select patients. As this topic is covered well in another area of this book, the primary focus of the present contribution will be discussion of the open management of these injuries.

Technique with Personal Tips

Operative Management: Carotid Injuries

The patient is placed in slight Trendelenburg with the neck extended and the head rotated away from the side of injury. The patient should be prepped from the chin down to the

knees in anticipating the need for a thoracic incision or saphenous vein harvest. The most common incision for exposure of the unilateral carotid artery is a vertical oblique incision made over the anterior border of the sternocleidomastoid muscle (SCM), from the angle of the mandible to the sternoclavicular joint. Retracting the SCM laterally will expose the internal jugular vein, with the carotid artery lying medial and deep to the vein. The vagus nerve is located in the posterior carotid sheath. Division of the facial vein exposes the carotid bifurcation and allows mobilization and control of the internal and external carotids. Simple lacerations of the internal jugular vein or external carotid artery may be repaired, but in most cases, non essential vessels can be ligated without sequela.

Some zone I injuries may be controlled and repaired through a cervical incision, but proximal zone I injuries may require extension inferiorly into a median sternotomy. Mobilization and superior retraction of the brachiocephalic veins will expose the aortic arch, brachiocephalic artery, and proximal common carotid arteries. Care should be taken to avoid the recurrent laryngeal nerves ascending posterior to the vessels.

Zone III carotid injuries are the most difficult to expose and get distal control. The cervical incision should be extended superiorly into the posterior auricular area and the digastric muscle divided, avoiding injury to the hypoglossal, glossopharyngeal, and facial nerves. Anterior subluxation of the mandible, and further improved by mandibular osteotomy, excision of the styloid process, and removal of the anterior clinoid process improve exposure (Fig. 16.2a, b). Temporary control of uncontrolled zone I or III hemorrhage may be obtained by insertion of an embolectomy catheter through the arterial defect or an arteriotomy and inflation of the balloon.

Most external carotid injuries may be ligated without consequence. Ligation of the common or internal carotid artery can result in devastating neurologic sequelae if collaterals are inadequate. Carotid ligation should be reserved for patients in whom repair is not technically possible, such as injuries at

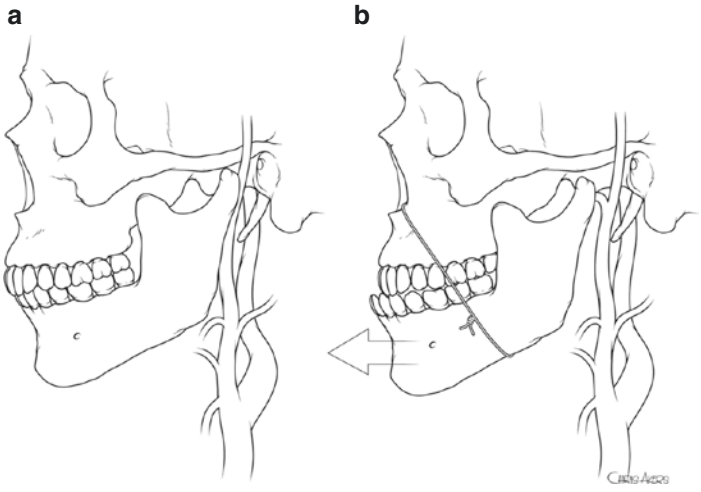

FIGURE 16.2 (**a**, **b**) Exposure of zone III carotid injuries

the base of the skull or patients with an established anemic cerebral infarction. In unstable patients, placement of a temporary intraluminal shunt and delayed reconstruction is an option.

Intravenous heparin should be administered if there are no other sites of hemorrhage or intracranial injury, preferably before clamping the artery. Alternatively, local administration of heparin at the site of injury may be used. Adequate collateral flow may not be present. Use of an intraluminal shunt to provide antegrade flow in complex repairs requiring a graft may be wise. Small lacerations may be primarily repaired using an interrupted or running suture after adequate debridement of wound edges. If primary repair is not possible, then a vein or prosthetic patch plasty of the defect is performed. Clean transections, such as stab wounds, may be repaired by mobilization of the proximal and distal artery and primary end-to-end anastomosis if this can be achieved without stenosis or tension.

Many carotid injuries, particularly from GSWs, are not amenable to primary repair or anastomosis after debridement. Reconstruction with either a vein or prosthetic interpo-

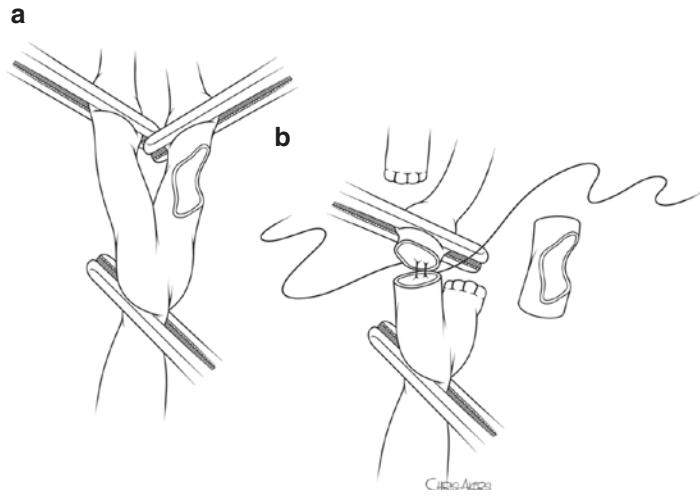

FIGURE 16.3 (**a**, **b**) Reconstruction of the proximal internal carotid by transecting the proximal external carotid artery and transposing it to the distal transected internal carotid

sition graft is needed. Saphenous vein is preferred for internal carotid artery reconstruction, with some evidence of improved patency and lower infection rates compared to prosthetic graft [25, 26]. Alternatively, reconstruction of the proximal internal carotid may be performed by transecting the proximal external carotid artery and transposing it to the distal transected internal carotid (Fig. 16.3a, b).

Common carotid artery injuries are best repaired using a thin-walled polytetrafluoroethylene graft, which has a better size match with the native artery and excellent long-term patency. An intraluminal shunt may be used here as well. If associated injuries to the aerodigestive tract have been repaired, well-vascularized tissue such as a sternocleidomastoid muscle flap should be placed between the repairs [27].

If the injury or dissection extends into the distal internal carotid artery (zone III), exposure and repair are significantly more difficult. Ligation or catheter-assisted thrombosis of the injured vessel should be considered in the asymptomatic

patient or if the appropriate expertise is not available to perform distal revascularization. Extracranial to intracranial carotid bypass may be performed but requires significant exposure of the intracranial carotid artery. Alternatively, saphenous vein bypass from the proximal internal carotid to the petrous carotid artery or middle cerebral artery has been reported. This technique avoids intracranial dissection of the carotid artery and has been associated with excellent associated long-term outcome and graft patency [25, 28].

Operative Management: Vertebral Arteries

Operative management is almost always necessary when there is severe active bleeding from the vertebral artery. The head is turned away from the injured site and the neck slightly extended. A generous incision is made on the anterior border of the SCM. The fascia is incised and the SCM retracted laterally. The omohyoid muscle is divided, and the carotid sheath is exposed and retracted, while the midline structures are retracted medially. A tissue plane anterior to the prevertebral muscles is opened, taking care to avoid the ganglia of the cervical sympathetic chain. Next the anterior longitudinal ligament is incised longitudinally. The transverse processes are palpated, and the overlying longus coli and the longissimus capitis muscle should be mobilized laterally with a periosteal elevator. The anterior aspect of the vertebral foramen is then best removed with rongeurs to expose the underlying vertebral artery. The artery can then be ligated. The cervical roots are just behind the artery, and care should be taken not to injure them. Blind clamping or clipping should be avoided. Although the artery can be identified between the transverse processes, this is technically challenging. In addition, the venous plexuses can be troublesome.

Another option for rapid control of the proximal vertebral artery is to approach it at the base of the neck where it comes off the subclavian artery. One method is to extend the incision toward the clavicle and transect the SCM off the clavicle,

retract the subclavian vein caudally, and transect or retract the anterior scalene muscle laterally. The first portion of the subclavian artery is medial, and it gives off the vertebral artery, the thyrocervical trunk, and the internal mammary muscle. The vertebral artery comes off the superior dorsal aspect of the ascending subclavian artery. When approaching the left vertebral artery, care should be taken not to injure the thoracic duct. The second method is to cut down directly on the clavicle and open the periosteum. The clavicle can be disarticulated at the sternal boarder and resected with towel clamps as a handle. This can be a rapid way of identifying the artery. Repair of the vertebral artery is extremely difficult and is not usually attempted. The collaterals are usually sufficient to not cause an ischemic stroke. When dealing with an active bleeding vertebral artery and obtaining vascular control is difficult, packing is an option if bleeding can be controlled in this manner [29].

Outcomes

Many patients with cervical vascular injuries die before reaching a hospital or present to the ED in cardiac arrest. Those surviving to reach the hospital may be completely asymptomatic and have subtle findings or active arterial hemorrhage, neck hematoma, and hemodynamic instability. Associated injuries may mask blunt cerebrovascular injury, but they should be suspected in any patient with a suspicious exam (such as the seatbelt sign—Fig. 16.4) concerning mechanism or neurologic deficits or deterioration not explained by head CT.

It should also be appreciated that a considerable percentage of patients with blunt cervical vascular injuries will be initially asymptomatic only to develop symptoms hours to days later, missing the window for intervention. In 1996, Fabian and colleagues found an average time to diagnosis of 53 h, with a range of 2–672 h [30]. The majority (78%) developed neurologic deficits prior to diagnosis. After initiating

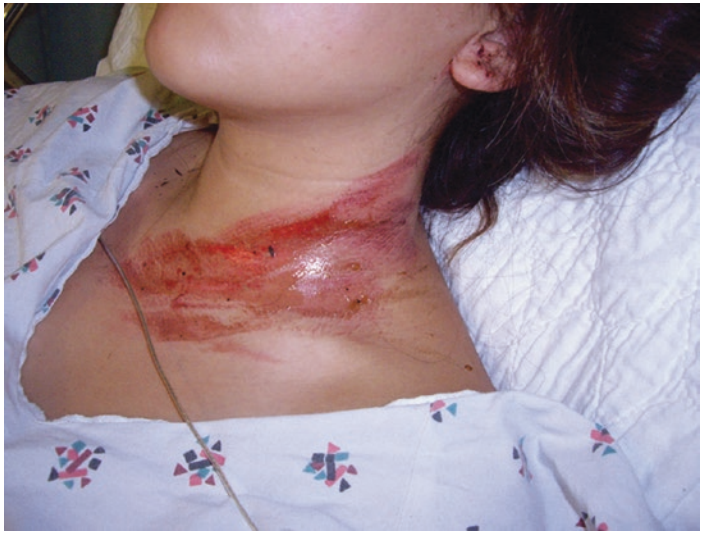

Figure 16.4 "Seatbelt sign" after motor vehicle accident

screening criteria, they demonstrated reduced mean time to diagnosis of 20 h, with 38% of injuries diagnosed based on the screening criteria alone. Only 34% of patients developed ischemic symptoms prior to diagnosis [29].

With the advancement of imaging capabilities, it has been suggested that the diagnosis of blunt cervical vascular injury is increasing. While management options for these injuries include observation, anticoagulation, antiplatelet therapy, endovascular stenting, and operation, the natural history of these lesions is not well defined. Biffl and colleagues [8, 9] have developed a widely used grading scale that can be utilized to both guide therapy and predict outcomes (Table 16.2). These investigators found that grade I injuries healed in the majority of cases with or without anticoagulation. Only 10% of grade II injuries healed with anticoagulation, with the majority (60%) progressing to grade III lesions (pseudoaneurysms) on repeat angiography. Almost all grade III lesions

Table 16.2 Grading scale for cerebrovascular injuries

Grade	Injury
Grade I	Luminal irregularity or dissection/intraluminal hematoma with <25% luminal narrowing
Grade II	Dissection or intraluminal hematoma of ≥25% of the lumen
Grade IIa	Dissection or intraluminal hematoma of 25–50% of the lumen
Grade IIb	Dissection or intraluminal hematoma of ≥50% of the lumen or intimal flap
Grade III	Pseudoaneurysm
Grade IV	Vessel occlusion
Grade V	Vessel transection

Data from References [8, 9]

(85%) remained unchanged, with 1 of 13 healing with the use of IV heparin. No grade III lesion healed without treatment.

Endovascular stents were used to treat the majority of persistent grade III lesions, with an 89% initial success rate. Grade IV injuries (occlusion) remained unchanged despite anticoagulation, but none of the patients treated with heparin developed a stroke. Grade V injuries were uniformly fatal in this series, despite attempts at angiographic embolization in two of the four patients. The authors also suggested categorizing arteriovenous or carotid-cavernous fistulae as grade II (insignificant) or grade V (hemodynamically significant).

Angiographic embolization, balloon occlusion or plasty, and stents may be used as a temporizing bridge to surgical repair for definitive treatment. Angiography with possible endovascular intervention should be considered in (1) hemodynamically stable patients with either physical exam or radiographic evidence of a distal internal carotid artery injury, (2) stable patients with evidence of an arteriovenous

or carotid-cavernous sinus fistula, (3) ongoing facial or intraoral hemorrhage from external carotid branches, and (4) small intimal defects or pseudoaneurysms in surgically inaccessible locations or high-risk surgical candidates. Stent/grafts may be particularly useful in patients with posttraumatic false aneurysms, arteriovenous fistulae, or arterial stenosis. Expanding experience with the use of interventional techniques for arterial injury is likely to better elucidate the optimal indications, timing, techniques, and outcomes [31].

There is class III evidence that systemic anticoagulation or antiplatelet therapy improves survival and neurological outcome after blunt carotid injury [28, 29], although anticoagulation may be more effective in carotid dissection than pseudoaneurysms [32]. Some studies, however, failed to show any obvious benefit from systemic heparin [33].

A recent study conducted at our own institution by Stein and colleagues highlighted the difficulties with any approach to therapy for BCVI as nearly one-third of patients were not candidates for therapy [6]. While treatment reduced the risk of infarction, strokes that did occur were not preventable. In the absence of prospective, randomized data regarding treatment, management decisions must be based on the injury pattern, associated injuries, clinical condition of the patient, and currently available literature. The algorithm for our present management approach of blunt cerebrovascular injury at R Adams Cowley Shock Trauma Center is shown in Fig. 16.5.

Complications with Treatment

Among patients who survive their initial injury, postoperative ischemic events remain the primary concern. For patients who present with or experience stroke in the setting of medical management, optimal response in the setting of coma or dense contralateral neurologic deficits remains controversial. While some earlier reports warned against revascularization in the presence of neurologic deficits due to the concern of converting an ischemic infarct to a hemorrhagic infarct

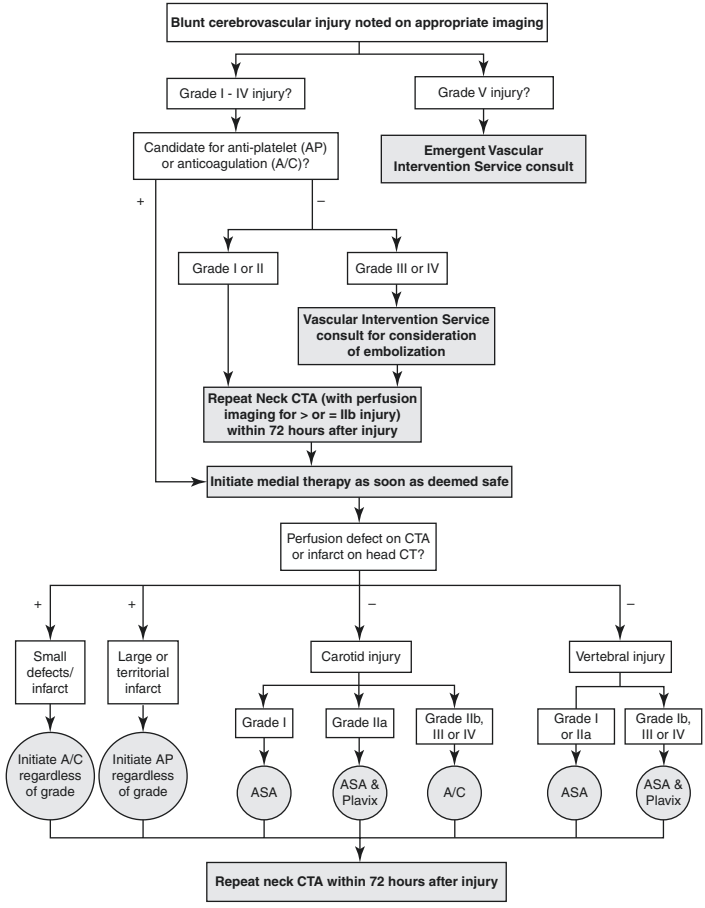

FIGURE 16.5 Initial management algorithm for blunt cerebrovascular injury at R Adams Cowley Shock Trauma Center, University of Maryland

[34], subsequent studies support that the best chance for neurologic improvement is early revascularization [35]. However, patients with coma (>4 h) have an extremely poor prognosis regardless of treatment, and revascularization often exacerbates cerebral edema and intracranial hypertension [36, 37].

Among patients undergoing endovascular or open vascular treatment of cervical neck injuries, concern for postoperative stroke or bleeding complications predominates. Post-repair ischemic events, if diagnosed early, should undergo emergent revision to restore cerebral perfusion. Postoperative hemorrhage due to repair disruption is most commonly due to technical error, but coagulopathy can also contribute to the onset of significant hematoma. Any suggestion of compressive symptoms or airway compromise demands immediate action via the establishment of a secure airway and re-exploration for hematoma evacuation and to address the cause of bleeding directly.

References

1. Demetriades D, Theodorou D, Cornwell E, Berne TV, Asensio J, Belzberg H, et al. Evaluation of penetrating injuries of the neck: prospective study of 223 patients. World J Surg. 1997;21(1):41–7; discussion 47–8.
2. Demetriades D, Theodorou D, Cornwell E, Asensio J, Belzberg H, Velmahos G, et al. Transcervical gunshot injuries: mandatory operation is not necessary. J Trauma. 1996;40(5):758–60.
3. Geddes AE, Burlew CC, Wagenaar AE, Biffl WL, Johnson JL, Peiracci FM, et al. Expanded screening criteria for blunt cerebrovascular injury: a bigger impact than anticipated. Am J Surg. 2016;212(6):1167–74.
4. Cothren CC, Moore EE, Biffl WL, Ciesla DJ, Ray CE Jr, Johnson JL, et al. Anticoagulation is the gold standard therapy for blunt carotid injuries to reduce stroke rate. Arch Surg. 2004;139(5):540–5; discussion 545–6.
5. Cothren CC, Moore EE, Biffl WL, Ciesla DJ, Ray CE Jr, Johnson JL, et al. Cervical spine fracture patterns predictive of blunt vertebral artery injury. J Trauma. 2003;55(5):811–3.
6. Stein DM, Boswell S, Sliker CW, Lui FY, Scalea TM. Blunt cerebrovascular injuries: does treatment always matter? J Trauma. 2009;66(1):132–43; discussion 143–4.
7. Miller PR, Fabian TC, Croce MA, Cagiannos C, Williams JS, Vang M, et al. Prospective screening for blunt cerebrovascular

injuries: analysis of diagnostic modalities and outcomes. Ann Surg. 2002;236(3):386–93; discussion 393–5.
8. Biffl WL, Ray CE Jr, Moore EE, Franciose RJ, Aly S, Heyrosa MG, et al. Treatment-related outcomes from blunt cerebrovascular injuries: importance of routine follow-up arteriography. Ann Surg. 2002;235(5):699–706; discussion 706–7.
9. Biffl WL, Moore EE, Offner PJ, Brega KE, Franciose RJ, Elliott JP, et al. Optimizing screening for blunt cerebrovascular injuries. Am J Surg. 1999;178(6):517–22.
10. Berne JD, Reuland KS, Villarreal DH, McGovern TM, Rowe SA, Norwood SH. Sixteen-slice multi-detector computed tomographic angiography improves the accuracy of screening for blunt cerebrovascular injury. J Trauma. 2006;60(6):1204–9; discussion 1209–10
11. Mayberry JC, Brown CV, Mullins RJ, Velmahos GC. Blunt carotid artery injury: the futility of aggressive screening and diagnosis. Arch Surg. 2004;139(6):609–12; discussion 612–3.
12. Apffelstaedt JP, Muller R. Results of mandatory exploration for penetrating neck trauma. World J Surg. 1994;18(6):917–9; discussion 920.
13. Meyer JP, Barrett JA, Schuler JJ, Flanigan DP. Mandatory vs selective exploration for penetrating neck trauma. A prospective assessment. Arch Surg. 1987;122(5):592–7.
14. Eddy VA. Is routine arteriography mandatory for penetrating injury to zone 1 of the neck? Zone 1 penetrating neck injury study group. J Trauma. 2000;48(2):208–13; discussion 213–4.
15. Demetriades D, Theodorou D, Cornwell E 3rd, Weaver F, Yellin A, Velmahos G, et al. Penetrating injuries of the neck in patients in stable condition. Physical examination, angiography, or color flow doppler imaging. Arch Surg. 1995;130(9):971–5.
16. Ordog GJ, Albin D, Wasserberger J, Schlater TL, Balasubramaniam S. 110 bullet wounds to the neck. J Trauma. 1985;25(3):238–46.
17. Hirshberg A, Wall MJ, Johnston RH Jr, Burch JM, Mattox KL. Transcervical gunshot injuries. Am J Surg. 1994;167(3):309–12.
18. van As AB, van Deurzen DF, Verleisdonk EJ. Gunshots to the neck: selective angiography as part of conservative management. Injury. 2002;33(5):453–6.
19. Inaba K, Munera F, McKenney M, Rivas L, de Moya M, Bahouth H, et al. Prospective evaluation of screening multislice helical computed tomographic angiography in the initial evaluation of penetrating neck injuries. J Trauma. 2006;61(1):144–9.

20. Osborn TM, Bell RB, Qaisi W, Long WB. Computed tomographic angiography as an aid to clinical decision making in the selective management of penetrating injuries to the neck: a reduction in the need for operative exploration. J Trauma. 2008;64(6):1466–71.
21. Nunez DB Jr, Torres-Leon M, Munera F. Vascular injuries of the neck and thoracic inlet: helical CT-angiographic correlation. Radiographics. 2004;24(4):1087–98; discussion 1099–100.
22. Armstrong WB, Detar TR, Stanley RB. Diagnosis and management of external penetrating cervical esophageal injuries. Ann Otol Rhinol Laryngol. 1994;103(11):863–71.
23. Fan ST, Lau WY, Yip WC, Poon GP, Yeung C, Wong KK. Limitations and dangers of gastrografin swallow after esophageal and upper gastric operations. Am J Surg. 1988;155(3):495–7.
24. Srinivasan R, Haywood T, Horwitz B, Buckman RF, Fisher RS, Krevsky B. Role of flexible endoscopy in the evaluation of possible esophageal trauma after penetrating injuries. Am J Gastroenterol. 2000;95(7):1725–9.
25. Vishteh AG, Marciano FF, David CA, Schievink WI, Zabramski JM, Spetzler RF. Long-term graft patency rates and clinical outcomes after revascularization for symptomatic traumatic internal carotid artery dissection. Neurosurgery. 1998;43(4):761–7; discussion 767–8.
26. Becquemin JP, Cavillon A, Brunel M, Desgranges P, Melliere D. Polytetrafluoroethylene grafts for carotid repair. Cardiovasc Surg. 1996;4(6):740–5.
27. Losken A, Rozycki GS, Feliciano DV. The use of the sternocleidomastoid muscle flap in combined injuries to the esophagus and carotid artery or trachea. J Trauma. 2000;49(5):815–7.
28. Rostomily RC, Newell DW, Grady MS, Wallace S, Nicholls S, Winn HR. Gunshot wounds of the internal carotid artery at the skull base: management with vein bypass grafts and a review of the literature. J Trauma. 1997;42(1):123–32.
29. Miller PR, Fabian TC, Bee TK, Timmons S, Chamsuddin A, Finkle R, et al. Blunt cerebrovascular injuries: diagnosis and treatment. J Trauma. 2001;51(2):279–85; discussion 285–6.
30. Fabian TC, Patton JH Jr, Croce MA, Minard G, Kudsk KA, Pritchard FE. Blunt carotid injury. Importance of early diagnosis and anticoagulant therapy. Ann Surg. 1996;223(5):513–22; discussion 522–5
31. DuBose J, Recinos G, Teixeira PG, Inaba K, Demetriades D. Endovascular stenting for the treatment of traumatic

internal carotid injuries: expanding experience. J Trauma. 2008;65(6):1561–6.
32. Biffl WL, Moore EE, Ryu RK, Offner PJ, Novak Z, Coldwell DM, et al. The unrecognized epidemic of blunt carotid arterial injuries: early diagnosis improves neurologic outcome. Ann Surg. 1998;228(4):462–70.
33. Eachempati SR, Vaslef SN, Sebastian MW, Reed RL 2nd. Blunt vascular injuries of the head and neck: is heparinization necessary? J Trauma. 1998;45(6):997–1004.
34. Thal ER, Snyder WH 3rd, Hays RJ, Perry MO. Management of carotid artery injuries. Surgery. 1974;76(6):955–62.
35. Ramadan F, Rutledge R, Oller D, Howell P, Baker C, Keagy B. Carotid artery trauma: a review of contemporary trauma center experiences. J Vasc Surg. 1995;21(1):46–55; discussion 55–6.
36. Bowley DM, Degiannis E, Goosen J, Boffard KD. Penetrating vascular trauma in Johannesburg, South Africa. Surg Clin North Am. 2002;82(1):221–35.
37. Teehan EP, Padberg FT Jr, Thompson PN, Lee BC, Silva M Jr, Jamil Z, et al. Carotid arterial trauma: assessment with the Glasgow Coma Scale (GCS) as a guide to surgical management. Cardiovasc Surg. 1997;5(2):196–200.

Chapter 17
Thoracic Vascular Injuries: Techniques

Benjamin J. Moran, Katherine Marie Kelley, and James V. O'Connor

Introduction of the Problem

Thoracic vascular trauma can present a significant clinical challenge. The relative inaccessibility of the vasculature, potential exsanguinating hemorrhage, and lack of operator experience all contribute to the difficulty in managing these potentially lethal injuries. To improve patient survival, it is

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key to recognize these injuries early and have familiarity with rapid exposure and repair techniques. This chapter will discuss the history of thoracic vascular trauma, evolution of treatment, operative techniques, management of complications, and outcomes. Emphasis will be placed on decision-making, clinical judgment, and operative techniques.

Thoracic vascular trauma is rare, occurring in less than 5% of traumatic vascular injuries, but associated with significant mortality. The majority of thoracic vascular injuries result from penetrating trauma, where all intrathoracic vessels are at risk and with significant mortality [1–5]. Blunt injuries most often involve the descending thoracic aorta, just distal to the left subclavian artery where the aorta is fixed. Patients with traumatic injuries often do not survive to the hospital; with penetrating mechanisms, there is a mortality greater than 50%, and with blunt aortic rupture, mortality is near the same [1, 6, 7]. Once these patients arrive, operative mortality is variable, ranging from 0 to 40% [8]. In contrast to penetrating trauma, there have been major advances in the treatment and survival of blunt aortic injury (BAI) with thoracic endovascular aortic repair (TEVAR), delayed operative management, and nonoperative management. Advances in computed tomographic angiography (CTA) define the nature, extent, and location of the vascular injury. Characterization of the injury is crucial in operative planning. Lastly, damage control strategies have been successfully employed in thoracic vascular trauma.

Penetrating Vascular Injury

The thorax, especially the mediastinum, contains several large arteries and veins. The aorta and its intrathoracic branches and the innominate, subclavian, and proximal carotids can all be injured from blunt or penetrating injury. Intrathoracic venous structures including the superior and inferior vena cava and right and left innominate, subclavian, proximal internal jugular, and azygos veins are all at risk. The

pulmonary artery and veins may also be injured, especially at the pulmonary hilum.

The vast majority of the thoracic great vessel injuries result from penetrating trauma, and many victims die before reaching definitive care [9]. Patients arriving with suspected great vessel injury demand rapid assessment and evaluation. Not surprisingly, those presenting in shock generally have a higher mortality, reinforcing its lethality. This subset of patients requires an immediate operation; only stable patients should undergo advanced imaging.

Initial Evaluation

All patients presenting with penetrating thoracic trauma are at risk for great vessel injury. Patients should undergo the standard ABCs of trauma care, vital signs, and a rapid physical exam with particular attention to external bleeding and expanding hematomas. An upper extremity pulse differential or absent radial pulse suggests an arterial injury, and an extremity neurologic deficit may be a result of a brachial plexus injury. A FAST examination will detect hemoperitoneum and hemopericardium. A chest radiograph will display invaluable information including a hemothorax, pneumothorax, or widened mediastinum. All trauma patients should have a type and cross match, and obtaining a lactate and arterial blood gas will prepare the patient for future transfusions and allow the clinician to understand the patient's physiology and depth of shock. Hypotension, generally defined as a systolic blood pressure < 90 mmHg, is an ominous sign and should be further worrisome if coupled with acidosis and a base deficit. Additionally, the clinician should not be fooled by hemodynamic stability, as these patients are often young, and can compensate for large volume hemorrhage, looking surprisngly good.

The crucial first decision in management is determination of shock and hemodynamic instability. Lactate and base deficit are excellent markers for presence and depth of shock. Unstable patients require immediate operative intervention.

Any additional delay in unstable treatments will only worsen mortality. Additional diagnostic studies and imaging should only be performed on stable patients. This point cannot be overemphasized; penetrating thoracic trauma with hemodynamic instability and shock requires surgery.

Hemodynamically stable patients may benefit from additional imaging. CTA has supplanted angiography as the imaging modality in thoracic trauma. It defines the nature, extent, and exact location of the injury. Information gleaned influences open versus endovascular approach and choice of incision if an open repair is indicated. CTA accurately diagnoses penetrating great vessel injuries, altering the operative approach in 25% of patients [8]. Chest CTA has been shown to be the definitive imaging study for penetrating mediastinal injuries. If trajectory warrants, patients may require bronchoscopy, esophagoscopy, or esophagram to exclude injury [10]. Blunt aortic injury is also accurately diagnosed with CTA [11].

Surgical Approach

The choice of the incision ("the incision decision") requires sound surgical judgment and is influenced by the patient's hemodynamics. Unlike abdominal exploration, which is virtually always performed through a midline laparotomy there are several surgical approaches to the thorax, each with advantages and disadvantages. Irrespective of the choice of incision, it must provide adequate surgical exposure and be versatile.

The thorax can be explored through an anterolateral incision, which can be extended across the midline as a bilateral thoracotomy or "clamshell" (Fig. 17.1a, c). This offers several advantages; it is rapid, allows excellent exposure of the anterior mediastinum and pleural spaces, and is an incision familiar to trauma surgeons. With the patient supine, a laparotomy may be concurrently performed. The disadvantages include inadequate access to posterior structures, and if a clamshell is performed,

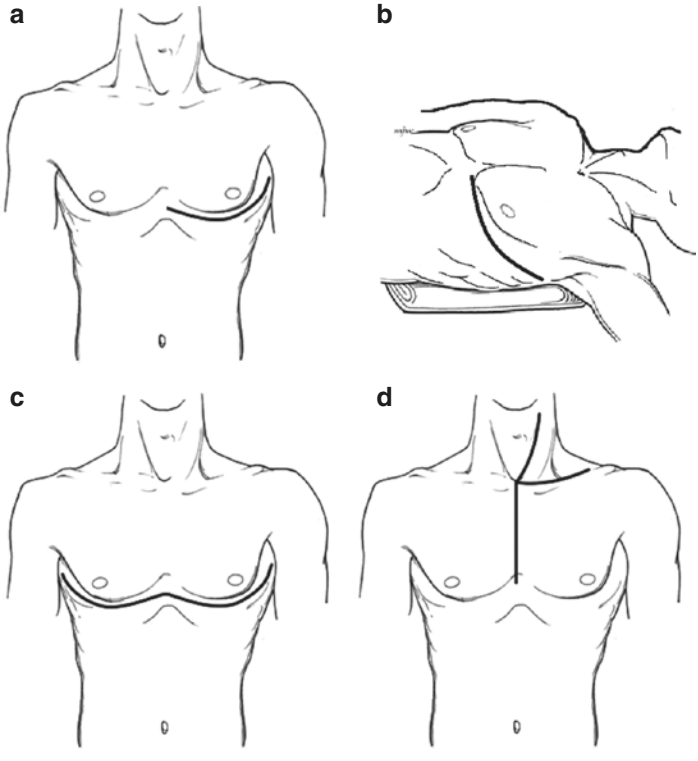

FIGURE 17.1 (**a**–**d**) Incision options for thoracic exposure. (**a**) Anterolateral thoracotomy. (**b**) Anterolateral thoracotomy with bump and extended ipsilateral arm to improve exposure of the pleural space. The incision can be extended across the sternum as a bilateral anterolateral thoracotomy ("clamshell"). (**c**) Anterolateral thoracotomy extended as a clamshell. For optimal exposure, the sternum must be divided as shown. Placing the incision more inferiorly will transect the xiphoid, thereby limiting exposure to the superior mediastinum. (**d**) Median sternotomy. It is the ideal approach to the heart and great vessels. This is a versatile incision as it can be extended to the neck or clavicle

the incision across the sternum may be placed too caudal, limiting superior mediastinal exposure. To improve exposure, extend the ipsilateral arm in a "taxi hailing" fashion, and place a bump under the back (Fig. 17.1b). When performing a clamshell thoracotomy, remember to ligate the internal mammary arteries. With profound hypotension, they may not initially be bleeding, but certainly will bleed once blood pressure is restored.

Median sternotomy is an excellent choice for mediastinal exposure (Fig. 17.1d). It is ideal for cardiac and great vessel injury and can be extended for neck or periclavicular exposure. The patient is positioned supine, so these approaches easily allow for laparotomy if indicated. A surgeon familiar with this incision can rapidly perform it, but less experienced operators may prefer the clamshell approach. As with the anterior lateral incision, sternotomy provides poor visualization of posterior structures and pleural structures. Accessing the aortic branch vessels can be accomplished through a sternotomy. The proximal right subclavian is best exposed with a sternotomy with periclavicular extension. The exposure of the left subclavian has been the subject of some debate. Our group's preference is a median sternotomy with left periclavicular extension which allows for excellent exposure of the left subclavian artery (Figs. 17.1d and 17.2) [12]. We have used this approach exclusively for left subclavian injuries, and of

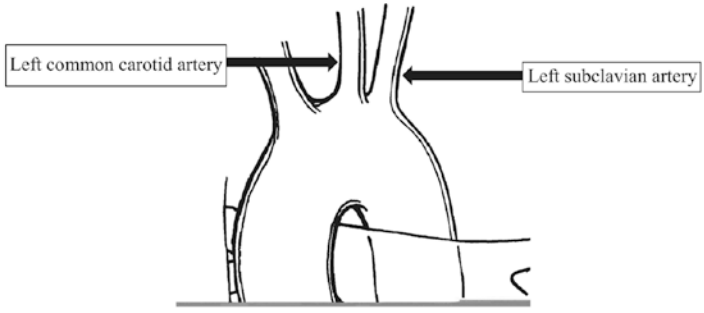

Figure 17.2 Aortic arch and branches. The left subclavian artery is adjacent to the left common carotid and can be exposed through a sternotomy with left periclavicular extension. This is the authors' preferred approach

note, clavicular resection is rarely needed [8]. We do not favor a high left thoracotomy or a trap-door incision. Sternotomy with or without cervical extension will allow for right and left carotid explorations as well.

Ultimately hemodynamically unstable patients require rapid evaluation with a thoughtful choice of incision and prompt surgical exploration. Anterolateral thoracotomy, clamshell, and sternotomy with various extensions are all acceptable incisions in the hemodynamically unstable patient.

The hemodynamically stable patient is best served with further surgical options. Unlike an emergent operation in unstable patients, this group of patients will have had imaging studies and have a defined location and nature of the vascular injury. This allows a more tailored operative approach. In addition to the anterolateral thoracotomy (unilateral or clamshell) and sternotomy, periclavicular, partial sternotomy and posterolateral thoracotomy approaches can be used. The "incision decision" is dependent on the specific injury, and again each incision has its inherent advantages and disadvantages.

A periclavicular incision has the advantages of being relatively rapid in experenced hands. It is versatile and can be extended into a sternotomy or a neck exploration (Fig. 17.3). The main disadvantage is limited exposure and, if clavicular resection is necessary, may prove more challenging than anticipated especially for surgeons with limited experience.

Posterolateral thoracotomy allows for excellent visualization of the pleural space and posterior structures and is the preferred incision for elective thoracic surgery (Fig. 17.4). Disadvantages include limited exposure of anterior mediastinum and lack of versatility. Because the patient is positioned laterally, there is no access for a laparotomy. Additionally, in lateral positioning, hypotension may be exacerbated. Single-lung ventilation will allow for excellent visualization of the pleural space. In stable patients, partial sternotomy is an attractive option for superior mediastinal exposure (Fig. 17.5). The manubrium is divided in the midline from the sternal notch passed the angle of Louis. It can be carried laterally as a "T" or "J," and a small sternal retractor

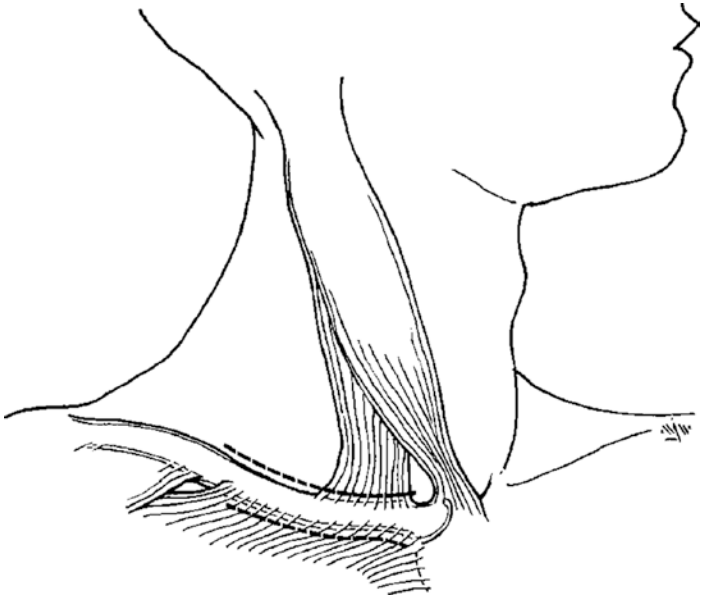

FIGURE 17.3 Periclavicular incision. Either supra- or infraclavicular approach can be used. Resecting the clavicle may be challenging and take more time than expected. Unroofing a hematoma with proximal control can be problematic

(pediatric sternal retractors work well) is placed. It affords excellent exposure of the superior mediastinum and is versatile, as it can be extended to the neck and clavicle or continued as a full sternotomy.

The ultimate choice of incision for exploring the hemodynamically stable patient will depend on the location of the injury, experience, and surgical judgment.

Intraoperative Management

Operative management of intrathoracic vascular trauma depends on the specific vessel injured and the patient's clinical condition. After large-volume blood loss in the chest, patients

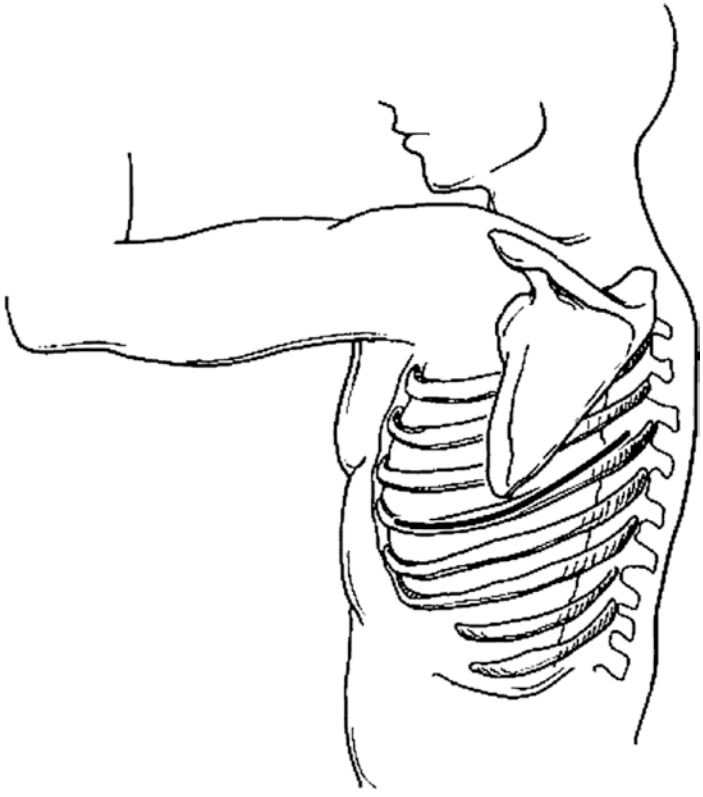

FIGURE 17.4 A posterolateral approach is preferred for elective thoracic operations. Limited exposure of the anterior mediastinum and lack of versatility are its disadvantages. Exposure of the hemithorax is greatly improved with double lumen tube and lung isolation

are often hypotensive, acidotic, hypothermic, and coagulopathic. In those patients, similar to patients with severe abdominal injury, damage control surgery is an attractive option. Using damage control thoracic techniques, hemorrhage is controlled, and the thorax is packed and temporarily closed. Resuscitation and rewarming are continued in the ICU. Once near-normal physiology is achieved, planned re-exploration and closure should be performed [13]. With the expanding role of catheter-

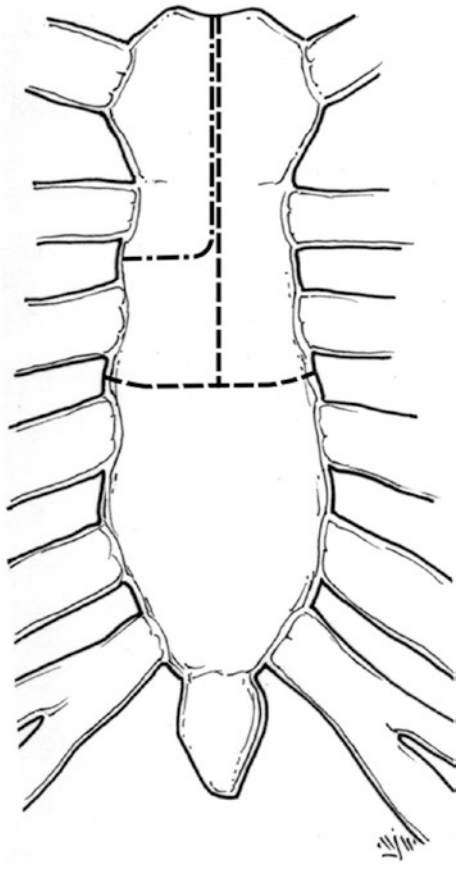

Figure 17.5 Partial sternotomy. This is an attractive option in stable patients. Carrying the sternotomy distal to the angle of Louis generally provides excellent exposure to the superior mediastinum. Either a "T" or "J" lateral division of the sternum can be utilized. This incision is versatile as it can be extended to the neck and clavicle or continued as a full median sternotomy

based therapy, both open and endovascular approaches should be considered. They may be done in conjunction with open operative care often as part of damage control thoracic surgery. Endovascular balloon occlusion is a sophisticated inflow control option, allowing control without extensive dissection in challenging anatomic locations. This is particularly helpful when treating proximal subclavian injuries.

Although thoracic venous injuries may be repaired, it can be time-consuming and often results in venous thrombosis

and possible embolization. The crucial exceptions are injuries of the superior and inferior vena cava, which must be repaired without lumen compromise, if at all possible. Thoracic veins can be ligated with little clinical consequence, but if ligation is performed, indwelling venous catheters must be removed prior to venous ligation. Be sure to coordinate with your anesthesia colleagues prior to ligation to verify removal and location of central lines. Facial or extremity edema is the sequela of venous ligation, which is managed by elevation and generally resolves within days.

Arterial injuries should always be repaired, if possible, with ligation reserved for life-saving measures for uncontrollable hemorrhage. There are multiple options for primary and delayed arterial repair. They include primary repair, patch angioplasty, graft interposition, and temporary shunting with delayed repair. Low-energy injuries (stab wounds) can often be repaired primarily with or without resection as indicated. Patch angioplasty can be accomplished with autologous vein or prosthetic material. Gunshot wounds result in significant tissue injury and generally necessitate resection with end-to-end interposition grafting. Contrary to the use of bypass grafting for diffuse atherosclerotic disease, penetrating injuries are localized and amenable to graft interposition. Formal bypasses are almost never used. Either autologous vein or prosthetic graft can be used. Size match and wound contamination will influence the choice of conduit. Vein is the preferred conduit in a grossly contaminated wound or if the graft traverses a joint. It is important to evaluate and assess the extent of injury, especially in high-energy mechanisms, and to resect back to healthy tissue so that the bypass graft will be sewn to healthy edges.

The principles of damage control surgery apply in vascular trauma [14]. Performing an arterial reconstruction in the face of profound acidosis, hypothermia, and coagulopathy is ill advised. The alternative approach is placing a temporary vascular shunt, physiologic resuscitation in the intensive care unit, and a delayed and planned definitive arterial reconstruction with restoration of normal physiologic parameters.

Preoperative planning, adherence to established principles, and a precisely performed technical repair are all crucial to a successful outcome. Patients should be widely prepped and draped, including the torso and extremities for vein harvesting. As discussed previously, the choice of incision is paramount. Proximal and distal vascular control is obtained directly or in combination with proximal balloon occlusion. Once the injury is isolated, devitalized tissue is excised; this point cannot be stressed enough when treating gunshot wounds. Resection back to visualized healthy and normal intima is imperative because a repair to damaged intima will fail. Next, inflow and back-bleeding must be assessed, and if inadequate, a thrombectomy should be performed. Since most individuals with penetrating trauma are young, inadequate inflow is not from atherosclerotic lesions but from a proximal thrombus. Back-bleeding may be more difficult to assess, and if poor, a thrombectomy should be performed. If thrombectomy fails to improve back-bleeding, an intraoperative angiogram will define the distal anatomy. An angiogram will show one of two things. If distal vessels are patent and poor retrograde flow is due to lack of collateral vessels, the arterial reconstruction may proceed. Conversely, if the distal vessels are occluded, additional procedures such as distal vessel exploration with thrombectomy are warranted. No matter the technique of arterial repair, the anastomosis must be tension-free.

At the conclusion of the arterial repair, vessel patency must be confirmed. While interrogation with a Doppler is often performed, the presence of a palpable distal pulse will confirm a patent vessel. An absent palpable pulse should prompt an intraoperative angiogram and, if abnormal, will allow for the rapid correction of any technical problem. A good rule following arterial reconstruction is the patient should leave the operating room with “a pulse or a picture.”

The role of anticoagulation in the management of vascular trauma is debated, especially among patients with head trauma or significant associated injuries. Systemic anticoagulation is ideal prior to the application of vascular clamps;

however, if systemic anticoagulation is contraindicated, flushing the arterial tree with heparinized saline is a viable option. Systemic anticoagulation should not be reversed. Similar to anticoagulation, the role of postoperative antiplatelet agents is not well defined. It is our practice, if no contraindication, to keep the patient on a low-dose aspirin for 1 month postoperatively.

The role of endovascular treatment of thoracic vascular trauma is rapidly evolving. Although these techniques are more commonly used in the treatment of blunt injuries, which are discussed below, there is a growing application in penetrating vascular trauma. Pseudoaneurysms, arterial-venous fistulae, occlusion, and transection, resulting from penetrating trauma, have all been successfully treated using endovascular techniques [15]. The advantages are obvious, definitive treatment without thoracotomy or sternotomy, thereby avoiding the associated operative complications. There is, however, attendant morbidity with endovascular therapy; the two most common are endoleaks and insertion site vascular complications. These will be discussed in more detail below. The role of endovascular therapies continues to expand as devices improve and more experience is gained using this modality. Please see the endovascular chapter (Chap. 19) for further information.

Personal Tips for Specific Vascular Injuries

The intercostal arteries are commonly injured arteries with penetrating trauma. Surgery to control hemorrhage from intercostal injury is the most common indication for thoracotomy following penetrating injury. Additionally, many return trips to the operating room are results from missed intercostal artery injuries. The intercostal vascular bundle runs on the inferior border of the rib, making it difficult for arterial isolation. Our preferred method to control these is placing a suture, usually 0 or #1 Vicryl suture, around the rib and reentering the chest at the top of the lower rib, similar to

a pericostal suture for chest closure. When the suture is tied down, the intercostal artery and vein are cinched up against the rib and ligated. It is important to ligate both proximally and distally from the point of injury, as the distal vessel may bleed from collaterals if not ligated. If this is difficult, exposure may be enhanced by simply opening the entrance wound and dissecting down onto the chest wall. The suture can be passed from the chest out into the wound and then back again into the chest with a greater degree of accuracy. If hemostasis is being attempted using a thoracoscopic approach, this same technique may be used with a suture passer inserted either through an enlarged tract or through separate stab incisions. We prefer using the orthopedic passer. In posterior intercostal injuries, the rib space is narrower, making the posterior area more difficult to control from an anterior exposure. In this scenario, temporizing and performing angiographic embolization may be helpful. Lastly, bleeding from an intercostal artery may stop when compressed following placement of a chest or sternal retractor. Removing the retractor and establishing exposure with handheld retractors is helpful and can allow visualization of the bleeding vessel.

Internal mammary arteries are also under systemic pressure and bleed accordingly. One of the most common reasons internal mammary arteries bleed is they are divided during a clamshell thoracotomy. As these patients are often in extremis, the internal mammary arteries may not bleed with arterial pressure. If resuscitation is successful, these arteries can bleed impressively as do the intercostals. Bleeding from the internal mammary may be minimized by placement of a sternal retractor. It is important to examine these with the retractor removed. As with the intercostals, treatment for a mammary artery is ligation.

Bleeding from the large mediastinal arteries can produce exsanguinating hemorrhage. Regardless of the exposure used, control is often difficult. Initial control with digital pressure can be very helpful until obtaining formal control. Additionally, a side-biting vascular clamp may stem hemorrhage until the vessels are dissected free. The innominate and right subcla-

vian arteries are best exposed through a sternotomy with clavicular extension if necessary. It is important to maintain flow via the right carotid artery, if possible, to avoid cerebral hypoperfusion.

Injuries to the pulmonary vascular system are fortunately rare but result in impressive bleeding. Because of the low pressure in the pulmonary circuit, and limited muscle in the vessel wall, bleeding from the pulmonary artery acts similarly to bleeding from a major vein. Due to the limited vessel media, the pulmonary system does not develop vasospasm. Several of the techniques mentioned in the vascular chapter can be helpful. In particular, injuries to the side of these vessels can often be controlled with intestinal Allis clamps. The clamps can be used to control hemorrhage from the injury without occluding the pulmonary artery or vein. The injury can then be repaired under the clamps. Inflow control for pulmonary artery or venous injuries may be expedited by opening the pericardium. Controlling the vessels within the pericardium allows the surgeon to gain inflow control without attempting to expose the area of injury.

Blunt Vessel Injury

One area which has seen a dramatic change in both diagnosis and management is blunt aortic injury (BAI). Historically, this highly lethal injury was suspected by mechanism, such as a motor vehicle collision with rapid horizontal deceleration, a wide mediastinum on plain chest radiograph, and the diagnosis confirmed by aortography. Except in special circumstances, such as an associated severe head injury, the treatment was operative repair, with cardiopulmonary support, distal arterial perfusion, or, without cardiopulmonary support, the "clamp and sew" technique.

In the current era, CTA is the screening modality of choice to define the extent of injury and determine if more selective management is warranted. Options include medical management without operative intervention, delayed intervention,

and the use of thoracic endovascular aortic repair (TEVAR) and open repair. This shift in practice is dramatically demonstrated by two studies, a decade apart, from the American Association for the Surgery of Trauma (AAST). In the first study published in 1997, no patient had a TEVAR, 65% had repair performed with cardiopulmonary support, and the remainder had the "clamp and sew" technique. Overall morality was 31% with a paraplegia rate of 8.7% [16]. The second AAST report in 2008 revealed a dramatic change in practice. Almost two-thirds of patients had TEVAR; the remainder were managed by open repair, and of those, 80% were performed with cardiopulmonary bypass support. Mortality and paraplegia were 13% and 1.6%, respectively [17]. The mortality and complication rates for TEVAR have continued to decrease as devices and techniques have improved [18]. While these data are encouraging, long-term TEVAR data with regard to endograft durability, patency, and complication rates are lacking.

The evaluation, management, and operative therapy for BAI will be thoroughly addressed in Chap. 19 on Endovascular Therapy in Trauma. In brief, the landmark study by Parmley defined the lethality of BAI [6]. Operative management with or without cardiopulmonary support was the accepted treatment. Subsequent studies describing initial medical management for blood pressure and heart rate control followed by delayed aortic repair showed improved outcomes [19]. This concept has expanded to include medical management alone for the management of minimal BAI [20–22]. Determining which injuries can be successfully managed medically requires high-quality imaging and sound surgical judgment. Blunt aortic injury in the multiply injured patient presents a challenge as there may be competing priorities. High-resolution CT scans now detect small aortic injuries previously undetected with older technology. Grading systems have been developed which guide the clinician [20, 22, 23]. Medical management alone, medical management followed by delayed repair, and urgent or emergent intervention either open or TEVAR are all options. Medical management is undertaken 32% of the

time although it rarely requires subsequent operation. TEVAR is the most common treatment choice and is utilized in 52% of cases. Open repair is only utilized in 16% of cases and is associated with a higher ISS and requiring a thoracotomy for another injury [18]. The combination of hemodynamics, associated injuries, and CT imaging will help guide a sound clinical decision.

Pediatrics represents a particularly challenging population in the area of BAI. Fortunately, this is a rare injury in pediatric patients [24]. Open repair remains the gold standard for aortic repair in children; however, the success seen with adults and similar concomitant injuries seen in pediatric patients have led to increasing interest in endovascular treatment of pediatric aortic injuries. One challenge specific to children is the smaller-size vessels both in the aorta and in the iliacs used for access. Another difficulty is ensuring that the graft will continue to match the size and shape of the aorta as the child grows [25–28]. Finally, as mentioned above, long-term graft patency and durability remain unknown. Attempts to surmount these technical challenges have included using devices designed for iliac vessels and novel devices [25, 28].

The main concern with medical management is the risk of aortic rupture. In one report, no mortality resulted from the aortic injury but rather was secondary to associated injuries [22]. However, a more recent study showed a 9.8% rate of aortic mortality in patients managed nonoperatively [18]. Complications associated with open repair are those common to any operation. A dreaded complication specific to the procedure is paraplegia, which is decreased when cardiopulmonary bypass or left atrial to femoral bypass is employed. Complications related to TEVAR can be divided into those which are device related, such as endoleaks, and vascular complications related to catheter insertion [20, 22].

While there is a larger experience with endovascular techniques to treat BAI, the same modality has been applied to traumatic injuries to peripheral arteries. Some published reports compare results of endovascular to open treatment [29, 30]. Care must be taken when comparing these non-

randomized reports, since there is a selection bias. Unstable patients are more likely to undergo open repair, while a catheter-based approach is used in the stable patient. Alternatively, there are observational studies describing the use of endovascular techniques [15, 31]. The promising results with endovascular therapy, device innovations, and increasing experience in treating traumatic vascular injuries will continue to expand the role for this modality.

Complications

Complications associated with the operative management of thoracic vascular trauma can be divided into those related to any operation and those specific to the individual procedure. Complications related to the former include bleeding, atelectasis, respiratory failure, and renal failure. Persistent chest tube drainage is an indication for re-exploration. Wound infections can be considered as a procedure-specific complication. Multiple sources of chest wall vasculature make thoracotomy infections infrequent and generally managed well by local wound care. The management of sternal wound infections and sternal dehiscence depends on the clinical setting. Dehiscence without a sternal infection may not require operative repair unless symptoms persist. Superficial sternal infections, without dehiscence, can be managed by local wound care and antibiotics. Deep sternal infections are a challenging problem. Reoperation, debridement of nonviable tissue including the sternum, culture-specific antibiotics, and wound care are appropriate. Vacuum-assisted wound care (VAC) has been used with excellent results, either as definitive therapy or as an intermediate step prior to muscle flap coverage [32]. The pectoralis is ideal if muscle flap coverage is needed [33].

Empyema, while infrequent, is a serious complication following thoracotomy. The diagnosis is suspected in the postoperative patient with fever, leukocytosis, a persistent opacity on chest radiograph, and difficulty weaning from the ventila-

tor. Enhancing parietal pleura on chest CT is suggestive of an empyema. The diagnosis is confirmed by thoracentesis and cultures, which guide antibiotic therapy. Interventional catheter-based techniques or decortication with VATS may be useful for early-stage empyema. The more advanced organized stages more often require thoracotomy and decortication [34]. Whichever technique is chosen, the principles of complete drainage, decortication, full lung expansion, and appropriate antibiotics are the principles to be followed.

Edema resulting from venous ligation is self-limiting and managed by elevation. Serious complications related to arterial repair include bleeding and limb ischemia from thrombosis, both of which often necessitate re-exploration. Frequent vascular checks are essential, and any decrease in pulse should be investigated with an imaging study, CTA, arterial duplex, or angiography, particularly if a catheter-based therapy is contemplated. An absent pulse and a threatened limb require intervention including lytic therapy, catheter-based therapy, and, more frequently, exploration and revision. Infections involving the vessel conduit can result in life-threatening hemorrhage. Pseudoaneurysm formation and anastomotic dehiscence are the dreaded sequela of infection. Vein grafts are more resistant, but not immune, to infection. Thorough debridement of devitalized tissue, adequate hemostasis, and covering the graft with viable tissue will lessen the chance of infection. Arterial duplex surveillance of the graft will detect a pseudoaneurysm.

In addition to the complications associated with an invasive procedure, catheter-based therapies have specific complications. A comprehensive discussion is found in Chap. 19 on Endovascular Therapy in Trauma, with a more limited review here. Endovascular complications may occur during the procedure or post-procedure. Endoleaks and access complications are among the more common morbidities encountered. Endoleaks may be an early or late complication and are classified as Type l, incomplete proximal or distal sealing of the graft; Type 2, flow from collateral vessels; Type 3, rupture of graft fabric or junctional separation; and Type 4, graft

porosity. Access complications are commonly discovered intraoperatively or shortly thereafter and require an open operative repair [35]. Another complication associated with TEVAR is coverage of the subclavian artery which occurs 41% of the time. Coverage of the subclavian artery is concerning for the development of steal syndrome and increased risk of stroke. Steal syndrome requiring intervention was not seen in the 2015 study by Dubose, but there was a 2.4% rate of stroke in patients with a covered subclavian artery [18]. Complications should decrease over time as devices become more sophisticated and clinicians gain more experience with this modality.

Conclusions

Trauma to thoracic vessels presents a formidable challenge. Life-threatening hemorrhage, the relative inaccessibility of the vessels, and difficulty obtaining adequate exposure culminate in a daunting clinical problem. The clinician is faced with several crucial decisions, which have a profound impact on the patient's outcome. First is determining hemodynamic instability and the presence of shock. While the presence of hypotension is an ominous finding, tissue hypoperfusion and shock may occur in its absence. In addition to initial vital signs, serum lactate and base deficit measurements are critically important. Hemodynamic instability or shock should prompt emergent exploration. Only those patients who are stable are suitable for advanced imaging. Once the determination is made for surgical exploration, the next important decision is the optimal incision ("the incision decision"). The surgeon must be familiar with all surgical approaches and their attendant advantages and disadvantages and, given the clinical situation, choose the appropriate incision. Having obtained appropriate exposure and identifying the vascular trauma, the next decision is the management of the specific injury. Again, the surgeon must understand the multiple options available and, in the context of the patient's overall

clinical condition, determine the best course of action. If the patient is in extremis, a damage control procedure may be optimal. Additionally, the surgeon must be familiar with endovascular techniques and their indications. Complications will not be infrequent among this patient population. Sound clinical judgment and adherence to fundamental surgical principles will improve survival and decrease morbidity.

References

1. Demetriades D, Rabinowitz B, Pezikis A, Franklin J, Palexas G. Subclavian vascular injuries. Br J Surg. 1987;74:1101–3.
2. Fulton JO, de Groot KM, Buckels NJ, von Oppell UO. Penetrating injuries involving the intrathoracic great vessels. S Afr J Surg. 1997;35:82–6.
3. Kalakuntla V, Patel V, Tahoe A, Weaver W. Six-year experience with management of subclavian artery injuries. Am Surg. 2000;66:927–30; discussion 930–1.
4. Lin PH, Koffron AJ, Guske PJ, Lujan HJ, Heilizer TJ, Yario RF, et al. Penetrating injuries of the subclavian artery. Am J Surg. 2003;185:580–4.
5. Demetriades D, Chahwan S, Gomez H, Peng R, Velmahos G, Murray J, et al. Penetrating injuries to the subclavian and axillary vessels. J Am Coll Surg. 1999;188:290–5.
6. Parmley LF, Mattingly TW, Manion WC, Jahnke EJ Jr. Nonpenetrating traumatic injury of the aorta. Circulation. 1958;17:1086–101.
7. Von Oppell UO, Bautz P, De Groot M. Penetrating thoracic injuries: what we have learnt. Thorac Cardiovasc Surg. 2000;48:55–61.
8. O'Connor JV, Scalea TM. Penetrating thoracic great vessel injury: impact of admission hemodynamics and preoperative imaging. J Trauma. 2010;68(4):834–7.
9. Demetriades D. Penetrating injuries to the thoracic great vessels. J Card Surg. 1997;12:173–9; discussion 179–80.
10. Burack JH, Kandil E, Sawas A, O'Neill PA, Sclafani SJ, Lowery RC, et al. Triage and outcome of patients with mediastinal penetrating trauma. Ann Thorac Surg. 2007;83(2):377–82; discussion 382.
11. Mirvis SE, Shanmuganathan K, Miller BH, White CS, Turney SZ. Traumatic aortic injury: diagnosis with contrast-enhanced

thoracic CT—five year experience at a major trauma center. Radiology. 1996;200:413–22.
12. Hajarizadeh H, Rohrer MJ, Cutler BS. Surgical exposure of the left subclavian artery by median sternotomy and left supraclavicular extension. J Trauma. 1996;41:136–9.
13. O'Connor JV, DuBose JJ, Scalea TM. Damage control thoracic surgery: management and outcomes. J Trauma Acute Care Surg. 2014;77(5):660–5.
14. Gifford SM, Aidinian G, Clouse WD, Fox CJ, Porras CA, Jones WT, et al. Effect of temporary shunting on extremity vascular injury: an outcome analysis from the global war on terror vascular injury initiative. J Vasc Surg. 2009;50(3):549–55.
15. Dubose JJ, Rajani R, Gilani R, Arthurs ZA, Morrison JJ, Clouse WD, et al. Endovascular Skills for Trauma and Resuscitative Surgery Working Group. Endovascular management of axillosubclavian arterial injury: a review of published experience. Injury. 2012;43(11):1785–92.
16. Fabian TC, Richardson JD, Croce MA, Smith JS Jr, Rodman G Jr, Kearney PA, et al. Prospective study of blunt aortic injury: multicenter trial of the American Association for the Surgery of Trauma. J Trauma. 1997;42(3):374–80; discussion 380–3.
17. Demetriades D, Velmahos GC, Scalea TM, Jurkovich GJ, Karmy-Jones R, Teixeira PG, et al. Operative repair or endovascular stent graft in blunt traumatic thoracic aortic injuries: results of an American Association for the Surgery of Trauma Multicenter Study. J Trauma. 2008;64(3):561–70; discussion 570–1.
18. DuBose JJ, Leake SS, Brenner M, Pasley J, O'Callaghan T, Luo-Owen X, et al. Contemporary management and outcomes of blunt thoracic aortic injury: a multicenter retrospective study. J Trauma Acute Care Surg. 2015;78(2):360–9.
19. Akins CW, Buckley MJ, Daggett W, McIlduff JB, Austen WG. Acute traumatic disruption of the thoracic aorta: a ten year experience. Ann Thorac Surg. 1981;31:305–9.
20. Neschis DG, Scalea TM, Flinn WR, Griffith BP. Blunt aortic injury. N Engl J Med. 2008;359:1708–16.
21. Malhotra AK, Fabian TC, Croce MA, Weiman DS, Gavant ML, Pate JW. Minimal aortic injury: a lesion associated with advancing diagnostic techniques. J Trauma. 2001;51:1042–8.
22. Rabin J, Dubose J, Sliker CW, O'Connor JV, Scalea TM, Griffith BP. Parameters for successful nonoperative management of traumatic aortic injury. J Thorac Cardiovasc Surg. 2014;147(1):143–9.

23. Mosquera VX, Marini M, Lopez-Perez JM, Muniz-Garcia J, Herrera JM, Cao I, et al. Role of conservative management in traumatic aortic injury: comparison of long-term results of conservative, surgical and endovascular treatment. J Thorac Cardiovasc Surg. 2011;142:614–62.
24. Heal ME, Chowdhury SM, Bandisode VM. Balloon-expandable covered stent implantation for treatment of traumatic aortic pseudoaneurysm in a pediatric patient. J Thorac Cardiovasc Surg. 2016;152(5):e109–11.
25. Menini Stahlschmidt CM, Von Bahten LC, Nicoluzzi L, Corvello A, Stahlschmidt FL, Guimaraes F. Successful endovascular management of a traumatic aortic rupture in a pediatric patient: case report and literature review. Ulus Travma Acil Cerrahi Derg. 2010;16:84–6.
26. Gunabushanam V, Mishra N, Calderin J, Glick R, Rosca M, Krishnasastry K. Endovascular stenting of blunt thoracic aortic injury in an 11-year-old. J Pediatr Surg. 2010;45:E15–8.
27. Martin MA, Barnatan M, Cole F, Long W, Hill J, Karmy-Jones R. A case report of traumatic aortic rupture in a pediatric patient: a possible role for endovascular management as a bridge to definitive repair. J Trauma. 2009;67:E136–9.
28. Keyhani K, Al E, Safi HJ, Azizzadeh A. Endovascular repair of traumatic aortic injury in a pediatric patient. J Vasc Surg. 2009;50(3):652–4.
29. Xenos ES, Freeman M, Stevens S, Cassada D, Pacanowski J, Goldman M. Covered stents for injuries of subclavian and axillary arteries. J Vasc Surg. 2003;38(3):451–4.
30. Carrick MM, Morrison CA, Pham HQ, Norman MA, Marvin B, Lee J, et al. Modern management of traumatic subclavian artery injuries: a single institution's experience in the evolution of endovascular repair. Am J Surg. 2010;199(1):28–34.
31. O'Connor JV, Byrne C, Scalea TM, Griffith BP, Neschis DG. Vascular injuries after blunt chest trauma: diagnosis and management. Scand J Trauma Resusc Emerg Med. 2009;17:42.
32. Agarwal JP, Ogilvie M, Wu LC, Lohman RF, Gottlieb LJ, Franczyk M, et al. Vacuum-assisted closure for sternal wounds: a first-line therapeutic management approach. Plast Reconstr Surg. 2005;116:1035–40.
33. Jones G, Jurkiewicz MJ, Bostwick J, Wood R, Bried JT, Culbertson J, et al. Management of the infected median sternotomy wound with muscle flaps: the Emory 20-year experience. Ann Surg. 1997;225:766–78.

34. O'Connor JV, Chi A, Joshi M, Dubose J, Scalea TM. Post- traumatic empyema: aetiology, surgery and outcome in 125 consecutive patients. Injury. 2013;44(9):1153–8.
35. Lee TC, Hughes GC. Complications of thoracic endovascular stent grafts. In: Franco KL, Thourani VH, editors. Cardiothoracic surgery review. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2012.

Chapter 18
Abdominal Vascular Injuries: Techniques

David V. Feliciano

Introduction of the Problem

The major cause of intraoperative or early postoperative deaths during and after laparotomies for trauma is hemorrhagic shock from injury to a major named vessel, the liver, or multiple organs/vessels. The unique problems when managing abdominal vascular injuries are listed in Box 18.1. These explain the significant mortality rates for abdominal arterial injuries (35–65%) and venous injuries (28–55%) [1].

Box 18.1: Unique Problems when Managing Abdominal Vascular Injuries

- Volume of flow
- Lack of tamponade (especially pelvis and mesentery after gunshot wounds)
- Difficulty in exposure
  - Overlying organs
  - Location in retroperitoneum
- Presence of multiple other intra-abdominal injuries
- Associated gastrointestinal contamination
- Surgeon's lack of open experience with vascular surgery

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History of Care of Abdominal Vascular Injuries

When compared to all vascular injuries in previous wars, abdominal vascular injuries accounted for only 2% in World War II, 2.3% in the Korean War, 2.9% in Vietnam War, and 2.8% in Operation Iraqi Freedom [2–5]. These low figures are explained by the high kinetic energy of missiles fired from military rifles and delays in transport to an operating room. And, in Iraq, the presence of truncal armor for American troops was surely a factor. With the low kinetic energy of civilian handguns (<1000 ft-lbs), abdominal vascular injuries are much more common in the United States.

Clinical Presentation

An abdominal vascular injury may present in one of the following ways: (1) intraperitoneal hemorrhage; (2) a contained mesenteric, retroperitoneal, or portal hematoma; (3) a leaking hematoma combined with hemorrhage; or (4) thrombosis of a vessel. Patients with hemorrhage or hematoma-hemorrhage, particularly from an arterial injury, usually present with hypotension and a failure to respond to the infusion of blood or crystalloid solutions. With a delay to operation, abdominal distension may develop. In the patient with a contained hematoma, particularly from a venous injury such as to an iliac vein or the inferior vena cava, modest hypotension may be reversed with resuscitation. In some patients, normotension after resuscitation may persist until the hematoma is opened at a laparotomy. The rare patient with thrombosis of a major named artery will have delayed symptoms related to the artery involved. Examples would be as follows: (1) loss of pulses in the lower extremities with blunt thrombosis of the abdominal aorta; (2) loss of ipsilateral pulses in a lower extremity with thrombosis of the common or external iliac artery; (3) severe abdominal pain with thrombosis of the proximal superior mesenteric artery; and (4) hematuria with thrombosis of the renal artery.

Resuscitation and Diagnosis

The presence of an abdominal vascular injury after trauma is usually not known except with the overt injuries mentioned above. Therefore, the extent of resuscitation and decision on the need for diagnostic imaging will depend on the patient's hemodynamic status and response to the infusion of blood and crystalloid solutions. Profoundly hypotensive patients (systolic blood pressure < 70–75 mmHg with patient in the supine position or ATLS Class III or IV shock) or those with only a transient response to blood and fluids should be moved to the operating room within 10 minutes of arrival to the trauma room. This time interval allows for application of an identification bracelet, insertion of intravenous catheters and infusion of blood and fluids, and insertion of a REBOA (resuscitative endovascular occlusion of the aorta) device through a common femoral artery [6]. Inflation of the balloon at the level of the diaphragm (zone I) after injury to the descending thoracic aorta has been ruled out by a chest x-ray (blunt trauma) will decrease significantly any arterial bleeding in the abdomen.

Patients with possible abdominal trauma who do not have peritonitis and who are normotensive or have a rapid response to the infusion of blood and crystalloid solutions undergo diagnostic testing as described elsewhere in this book. This would include local wound exploration (stab wound), surgeon-performed ultrasound, computed tomography (CT) with contrast, and, on rare occasions, diagnostic peritoneal tap/lavage.

Preparation for Operation

Skin Preparation and Draping

All patients with symptomatic abdominal trauma should have skin preparation and draping from the clavicles to the knees bilaterally. This allows for extension of the midline abdominal incision into a median sternotomy, addition of an

anterolateral thoracotomy, or access to the femoral vessels and greater saphenous vein.

Equipment Needed if an Abdominal Vascular Injury Is Present

In addition to the usual surgical instruments and retractors, vascular equipment that should be available in the operating room during every trauma laparotomy would include the following: (1) aortic compression device if REBOA is not inserted; (2) vascular instrument tray; (3) polypropylene sutures (3-0→6-0); (4) woven Dacron and ringed polytetrafluoroethylene vascular grafts in appropriate sizes; and (5) unfractionated heparin (used systemically, on occasion, and "regionally" at 50 units/mL).

Hematoma or Hemorrhage in the Supramesocolic Midline Area (Zone I)

Anatomy

A midline retroperitoneal hematoma or area of hemorrhage superior to the transverse mesocolon may be from an injury to the diaphragmatic/supraceliac abdominal aorta, visceral abdominal aorta, celiac axis, proximal superior mesenteric artery, or proximal renal artery. Should the hematoma or area of hemorrhage be more to the right side in the supramesocolic area, an injury to the suprarenal infrahepatic inferior vena cava should be suspected. And, if the hematoma or area of hemorrhage is directly inferior to the transverse mesocolon and the midgut appears ischemic, an injury to the superior mesenteric artery or vein inferior to the pancreas should be suspected.

Exposure and Vascular Control

A contained hematoma is approached by a left-sided medial visceral rotation. This involves mobilization of the left colon,

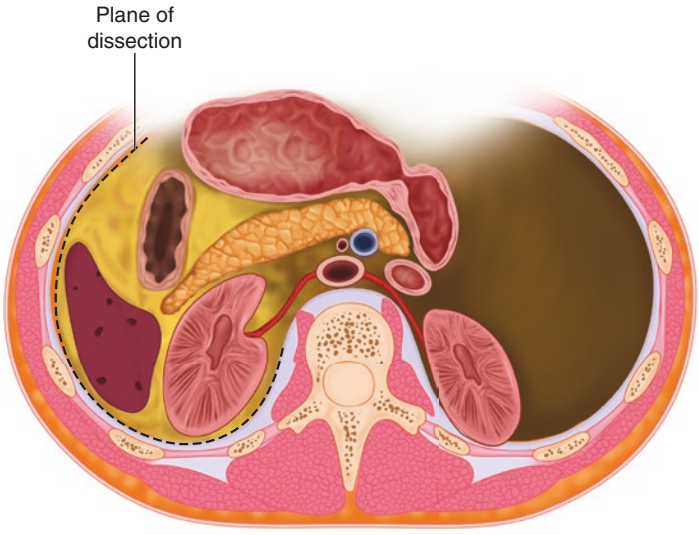

FIGURE 18.1 Left medial visceral mobilization is performed in the retroperitoneal plane behind all left-sided intra-abdominal viscera in a patient with a supramesocolic hematoma in the midline

splenic flexure, spleen, tail of the pancreas, fundus of the stomach, and, in select circumstances, left kidney (Fig. 18.1). This maneuver is best accomplished by the surgeon on the left side of the operating table with scissors, while the surgeon on the right side applies elevation and traction. After this maneuver, the supraceliac and visceral segments of the abdominal aorta will still not be visualized due to the overlying lymphatics and celiac ganglia. There structures must be dissected away to allow for proper visualization of the left side of the aorta for cross-clamping. If the hematoma is in the aortic hiatus of the diaphragm, the hiatus should be divided in a radial fashion at the 2 o'clock position for 8–10 cm to allow for cross-clamping of the distal descending thoracic aorta in the posterior mediastinum.

While the left medial mobilization maneuver allows for safe proximal control of any major abdominal arterial injury, it has some disadvantages. The main ones are the time

required to complete the maneuver (5–7 minutes) and the distortion of the juxtarenal aorta (a fold) if the left kidney is a part of the mobilization.

Once the descending thoracic aorta or diaphragmatic/supraceliac aorta is clamped, rapid dissection on the anterior and left lateral aorta is performed. This will allow for visualization of whether the aorta or a visceral vessel is injured. Of note, the origins of the celiac axis and superior mesenteric artery have the appearance of the letter "V" as they are so close in some patients. Also, the left-sided exposure will not allow for appropriate visualization of the proximal right renal artery because of the overlying mesentery of the small bowel; hence, a right-sided medial mobilization maneuver (to be described) will be necessary.

An area of hemorrhage in the midline supramesocolic area is approached by putting left-sided traction on the lesser curve of the stomach, manually opening the lesser omentum, and palpating the supraceliac aorta on the spine. The surgeon's left 2nd and 3rd fingers are then insinuated inside the fibers of the muscular aortic hiatus of the diaphragm. Finally, the surgeon uses his or her right hand to place a DeBakey aortic clamp straight down over the left fingers until the tips of the clamp touch a lumber vertebra and it can be closed. When there is difficulty insinuating the fingers inside the muscular aortic hiatus, the hiatus is divided at the 2 o' clock position as previously described to allow for cross-clamping of the descending thoracic aorta.

Diaphragmatic/Supraceliac Aorta

An isolated perforation is repaired with 3-0 or 4-0 polypropylene sutures in an interrupted or continuous fashion. Adjacent perforations are connected with Potts scissors and closed in a transverse or oblique fashion. When there is a loss of the aortic wall, segmental resection is followed by insertion of a 12, 14, or 16 mm woven Dacron or polytetrafluoroethylene (PTFE) graft. The following steps will lower the risk of a

postoperative graft infection: (1) separate gastrointestinal injuries from field of aortic repair; (2) irrigate retroperitoneal space around graft with saline-antibiotic solution; and (3) close retroperitoneum with absorbable suture.

With prolonged cross-clamping of the proximal abdominal aorta, a washout "hyperkalemia-acidosis" and hypotension can be expected as the cross-clamp is released. Preliminary administration of intravenous bicarbonate, crystalloid solutions and warm packed red blood cells will minimize these effects.

Celiac Axis

A proximal injury to the left gastric or splenic artery should be ligated. On occasion, an injury to the proximal hepatic artery may be amenable to a lateral arteriorrhaphy, an end-to-end anastomosis, or the insertion of a saphenous vein interposition graft. If necessary, the celiac axis may be ligated and divided to obtain improved exposure of an injury to the diaphragmatic/supraceliac aorta or visceral aorta.

Superior Mesenteric Artery (SMA)

The rare Fullen zone I injury beneath the neck of the pancreas is approached by dividing the neck of the pancreas between Glassman intestinal clamps or by using a gastrointestinal anastomosis (GIA) stapler. Fullen zone II injuries between the inferior pancreaticoduodenal and middle colic arteries are approached by entering the lesser sac and exposing and elevating the inferior border of the pancreas.

After a laceration or perforation in Fullen zones I, II, and III is isolated, a lateral arteriorrhaphy is performed with 5-0 polypropylene suture. Significant injuries (transection) in the small bowel mesentery (Fullen zone IV) are ligated. The small bowel or colon distal to the ligation is then observed intraoperatively to see if immediate resection is indicated [7].

There are two rules when dealing with significant injuries to the SMA in Fullen zones I, II, and III [8]. First, an injury to the SMA in these locations should NEVER be ligated unless a separate vascular bypass is performed. Second, any complex repair of the SMA in this location in association with an overlying or adjacent injury to the pancreas will be at risk for a blowout if a pancreatic leak occurs in the postoperative period. The ideal operative approach in either circumstance is to insert a temporary intraluminal shunt to perfuse the midgut, and an extra-anatomic vascular bypass would be deferred until a reoperation. At reoperation in a stable patient, the infrarenal abdominal aorta and inferior mesenteric artery are exposed in the retroperitoneum. Then, the SMA in Fullen zone III is exposed through the posterior aspect of the mesentery of the small bowel. After retrieval of an autogenous saphenous vein graft and initiation of systemic heparinization, the graft is sewn in an end-to-side fashion to the infrarenal abdominal aorta using 4-0 polypropylene suture. The graft is flushed, and the appropriate length is cut after the small bowel mesentery is allowed to lay back on the retroperitoneal location of the graft. The bypass is completed by an end-to-side anastomosis to the posterior SMA using 5-0 polypropylene suture.

Proximal Renal Arteries

See Section “Hematoma or Hemorrhage in the Perirenal Area (Zone II).”

Superior Mesenteric Vein (SMV)

An injury to the SMV lying to the right of the proximal SMA will present with a hematoma or area of hemorrhage at the base of the mesentery. As with the SMA, exposure is obtained under the neck of the pancreas, in the lesser sac inferior to the pancreas, or in the mesentery itself. Because of the many small veins entering the proximal SMV, obtaining proximal and distal control around a significant injury is tedious. On occa-

sion, a small Satinsky or Cooley vascular clamp may be applied around the injury to allow for a lateral venorrhaphy with 5-0 polypropylene suture. Resection and an end-to-end anastomosis can be performed for a significant injury or when venorrhaphy has narrowed the lumen by >50%. During the performance of the anastomosis, it is helpful to place the patient in a head-down position and have an assistant push the small bowel toward the patient's head to relieve tension.

Ligation of a significant injury is appropriate in the patient with near exsanguination or multiple associated injuries. At the Shock Trauma Center, results have been poor with this approach; however, survival was 40% in one large series from another institution published in 2007 [9]. Ligation causes immediate edema and dark discoloration of the midgut, and venous infarction may appear imminent at a reoperation. A saphenous vein bypass from an engorged mesenteric vein to the splenic vein was used for decompression successfully at the Shock Trauma Center in 2018.

There is "splanchnic hypervolemia and systemic hypovolemia" as described by H. Harlan Stone after ligation of the SMV (or portal vein). Infusions of large amounts of balanced crystalloid solutions are often needed to maintain the patient's blood pressure till the splanchnic hypervolemia reverses over several days.

Hematoma or Hemorrhage in the Inframesocolic Midline Area (Zone I)

Anatomy

A retroperitoneal midline hematoma or area of hemorrhage inferior to the transverse mesocolon may be from an injury to the infrarenal abdominal aorta or inferior vena cava (IVC). A true midline hematoma is almost always from an injury to the aorta, with its highest point directly over the area of injury ("Mt. Everest phenomenon"). A hematoma

toward the right side, particularly one that elevates the ascending colon and its mesentery, is almost always from an injury to the IVC.

Exposure and Vascular Control

A contained true midline inframesocolic hematoma is exposed by eviscerating the transverse colon superiorly and the small bowel to the right of the midline incision and mobilizing the ligament of Treitz. With the surgeon's left hand on the aortic pulsations at the base of the mesocolon, the retroperitoneum is opened till the crossover left renal vein is visualized. A curved DeBakey aortic cross-clamp is then applied for proximal control. The large hematoma can then be split manually in the retroperitoneum to allow for cross-clamping of the infrarenal aorta distal to the area of injury.

When no injury to the infrarenal aorta is found or the hematoma is thought to be related to an injury to the IVC, a right-sided medial mobilization maneuver is performed (Fig. 18.2). The cecum and the proximal ascending colon are mobilized to the midline to allow for visualization of the confluence of the common iliac veins and the proximal IVC. Immediate cross-clamping of the cava is appropriate if the patient is bleeding in addition to the presence of a large hematoma or if the patient is profoundly hypotensive. Should the cross-clamp on the IVC aggravate hypotension (loss of venous return), the infrarenal abdominal aorta will have to be cross-clamped, as well. The remainder of the ascending colon and hepatic flexure is then mobilized, and a rapid Kocher maneuver is performed. The rest of the retroperitoneal hematoma is split manually over the IVC till the area of injury is localized. A distal cross-clamp is then placed on the IVC if the injury is in the infrarenal segment. An injury to the juxtarenal or suprarenal infrahepatic IVC will mandate cross-clamping of both renal veins and the IVC at the edge of the liver.

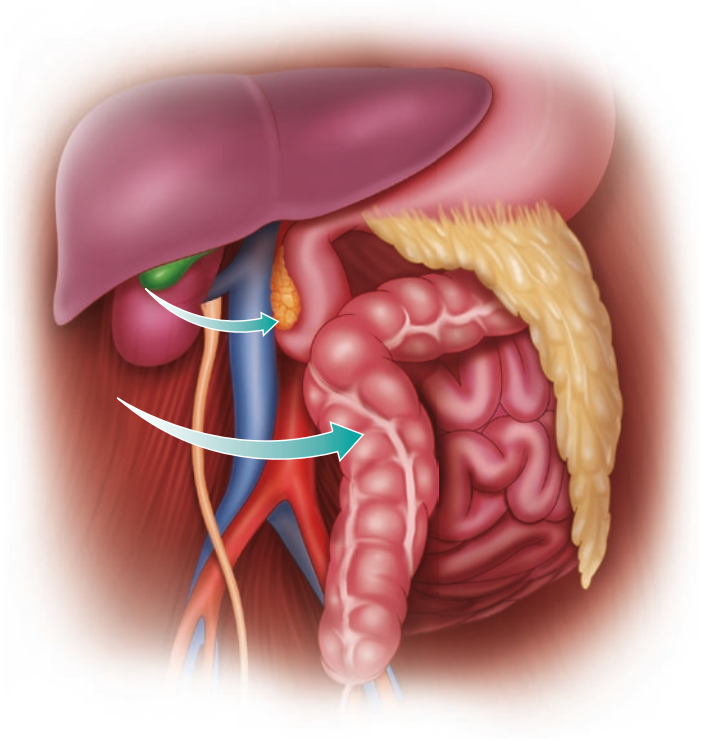

Figure 18.2 Right medial visceral mobilization is performed in the retroperitoneal plane behind all right-sided intra-abdominal viscera EXCEPT the kidney in a patient with a supramesocolic or inframesocolic hematoma or bleeding in the area of the inferior vena cava

An area of hemorrhage in the true midline retroperitoneum inferior to the mesocolon is approached the same as a hematoma with cross-clamping of the intrarenal aorta. Active hemorrhage from a suspected injury to the IVC mandates a rapid right-sided medial mobilization as described above. A line of Judd-Allis clamps can then be used to control a long anterior or lateral laceration in the IVC. A routine perforation is elevated with a DeBakey tissue forceps, and a Satinsky clamp is applied underneath.

Infrarenal Abdominal Aorta

Repairs are similar to those described for the diaphragmatic/supraceliac abdominal aorta. A viable pedicle of omentum is then placed between any anterior suture repair or graft in the infrarenal aorta and the third and fourth portions of the duodenum to prevent a postoperative aortoduodenal fistula.

Inferior Vena Cava

An isolated anterior or lateral perforation or connected perforation is repaired in a transverse or oblique fashion with 4-0 or 5-0 polypropylene suture. Repair of a posterior perforation through an enlarged anterior perforation results in significant narrowing. A better choice is to rotate the IVC and fix the posterior perforation from the outside. When a suture repair results in greater than 50% narrowing of the IVC or when near exsanguination has occurred (need for "damage control"), the infrarenal IVC should be ligated [10]. This is performed between two vascular clamps to collapse the IVC, and two 0-silk ties are used. It is worthwhile to measure the pressures in the below-knee anterior compartments in the operating room after ligation of the IVC. A compartment pressure > 30–35 mm Hg should prompt consideration for a bilateral below-knee two-skin incision four-compartment fasciotomy.

Ligation of the suprarenal infrahepatic IVC should be followed by an early (<6 hours) reoperation to insert a large ringed PTFE prosthesis. Both renal veins should be reimplanted into the graft if they were compromised by the original ligation.

Hematoma or Hemorrhage in the Perirenal Area (Zone II)

Anatomy

A perirenal hematoma or hemorrhage may be from an injury to the renal artery, renal vein, both, or the kidney itself.

In a hemodynamically stable patient with blunt trauma or with a penetrating wound to the flank in proximity to the kidney, a CT is performed to assess the magnitude if a renal injury is present. An injured, but reasonably intact kidney without extravasation of intravenous contrast does not mandate a laparotomy. If a laparotomy is to be performed for other reasons, the perirenal hematoma does not have to be opened unless, as previously noted, it is pulsatile, rapidly expanding, or ruptured. Should the CT document blunt occlusion of one renal artery 2–3 cm from the juxtarenal aorta, there is little enthusiasm for open revascularization if the contralateral kidney is normal. An endovascular approach would be preferred in the modern era.

Exposure and Vascular Control

A juxtarenal (rather than perirenal) hematoma or area of hemorrhage is approached with a left medial mobilization maneuver leaving the left kidney down to obtain proximal control of the supraceliac aorta. On the left side, dissection continues inferiorly until the origin of the left renal artery at the 4 o'clock position is visualized. Distal control of the left renal artery would be obtained by dividing the left lateral retroperitoneum and manually elevating the kidney. On the right side, a right medial mobilization maneuver leaving the kidney down is necessary to visualize the origin of the right renal artery medial to the IVC at the 4 o'clock position.

A perirenal or renal hematoma that needs to be opened or area of hemorrhage is approached directly in the modern era. The hepatic flexure and C-loop of the duodenum on the right or the splenic flexure, spleen, and tail of the pancreas on the left are first mobilized medially. As noted previously, the retroperitoneum lateral to the injured kidney or renal vessels is divided, and the kidney is elevated out of the retroperitoneum. Vascular clamps can be applied to any injured renal vessel in the hilum.

Renal Artery

Lateral arteriorrhaphy with 5-0 or 6-0 polypropylene suture is appropriate for a small perforation or laceration. While segmental resection and an end-to-end anastomosis is rarely performed, it is possible if the kidney is moved medially. In the presence of a palpably normal contralateral kidney, many significant injuries to the renal artery are treated with ligation and ipsilateral nephrectomy. Injury to the renal artery to a solitary kidney or to bilateral renal arteries or kidneys mandates repair of the renal vessels or autotransplantation of the least-injured kidney to the contralateral pelvis by the transplant team.

Renal Vein

Lateral repair of the renal vein is performed with 5-0 or 6-0 polypropylene suture. Ligation of the injured right renal vein mandates a right nephrectomy at an early reoperation. Central ligation of the left renal vein (medial to the gonadal and adrenal veins) is tolerated (kidney is preserved) without sequelae in approximately 70% of patients in elective vascular series.

Hematoma or Hemorrhage in the Pelvic Retroperitoneum (Zone III)

Anatomy

A lateral pelvic hematoma or area of hemorrhage may be from an injury to the iliac artery, iliac vein, both, the ureter, or pelvic bones.

As noted previously, a blunt stable hematoma related to a pelvic fracture is not opened at a laparotomy performed for other reasons. A blunt rapidly expanding, pulsatile, or ruptured hematoma is, however, opened, as is any lateral pelvic hematoma or area of hemorrhage from a penetrating wound. There continues to be controversy regarding the need to

open deep stable pelvic hematomas in the small pelvis of men after penetrating trauma.

Exposure and Vascular Control

A lateral pelvic hematoma is first approached by eviscerating the small bowel to the right and exposing the bifurcation of the abdominal aorta. Careful dissection will allow for rapid passage of a vessel loop around the ipsilateral common iliac artery and exposure of the most distal aspect of the ipsilateral common iliac vein. The ipsilateral distal external iliac artery and proximal external iliac vein are then exposed and looped under the inguinal ligament beyond the hematoma. Prior to opening the hematoma and finding the area of injury and the ureter, proximal and distal vascular clamps are applied to the iliac artery and vein.

The iliac vessel responsible for an area of hemorrhage on one side of the pelvis is compressed with a laparotomy pad or grabbed with the surgeon's hand if it is easily visible. Then, proximal and distal vascular control around the area of injury is obtained.

One special technique to expose an injury to the confluence of the common iliac veins or the proximal IVC is to intentionally divide an uninjured right common iliac artery. This allows for mobilization of the aortoiliac bifurcation to the left and significantly improves visualization of the iliac-IVC junction. Following venous repair or ligation, the right common iliac artery is reanastomosed.

For injuries of the right internal iliac vein in the narrow male pelvis, the overlying internal iliac artery is divided and ligated to improve visualization.

Common and External Iliac Artery

In addition to all the standard repairs, transposition of the end of the proximal common iliac artery (after segmental resection) to the side of the contralateral common iliac artery can be performed to create a new "bifurcation." Another option is

to mobilize the internal iliac artery to replace the adjacent injured external iliac artery after a segmental resection.

The most important operative principle when dealing with injuries to these vessels is to NEVER perform a ligation as there is a 50–75% risk of a major amputation in the ipsilateral lower extremity postoperatively [11]. A temporary intraluminal arterial shunt is the appropriate choice in the patient needing "damage control."

The other major risk after a complex repair (end-to-end anastomosis or interposition graft) of the common or external iliac artery is a postoperative blowout of the suture line or dissolution of an autogenous graft caused by pelvic sepsis from associated injuries. To lower the risk, any end-to-end anastomosis or interposition graft should be covered or wrapped with a viable pedicle of omentum before closure of the pelvic retroperitoneum. In the patient with extensive fecal soilage from an associated injury to the colon or rectum, an extra-anatomic vascular bypass rather than an in situ complex repair should be considered. After removal of the shunt at a reoperation, the proximal end of the injured common or external iliac artery is doubly oversewn with a 4-0 or 5-0 polypropylene suture. It is then covered with a viable pedicle of omentum and buried under a retroperitoneal closure. The extra-anatomic crossover femorofemoral bypass is then performed with a ringed 8 mm PTFE graft passed inferior to the midline abdominal incision.

Of interest, blunt injuries to the common or external iliac artery have the highest amputation rate for all iliac vascular injuries (50%) [12]. In the absence of bleeding, blunt thrombosis of either artery may be treated by the insertion of an endovascular stent. If stenting is not possible, performing a crossover femorofemoral bypass graft would be appropriate if there is no other indication for a laparotomy.

Common and External Iliac Vein

Lateral venorrhaphy with 5-0 polypropylene suture is preferred, while ligation is performed as part of a "damage control" procedure. After ligation, the pressure in the anterior

compartment of the ipsilateral leg should be measured. A pressure in the 30–35 mm Hg range should prompt consideration to perform a below-knee two-skin incision four-compartment fasciotomy.

A venous repair that narrows the lumen of the common or external iliac vein greater than 50% is at risk for thrombosing postoperatively or becoming a source for a pulmonary embolus [13, 14]. Postoperative anticoagulation with enoxaparin or dalteparin is appropriate, though the length of time this should be continued is unknown.

Hematoma or Hemorrhage in the Porta Hepatis

Anatomy

A portal hematoma or area of hemorrhage may be from an injury to the portal vein, hepatic artery, both, or the extrahepatic biliary ducts.

A stable hematoma or area of hemorrhage is approached by placing an angled or curved vascular clamp around all three structures in the hepatoduodenal ligament, as close to the duodenum as possible. If the ligament is long enough, a vascular clamp may be placed on the distal porta at the edge of the liver, as well. With proximal and distal portal control, the hematoma is opened, the common bile duct is looped and pulled to the right, and the common hepatic artery (to the left) and portal vein (posterior) are visualized.

Portal Vein

Lateral venorrhaphy or a transverse or oblique repair of two connected perforations is performed with 4-0 or 5-0 polypropylene suture. Other repairs reported in the literature have included the following: resection with an end-to-end anastomosis or PTFE interposition graft; transposition of the splenic vein down to replace the proximal portal vein; or an autoge-

nous vein graft from the superior mesenteric vein to the distal portal vein. Neither a portacaval shunt nor a vein graft from the proximal portal vein to the IVC is recommended because of the lifetime risk of hepatic encephalopathy.

Ligation is appropriate during "damage control" and has the same disadvantages as previously described after ligation of the superior mesenteric vein. Those include distension and discoloration of the midgut, risk of venous infarction, and transient splanchnic hypervolemia and systemic hypovolemia [15].

Hepatic Artery

A repair of the hepatic artery in the porta is uncommon but should be performed if the portal vein has been ligated. Injuries to the right or left hepatic artery are usually ligated, and a cholecystectomy is mandatory on the right. Ligation of the lobar hepatic artery and portal vein will lead to necrosis of the lobe, and lobectomy or extensive resectional debridement will have to be performed at a reoperation.

Endovascular Approaches

Endovascular stents and stent grafts have been used in the management of abdominal vascular injuries since the first report by Juan Parodi in 1993. Primary indications for endovascular approaches are listed in Box 18.2.

Box 18.2: Indications for Endovascular Approaches

- Blunt intimal injury, thrombosis, or contained extravasation from abdominal aorta, visceral artery, iliac artery, inferior vena cava, iliac vein
- Hostile abdomen
- Late traumatic false or true aneurysm or arteriovenous fistula

Complications

Complications after abdominal vascular repairs include thrombosis, infection-dehiscence, and vascular-enteric fistulas. Thrombosis most commonly occurs after venous repairs but may occur after repair of a vasoconstricted superior mesenteric or renal artery, as well. A "second-look" operation or a CT arteriogram is appropriate when the surgeon has a concern about this complication or the patient has signs of the same.

Dehiscence-infection occurs most commonly with associated injuries to the pancreas and proximal superior mesenteric artery or to the gastrointestinal tract and common or external iliac artery as previously noted. Prevention involves the following: (1) isolating the arterial repair from local contamination; (2) covering the arterial repair with mesenteric tissue, retroperitoneum, or a viable omental pedicle; or (3) considering an extra-anatomic bypass as previously described.

Vascular-enteric fistulas most commonly occur after repair of the infrarenal aorta or common or external iliac artery. Prevention is by complete coverage of suture lines or interposition grafts using the options listed above.

Survival

Survival figures after repair or ligation of abdominal vascular injuries are listed in Tables 18.1 and 18.2 [1, 16–18].

Table 18.1 Survival after abdominal arterial injuries

	Historic[a]	2001[b]
Suprarenal aorta	34.8%	8.3%
Infrarenal aorta	46.2%	34.2%
SMA[c]	57.7%	54.3%
Renal artery	87%	65.1%
Common iliac artery	61% (45–81%)	46.8%
External iliac artery		64.1%

[a]Data from Reference [1]
[b]Data from References [16–18]
[c]*SMA* superior mesenteric artery

Table 18.2 Survival after abdominal venous injuries

	Historic[a]	2001[b]
Infrarenal IVC[£]	76.1%	46.1%
SMV[c]	72%	58.3%
Common iliac vein	70–95%	58.6%
External iliac vein		72.2%
Portal vein	50%	–

[a]Data from Reference [1]
[b]Data from References [16–18]
[c]*IVC* inferior vena cava, *SMV* superior mesenteric vein

References

1. Asensio JA, Feliciano DV. Abdominal vascular injury. In: Moore EE, Feliciano DV, Mattox KL, editors. Trauma, vol. 651. 8th ed. New York: McGraw-Hill; 2017.
2. DeBakey ME, Simeone FA. Battle injuries of the arteries in World War II: an analysis of 2,471 cases. Ann Surg. 1946;123:534–79.
3. Hughes CW. Arterial repair during the Korean War. Ann Surg. 1958;147:555–61.
4. Rich NM, Baugh JH, Hughes CW. Acute arterial injuries in Vietnam: 1,000 cases. J Trauma. 1970;10:359–69.

5. Beekley AC, Blackbourne LH, Sebesta JA, McMullin N, Mullenix PS, Holcomb JB, et al. Selective nonoperative management of penetrating injury from combat fragmentation wounds. J Trauma. 2008;64:S108.
6. Brenner ML, Moore LJ, DuBose JJ, Tyson GH, McNutt MK, Albarado RP, et al. A clinical series of resuscitative endovascular balloon occlusion of the aorta for hemorrhage control and resuscitation. J Trauma. 2013;75:506–11.
7. Asensio JA, Britt LD, Borzotta A, Peitzman A, Miller FB, Mackersie RC, et al. Multi-institutional experience with the management of superior mesenteric artery injuries. J Am Coll Surg. 2001;193:354–66.
8. Feliciano DV, Asensio JA. Injuries to great vessels of the abdomen in Scientific American Surgery (online), edited by SW Ashley. Hamilton: Decker Intellectual Properties; 2016. https://doi.org/10.2310/7800.2138
9. Asensio JA, Petrone P, Garcia-Nunez L, Healy M, Martin M, Kuncir E. Superior mesenteric venous injuries: to ligate or repair remains the question. J Trauma. 2007;62:668–75.
10. Sullivan PS, Dete CJ, Patel S, Carmichael M, Srinivasan JK, Wyrzykowski AD, et al. Outcome of ligation of the inferior vena cava in the modern era. Am J Surg. 2010;199:500–6.
11. Ball CG, Feliciano DV. Damage control techniques for common and external iliac artery injuries: have temporary intravascular shunts replaced the need for ligation? J Trauma. 2010;68:1117–20.
12. Harris DG, Drucker CB, Brenner ML, Narayan M, Sarkar R, Scalea TM, et al. Management and outcomes of blunt common and external iliac artery injuries. J Vasc Surg. 2014;59:180–5.
13. Lauerman MH, Rybin D, Doros G, Kalish J, Hamburg N, Eberhardt RT, et al. Characterization and outcomes of iliac vessel injury in the 21st century: a review of the National Trauma Data Bank. Vasc Endovasc Surg. 2013;293(47):325–30.
14. Magee GA, Cho J, Matsushima K, Strumwasser A, Inaba K, Jazaeri O, et al. Isolated iliac vascular injuries and outcome of repair versus ligation of isolated iliac vein injury. J Vasc Surg. 2018;67:254–61.
15. Stone HH, Fabian TC, Turkelson ML. Wounds of the portal venous system. World J Surg. 1982;6:335.
16. Asensio JA, Chahwan S, Hanpeter D, Demetriades D, Forno W, Gambaro E, et al. Operative management and outcome of 302 abdominal vascular injuries. Am J Surg. 2001;180:528–33.

17. Davis TP, Feliciano DV, Rozycki GS, Bush JB, Ingram WL, Salomone JP, et al. Results with abdominal vascular trauma in the modern era. Am Surg. 2001;67:565–71.
18. Tyburski JG, Wilson RF, Dente C, Steffes C, Carlin AM. Factors affecting mortality rates in patients with abdominal vascular injuries. J Trauma. 2001;50:1020–6.

Chapter 19
Endovascular Therapy in Trauma

Rishi Kundi

Abbreviations

BAAI	Blunt Abdominal Aortic Injury
BTAI	Blunt Thoracic Aortic Injury
CT	Computerized Tomography
cTAG	Conformable Thoracic Aortic Graft
EVAR	Endovascular Aneurysm Repair
FACS	Fellow of the American College of Surgeons
FDA	Food and Drug Administration
IVC	Inferior Vena Cava
IVUS	Intravascular Ultrasonography
MD	Medical Doctor
REBOA	Resuscitative Endovascular Balloon Occlusion of the Aorta
SVS	Society for Vascular Surgery
TEVAR	Thoracic Endovascular Aneurysm Repair
V1, V2, V3, V4	First Through Fourth Segments of the Vertebral Artery

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Introduction

The last 25 years have seen a transformation in vascular surgery practice as the specialty has incorporated endovascular and interventional techniques. The treatment of vascular trauma has undergone an identical and parallel change; multiple National Trauma Data Bank analyses have consistently demonstrated an increasing shift in operative treatment of vascular trauma from open surgery to a treatment scheme that relies heavily on endovascular therapies [1–5]. At least some of these studies have also shown a concomitant decrease in the morbidity and mortality associated with these injuries [4, 6]. Although a direct causative relationship between endovascular intervention and improved survival is unlikely, a related improvement in the speed of hemorrhage control may be involved.

As endovascular surgery has increased in prevalence, so, too, has the construction and use of hybrid operating rooms with full angiographic capabilities. These rooms allow the simultaneous performance of open surgery and endovascular intervention [7]. This has eliminated transit delay between the angiography suite and the operating room. Additionally, the trauma surgeon no longer has to choose initial angiography or laparotomy for the unstable, multiply injured patient. Finally, the presence of hybrid rooms has allowed the development of novel techniques combining both endovascular and open methodologies.

A full explication of endovascular trauma techniques is outside the scope of this chapter. It is our hope that a description of the conceptual basis underlying endovascular intervention and a review of specific methods and contexts will enable the trauma surgeon to recognize the possibility of an endovascular solution to their patient's injuries.

Endovascular Principles

All endovascular trauma surgery is divisible into four parts: intra-arterial catheterization and injection, balloon occlusion, embolization, and stent emplacement.

Catheterization and Intra-arterial Injection

Catheter angiography is the intraluminal instillation of radiopaque dye under fluoroscopic visualization, whether by direct radiography or subtraction angiography, using a catheter that may be advanced far from the point of vascular access. Injection may be from any vessel into which the catheter has been positioned, whether the aorta or a subsegmental pulmonary artery. It is a dynamic study that can reveal flow, occlusion, or extravasation.

As multidetector CT angiography has increased tremendously in both sensitivity and specificity over the last decade, the advantage of catheter-based angiography in detecting injury is no longer overwhelming. Rather, it is the dynamic depiction of flow combined with the ability to select vessels that makes angiography valuable in selected patients. This becomes particularly important in well-collateralized beds such as the pelvis, where even injury laterality can be obscure.

An additional advantage of angiography is the ability to identify the site of vascular injury or obtain vascular control before and after other, nonvascular intervention. Identification of arterial continuity before and after reduction and fixation, for example, allows orthopedic and trauma teams to work in coordination. Angiography also facilitates open exploration and repair of vascular injury, particularly in the extremities.

In addition to contrast, intra-arterial injection of agents such as nitroglycerin may be useful in distinguishing true vascular injury from the severe vasospasm that is frequent in extremity trauma in the younger population [8].

Balloon Occlusion

Endovascular access allows obstruction of blood flow from within the lumen but has some advantages over traditional extrinsic clamping.

Balloon occlusion has been employed for vascular control before the advent of endovascular surgery. In the past, this

was usually achieved using balloon catheters inserted through the injury itself and advanced blindly. Through image-guided balloon occlusion with access distant to the injury, the surgeon is able to treat a broader range of vascular injury without extensive proximal exposure. This, in turn, allows open repair of axillosubclavian, common carotid, and common femoral injury without the need to enter the thorax or pelvis for proximal control. The use of aortic balloon occlusion is discussed later in this chapter.

A critical point when performing balloon occlusion is the sizing of the occlusion balloon to the inflow vessel. The principles of angioplasty do not apply – applying significant radial force can prove catastrophic. Insufflation to the point of control and no further is the guiding principle.

Embolization

If balloon occlusion is endovascular clamping, embolization is endovascular vessel ligation. Embolization involves the selective delivery of permanent or semipermanent occlusive material through catheter or sheath. Four methods of embolization are widely available: large-diameter devices, coils, hemostatic slurry, and liquid/particulate agents. The injury and anatomy determines the optimal modality.

Pseudoaneurysm sacs from arterial injury may be embolized, but care must be taken to avoid migration of coils from the sac into the adjacent flow stream. A "coil-and-cage" strategy may be employed in which an uncovered stent is deployed across the injury and coils are delivered through its cells. Slurry and liquid-phase agents are generally inappropriate.

End-vessel bleeding is often amenable to treatment with embolization; this is most often seen in pelvic injury. The ability to select and embolize as distally as possible while arresting bleeding reduces the chances of ischemia and minimizes the likelihood that collateral flow will cause bleeding to recur. Occasionally, vessel transection is best treated with embolization, for instance, distal cervical vertebral artery transaction [9].

In solid organ injury, selective distal embolization can preserve organ viability while controlling bleeding [10]. Splenic injury warrants special mention. Blunt splenic injury often involves diffuse bleeding. Selective embolization is not possible in such a wide area and, even if performed, would result in significant splenic infarction. Traditional nonoperative management has a substantial risk of failure and even recurrent bleeding. Proximal occlusion of the splenic artery, however, increases the rate of successful nonoperative management and allows resolution of hemorrhage with preserved splenic function. This is likely due to the preservation of perfusion from collateral supply and the decrease in perfusion pressure [11].

Stenting

The proliferation of elective stenting in the coronary and peripheral vasculature in the last 25 years has provided the endovascular surgeon with myriad options for treatment. In the context of trauma, only two categories need to be remembered: uncovered and covered stents, with the latter including stent grafts.

Uncovered stents are used in trauma when durable radial force is desired without the need for control of extravasation. This is a rare occurrence, usually with "cage and coil" embolization or when intimal flaps require aggressive reapposition to the arterial wall.

Covered stents, including both peripheral and aortic devices, are used frequently in trauma. They involve a metal framework with a fabric or polymer sheath. The covered stent is used to exclude extraluminal flow. This includes both pseudoaneurysm and free extravasation but also includes branch vessels of the target artery. Use of covered stents, whether in the superficial femoral artery or the aorta, must include awareness of potential branch occlusion.

In addition to branch occlusion, covered stents must be correctly sized. Undersized stents are prone to migration, and

oversized stents may collapse, thrombose or, if balloon-expandable, exacerbate injury.

Exhaustive study of medium-term and long-term patency of stents, both covered and uncovered, has been made, but only in the elective context and in patients with chronic occlusive or aneurysmal disease who suffer from the expected comorbidities. Long-term performance in patients composing the trauma demographic is essentially unknown aside from case series, but in-stent stenosis, distal embolization, and stent occlusion must always be a consideration after implantation.

Practical Endovascular Trauma Surgery

Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA)

Emergent thoracotomy was described as early as the 1870s, with resuscitative thoracotomy advocated in the 1960s [12–14]. Clamping the descending thoracic aorta drastically reduces hemorrhage pressure distal to the clamp allowing exposure and treatment of the patient in extremis. Endovascular occlusion of the descending thoracic aorta with a Foley catheter was first described in the Korean conflict [15].

As endovascular aneurysm repair increased and "modelling" balloons designed for aortic diameters became commercially available, interest in endovascular balloon occlusion for resuscitation returned. REBOA appears to have equivalent efficacy to emergency thoracotomy but offers several advantages [16, 17]. Transfemoral arterial access is rapid and requires only standard trauma bay equipment. Placement of the balloon itself can be done at the bedside using estimated distances for positioning. Confirmation can be done with plain portable radiography [18].

Unlike open thoracotomy, REBOA allows positioning of the occlusive balloon in either the thoracic or the infrarenal aorta. In the case of distal injury, this allows simultaneous temporizing hemorrhage control while maintaining renal and

mesenteric perfusion [19]. This flexibility has allowed REBOA to be used in a variety of non-trauma patients, including elective hepatobiliary, orthopedic, and oncologic pelvic surgery as well as ruptured aortic aneurysm and postpartum hemorrhage (Fig. 19.1) [20–26].

Further endovascular intervention for hemorrhage is possible while the REBOA is present. The periodic and partial deflation of the balloon to identify, localize, and treat vascular injury while maintaining hemodynamic stability is a subtle art, as is maneuvering wires, catheters, and sheaths around the REBOA catheter [27].

Cerebrovascular Injury

Intracranial

Though the methods of intracranial endovascular trauma surgery are not dissimilar from those described here, neurosurgical practices are outside the scope of this review. The changing practices in this area are of significant interest, however, as the advances in military medical treatment of traumatic brain injury, both blunt and penetrating, increasingly involve endovascular surgery [28].

Cervical Carotid Artery

The potential for endovascular treatment of cervical carotid injury varies by both mechanism of injury and zone of the neck.

Antiplatelet or anticoagulant therapy is sufficient for the majority of blunt cerebrovascular injury [29]. In selected dissections with significant luminal encroachment or with pseudoaneurysm, intervention may be considered. Case series of covered stent placement demonstrate that this is a possible modality, but compliance with postoperative anticoagulation or antiplatelet agents is critical [30–32]. Coil embolization of the pseudoaneurysm sac carries the risk of catastrophic distal

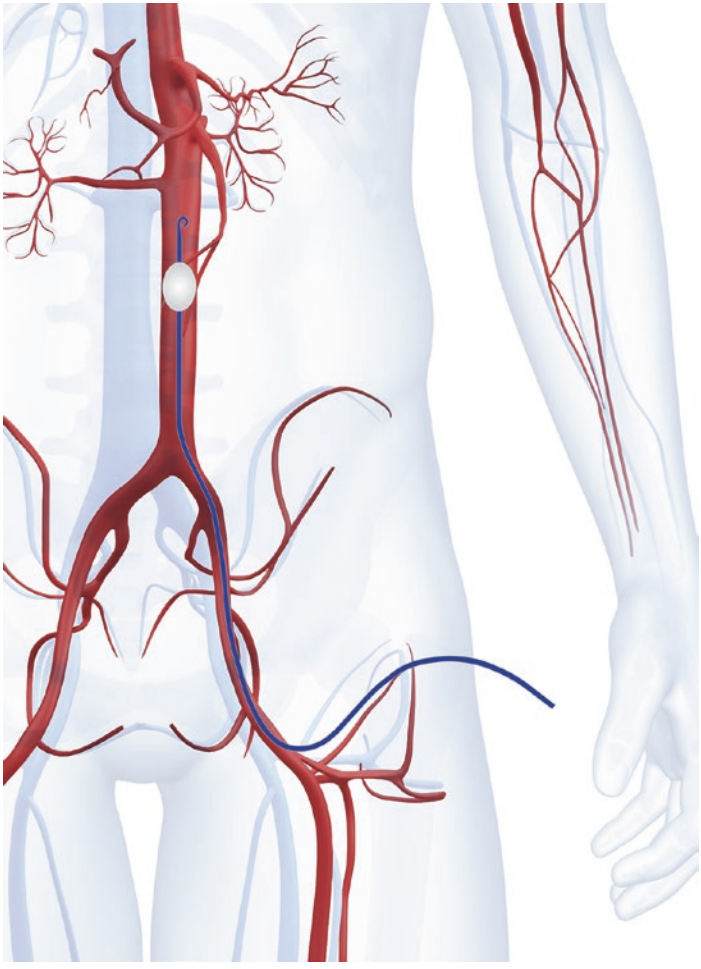

Figure 19.1 Diagram illustrating position of deployed and inflated REBOA catheter. Initial efforts utilized guidewire placement, but proprietary, dedicated devices are now designed for wireless advancement. As shown, the REBOA balloon is positioned in Zone III. This would be ideal placement for control of pelvic hemorrhage. (Courtesy of Prytime Medical Devices, Inc., Boerne, TX, USA)

migration of the embolization medium. Intraprocedural balloon exclusion or a "coil-and-cage" strategy can be used to address this risk [33, 34].

In penetrating carotid trauma, endovascular instrumentation is of greatest benefit in Zones I and III. Penetrating injuries to Zone II are best addressed through open surgery.

Open repair of Zone I injury may benefit from preoperative aortography or selective angiography to determine the location of disease or intraoperative balloon occlusion if patient anatomy otherwise requires intrathoracic proximal control. Stent graft repair of a common carotid injury has been reported [35].

Penetrating Zone III injuries have been treated by both stenting and, in the case of complete transection, coil embolization. The difficult exposure of the carotid artery at the skull base makes successful endovascular treatment of injuries at this level appealing.

Vertebral Artery

Blunt vertebral artery injuries are rarely candidates for surgical treatment. A small proportion of these injuries are immediately catastrophic, and these are usually associated with severe basilar skull or high cervical spine fracture [36–38]. The overwhelming majority are adequately treated with medical management. Coil embolization is used for selective high-grade injuries.

Penetrating vertebral artery injuries are more common. The V2 and V3 segments are enclosed by the vertebrae, and open exposure is arduous, but endovascular repair is also challenging. Tortuosity and small vessel diameter are prohibitive of stent grafting the vertebral arteries, and the vessels' mobility and tendency to retract after transection make successful bridging unlikely. Significant hemorrhage is thus best treated with coil embolization of the area of extravasation distally and the stump lumen proximally. Injuries in the V1 and V4 segments are hostile to endovascular intervention.

Upper Extremity

Subclavian and Axillary Arteries

Injury to the intrathoracic and proximal extrathoracic portions of the subclavian and axillary arteries carries substantial mortality. In their seminal review of penetrating trauma to these vessels, Demetriades and colleagues demonstrated a mortality of over 34% [39].

The anatomy of these vessels presents several challenges. The retroclavicular location of the proximal artery makes temporizing direct pressure difficult, necessitating practices such as extraluminal Foley balloon compression through the wound tract [40]. Exposure of the subclavian and axillary arteries is challenging in the elective, controlled environment. In the context of trauma, the need for proximal control can require progressive extension of exposure to median sternotomy or trapdoor or even clamshell thoracotomy [41]. Open exposure also carries a significant risk of iatrogenic injury to the densely packed adjacent arterial, venous, and neural structures.

Endovascular surgery offers the potential for definitive endoluminal repair of both penetrating and blunt injuries of the subclavian and axillary arteries without the need for open exposure. In a retrospective, propensity-matched, 10-year experience, Branco and colleagues showed that endovascular treatment of axillosubclavian injury was associated with a nearly fivefold lower mortality over open repair with a tendency toward less complications postoperatively [42]. Smaller case reports have found shorter operative times and lower transfusion requirements as well [43–45].

Definitive endovascular repair of axillosubclavian injury can consist of stent grafting of an injured artery or coil embolization of avulsed branches (Fig. 19.2a, b). The vessel's large diameter and tendency to maintain patency distal to injury facilitates wire crossing and stent graft shunting of the transected artery, allowing open repair after resuscitation and resolution of other injuries [46]. Definitive repair of the proximal subclavian artery is anatomically limited by the ves-

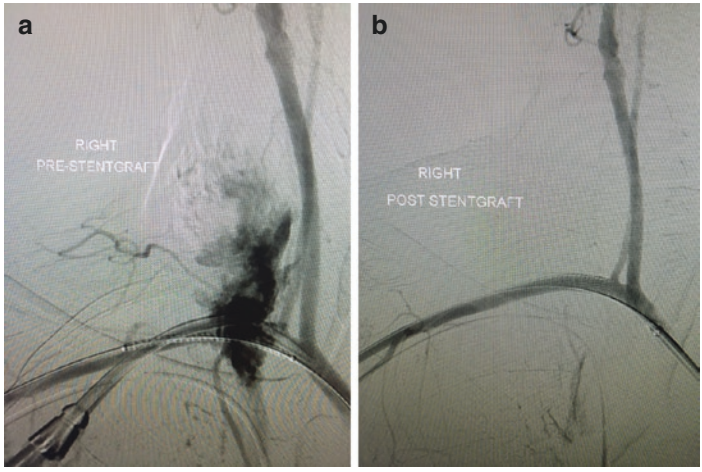

FIGURE 19.2 (**a**, **b**) Definitive endovascular repair of penetrating injury to the right subclavian artery. (**a**) Massive extravasation is evident. Brachial access has been obtained and a Gore Viabahn stent graft advanced but not deployed across the injury. (**b**) After deployment, extravasation is not seen. Note the preservation of the vertebral artery and occlusion of the internal thoracic artery and thyrocervical trunk

sel's flexion point and by the vertebral artery. Occlusion of other small branches presents less of a dilemma because of copious collateral flow. Even if open repair is necessary, a hybrid approach involving angiography and balloon occlusion for antegrade control may be advantageous [47–49]. Access may be obtained from femoral or brachial points. Radial access is not advised because of the larger sheath sizes required for covered stent placement.

Brachial Artery and Beyond

The presence of ischemia or compressive hematoma combined with the speed and ease of exposure usually makes open treatment of vascular injury distal to the shoulder preferable. Asymptomatic dissection of the brachial artery may be appropriately treated with low-pressure balloon inflation to reap-

pose the intimal flap to the arterial wall [50]. Balloon tamponade may be used to treat small perforations in the forearm vasculature, and selective embolization is useful in small branches such as the common interosseous artery and its branches [51].

Aorta

Descending Thoracic Aorta

The endovascular treatment of blunt injury to the descending thoracic aorta (BTAI) has progressed rapidly in the last decade. A review of the National Trauma Data Bank by Grigorian and colleagues confirms that endovascular repair was performed for 25.7% of all BTAI in 2015, compared with 12.1% in 2007; over that same period, open repair of BTAI decreased from 7.4% to 1.9%. Endovascular repair was associated with decreased mortality, renal injury, and length of stay compared to open aortic repair [52]. The Society for Vascular Surgery (SVS) guidelines recommend that endovascular repair be performed preferentially over open repair or nonoperative management.

Treatment of BTAI is generally protocolized and is contingent upon grade. The SVS classification system works from the lumen outward: Grade I, intimal tear; Grade II, intramural hematoma or extensive intimal flap; Grade III, pseudoaneurysm; and Grade IV, free intrathoracic rupture. The guidelines derived from this system mandate observation and repeat CTA of Grade I injury and immediate repair of Grades II–IV [53]. Starnes and colleagues have proposed a simpler, modified version involving minimal (intimal injury), moderate (anything distorting the normal aortic contour), and severe (extravasation) classes and recommends observation, semi-elective repair, and emergent repair, respectively [54]. DuBose and associates performed a multi-institutional analysis demonstrating no advantage to endovascular repair in patients with Grade I–II injury over observation [55].

Patients who are managed nonoperatively should be treated, if possible, with β-blockade and an antiplatelet agent.

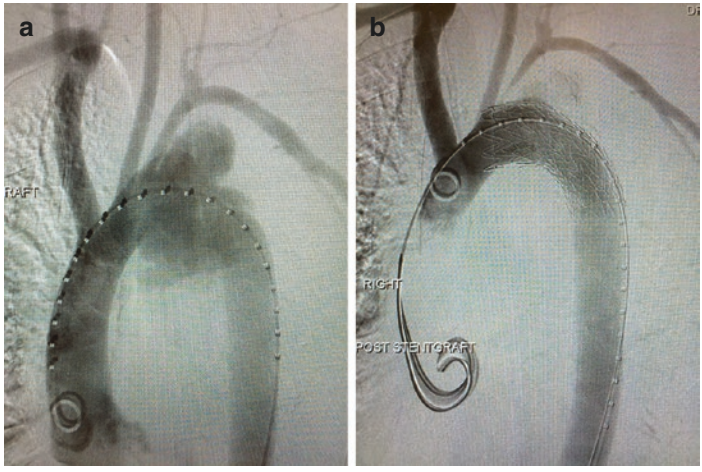

FIGURE 19.3 (**a**, **b**) Endovascular repair of Grade IV blunt thoracic aortic injury. (**a**) Diagnostic aortography showing classic injury at the location of the ligamentum arteriosum. Advancement of the marking pigtail catheter and a left anterior oblique projection allows maximal visualization of the arch. (**b**) After deployment, exclusion of the injury is obvious. A double-curved Lunderquist wire has been advanced until it rests against the cusps of the aortic valve, as seen by the organized motion artefact. The orifice of the left subclavian artery has been covered, but preserved flow via collaterals is seen. The origin of the left common carotid artery is encroached upon by uncovered wireforms, and flow is not impeded

Repeat imaging with CT angiography is generally performed within 7 days. Progression should prompt endovascular repair (Fig. 19.3a, b).

Preparation for endovascular repair should entail careful review of CT angiography, preferably with workstation reconstructions. Careful measurement of required coverage length, as well as proximal and distal landing zones, must be performed. In the emergent setting, the left subclavian artery origin may be covered with impunity. The left common carotid artery, however, cannot be encroached upon, and so, taking proximal sealing zone into account, aortic injury less

than 15 mm from the left common carotid artery is generally not reparable through endovascular surgery. CTA measurement of aortic diameter in the hyperdynamic, hypovolemic patient is exceedingly unreliable. Wallace and associates have demonstrated significant variation in aortic diameters on admission CT angiography and intraoperative, pre-TEVAR intravascular ultrasound. IVUS is strongly recommended for confirmation of proximal and distal diameter measurement prior to deployment of endograft [56].

There are currently four thoracic endografts approved by the FDA for repair of aneurysmal disease. Only the Medtronic Valiant and the Gore cTAG have been approved by the FDA for treatment of BTAI, but off-label use of the Bolton and Cook devices is common [57]. Thoracic endografts with branches to preserve flow through the great vessels will likely become available in the near future, and their suitability in trauma will become apparent with time (Fig. 19.4a, b) [58].

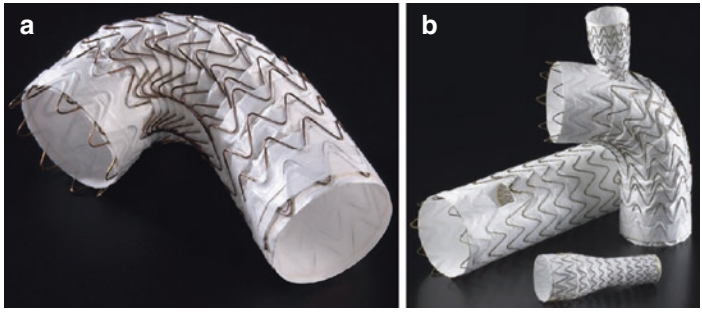

Figure 19.4 (**a**, **b**) Thoracic endografts. (**a**) Gore Conformable TAG® (cTAG) endoprosthesis. This and the Medtronic Valiant have FDA approval for treatment of BTAI, and the size ranges are accordingly consistent with aortic sizes of young patients. Note the proximal extent of the wireforms compared with the extent of the fabric. (**b**) The Gore TAG® thoracic branch endoprosthesis is an investigational device that theoretically allows continued perfusion of the left subclavian artery and therefore more proximal aortic coverage. It is investigational and not approved for use. (Both: Courtesy of W.L. Gore and Associates, Inc., Neward, DE, USA)

In experienced hands, percutaneous access for both TEVAR and EVAR can be performed rapidly. In the hypotensive patient with non-palpable pulses, however, direct femoral exploration and arterial cutdown may be faster.

Complications of endograft repair include poor apposition of the proximal stent graft to the aortic arch because of poor conformation, undersizing, and infolding. Consequences may be minimal or catastrophic. Thorough planning and familiarity with device deployment is essential.

Long-term results of endovascular treatment of BTAI are unknown. Normal growth of the thoracic aorta has been observed in young patients in the years after endograft placement, but this growth appears to be attenuated in the grafted segment [56].

Abdominal Aorta

Abdominal aortic injury is rarely compatible with definitive endovascular intervention both because of presentation and because of anatomy. Penetrating injury to the aorta by definition removes the ability of the retroperitoneum to tamponade bleeding, usually resulting in hemodynamic instability and immediate laparotomy. Blunt abdominal aortic injury (BAAI) is exceedingly rare. Retrospective studies report an annual incidence of 0.02% to 0.1% of all trauma admissions [59, 60]. Even among autopsy series, BAAI was found in only 0.2% of blunt trauma mortalities.

BAAI may be graded in the same way as BTAI, ranging in severity from intimal flap to intramural hematoma, pseudoaneurysm, and free rupture. Intimal injury, however, carries greater risk in the abdominal aorta than in the thoracic. Evolution to overt dissection carries significant potential for occlusion of critical mesenteric and renal branches. Circumferential dissection, seen most often with flexion, extension, or distraction spinal injury, usually presents as acute aortic occlusion, a condition with extraordinarily high mortality. Early endovascular intervention for circumferential intimal injury has been described with successful preser-

vation of aortic patency [61]. As with penetrating injuries, free rupture is almost always treated with immediate laparotomy.

BAAI is classified by anatomic location into three zones. Zone I extends from the diaphragm to the superior mesenteric artery and Zone II from there to the renal arteries. The infrarenal aorta is Zone III. When considering endovascular repair, the location of the injury relative to branches of the aorta is the primary consideration. A 20-year retrospective review by Deree and associates found that more than 55% of all abdominal aortic injury fell into Zones I or II [62].

Acute occlusion of the superior mesenteric artery or celiac artery will result in fatal visceral ischemia. A critically stenosed orifice in a patient with chronic mesenteric occlusion, however, can often be sacrificed without consequence. Acute occlusion of the renal arteries will result in acute and permanent renal failure, but in the face of lethal exsanguination, this may be preferable. Coverage of one renal artery by aortic endograft does not necessarily result in clinically significant renal insufficiency.

Elective repair of paravisceral aneurysmal disease currently requires fenestrated endografts created for individual patients. Standardized fenestrated and branched grafts may become available in the next several years, and these devices may allow endovascular repair of the aorta in Zone I and Zone II.

Most injuries to the abdominal aorta may be treated with tube stent grafts, but Zone III injuries, if sufficiently close to the aortic bifurcation, will require either a bifurcated endograft or an aorto-uni-iliac endograft with femorofemoral bypass.

Abdominal aortic injury, both penetrating and blunt, with hemodynamic instability classically requires laparotomy for hemorrhage control with no opportunity for endovascular repair. The increasing prevalence of REBOA may allow a more frequent definitive endovascular repair of abdominal aortic injuries.

Lower Extremity

Pelvis

The utility of endovascular intervention in the treatment of blunt pelvic trauma has been very well established. The combination of orthopedic fixation and angiography with embolization has been in widespread use since the 1970s [63–67].

Historically, interventional radiologists have performed angioembolization. The distinction between the operating room and radiology suite has resulted in a temporal and spatial separation between pelvic fixation and angiography, both of which contribute to hemorrhage control [68]. This results not only in a delay in addressing pelvic bleeding but results in an unstable patient being outside of a resuscitative setting.

A drive to combine fixation and embolization has led to development of both temporary pelvic fixation systems and preperitoneal packing, both of which are designed to compensate for a delay that may not be necessary.

Similar to the upper extremity, penetrating injury to the iliac arterial system carries significant morbidity and mortality. The 30-day mortality of iliac vascular trauma ranges from approximately 25% to 40% depending upon accompanying injuries [69].

The use of endovascular strategies to treat penetrating iliac arterial injuries is particularly appealing given the frequency of accompanying bowel spillage and infectious risk of open prosthetic repair as well as the progressive difficulty presented by internal iliac arterial exposure.

Stent grafting of the common or external iliac arteries is made easier by the widespread availability of iliac arterial stents in a wide range of sizes.

The prolific collateralization across the pelvis as well as from the external iliac and common femoral arteries makes internal iliac artery embolization for penetrating trauma a definitive hemorrhage control procedure worth consideration. The associated risks include colon, buttock, and spinal cord ischemia but decrease as embolization is performed

more proximally [70]. Immediate or delayed crossover revascularization may preserve perfusion to the leg or to the internal iliac system enabling permanent occlusion of the common or external iliac arteries.

Infrainguinal

The ubiquity of peripheral arterial disease has resulted in a larger armamentarium and volume of experience with endovascular surgery in the lower extremity compared to the upper. The greater challenge of vascular exposure in the leg compared to the arm makes endoluminal intervention more appealing, but caution must be exercised.

The utility of contralateral retrograde access and placement of a working sheath cannot be overstated whether open or endovascular repair is performed. Angiographic localization of injury, intra-arterial instillation of vasodilators, and balloon control of proximal inflow are all enabled by sheath placement.

The treatment of common femoral arterial disease should be performed using an open technique but may be performed with proximal endoluminal balloon occlusion. This will allow repair of the entire vessel without entering the pelvis. Endovascular repair is not advised. The epigastric and circumflex branches of the common femoral artery are easily exposed and ligated. The vessel lies at a flexion point, and stents therein may kink, fracture, or thrombose. If a stent graft acutely occludes the profunda femoris, catastrophic thigh necrosis will result.

The most frequent etiology of profunda femoral injury is orthopedic and iatrogenic. Penetrating traumatic injury, however, is not unknown [71–76]. The root and proximal profunda are readily uncovered and repaired, but the distal branches are not easily exposed and extremely friable. They are easily embolized, however, and occlusion of any given branch is unlikely to have adverse effect [77]. In a patient with peripheral arterial disease, the profunda may provide the entire lower extremity arterial supply, and its branches

must be embolized with care. Repair of the midportion of the profunda with a stent graft has been reported [78].

The superficial femoral and popliteal arteries, whether injured through blunt or penetrating means, provide attractive targets for endovascular repair, but several factors must be taken into consideration before pursuing aggressive treatment.

The only information provided by angiography is endoluminal profile. The extent and degree of traction, thermal, blast, or crush damage is impossible to determine. If angiography demonstrates vessel occlusion, this may reflect thrombosis of a dissection, transection, or even extrinsic compression. Pharmacomechanical thrombectomy, angioplasty, or primary stent grafting is not advisable in any of these cases. The popliteal artery is also subject to flexion stress, and even elective stenting in this vessel is undertaken knowing the increased risk of kink, fracture, or occlusion [79, 80].

Endovascular repair of the femoropopliteal segment should only be considered if preoperative CT angiography confirms that the vessel has not been completely transected and if luminal patency is confirmed on initial angiography. Suction catheter thrombectomy has been performed in this setting, but the danger of worsening an injury or causing distal embolization is enormous [81]. Definitive placement of a stent, particularly a stent-graft, should be performed only if the patient is appropriate for postoperative anticoagulant or antiplatelet therapy.

Tibial endovascular intervention is not often performed for trauma due to the relative rarity of treatable injury. Tibial artery injury rarely results in life-threatening hemorrhage rather than contained pseudoaneurysm, but loss of patency correlates with the need for amputation [82]. While there are no stents approved for use below the knee in the United States, use of covered, drug-eluting, and bare metal coronary stents for injury has been described in several case reports and series [83–86]. The well-established poor patency of tibial stents placed for occlusive disease probably does not represent their performance in younger, healthy arteries. Coil

embolization of traumatic tibial pseudoaneurysm is well described [87–90].

Endovascular Therapy of Venous Trauma

Although the above discussion relates to arterial injury, endovascular treatment of venous injury is widely performed, if less frequently [91–98]. When formulating an endovenous trauma surgical plan, several critical differences from arterial therapy should be noted.

The variability in vessel size in the venous circulation is tremendous. Moreover, venous capacitance means that in the setting of trauma, the size of a vessel on venography is representative of neither the usual nor the maximal sizes of the target. For example, radiography studies have shown an average normal IVC diameter of around 20 mm. Similar studies on patients in hypovolemic shock demonstrated an average diameter of less than 7 mm [99, 100].

Flow in the venous system is directed centrally rather than peripherally. Inadvertent migration of arterial coils may result in ischemia requiring snare retrieval; migrated venous stents may result in foreign bodies in the heart or pulmonary circulation with immediate and detrimental physiologic effect [101–104].

The endovascular exclusion of arterial trauma with stent grafting is significantly aided by intraluminal pressure. Venous stents are not "pinned" to the vessel wall in the same way and rely much more upon the radial force of the stent to prevent "floating" of the conduit. This makes proper sizing all the more important.

Conclusion

The last 30 years have seen a revolution in not only the scope and techniques of vascular surgery. The principles of the field remain inviolate, but their application from within and with-

out the vessel lumen has changed the conceptual understanding of vascular surgery. Endovascular techniques afford rapid diagnostic and therapeutic maneuvers that supplement open surgery and enlarge the potential for treatment of all vascular conditions. Vascular trauma is no exception. For the trauma surgeon, a familiarity with the technical principles of endovascular surgery and with their application in various anatomical and injury contexts will enable assessment of the injured patient and consideration of open, endovascular, or hybrid treatment. As hybrid operating rooms become more widespread, endovascular and open procedures performed simultaneously will enable the patient to receive coordinated care without delays. Basic vascular surgical principles have not changed since Hallowell's brachial artery repair in 1759; endovascular trauma surgery simply allows their application more quickly and efficiently.

References

1. Avery LE, Stahlfeld KR, Corcos AC, Scifres AM, Ziembicki JA, Varcelotti J, et al. Evolving role of endovascular techniques for traumatic vascular injury: a changing landscape? J Trauma Acute Care Surg. 2012;72(1):41–6; discussion 6-7.
2. Branco BC, DuBose JJ, Zhan LX, Hughes JD, Goshima KR, Rhee P, et al. Trends and outcomes of endovascular therapy in the management of civilian vascular injuries. J Vasc Surg. 2014;60(5):1297–307.e1.
3. Branco BC, Naik-Mathuria B, Montero-Baker M, Gilani R, West CA, Mills JL Sr, et al. Increasing use of endovascular therapy in pediatric arterial trauma. J Vasc Surg. 2017;66(4):1175–83.e1.
4. Shalhub S, Starnes BW, Hatsukami TS, Karmy-Jones R, Tran NT. Repair of blunt thoracic outlet arterial injuries: an evolution from open to endovascular approach. J Trauma. 2011;71(5):E114–21.
5. Trellopoulos G, Georgiadis GS, Aslanidou EA, Nikolopoulos ES, Pitta X, Papachristodoulou A, et al. Endovascular management of peripheral arterial trauma in patients presenting in hemorrhagic shock. J Cardiovasc Surg. 2012;53(4):495–506.

6. Reuben BC, Whitten MG, Sarfati M, Kraiss LW. Increasing use of endovascular therapy in acute arterial injuries: analysis of the National Trauma Data Bank. J Vasc Surg. 2007;46(6):1222–6.
7. Kirkpatrick AW, Vis C, Dube M, Biesbroek S, Ball CG, Laberge J, et al. The evolution of a purpose designed hybrid trauma operating room from the trauma service perspective: the RAPTOR (Resuscitation with Angiography Percutaneous Treatments and Operative Resuscitations). Injury. 2014;45(9):1413–21.
8. Shah SR, Wearden PD, Gaines BA. Pediatric peripheral vascular injuries: a review of our experience. J Surg Res. 2009;153(1):162–6.
9. Leiderman DBD, Zerati AE, Wolosker N, Hoffmann Melo HA, da Silva ES, De Luccia N. Endovascular treatment of penetrating injury to the vertebral artery by a stab wound: case report and literature review. Ann Vasc Surg. 2017;45:267.e1–5.
10. Rao D, Yu H, Zhu H, Yu K, Hu X, Xie L. Superselective transcatheter renal artery embolization for the treatment of hemorrhage from non-iatrogenic blunt renal trauma: report of 16 clinical cases. Ther Clin Risk Manag. 2014;10:455–8.
11. Foley PT, Kavnoudias H, Cameron PU, Czarnecki C, Paul E, Lyon SM. Proximal versus distal splenic artery embolisation for blunt splenic trauma: what is the impact on splenic immune function? Cardiovasc Intervent Radiol. 2015;38(5):1143–51.
12. Beall AC Jr, Diethrich EB, Cooley DA, DeBakey ME. Surgical management of penetrating cardiovascular trauma. South Med J. 1967;60(7):698–704.
13. Hermreck AS. The history of cardiopulmonary resuscitation. Am J Surg. 1988;156(6):430–6.
14. Moore EE, Moore JB, Galloway AC, Eiseman B. Postinjury thoracotomy in the emergency department: a critical evaluation. Surgery. 1979;86(4):590–8.
15. Hughes CW. Use of an intra-aortic balloon catheter tamponade for controlling intra-abdominal hemorrhage in man. Surgery. 1954;36(1):65–8.
16. Aso S, Matsui H, Fushimi K, Yasunaga H. Resuscitative endovascular balloon occlusion of the aorta or resuscitative thoracotomy with aortic clamping for noncompressible torso hemorrhage: a retrospective nationwide study. J Trauma Acute Care Surg. 2017;82(5):910–4.
17. Moore LJ, Brenner M, Kozar RA, Pasley J, Wade CE, Baraniuk MS, et al. Implementation of resuscitative endovascular balloon occlusion of the aorta as an alternative to resuscitative

thoracotomy for noncompressible truncal hemorrhage. Journal Trauma Acute Care Surg. 2015;79(4):523–30; discussion 30–2.
18. Pezy P, Flaris AN, Prat NJ, Cotton F, Lundberg PW, Caillot JL, et al. Fixed-distance model for balloon placement during fluoroscopy-free resuscitative endovascular balloon occlusion of the aorta in a civilian population. JAMA Surg. 2017;152(4):351–8.
19. Stannard A, Eliason JL, Rasmussen TE. Resuscitative endovascular balloon occlusion of the aorta (REBOA) as an adjunct for hemorrhagic shock. J Trauma. 2011;71(6):1869–72.
20. Yang L, Chong-Qi T, Hai-Bo S, Lan Z, Tian-Fu Y, Hong D, et al. Appling the abdominal aortic-balloon occluding combine with blood pressure sensor of dorsal artery of foot to control bleeding during the pelvic and sacrum tumors surgery. J Surg Oncol. 2008;97(7):626–8.
21. Veith FJ, Gupta S, Daly V. Technique for occluding the supraceliac aorta through the abdomen. Surg Gynecol Obstet. 1980;151(3):426–8.
22. Miura F, Takada T, Ochiai T, Asano T, Kenmochi T, Amano H, et al. Aortic occlusion balloon catheter technique is useful for uncontrollable massive intraabdominal bleeding after hepato-pancreato-biliary surgery. J Gastrointest Surg. 2006;10(4):519–22.
23. Siebler J, Dipasquale T, Sagi HC. Use of temporary partial intrailiac balloon occlusion for decreasing blood loss during open reduction and internal fixation of acetabular and pelvis fractures. J Orthop Trauma. 2012;26(6):e54–7.
24. Harma M, Kunt AS, Andac MH, Demir N. Balloon occlusion of the descending aorta in the treatment of severe post-partum haemorrhage. Aust N Z J Obstet Gynaecol. 2004;44(2):170–1.
25. Matsuda H, Tanaka Y, Hino Y, Matsukawa R, Ozaki N, Okada K, et al. Transbrachial arterial insertion of aortic occlusion balloon catheter in patients with shock from ruptured abdominal aortic aneurysm. J Vasc Surg. 2003;38(6):1293–6.
26. Malina M, Veith F, Ivancev K, Sonesson B. Balloon occlusion of the aorta during endovascular repair of ruptured abdominal aortic aneurysm. J Endovasc Ther. 2005;12(5):556–9.
27. Conti BM, Richards JE, Kundi R, Nascone J, Scalea TM, McCunn M. Resuscitative endovascular balloon occlusion of the aorta and the anesthesiologist: a case report and literature review. A A Case Rep. 2017;9(5):154–7.
28. Bell RS, Ecker RD, Severson MA 3rd, Wanebo JE, Crandall B, Armonda RA. The evolution of the treatment of trau-

matic cerebrovascular injury during wartime. Neurosurg Focus. 2010;28(5):E5.
29. Shahan CP, Sharpe JP, Stickley SM, Manley NR, Filiberto DM, Fabian TC, et al. The changing role of endovascular stenting for blunt cerebrovascular injuries. J Trauma Acute Care Surg. 2018;84(2):308–11.
30. Choi HC, Park SE, Choi DS, Shin HS, Kim JE, Choi HY, et al. Ruptured extracranial carotid artery: endovascular treatment with covered stent graft. J Neuroradiol. 2018;45(4):217–23.
31. Kreiser K, Grober I, Zimmer C, Storck K. Stent grafts in patients with carotid blowout syndrome: outcome and antiplatelet therapy in preventive versus emergency cases. Head Neck. 2018;40(11):2521–7.
32. Tsai YH, Wong HF, Weng HH, Chen YL. Stent-graft treatment of traumatic carotid artery dissecting pseudoaneurysm. Neuroradiology. 2010;52(11):1011–6.
33. Chaer RA, Derubertis B, Kent KC, McKinsey JF. Endovascular treatment of traumatic carotid pseudoaneurysm with stenting and coil embolization. Ann Vasc Surg. 2008;22(4):564–7.
34. Seth R, Obuchowski AM, Zoarski GH. Endovascular repair of traumatic cervical internal carotid artery injuries: a safe and effective treatment option. AJNR Am J Neuroradiol. 2013;34(6):1219–26.
35. Richard SA, Zhang CW, Wu C, Ting W, Xiaodong X. Traumatic penetrating neck injury with right common carotid artery dissection and stenosis effectively managed with stenting: a case report and review of the literature. Case Rep Vasc Med. 2018;2018:4602743.
36. Yoshihara H, Vanderheiden TF, Harasaki Y, Beauchamp KM, Stahel PF. Fatal outcome after brain stem infarction related to bilateral vertebral artery occlusion – case report of a detrimental complication of cervical spine trauma. Patient Saf Surg. 2011;5:18.
37. Berne JD, Cook A, Rowe SA, Norwood SH. A multivariate logistic regression analysis of risk factors for blunt cerebrovascular injury. J Vasc Surg. 2010;51(1):57–64.
38. Cothren CC, Moore EE, Biffl WL, Ciesla DJ, Ray CE Jr, Johnson JL, et al. Cervical spine fracture patterns predictive of blunt vertebral artery injury. J Trauma. 2003;55(5):811–3.
39. Demetriades D, Chahwan S, Gomez H, Peng R, Velmahos G, Murray J, et al. Penetrating injuries to the subclavian and axillary vessels. J Am Coll Surg. 1999;188(3):290–5.

40. Wall MJ Jr, Granchi T, Liscum K, Mattox KL. Penetrating thoracic vascular injuries. Surg Clin North Am. 1996;76(4):749–61.
41. Graham JM, Feliciano DV, Mattox KL, Beall AC Jr, DeBakey ME. Management of subclavian vascular injuries. J Trauma. 1980;20(7):537–44.
42. Branco BC, Boutrous ML, DuBose JJ, Leake SS, Charlton-Ouw K, Rhee P, et al. Outcome comparison between open and endovascular management of axillosubclavian arterial injuries. J Vasc Surg. 2016;63(3):702–9.
43. Carrick MM, Morrison CA, Pham HQ, Norman MA, Marvin B, Lee J, et al. Modern management of traumatic subclavian artery injuries: a single institution's experience in the evolution of endovascular repair. Am J Surg. 2010;199(1):28–34.
44. Shalhub S, Starnes BW, Tran NT. Endovascular treatment of axillosubclavian arterial transection in patients with blunt traumatic injury. J Vasc Surg. 2011;53(4):1141–4.
45. Xenos ES, Freeman M, Stevens S, Cassada D, Pacanowski J, Goldman M. Covered stents for injuries of subclavian and axillary arteries. J Vasc Surg. 2003;38(3):451–4.
46. Rohlffs F, Larena-Avellaneda AA, Petersen JP, Debus ES, Kolbel T. Through-and-through wire technique for endovascular damage control in traumatic proximal axillary artery transection. Vascular. 2015;23(1):99–101.
47. Stedman HH, Carpenter JP, Shlansky-Goldberg RD. Percutaneous balloon catheter vascular control for infected axillary artery pseudoaneurysm. J Cardiovasc Surg. 1994;35(6):529–31.
48. Sommer C, Frutiger A, Lutolf M, Mark G, Ruedi T. Temporary balloon catheter occlusion for control of bleeding of a blunt injury of the proximal axillary artery. Case report and review of the literature. Unfallchirurg. 1992;95(10):498–500.
49. Gordon RL, Landau EH, Shifrin E, Romanoff H. The use of a balloon catheter in the treatment of an iatrogenic pseudo-aneurysm of the subclavian artery. J Cardiovasc Surg. 1983;24(2):178–80.
50. Lonn L, Delle M, Karlstrom L, Risberg B. Should blunt arterial trauma to the extremities be treated with endovascular techniques? J Trauma. 2005;59(5):1224–7.
51. George S, Mamas M, Nolan J, Ratib K. Radial artery perforation treated with balloon tracking and guide catheter tamponade – a case series. Cardiovasc Revasc Med. 2016;17(7):480–6.
52. Grigorian A, Spencer D, Donayre C, Nahmias J, Schubl S, Gabriel V, et al. National trends of thoracic endovascular aortic

repair versus open repair in blunt thoracic aortic injury. Ann Vasc Surg. 2018;52:72–8.
53. Lee WA, Matsumura JS, Mitchell RS, Farber MA, Greenberg RK, Azizzadeh A, et al. Endovascular repair of traumatic thoracic aortic injury: clinical practice guidelines of the Society for Vascular Surgery. J Vasc Surg. 2011;53(1):187–92.
54. Heneghan RE, Aarabi S, Quiroga E, Gunn ML, Singh N, Starnes BW. Call for a new classification system and treatment strategy in blunt aortic injury. J Vasc Surg. 2016;64(1):171–6.
55. DuBose JJ, Leake SS, Brenner M, Pasley J, O'Callaghan T, Luo-Owen X, et al. Contemporary management and outcomes of blunt thoracic aortic injury: a multicenter retrospective study. J Trauma Acute Care Surg. 2015;78(2):360–9.
56. Wallace GA, Starnes BW, Hatsukami TS, Sobel M, Singh N, Tran NT. Intravascular ultrasound is a critical tool for accurate endograft sizing in the management of blunt thoracic aortic injury. J Vasc Surg. 2015;61(3):630–5.
57. Bodell BD, Taylor AC, Patel PJ. Thoracic endovascular aortic repair: review of current devices and treatments options. Tech Vasc Interv Radiol. 2018;21(3):137–45.
58. Haulon S, Soler R, Eliason J, Ramadan R, Guihaire J, Fabre D. Commentary: extending the boundaries of total endovascular aortic arch repair. J Endovasc Ther. 2018;25(5):547–9.
59. Harris DG, Drucker CB, Brenner ML, Sarkar R, Narayan M, Crawford RS. Patterns and management of blunt abdominal aortic injury. Ann Vasc Surg. 2013;27(8):1074–80.
60. Michaels AJ, Gerndt SJ, Taheri PA, Wang SC, Wahl WL, Simeone DM, et al. Blunt force injury of the abdominal aorta. J Trauma. 1996;41(1):105–9.
61. Kalsi R, Drucker CB, Salazar JH, Luther LI, Diaz JJ, Kundi R. Blunt multifocal aortic injury with abdominal aortic intimointimal intussusception. J Vasc Surg Cases Innov Tech. 2018;4(1):37–40.
62. Deree J, Shenvi E, Fortlage D, Stout P, Potenza B, Hoyt DB, et al. Patient factors and operating room resuscitation predict mortality in traumatic abdominal aortic injury: a 20-year analysis. J Vasc Surg. 2007;45(3):493–7.
63. Margolies MN, Ring EJ, Waltman AC, Kerr WS Jr, Baum S. Arteriography in the management of hemorrhage from pelvic fractures. N Engl J Med. 1972;287(7):317–21.
64. Kerr WS Jr, Margolies MN, Ring EJ, Waltman AC, Baum SN. Arteriography in pelvic fractures with massive hemorrhage. Trans Am Assoc Genitourin Surg. 1972;64:14–7.

65. Ring EJ, Waltman AC, Athanasoulis C, Smith JC Jr, Baum S. Angiography in pelvic trauma. Surg Gynecol Obstet. 1974;139(3):375–80.
66. Ring EJ, Athanasoulis C, Waltman AC, Margolies MN, Baum S. Arteriographic management of hemorrhage following pelvic fracture. Radiology. 1973;109(1):65–70.
67. Kerr WS Jr, Margolies MN, Ring EJ, Waltman AC, Baum SN. Arteriography in pelvic fractures with massive hemorrhage. J Urol. 1973;109(3):479–82.
68. Thorson CM, Ryan ML, Otero CA, Vu T, Borja MJ, Jose J, et al. Operating room or angiography suite for hemodynamically unstable pelvic fractures? J Trauma Acute Care Surg. 2012;72(2):364–70; discussion 71–2.
69. Magee GA, Cho J, Matsushima K, Strumwasser A, Inaba K, Jazaeri O, et al. Isolated iliac vascular injuries and outcome of repair versus ligation of isolated iliac vein injury. J Vasc Surg. 2018;67(1):254–61.
70. Chitragari G, Schlosser FJ, Ochoa Chaar CI, Sumpio BE. Consequences of hypogastric artery ligation, embolization, or coverage. J Vasc Surg. 2015;62(5):1340–7.e1.
71. Singh S, Arora S, Thora A, Mohan R, Sural S, Dhal A. Pseudoaneurysm of profunda femoris artery following dynamic hip screw fixation for intertrochanteric femoral fracture. Chin J Traumatol. 2013;16(4):233–6.
72. Karkos CD, Karamanos DG, Papazoglou KO, Papadimitriou DN, Gerogiannis IN, Demiropoulos FP, et al. Ruptured pseudoaneurysm of the profunda femoris artery due to pellet injury: endovascular treatment by coil embolization. Cardiovasc Intervent Radiol. 2009;32(4):837–9.
73. Cantasdemir M, Kantarci F, Mihmanli I, Numan F. Embolization of profunda femoris artery branch pseudoaneurysms with ethylene vinyl alcohol copolymer (onyx). J Vasc Interv Radiol. 2002;13(7):725–8.
74. Potenza V, Saputo U, Catellani F, Farsetti P, Caterini R. Laceration of a branch of the profunda femoris artery caused by a spike of the displaced lesser trochanter in an intertrochanteric femoral fracture. A case report. Int J Surg Case Rep. 2016;24:195–8.
75. Lee PYF, Rao PVR, Golding DM, Brock J. Delayed profunda femoris artery bleeding after intramedullary nailing of an unstable intertrochanteric fracture: a case report. JBJS Case Connect. 2017;7(3):e60.

76. Harper K, Iorio J, Balasubramanian E. Profunda femoris pseudoaneurysm following total hip arthroplasty revision. Case Rep Orthop. 2015;2015:301949.
77. Biswas S, McNerney P, Kiproff P. Pseudoaneurysm of the profunda femoris artery following blunt trauma treated by endovascular coil embolization: review of two cases and relevant literature. Case Rep Emerg Med. 2017;2017:8079674.
78. Pecoraro F, Dinoto E, Bracale UM, Badalamenti G, Farina A, Bajardi G. Symptomatic deep femoral artery pseudoaneurysm endovascular exclusion. Case report and literature review. Ann Vasc Surg. 2017;42:303.e5–9.
79. Eslami MH, Rybin D, Doros G, Farber A. Open repair of asymptomatic popliteal artery aneurysm is associated with better outcomes than endovascular repair. J Vasc Surg. 2015;61(3):663–9.
80. Leake AE, Segal MA, Chaer RA, Eslami MH, Al-Khoury G, Makaroun MS, et al. Meta-analysis of open and endovascular repair of popliteal artery aneurysms. J Vasc Surg. 2017;65(1):246–56.e2.
81. Hutto JD, Reed AB. Endovascular repair of an acute blunt popliteal artery injury. J Vasc Surg. 2007;45(1):188–90.
82. Scalea JR, Crawford R, Scurci S, Danquah J, Sarkar R, Kufera J, et al. Below-the-knee arterial injury: the type of vessel may be more important than the number of vessels injured. J Trauma Acute Care Surg. 2014;77(6):920–5.
83. Spirito R, Trabattoni P, Pompilio G, Zoli S, Agrifoglio M, Biglioli P. Endovascular treatment of a post-traumatic tibial pseudoaneurysm and arteriovenous fistula: case report and review of the literature. J Vasc Surg. 2007;45(5):1076–9.
84. Rabellino M, Shinzato S, Aragon-Sanchez J, Peralta O, Marenchino R, Garcia-Monaco R. Leg ulcer as a complication of a posttraumatic tibial arteriovenous fistula treated by endovascular approach with stent-graft placement. Int J Low Extrem Wounds. 2012;11(3):147–51.
85. Jones A, Kumar S. Successful stenting of iatrogenic anterior tibial artery pseudoaneurysm. EJVES Short Rep. 2016;30:4–6.
86. Biagioni RB, Burihan MC, Nasser F, Biagioni LC, Ingrund JC. Endovascular treatment of penetrating arterial trauma with stent grafts. VASA. 2018;47(2):125–30.
87. Yu PT, Rice-Townsend S, Naheedy J, Almodavar H, Mooney DP. Delayed presentation of traumatic infrapopliteal arteriovenous fistula and pseudoaneurysm in a 10-year-old boy managed by coil embolization. J Pediatr Surg. 2012;47(2):e7–10.

88. Verma R, Seymour R, Hockings M. Endovascular coil embolization of pseudoaneurysm of a branch of the anterior tibial artery following total knee replacement. J Knee Surg. 2009;22(3):269–71.
89. Singh D, Ferero A. Traumatic pseudoaneurysm of the posterior tibial artery treated by endovascular coil embolization. Foot Ankle Spec. 2013;6(1):54–8.
90. Jain M, Naregal A, Kasat L, Bajaj R, Borwankar SS. Anterior tibial artery pseudoaneurysm. Indian J Pediatr. 1999;66(2):298–300.
91. Zahradnik V, Kashyap VS. Alternative management of iliac vein injury during anterior lumbar spine exposure. Ann Vasc Surg. 2012;26(2):277.e15–8.
92. Yamanaka K, Yamamoto A, Ishida K, Matsuzaki J, Ozaki T, Ishihara M, et al. Successful endovascular therapy of a penetrating zone III jugular bulb injury. A case report. Interv Neuroradiol. 2012;18(2):195–9.
93. Willaert W, Van Herzeele I, Ceelen W, Van De Putte D, Vermassen F, Pattyn P. Endovascular treatment of an iatrogenic perforation of the internal iliac vein. Ann Vasc Surg. 2012;26(5):733.e1–4.
94. Sofue K, Sugimoto K, Mori T, Nakayama S, Yamaguchi M, Sugimura K. Endovascular uncovered Wallstent placement for life-threatening isolated iliac vein injury caused by blunt pelvic trauma. Jpn J Radiol. 2012;30(8):680–3.
95. Qi Y, Gillespie DL. Venous trauma: new lessons and old debates. Perspect Vasc Surg Endovasc Ther. 2011;23(2):74–9.
96. Merchant M, Pallan P, Prabhakar N, Saker M, Resnick SA. Treatment of traumatic thoracic and iliac venous injury with endovascular stent-grafts. J Vasc Interv Radiol. 2013;24(12):1920–3.
97. Briggs CS, Morcos OC, Moriera CC, Gupta N. Endovascular treatment of iatrogenic injury to the retrohepatic inferior vena cava. Ann Vasc Surg. 2014;28(7):1794.e13–5.
98. Garcarek J, Watorek E, Kurcz J, Kusztal M, Golebiowski T, Letachowicz K, et al. Endovascular reparation of central vein injury with balloon-protected embolization. Cardiovasc Intervent Radiol. 2015;38(4):1057–9.
99. Yanagawa Y, Sakamoto T, Okada Y. Hypovolemic shock evaluated by sonographic measurement of the inferior vena cava during resuscitation in trauma patients. J Trauma. 2007;63(6):1245–8.. discussion 8

100. Prince MR, Novelline RA, Athanasoulis CA, Simon M. The diameter of the inferior vena cava and its implications for the use of vena caval filters. Radiology. 1983;149(3):687–9.
101. Rastogi N, Kabutey NK, Kim D. Unintended coil migration into the right ventricle during the right ovarian vein coil embolization. Vasc Endovasc Surg. 2011;45(7):660–4.
102. Moriel EZ, Mehringer CM, Schwartz M, Rajfer J. Pulmonary migration of coils inserted for treatment of erectile dysfunction caused by venous leakage. J Urol. 1993;149(5 Pt 2):1316–8.
103. Toyoda N, Torregrossa G, Itagaki S, Pawale A, Reddy R. Intracardiac migration of vena caval stent: decision-making and treatment considerations. J Card Surg. 2014;29(3):320–2.
104. Balasubramaniyam N, Garg J, Rawat N, Chugh S, Mittal V, Baby B, et al. Dual stent migration to the heart and pulmonary artery. Am J Ther. 2014;21(6):e199–203.

Chapter 20
Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA)

Stephen E. Varga

History

Non-compressible torso hemorrhage (NCTH) is a leading cause of potentially preventable death in trauma [1]. Resuscitative thoracotomy has traditionally been employed to provide temporary hemostasis through aortic cross-clamping, but its indications are controversial; such a course of action is highly morbid and carries potential risk to the provider [2]. Resuscitative endovascular balloon occlusion of the aorta (REBOA) represents a less invasive adjunct, designed to sustain central circulation until hemostasis can be achieved with potential for improved outcomes.

Temporary occlusion of the aorta with an endovascular balloon to increase cerebral and coronary perfusion while controlling distal hemorrhage is not a new concept. The use of an open technique to insert an intra-aortic occlusive balloon to control aortic hemorrhage was first described in the Korean War [3], but it did not become popular in trauma due to inex-

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perience, the lack of easily useable devices, and high possibility for complications. With advances in vascular surgery, interest in REBOA was reinvigorated, particularly due to its well-reported applications in controlling bleeding from ruptured abdominal aortic aneurysm [4, 5], complicated pelvic operative procedures [6, 7], orthopedic procedures [8], and postpartum hemorrhage [9]. The use of REBOA in animal models with hemorrhagic shock demonstrated improvement in lactate, PH, pCO_2, and central circulation, paving the way for its application to trauma [10–13]. With continued evolution of endovascular techniques and improved devices, REBOA is increasingly used as an adjunct in trauma resuscitation. Through the development of a minimally invasive approach through the common femoral artery (CFA) [5, 14, 15], REBOA has become a practical tool that can be used to control NCTH in trauma patients and should now be considered as an alternative to a resuscitative thoracotomy in traumatic arrest for intra-abdominal, pelvic, or junctional hemorrhage [16–21].

Patient Selection

Patient selection is a key factor when making a decision about who may benefit from REBOA placement. Currently, there are no randomized studies to help guide this decision, but there are several strong relative contraindications to consider. Any patient with penetrating thoracic injury or concern for bleeding proximal to balloon deployment site should not be considered a candidate for REBOA use as this may worsen the bleeding. A chest X-ray (CXR) should be obtained, if possible before considering REBOA after blunt injuries. Patients with a widened mediastinum on initial CXR may have a blunt thoracic aorta injury which may rupture after the REBOA balloon is inflated [22]. If there are no contraindications for REBOA, our institution's current indication for REBOA is any patient with hypotension (systolic blood pressure less than 90) and concern for hemorrhage below the diaphragm who is a partial or nonresponder to a

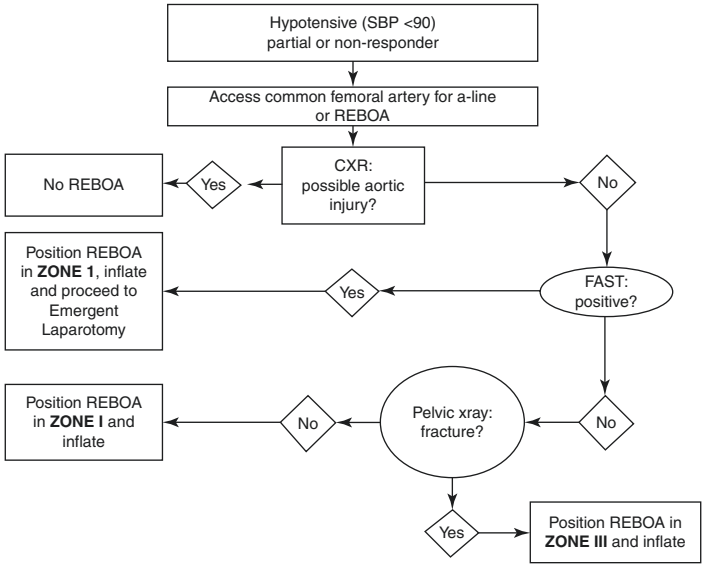

FIGURE 20.1 R Adams Cowley Shock Trauma Center (University of Maryland) clinical algorithm for REBOA placement

fluid or blood transfusion challenge. The Shock Trauma Center clinical algorithm for REBOA is outlined in Fig. 20.1.

Arterial Access

The first step in placing a REBOA begins with cannulation of the CFA. It is essential that CFA access is achieved rapidly and correctly, as achieving vascular access is the rate-limiting step of REBOA [18]. In addition, using the superficial femoral aretry for sheath placemnet may increase the rate of access site complications. Our current practice is to obtain percutaneous CFA access in any patient in shock and those with high-risk mechanisms. We place an arterial line which allows continuous blood pressure measurement. This may help guide the decision to place a REBOA.

CFA access can be accomplished using one of the three techniques: percutaneous, open, or exchange over a guidewire from an existing femoral arterial line. Occasionally, the CFA can be accessed using only palpation as a guide. In most cases, ultrasound guidance for percutaneous access is ideal because it enhances the success rate in achieving arterial access in patients with severe hypotension or no palpable pulse to guide needle insertion. Either a standard femoral arterial line kit or a 5 Fr micropuncture set can be used to initially gain access to the CFA. Once the artery is identified with ultrasound guidance, we prefer to access the CFA under direct visualization using the 5 Fr micropuncture set's hollow 21 gauge at a 45-degree angle through which a 0.018 inch wire can be passed. After the wire has been passed into the artery, the needle is removed, and a small incision is made in the skin at the wire insertion site. Next, the 5 Fr sheath with dilator is placed over the wire through the skin and into the artery. The dilator and wire are then removed, leaving the sheath in place for arterial monitoring and access if REBOA is indicated.

Regardless of whether the CFA is accessed with ultrasound guidance or not, it is important to remember to puncture the skin more distally than normal. The CFA must be accessed using a gentle angle, as parallel as possible to the CFA. If the CFA is punctured at a right angle, the guidewire may pass easily, but the sheath will have trouble making the angle into the artery. As the tip of the introducer is pointed, this may cause injury to the posterior wall of the CFA.

If percutaneous access of the CFA is not successful, an open cutdown can be used. The following steps are recommended in achieving CFA access with the open technique. The CFA is approximately two fingers' breadth lateral to the pubic tubercle, starts superficially, and progresses deep and medially. The incision to access the CFA should be made longitudinally along the medial boarder of the Sartorius and should be carried above the inguinal ligament (Fig. 20.2). The femoral sheath is then identified and opened to expose the anterior surface of the CFA. It is often helpful to retract the CFA up in to the wound to ensure the needle engages the artery smoothly. Place a hollow 18 gauge needle at a 45-degree angle

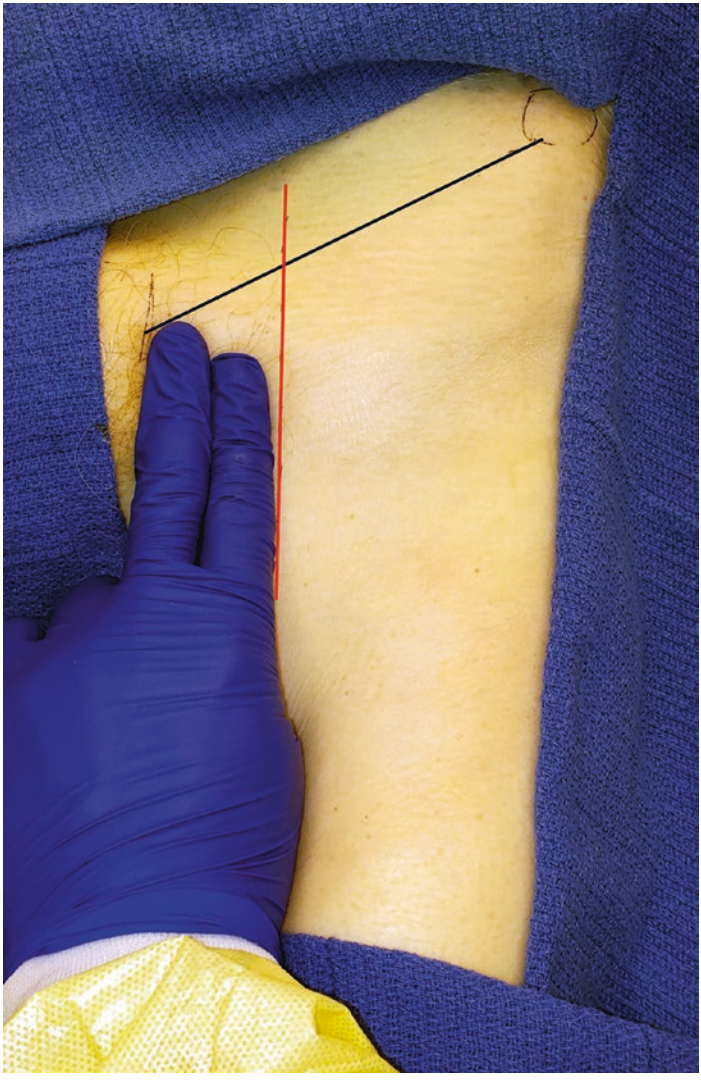

FIGURE 20.2 Incision (red line) to access CFA should be made approximately two fingers' breadth lateral to the pubic tubercle and should be carried above the inguinal ligament (black line)

into the artery, and then pass a standard 0.025 inch J-tip guidewire through the needle. After the wire has been passed, remove the needle. Next, ensure that the 7 Fr sheath internal dilator is firmly in place through the lumen of the 7 Fr sheath, and then advance the dilator and sheath as a single unit over the guidewire through the artery. The wire and dilator are then removed, leaving the 7 Fr sheath in place. Flush the sheath with normal saline, and assure that the stopcock on the side port of the sheath is in the off position to avoid bleeding.

REBOA Device and Sheath Upsizing

Our institution's device of choice is the Prytime ER-REBOA™ catheter (Boerne, TX, USA) (Fig. 20.3). This device was specifically designed for trauma application, as it can be placed through a 7 Fr sheath and has an atraumatic tip allowing it to be placed without a guidewire. In addition, it has an arterial monitoring port distal to the balloon that can be used to measure arterial response to REBOA and

Figure 20.3 Prytime ER-REBOA. (Used with permission of Prytime Medical Devices, Inc. Boerne, TX, USA. Available at: http://prytimemedical.com/wp-content/uploads/2018/06/ADV-006-Rev-H_ER-REBOA-Catheter-Quick-Reference-Guide-Wall-Poster_8.5x11_EN-2.pdf)

external markings on the catheter to facilitate appropriate depth of insertion.

Once the decision has been made to place the ER-REBOA, the 5 Fr sheath must be upsized to a 7 Fr sheath. This is accomplished by placing a standard 0.035 inch J-tip guidewire through the lumen of the 5 Fr sheath into the artery, allowing the 5 Fr sheath to be removed while maintaining arterial access. The small incision over the wire is then enlarged to facilitate placement of the larger sheath. Next ensure that the 7 Fr sheath internal dilator is firmly in place through the lumen of the 7 Fr sheath, and then advance as a single unit over the guidewire through the skin and into the artery. It is usually necessary to rotate the sheath during placement to allow it to slip into the artery, particularly in spastic CFAs often seen in trauma patients in shock. The wire and dilator are then removed leaving the 7 Fr sheath in place. Flush the sheath with normal saline, and assure that the stopcock on the side port of the sheath in in the off position to avoid bleeding.

REBOA Positioning, Insertion, and Inflation

Using the Shock Trauma Center Algorithm for REBOA, (Fig. 20.1) the clinician must decide which aortic zone (Fig. 20.4) is to be occluded. Zone I is the descending thoracic aorta between the origin of the left subclavian artery and the celiac arteries. Zone II is the descending aorta between the celiac and the lowest renal artery and is a "no-occlusion zone". Zone III is the infrarenal abdominal aorta between the lowest renal artery and the aortic bifurcation. The decision about which zone is to be occluded is facilitated by the Focused Assessment with Sonography for Trauma (FAST) exam and the presence of pelvic fractures. If the FAST exam is positive, suggestive of intra-abdominal hemorrhage, position the REBOA in Zone I, and proceed to emergent laparotomy. If the FAST exam is negative and there is suspicion for a pelvic source of hemorrhage, first obtain a pelvic radiograph. If the pelvic radiograph is negative for fracture, position the

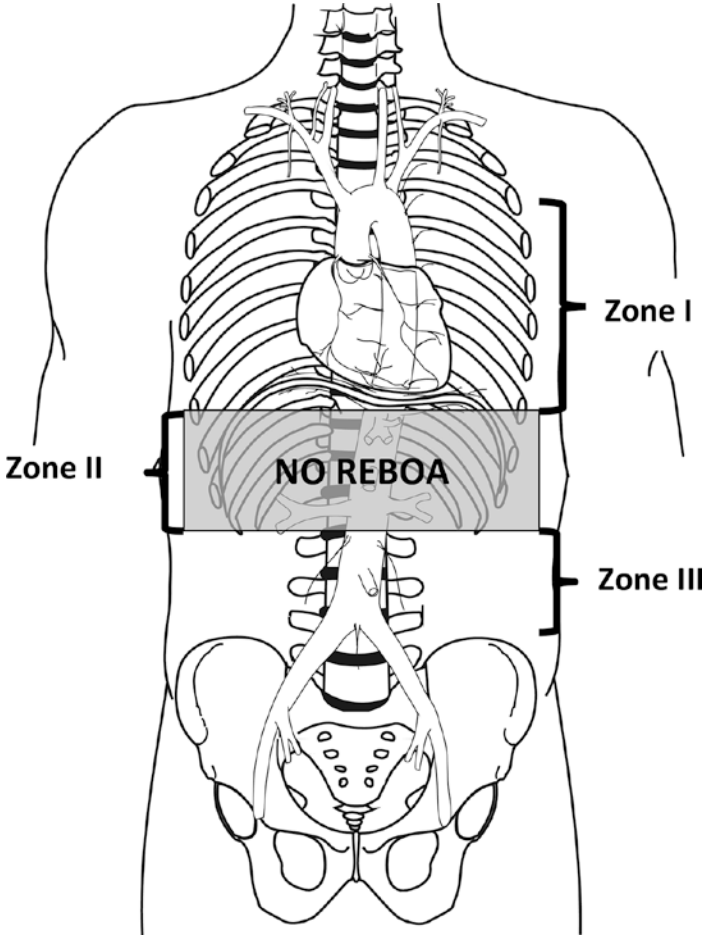

FIGURE 20.4 Aortic ones for REBOA placement. (Used with permission of Wolters Kluwer Health, Inc., from Stannard et al. [25])

REBOA in Zone I; if the pelvic radiograph is positive for fracture, position the REBOA in Zone III (Fig. 20.4). Appropriate depth of advancement for REBOA can be derived from epidemiologic studies [23], radiographic visualization, or external measurement using landmarks. External

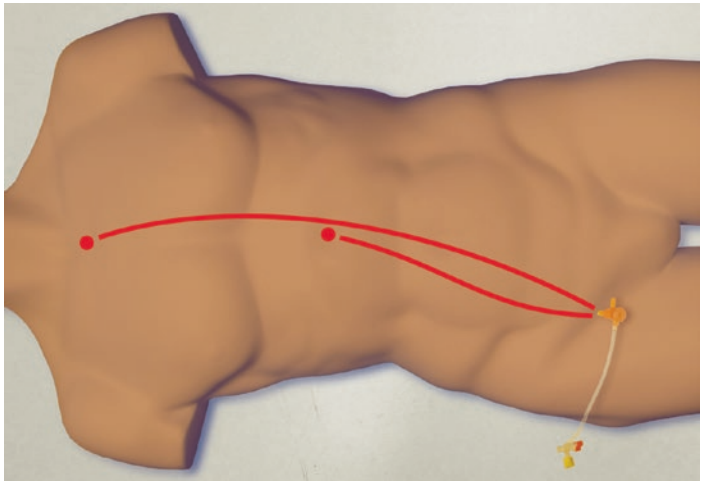

FIGURE 20.5 External landmarks for REBOA aortic zone placement. Sternal notch to the femoral artery sheath for Zone I (superior line). Xiphoid to the femoral artery sheath for Zone III (inferior line). (Used with permission of Prytime Medical Devices, Inc. Boerne, TX, USA. Available at: http://prytimemedical.com/wp-content/uploads/2018/06/ADV-006-Rev-H_ER-REBOA-Cathseter-Quick-Reference-Guide-Wall-Poster_8.5x11_EN-2.pdf)

measurement our preferred method, as patients with indications for REBOA placement are often in extremis and timely radiographic images are rarely available.

Zone I will be the initial zone of placement in most patients who present in hemorrhagic shock from a suspected hemorrhage below the diaphragm. To determine the depth of deployment of Prytime ER-REBOA using external landmarks for Zone I, place the atraumatic tip of the catheter at the sternal notch, and then make note of the distance to the femoral artery sheath opening (Fig. 20.5). Zone III occlusion may be helpful in cases with pelvic or junctional femoral hemorrhage [24]; to determine the depth of deployment in Zone III, place the atraumatic tip of the catheter just below

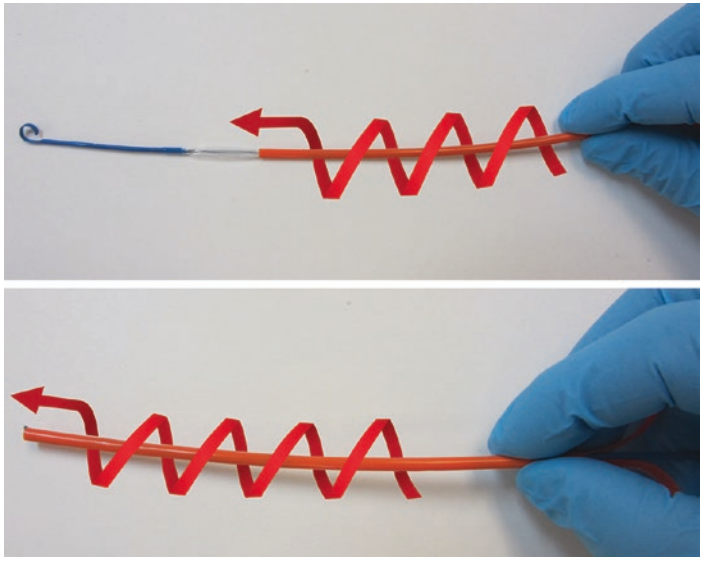

Figure 20.6 Corkscrew twisting technique to wrap the balloon with the peel-away cover. (Used with permission of Prytime Medical Devices, Inc. Boerne, TX, USA. Available at: http://prytimemedical.com/wp-content/uploads/2018/06/ADV-006-Rev-H_ER-REBOA-Catheter-Quick-Reference-Guide-Wall-Poster_8.5x11_EN-2.pdf)

the xiphoid process, and then make note of the distance to the femoral artery sheath opening (Fig. 20.5) [23–28].

Once the desired depth of insertion is established, prepare the Prytime ER-REBOA by attaching a 30 cc syringe filled to 24 cc (with 16 cc saline and 8 cc contrast or 24 cc saline if contrast is not readily available) to the balloon port. Apply negative pressure to the 30 cc for 5 seconds to remove any remaining air from the balloon, and close the stopcock on the balloon port. Next advance the orange peel-away cover over the balloon and the atraumatic tip using a corkscrew twisting technique to wrap the balloon tightly and avoid damaging the balloon (Fig. 20.6). Finally, flush the arterial monitoring port, and attach it to the standard arterial line set up with a transducer.

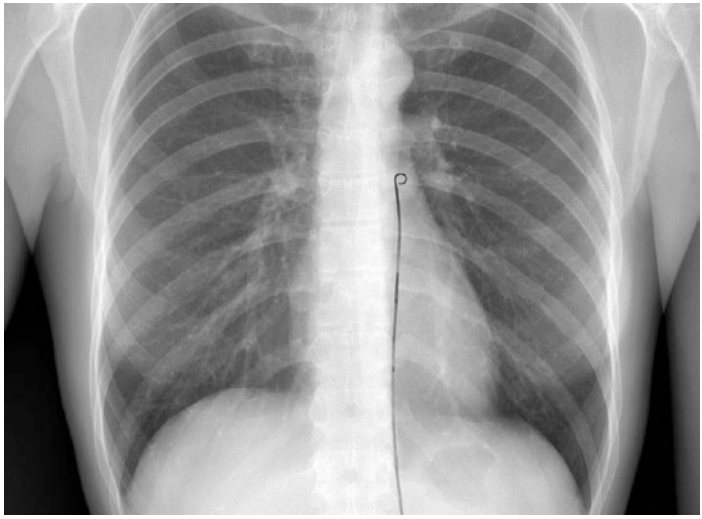

FIGURE 20.7 CXR demonstrating Zone I placement of REBOA. (Used with permission of Prytime Medical Devices, Inc. Boerne, TX, USA. Available at: http://prytimemedical.com/wp-content/uploads/2018/06/ADV-006-Rev-H_ER-REBOA-Catheter-Quick-Reference-Guide-Wall-Poster_8.5x11_EN-2.pdf)

Insert the orange peel-away cover into the 7 Fr sheath to approximately 5 mm to open the valve. There is a characteristic pop when thge catheter engages the sheath. Advanong the catheter through the sheath can take some effort. As the catheter exits the sheath, advancing it become easier. Once the valve is opened, use the blue catheter shaft to advance the catheter to the desired depth. While holding the blue catheter, peel the orange cover away in order to visualize the catheter markings to insert the catheter to the appropriate depth as previously measured.

Once the catheter has been placed, obtain a CXR or pelvic X-ray, depending on the zone selected, if immediately available, to confirm position using the radiopaque markers on each end of the balloon (Fig. 20.7). Once in position, hold the catheter at its insertion site in the femoral sheath to prevent

balloon migration during inflation. Inflate the balloon in a controlled fashion until resistance is met and there is an increase in the arterial waveform from the arterial line above the balloon. In Zone I, start with 8 cc of saline, and re-evaluate for response; in Zone III, start with 2 cc and re-evaluate, ensuring not to over-inflate [23–28]. As one gains experience, there is a feel to knowing the balloon is properly inflated. Once the desired response is achieved, close the stopcock on the balloon port, and secure the catheter and the sheath to the patient to avoid migration during transport to a location where definitive control of bleeding can be obtained. As central aortic pressure returns with resuscitation, it will push the balloon caudal; therefore, even when the catheter and sheath are secured, they need to be continuously observed to assure there is no caudal migration which could result in intimal injury.

REBOA Deflation and Removal

The length of time the patient can tolerate balloon occlusion is not well known. Based on the literature, the current recommended time for aortic occlusion is less than 60 minutes, particularly in Zone I [18, 22]. Be sure to make note of the time the balloon was inflated, and then make every effort to obtain control of the hemorrhage as quickly as possible to minimize the ischemic burden. After hemorrhage control is obtained, an attempt at balloon deflation can be made. Communication between the anesthesia team and the person controlling the balloon at this step is critical for successful deflation because it may result in a significant decrease in afterload and rebound hypotension. The balloon should be deflated slowly over several minutes while observing the patient's response, with the goal of minimizing hypotension and potential reperfusion response. Use the patient's response to further guide resuscitation and balloon deflation. Intermittent balloon inflation and deflation may be necessary until hemodynamic stability is restored. After the REBOA is no longer required, it may be removed. To remove the cath-

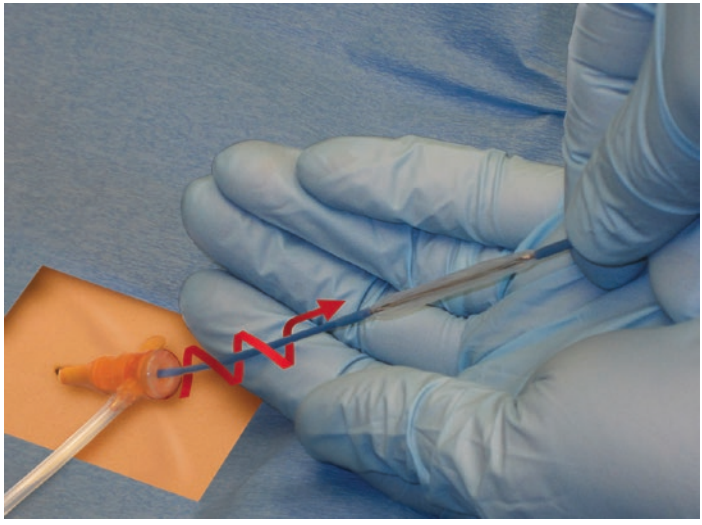

Figure 20.8 Corkscrew twisting technique to remove the catheter and balloon. (Used with permission of Prytime Medical Devices, Inc. Boerne, TX, USA. Available at: http://prytimemedical.com/wp-content/uploads/2018/06/ADV-006-Rev-H_ER-REBOA-Catheter-Quick-Reference-Guide-Wall-Poster_8.5x11_EN-2.pdf)

eter, apply negative pressure to the 30 cc for 5 seconds to remove any remaining saline from the balloon, and close the stopcock on the balloon port. Then using the corkscrew twisting technique (Fig. 20.8), remove the catheter and balloon from the sheath, and flush the 7 Fr sheath with 100 mL of heparinized saline (1000 units of heparin in 1 L of saline).

After the patient has stabilized and the coagulation parameters are corrected, the sheath may be removed. It is highly recommended to obtain an angiogram through the sheath prior to removal to prove there is no thrombus formation and that there is appropriate runoff to the foot. We often flush the sheath with dilute hepatinized saline before removing it. If angiography is not available, consider a detailed Doppler examination prior to sheath removal. If the percutaneous technique was used to gain access for the

7 Fr sheath, simple pressure can be used to achieve closure. Simply remove the sheath from the groin, and apply manual compression for at least 30 minutes. Then keep the patient supine, without any hip or knee flexion for 6 hours. If the open technique was used to gain access to the CFA, the arteriotomy should be closed surgically. This can be accomplished by first exposing the CFA proximal and distal to the sheath entry site to allow for control. This often requires dissection proximally under the inguinal ligament, as well as identification and control of both the superficial femoral artery and profunda femoris for distal control. After obtaining proximal and distal control, the sheath may be removed, and the arteriotomy should be examined and tailored if necessary to allow primary transverse closure. The closure should be performed using 5-0 permanent monofilament suture in either an interrupted or running fashion, ensuring fore and back bleeding prior to completion of the closure. Confirm restoration of blood flow with manual palpation of distal pulses, and then close the soft tissues and skin in layers using absorbable suture. It is recommended to obtain an arterial duplex of the femoral artery between 24 and 72 hours after sheath removal.

Outcomes

The use of REBOA in trauma has been increasing, and studies to investigate outcomes are still ongoing. The technique of REBOA continues to evolve, and the exact indications for REBOA remain uncertain [19]. The American Association for the Surgery of Trauma (AAST) Aortic Occlusion in Resuscitation for Trauma and Acute Care Surgery (AORTA) multi-institutional database was designed to prospectively compare outcomes from open and endovascular aortic occlusion; this as well as other studies has shown that REBOA is a viable alternative to open aortic occlusion [17–21]. Initial data from this database demonstrated no significant difference in overall mortality between patients undergoing resus-

citative thoracotomy and those undergoing REBOA for NCTH [19]. A more recent review of the AORTA study group demonstrates a potential overall survival benefit over resuscitative thoracotomy, particularly in patients not requiring cardiopulmonary resuscitation [19]. Considerable research is still needed to help further define specific indications, compare outcomes, and assess complications.

Complications

Complications of REBOA are significant, and the inherent morbidity and mortality of REBOA is often compounded by coexisting injury and hemorrhagic shock [29]. Most complications are due to complications with vascular access, ischemia, and reperfusion with an overall vascular complication rate as high as 13% [30]. Complications related to groin access occurred in approximately 5% of patients [31], and an overall rate of morbidity reported in the literature was 3.7% [32]. These include accessing the wrong part of the vascular tree, misplacement of the wire, arterial thrombus, distal embolization, vessel injury, and amputation (2%) [31]. Over-inflation of the balloon and consequent balloon rupture or aortic injury can result in significant morbidity and mortality [32]. Another complication that must be avoided is the profound ischemia related to a long-term occlusion, causing irreversible organ damage, significant reperfusion injury, and death.

Translational research suggests that Zone I REBOA is survivable for 60 minutes and Zone III for 90 minutes [33], and preliminary clinical data suggest Zone I occlusion times less than 60 minutes appear to be tolerated in most patients [18]. The attempt to minimize distal ischemia while still maintaining critical organ perfusion above the level of occlusion has led to the development of partial REBOA in which the balloon is deflated slightly, allowing hypotensive resuscitation below the level of balloon [22, 34]. Partial REBOA has been shown to maintain normal physiology better than complete occlusion, reduce hemodynamic instability, and lessen

distal organ ischemia and may offer a way to extend the duration of intervention beyond 60 minutes [1].

When larger sheaths are required for arterial access (12–14 FR), distal thrombus and arterial dissection are common occurrences associated with sheath removal, but, with the evolution of the 7 Fr sheath, the incidence of these complications is expected to decrease [35]. More studies are still needed to fully understand the incidence and consequence of complications associated with REBOA, the exact indications, and the time frame in which REBOA is most effective to improve its role in trauma resuscitation.

References

1. Kauvar D, Lefering R, Wade C. Impact of hemorrhage on trauma outcomes: an overview of epidemiology, clinical presentations, and therapeutic considerations. J Trauma Acute Care Surg. 2006;60(6 Suppl):S3–11.
2. Seamon M, Pathak A, Bradley K, Fisher CA, Gaughan JA, Kulp H, et al. Emergency department thoracotomy: still useful after abdominal exsanguination? J Trauma Acute Care Surg. 2008;64(1):1–7.
3. Hughes CW. Use of an intra-aortic balloon catheter to tamponade for controlling intraabdominal hemorrhage in man. Surgery. 1954;36(1):65–8.
4. Matsuda M, Tanaka Y, Hino Y, Matsukawa R, Ozaki N, Okada K, et al. Transbrachial arterial insertion of aortic occlusion balloon catheter in patients with shock from ruptured abdominal aortic aneurysm. J Vasc Surg. 2003;38(6):1293–6.
5. Malina M, Veith F, Ayyash K, Sonesson B. Balloon occlusion of the aorta during endovascular repair of ruptured abdominal aortic aneurysm. J Endovasc Ther. 2005;12(5):556–9.
6. Yang L, Chong-Qi T, Hai-Bo S, Lan Z, Tian-Fu Y, Hong D, et al. Applying the abdominal aortic balloon occluding combined with blood pressure sensor of dorsal artery of foot to control bleeding during the pelvic and sacrum tumor surgery. J Surg Oncol. 2008;97(7):626–8.
7. Veith FJ, Gupta S, Daly V. Techniques for occluding the supraceliac aorta through the abdomen. Surg Gynecol Obstet. 1980;151(3):426–8.

8. Seibler J, DiPascuale T, Sagi HC. Use of temporary partial intra-iliac balloon occlusion for decreasing blood loss during open reduction and internal fixation of acetabular and pelvic fractures. J Orthop Trauma. 2012;26(12):e54–7.
9. Harma M, Kunt AS, Andac MH, Andac MH, Demir N. Balloon occlusion of the descending aorta in the treatment of severe post-partum hemorrhage. Aust N Z J Obstet Gynaecol. 2004;44(2):170–1.
10. Spence P, Lust R, Chitwood IH, Sun YS, Austin EH 3rd. Transfemoral balloon aortic occlusion during open CPR improves myocardial and cerebral blood flow. J Surg Res. 1990;49(3):217–21.
11. Sesman J, Labandeira J, Sara M, Espila JL, Arteche A, Saez MJ. Effects of intra-aortic occlusion balloon in external thoracic compressions during CPR in pigs. Am J Emerg Med. 2002;20(5):453–62.
12. White J, Cannon J, Stannard A, Markov NP, Spencer JR, Rasmussen TE. Endovascular balloon occlusion of the aorta is superior to resuscitative thoracotomy with aortic clamping in a porcine model of hemorrhagic shock. Surgery. 2011;150(3):400–9.
13. Avaro J, Mardelle V, Roch A, Gil C, de Biasi C, Oliver M, et al. Forty minute endovascular occlusion increases survival in an experimental model of uncontrolled hemorrhagic shock caused by abdominal trauma. J Trauma Acute Care Surg. 2011;71(3):720–5.
14. Gupta BK, Khaneja SC, Flores L, Eastlick L, Longmore W, Shaftan GW. The role of intra-aortic balloon occlusion in penetrating abdominal trauma. J Trauma. 1989;29(6):861–5.
15. Low RB, Longmore W, Rubinstein R, Flores L, Wolvek S. Preliminary report on the use of the Percluder occluding aortic balloon in human beings. Ann Emerg Med. 1986;15(12):1446–69.
16. Moore LJ, Brenner M, Kozar RA, Pasley J, Wade CE, Baraniuk MS, et al. Implementation of resuscitative endovascular balloon occlusion of the aorta as an alternative to resuscitative thoracotomy for noncompressible truncal hemorrhage. J Trauma Acute Care Surg. 2015;79(4):523–30.
17. Brenner M, Teeter W, Hoehn M, Pasley J, Hu P, Yang S, et al. Use of resuscitative endovascular balloon occlusion of the aorta for proximal aortic control in patients with severe hemorrhage and arrest. JAMA Surg. 2018;153(2):130–5.
18. Brenner M, Inaba K, Aiolfi A, DuBose J, Fabian T, Bee T, et al. Resuscitative endovascular balloon occlusion of the aorta and resuscitative thoracotomy in select patients with hemorrhagic

shock: early results from the American association for the surgery of trauma's aortic occlusion in resuscitation for trauma and acute care surgery registry. J Am Coll Surg. 2018;226(5):730–40.
19. DuBose J, Scalea T, Brenner M, Skiada D, Inaba K, Cannon J, et al. The AAST prospective aortic occlusion for resuscitation in trauma and acute care surgery (AORTA) registry: data on contemporary utilization and outcomes of aortic occlusion and resuscitative balloon occlusion of the aorta (REBOA). J Trauma Acute Care Surg. 2016;81(3):409–19.
20. Manzano Nunez R, Naranjo M, Foianini E, Ferrada P, Rincon E, García-Perdomo HA, et al. A meta-analysis of resuscitative endovascular balloon occlusion of the aorta (REBOA) or open aortic cross-clamping by resuscitative thoracotomy in non-compressible torso hemorrhage patients. World J Emerg Surg. 2017;12:30.
21. Brenner M, Moore L, DuBose J, Tyson GH, McNutt MK, Albarado RP, et al. A clinical series of resuscitative endovascular balloon occlusion of the aorta for hemorrhage control and resuscitation. J Trauma Acute Care Surg. 2013;75(3):506–11.
22. DuBose J. How I do it: partial resuscitative endovascular balloon occlusion of the aorta (P-REBOA). J Trauma Acute Surg. 2017;83(1):197–9.
23. Pezy P, Flaris A, Prat N, Cotton F, Lundberg PW, Caillot JL, et al. Fixed-distance model for balloon placement during fluoroscopy-free resuscitative endovascular balloon occlusion of the aorta in a civilian population. JAMA Surg. 2017;70(3):652–63.
24. Martinelli T, Thony F, Declety P, Sengel C, Broux C, Tonetti J, et al. Intra-aortic balloon occlusion to salvage patients with life-threatening hemorrhagic shock from pelvic fractures. J Trauma Acute Care Surg. 2010;68(4):942–8.
25. Stannard A, Eliason J, Rasmussen T. Resuscitative endovascular balloon occlusion of the aorta (REBOA) as an adjunct for hemorrhagic shock. J Trauma Acute Care Surg. 2011;71(6):1869–72.
26. Linnebur M, Inaba K, Haltmeier T, Rasmussen TE, Smith J, Mendelsberg R, et al. Emergent non-image-guided resuscitative endovascular balloon occlusion of the aorta (REBOA) catheter placement: a cadaver-based study. J Trauma Acute Care Surg. 2016;81(3):453–7.
27. Morrison J, Stannard A, Mildwinter M, Sharon DJ, Eliason JL, Rasmussen TE, et al. Prospective evaluation of the correlation between torso height and aortic anatomy in respect of a fluoroscopy free aortic balloon occlusion system. Surgery. 2014;155(6):1044–51.

28. MacTaggart J, Poulson W, Akhter M, Seas A, Thorson K, Phillips NY, et al. Morphometric roadmaps to improve accurate device deliver y for fluoroscopy-free resuscitative endovascular aortic balloon occlusion of the aorta. J Trauma Acute Care Surg. 2016;80(6):941–6.
29. Davidson A, Russo R, Reva V, Brenner ML, Moore LJ, Ball C, et al. The pitfalls of resuscitative endovascular balloon occlusion of the aorta: risk factors and mitigation strategies. J Trauma Acute Care Surg. 2018;84(1):192–202.
30. Taylor J, Harvin J, Martin C, Holcomb JB, Moore LJ. Vascular complications from resuscitative endovascular balloon occlusion of the aorta: life over limb? J Trauma Acute Care Surg. 2017;83(1 Suppl 1):S120–3.
31. Manzano Nunez R, Orlas C, Herrerra Escobar J, Galvagno S, DuBose J, Melendez JJ, et al. A meta-analysis of the incidence of complications associated with groin access after the use of resuscitative endovascular balloon occlusion of the aorta in trauma patients. J Trauma Acute Care Surg. 2018;85(3):626–34.
32. Morrison J, Galgon R, Jansen J, Cannon JW, Rasmussen TE, Eliason JL. A systematic review of the use of resuscitative endovascular balloon occlusion of the aorta in the management of hemorrhagic shock. J Trauma Acute Care Surg. 2016;80(2):324–34.
33. Ribeiro M, Feng C, Nguyen A, Rodrigues VC, Bechara GEK, de-Moura RR, et al. The complications associated with Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA). World J Emerg Surg. 2018;13:20.
34. Johnson A, Neff L, Williams K, JJ DB, EVAC Study Group. Partial resuscitative balloon occlusion of the aorta (P-REBOA): clinical technique and rationale. J Trauma Acute Care Surg. 2016;81(5 Suppl):S133–7.
35. Russo R, Neff L, Lamb C, Cannon JW, Galante JM, Clement NF, et al. Partial resuscitative endovascular balloon occlusion of the aorta in swine model of hemorrhagic shock. J Am Coll Surg. 2016;223(2):359–68.

Part VI
Techniques for Bony and Soft Tissue Injury

Chapter 21
Extremity Fasciotomies

Sharon M. Henry and Habeeba Park

Abbreviations

6 Ps	Pain, pallor, paresthesias, paralysis, poikilothermia, pulselessness
ACS	Acute compartment syndrome
APL	Abductor pollicis longus
BR	Brachioradialis
CK	Creatine kinase
DPC	Delayed primary closure
ECRB	Extensor carpi radialis brevis
ECRL	Extensor carpi radialis longus

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ECU	Extensor carpi ulnaris
ED	Extensor digiti
EDC	Extensor digitorum communis
EPB	Extensor pollicis brevis
EPL	Extensor pollicis longus
FCS	Foot compartment syndrome
FCU	Flexor carpi ulnaris
FDP	Flexor digitorum profundus
FDS	Flexor digitorum superficialis
Hg	Mercury
ICP	Intracompartmental pressure
ISS	Injury severity score
IV	Intravenous
IVC	Inferior vena cava
L	Liter
LDH	Lactate dehydrogenase
MC	Metacarpal
Mm	Millimeters
MT	Metatarsal
N	Nerve
NPWT	Negative pressure wound therapy
U	Units

Introduction of the Problem

Richard von Volkmann in 1881 described the sequelae of applying overly restrictive dressings or casts to an injured limb–Volkmann's contracture. In 1906 Hildebrand related Volkmann's ischemic contracture to elevated tissue pressure. Murphy described the technique of fasciotomy for the treatment of elevated intracompartmental pressure (ICP) in 1910 [1]. Elevated ICP impairs the microcirculation of the involved compartment. The elevated pressure produces ischemia that affects muscles and nerves leading to muscle and nerve dysfunction [2].

The most common indication for fasciotomy is for the diagnosis of acute compartment syndrome (ACS). ACS is

caused by raised ICP within a closed osteo-fascial or myofascial space. The overall incidence of compartment syndrome is estimated to be 0.7 per 100,000 in women and 7.3 per 100,000 in men [3]. The true incidence is not known because of variations in clinical presentation. Vigilance is therefore necessary to avoid the consequences of delayed treatment. Trauma is the most common etiology for extremity compartment syndrome. A 1.3% prevalence was found among trauma admissions at a level 1 trauma center [4]. Familiarity with the trauma-related etiologies helps to identify the patient populations at risk. Extremity ACS following trauma occurs most commonly after high-energy injury to the limbs. Fractures account for up to 75% of cases of ACS [2]. It is important to keep in mind that ACS can follow both open and closed fractures. It should not be assumed that decompression of the compartment has occurred because of the small fascial tears that accompany open fractures. It is estimated that 5.9% of open and 2.2% of closed tibial fractures result in ACS [5]. About 30% of extremity ACS occurs following soft tissue injury without associated fracture. Patients younger than 35 years are at higher risk of developing ACS and males at higher risk than females, perhaps because of increased muscle bulk. Older patients are at lower risk and patients with diastolic hypertension are at lower risk [6]. The site of fasciotomy varies as well. Sixty-eight percent involve the leg, 14% the forearm, and 9% the thigh. Gluteal, foot, and hand ACS occur less frequently [5].

Acute Compartment Syndrome

In addition to fracture and soft tissue injury, ACS may occur following compression with tight bandages or casts, vascular injury especially combined venous and arterial, reperfusion after prolonged ischemia, crush injuries, gunshot wounds, circumferential or electrical burns, extensive venous thrombosis, and prolonged immobilization (as can occur with alcohol or drug intoxication, or prolonged operative cases). Less common

causes include envenomation and hematomas resulting from trauma, and/or coagulopathy, either congenital or acquired. Fasciotomy is sometimes performed in patients at high risk for the development of ACS before overt signs develop. Candidates for this approach have had vascular injury with shunting or repair after prolonged ischemia, have combined venous and arterial vascular injury, or have undergone venous ligation. Combined venous and arterial injury increases the incidence of compartment syndrome by 41.8% [5]. ACS can also be a sequela of soft tissue infection. Fasciotomy along with drainage and debridement is necessary to treat the soft tissue infection. Examples include pyomyositis and necrotizing fasciitis. Box 21.1 lists indications for fasciotomy.

Box 21.1: Indications for Fasciotomy

- Acute compartment syndrome
  - High-energy blunt trauma to extremity with or without fracture
  - Penetrating wound to the extremity
  - Blast injury
  - Reperfusion following ischemia
  - Crush injury
  - Prolonged immobilization with intoxication or drug overdose
  - Prolonged operation
  - Prolonged lithotomy or Trendelenburg positioning
  - Envenomation
  - Infection (necrotizing soft tissue infections, severe pyomyositis, or cellulitis)
  - Circumferential burns or electrical burns
  - Vascular injury (especially combined arterial and venous)
  - Deep venous thrombosis
  - IV infiltration with extravasation of fluids or medicines

  - Vigorous crystalloid resuscitation
  - Coagulopathy
  - Tight casts or bandages
  - Strenuous exercise
  - High-pressure injections
- Prophylaxis against development of compartment syndrome
- Prolonged ischemia
- Exploration for infection

Fasciotomy is performed to decrease the elevated ICP restoring perfusion. Unrelieved elevation of the pressure within a compartment above a threshold for a prolonged period can result in painful tissue ischemia, venous congestion, and neuropraxia. Irreversible ischemic damage to multiple compartments may make amputation necessary. The best strategy available to avoid delayed treatment of ACS is to recognize the patient at risk, closely monitor them for evolution of their exam, and to initiate surgical treatment when recognized. Box 21.2 lists factors that increase risk of ACS among trauma patients.

Box 21.2: Factors that Increase Risk of ACS in Trauma Patients

- Demographics
  - Young age < 35 years [17]
  - Male sex
  - Injury patterns
  - Gunshot wounds
  - Blast injuries
  - Crush injuries
  - Combined arterial and venous injuries

  - Major vascular injury below the aortic bifurcation
  - Tibial fractures
  - Open fractures
  - Joint dislocations
- Systemic factors
  - Need for massive transfusion
  - Large-volume crystalloid resuscitation
  - Admission hypotension
  - High injury severity score (ISS)

ACS Pathophysiology

Perfusion of a tissue compartment is related to the arteriolar and capillary perfusion gradients. Normal ICP is 0–8 mm Hg [7]. When pressures exceed capillary filling pressure, nutrient tissue perfusion is prevented resulting in tissue ischemia. At the same time, venous and lymphatic outflow are also compromised, producing further pressure buildup within the compartment. Blood flow at the arteriolar level is compromised by the higher compartmental pressure, and further ischemia results. Perfusion pressure is related to blood pressure. In experimental models, muscle ischemia results when intracompartmental pressure is within 10 mm Hg of the diastolic blood pressure (BP). The ischemia triggers the release of vasoactive chemicals and cytokines. The endothelium becomes permeable causing more tissue edema compounding the ICP increase. Muscle cells release myoglobin causing rhabdomyolysis. Rhabdomyolysis may accompany ACS in up to 23% of cases [3]. High circulating levels of myoglobin cause renal tubular obstruction and acute kidney injury. Ultimately, cell death occurs, and substances are released that amplify the production of edema within the compartment. Figure 21.1 illustrates the cycle that without timely interruption can result in tissue loss. Sustained ICP elevation

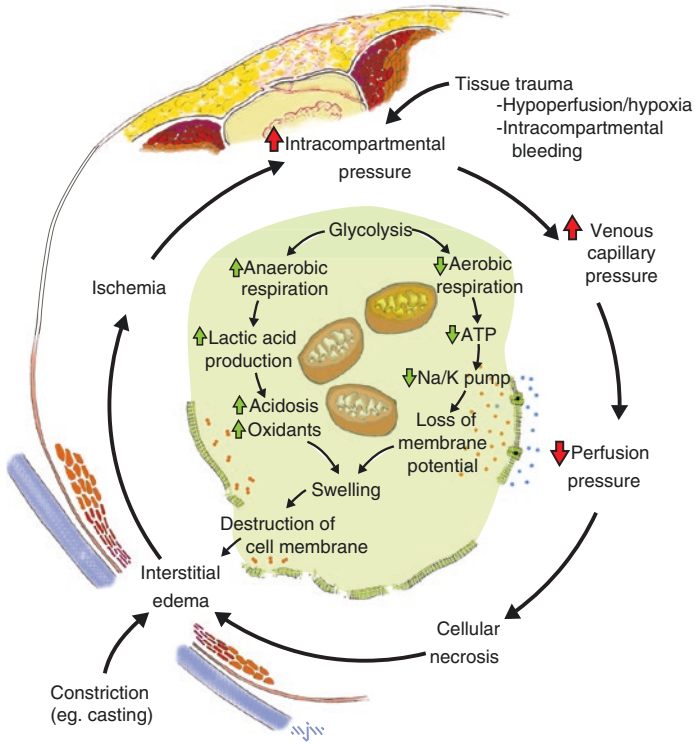

FIGURE 21.1 ACS pathophysiology. Constriction, tissue trauma, edema, or bleeding causes increased pressure within a myo- or osteo-fascial compartment. As the intracompartmental pressure increases, the venous capillary pressure rises leading to hypoperfusion (red arrows). Intracellular metabolism converts from aerobic to anaerobic. The cell becomes acidotic and oxidants accumulate. The cell swells as the Na/K pump fails. The cell membrane is destroyed and intracellular contents enter the interstitium resulting in more edema. When perfusion is decreased below critical thresholds for extended time, cell death and necrosis result

above 30 mm Hg for 4–8 hours leads to irreversible tissue injury. Complete recovery of function occurs in nearly all patients undergoing fasciotomy within 6 hours, but only in 68% of patients when within 12 hours, and only 8% recover after 12 hours [8].

ACS Diagnosis

The diagnosis is heavily dependent on clinical evaluation. Swelling of the extremity will be present. Though swelling following injury is common, the swelling associated with compartment syndrome is tense, and the skin may be shiny and taut. Pain is the earliest and most sensitive–though least specific–indication of compartment syndrome. Trauma patients are expected to have pain, but the pain associated with compartment syndrome is often described as a change in intensity or to exceed that which would be expected for the injury. In addition, pain with passive stretch of the muscles within the compartment will produce excruciating pain. The six clinical signs of ischemia (6 Ps) are often associated with the diagnosis of compartment syndrome (Box 21.3). Since compartment syndrome results from changes in arteriolar and capillary perfusion gradients, ACS occurs even with palpable extremity pulses and without pallor or capillary refill delays. Muscle paralysis is also a late indication of compartment syndrome. Moreover, muscle strength is often difficult to elicit in an injured limb. However, a change in clinical examination would be an important clue. If fasciotomy is performed once all 6 Ps are present, functional recovery is unlikely. Patients must be alert and awake in order to elicit the most useful clinical findings. When the prognostic value of pain, pain on passive stretch, paresthesia, and paralysis (4 Ps) are compared, they individually have a prognostic value between 13 and 19%. When pain and pain on passive stretch are both present, the prognostic value rises to 68%. The addition of paresthesias and paralysis increases the probability to 93% and 98%, respectively. However, it has been reported that when foot drop is present at the time of diagnosis, only 13% of patients recover function following fasciotomy [9]. Though major sensory and motor deficits are late signs of ACS, diminished two-point discrimination and loss of vibratory sense are considered early markers of ACS [8].

Box 21.3: Signs of Vascular Ischemia
- Pain
- Paresthesias
- Pallor
- Poikilothermia
- Paralysis
- Pulselessness

The diagnosis can be difficult to make even in patients that are awake and alert. Patients receiving regional block and continuous analgesic drips and with altered mental status lack even the usual nonspecific physical findings. Repeated assessments continue to be essential to trigger the timely and accurate diagnosis of ACS. A warm extremity with palpable peripheral pulses is reassuring but does not exclude the diagnosis of ACS, particularly when worsening pain and paresthesias are also present.

Objective measures are added when the diagnosis is unclear. Unfortunately, there is no pathopneumonic diagnostic laboratory test. Myoglobin is released as cell death occurs, so elevation of serum myoglobin is associated with ACS. Myoglobin has a short half-life. Creatine kinase (CK), aldolase, and lactate dehydrogenase (LDH) are also released. No specific threshold is recognized especially in trauma patients who may have sustained direct muscle injury. CK and myoglobin levels should be serially measured though. CK levels >2000 U/L may be associated with compartment syndrome in the absence of fracture and >4000 when fracture is present [10]. A rising level is concerning. CK levels remain elevated for 1–3 days [11]. Urine myoglobin is also frequently measured especially in cases of crush or prolonged immobilization. Acute kidney injury (AKI) requiring dialysis results from precipitation of myoglobin in the renal tubules. Hydration, alkalinization of the urine, and early continuous renal replacement therapy are strategies to minimize renal damage.

ICP measurements supplement the physical examination. Measurements can be repeated to identify progression. This test is particularly useful in patients who are comatose, paralyzed, anesthetized, or under the effect of nerve blocks. Whiteside described a technique to measure ICP. His technique utilized a pressure manometer, syringe, two stopcocks, and a needle. Using his technique, saline is aspirated into an extension that is connected to a stopcock through a needle, taking care not to aspirate air. Another extension tube connects to a stopcock and to a manometer and the saline-filled extension tube. A syringe with 15 cc of air is connected to the unused stopcock port. The needle is advanced into the compartment, and the plunger of the air-filled syringe is depressed. When the saline meniscus changes from convex to flat, the manometer reading indicates the compartmental pressure [6]. Figure 21.2 illustrates the setup. Commercial devices are also available that utilize a similar principle. The most commonly used is made by Stryker (Kalamazoo, MI, USA) and is shown in Fig. 21.3a,

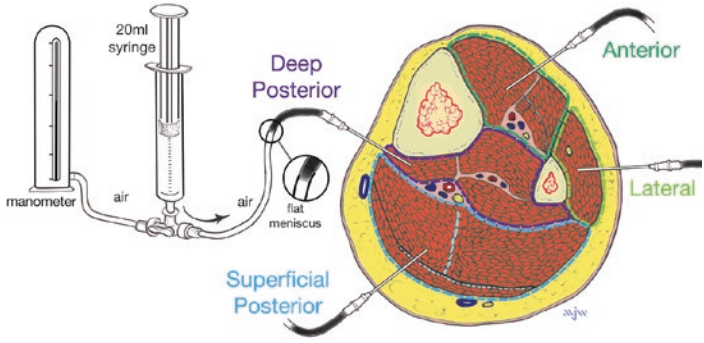

FIGURE 21.2 Whitesides' compartment pressure setup. Saline is introduced into intravenous tubing connected to a needle. A stopcock with a 20 ml syringe filled with air and a manometer connect to the tubing. The needle is introduced into the compartment. The plunger on the air-filled syringe is depressed. When the saline meniscus changes from convex to flat, the manometer reading will indicate the compartment pressure. The needles indicate the access points to anterior, lateral, superficial, and deep posterior compartments of the leg

FIGURE. 21.3 (**a**, **b**) Stryker device. Needle, monitor, syringe, and diaphragm. Needle monitor and syringe separate (**a**) and assembled (**b**)

b. Arterial pressure monitoring devices can be adapted to measure compartment pressures using appropriate needles or catheters, stopcocks, and extension tubing. Attach an 18 gauge needle to arterial line tubing. Flush and zero the setup. Advance the needle into the desired compartment, and note the mean arterial pressure (MAP). The MAP represents the ICP. Side port and slit catheters improve the accuracy over straight needles [12]. Several principles are important to remember. Pressures are higher within 5 cm of a fracture site and at the center of a compartment. Compartment pressures are not necessarily uniform throughout an extremity [2]. No precise best location to measure ICP has been determined. Multiple sites should be tested [13]. Serial measurements may be required to make the diagnosis. Continuous measurements are advocated by some to assure prompt diagnosis. An absolute measurement of 30–45 mm Hg is frequently used to diagnose compartment syndrome in the appropriate clinical setting. Some recognize the relationship between systemic blood pressure and ICP and prefer to use a calculated delta p. This is derived from subtracting the measured pressure from either the MAP or diastolic BP. Differences of less than 30 mm Hg indicate ACS [14]. Useful measurements are dependent on correct placement within the affected compartments.

ACS Treatment

General

Remove any constricting bandages or casts. Do not elevate the limb. Maintain it at the level of the heart except in cases of phlegmasia. Resuscitate to assure euvolemia. ACS is a surgical emergency. Operation should commence rapidly after diagnosis. Hyperbaric oxygen therapy may be a useful adjunct to fasciotomy to treat ACS and tissue ischemia.

Fasciotomy

Technique

General Principles

Several principles apply generally to fasciotomy regardless of the site involved. They include the following:

- Make longitudinal incision over the entirety of the investing fascia of each compartment. The skin overlying the affected compartment should be completely opened.
- Release the compartment contents.
- Protect critical neurovascular structures.
- Carefully evaluate the muscle for viability, remove frankly necrotic tissue, and reevaluate ischemic tissue with potential for reversibility.
- Delayed closure.

Table 21.1 outlines the compartments, contents along with clinical symptoms observed, and outcome of delayed treatment.

Table 21.2 outlines the compartments and incisions used to approach them.

Buttock

Trauma is not a common indication for buttock fasciotomy. Prolonged immobilization following alcohol or drug intoxication, intramuscular injection, or prolonged operative procedures are indications that are more common. Case reports have described the condition following trauma associated with gluteal artery bleeding from aneurysmal rupture or injury and high-energy direct trauma to the buttock. Trauma-associated etiologies include contusion, gluteal artery injury, hip dislocation, acetabular fracture, pelvic fracture, and following vascular procedures. Infection and spontaneous hemorrhage are other potential etiologies [15]. Patients

Table 21.1 Compartment, contents, CS clinical findings, and CS sequelae

Site	Compartment and contents					Clinical findings	Sequelae untreated CS
Foot	Medial	Lateral	Central (superficial and deep)	Interosseous [4]	Adductor	Tense swelling, shiny taut skin, blisters, pain out of proportion to injury, pain on passive dorsiflexion of the toes, decreased two-point discrimination, and light touch on plantar foot and toes	Pain, ischemic contracture (claw toe and hammer toe), insensate foot, neuropathic ulcers
	Flexor hallucis brevis and abductor hallucis superficial veins and nerves	Abductor digiti minimi, flexor digiti minimi brevis Superficial veins and nerves	Flexor digitorum brevis, flexor digitorum longus, quadratus plantae, and plantar aponeurosis Posterior tibial nerves and artery, medial and lateral plantar nerve artery and vein	Doral and plantar interossei Superficial veins and nerves	Adductor hallucis		

Lower leg	Anterior	Lateral	Superficial posterior	Deep posterior	Pain on passive stretch ankle plantar or dorsiflexion, muscle weakness, tense compartment, hypoesthesia	Rhabdomyolysis, muscle loss, amputation, foot drop, chronic pain, numbness
	Tibialis anterior extensor digitorum longus, extensor halluces longus, peroneus tertius, anterior tibial artery	Peroneus longus, peroneus brevis, common peroneal nerve, and superficial peroneal nerve	Gastrocnemius, soleus, plantaris, superficial veins, sural nerve	Popliteus, flexor halluces longus, flexor digitorum longus, tibia and posterior tibial artery, tibial n and deep veins		
Thigh	Anterior		Posterior	Medial	Tense edema, pain and paresthesias, pain with passive range of movement of the knee	Rhabdomyolysis, myonecrosis, chronic pain
	Vastus medialis, sartorius, rectus femoris, vastus intermedius, vastus lateralis, femoral artery, vein and nerve		Semimembranosus, semitendinosus, biceps femoris, sciatic nerve	Gracilis, adductor magnus, deep femoral artery and vein		
Buttock	Gluteus maximus	Gluteus medius and minimus		Tensor fascia lata	Tense swelling, buttock pain, sciatica	Rhabdomyolysis, myonecrosis, chronic pain

(continued)

Table 21.1 (continued)

Site	Compartment and contents						Clinical findings	Sequelae untreated CS
Arm	Anterior	Posterior			Deltoid		Tense swelling, intense pain	Myonecrosis, chronic pain, amputation
	Biceps brachii, brachialis and coracobrachialis, brachial artery, median and ulnar nerves	Triceps brachii and anconeus muscles and radial nerve			Deltoid muscle			
Forearm	Volar superficial	Volar deep		Mobile wad	Dorsal		Tightness and swelling, pain with movement especially passive stretch, decreased 2-point discrimination, blisters	Loss of function, ischemic contracture, neurologic deficit, amputation
	FDS, FCR, pollicis longus, pronator teres, ulnar nerve	FCU, FDP, FPL, common interosseous artery, ulnar artery, median and ulnar nerves		Brachioradialis, ECR longus and brevis, radial artery, and superficial radial nerve	Supinator, ECU, EPL, EDC			
Hand	Carpal tunnel	Dorsal interosseous 4 dorsal interosseous muscles	Plantar interosseous 3 palmar interosseous muscles	Adductor Adductor pollicis	Thenar Thenar muscles	Hypothenar Hypothenar muscles	Pain out of proportion to injury and pain on passive stretch Tense swelling with extension of the metacarpophalangeal joints and flexion the intercarpal joints	Loss of function, amputation neurologic injury

Data from References [1, 4, 27, 40]

Table 21.2 Compartments and incisions

Region	Compartments	Incision
Buttock	3 Tensor Gluteus maximus Gluteus medius and minimus	From just below the iliac crest lateral to the posterior superior iliac spine to the greater trochanter and below
Lower extremity		
Thigh	3 Anterior Posterior Medial	Anterior and posterior through incision from the intertrochanteric line extending to the lateral condyle. Retract the vastus lateralis, and divide the intermuscular septum to open the posterior compartment When needed add a medial incision to decompress the adductor muscles
Leg	4 Anterior Lateral Superficial posterior Deep posterior	Anterior and lateral compartment through incision 2 cm anterior to the fibula. An H shaped incision across the muscular septum connects the compartments. Superficial and deep posterior 2 cm medial posterior to the tibia release gastrocnemius and soleus to open superficial posterior compartment. Separate the soleus from the tibia to enter the deep posterior compartment

(continued)

Table 21.2 (continued)

Region	Compartments	Incision
Foot	9 Medial Lateral Superficial central Deep central (calcaneal) Interosseous [4] Adductor	Dorsal incisions to approach the interossei and adductor compartments medial to the 2nd metatarsal and lateral to the 4th metatarsal dissection of the muscle medially from the 2nd MT expose the adductor fascia Medial plantar incision Beginning 4 cm from the posterior aspect of the heel and 3 cm superior to the plantar surface paralleling the plantar surface for 6 cm to access the superficial and deep compartments
Upper extremity		
Upper arm	3 Anterior Posterior Deltoid	Single incision lateral arm extends proximally over the deltoid if that compartment is involved Medial incision when access to brachial artery is needed
Forearm	3 Volar Mobile wad Dorsal	Above the elbow medial to biceps tendon angle across the antecubital fossae to the mobile wad radially continuing distally across the wrist crease at an angle on the ulnar side of palmaris longus on to the palm
Hand	11 Dorsal [4] Volar [3] interosseous Adductor Thenar Hypothenar Carpal tunnel	Dorsal incisions over the 2nd and 4th metacarpals decompress the dorsal, volar, and adductor compartments Incisions over the thenar and hypothenar areas at the junction of the palmar with the dorsal skin

Data from References [1, 8, 18, 19, 30]

report buttock pain and will avoid putting pressure on the affected side. The hip is often held flexed and extension is avoided. Knee extension will elicit severe discomfort. Tense swelling is apparent on physical examination. Buttock numbness and sciatic nerve compression symptoms are often present. The large muscle burden of the area combined with the usual delay in presentation make rhabdomyolysis likely. Renal function, electrolytes, CK, and myoglobin levels should be followed. Should compartment pressure measurement be necessary, place the needle to minimize the risk of neurovascular injury. To measure pressure in the gluteus maximus, identify the proximal inner quadrant of the buttock 2 cm inferior and lateral to the posterior superior iliac spine (PSIS). To measure pressure in the gluteus medius and minimus, insert the needle 2 cm below the iliac crest over the middle third of the iliac wing. The tensor compartment is accessed 2 cm anterior to and 3 cm distal to the greater trochanter [16]. The pressure associated with ACS of the buttock is not precisely known, and the numbers utilized for the lower and upper extremity are adopted for this region. Normal pressures are thought to range from 13 to 14 mm Hg [17].

Three compartments are recognized in the buttock. These include the tensor, the gluteus medius and minimus muscle, and the gluteus maximus muscle. The sciatic nerve does not lie within the gluteal compartments, but it is frequently compressed by swelling of the gluteus maximus above it [17].

To perform a buttock fasciotomy, position the patient in the lateral decubitus position with the affected side up. The hip is flexed. Prone positioning is also possible, particularly if there is bilateral involvement. Mark an incision that extends from just below the iliac crest lateral to the posterior superior iliac spine to the greater trochanter and below (posterior or Kocher-Langenbeck incision). The incision can be extended down the femur should there be a need to decompress the thigh. Incise the skin and deepen the incision to the gluteal fascia. Separate the superolateral edge of the gluteus maximus from the iliotibial tract [18]. The fascia is incised and the

muscle split to assure decompression. Avoid injury to the gluteal artery. Split the gluteus maximus muscle in the direction of its fibers. The gluteus medius is exposed with superior retraction of the gluteus maximus, and the fascia is incised. The gluteus minimus is located deep to the medius. The muscles are inspected and evaluated for consistency, contractility, color, and perfusion. Any hematoma identified is evacuated, and frankly dead tissue is removed. Non-contractile muscle that is perfused is preserved as function may return after decompression. If arterial injury caused the compartment syndrome, ligation or embolization to control further bleeding will be necessary [16].

Leave the wound open. It can be dressed with moist gauze dressings. Negative pressure wound therapy (NPWT) is another option to manage the wound. Reassess the tissues in 24–48 hours.

Lower Extremity

Thigh

Due to the large volume spaces in the compartments, thigh compartment syndrome is not a common finding. However, patients at risk for developing it include those with femur/pelvic fractures and ligation of major venous structures such as the inferior vena cava (IVC) and those who have suffered blast injuries.

There are three compartments in the thigh (Fig. 21.4). These include the anterior comprising the quadriceps muscles, the posterior comprising the hamstring muscles, and the medial comprising the adductor muscles. In most cases, effective decompression of the anterior and posterior compartments is performed through a single laterally placed incision. To open the quadriceps compartment, an anterolateral longitudinal skin incision is created along the iliotibial tract, extending from the lateral condyle to the intertrochanteric space. A long skin incision along the

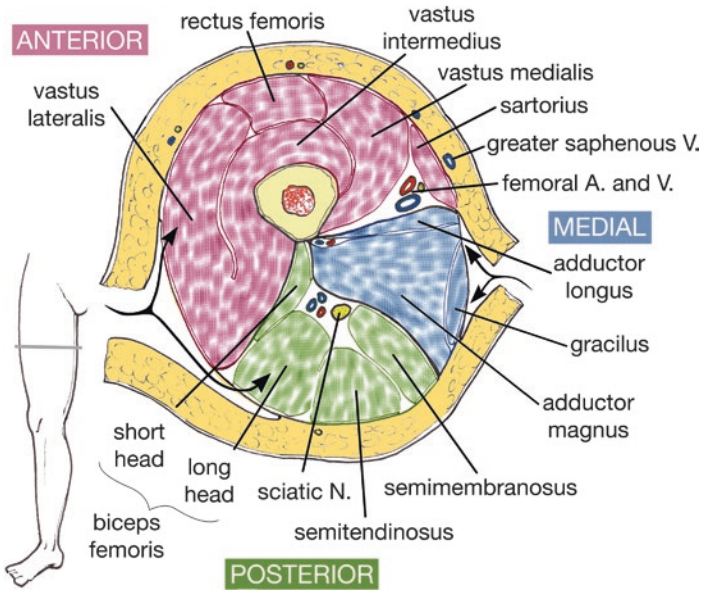

FIGURE 21.4 Thigh compartments. The anterior (shaded in purple) including the rectus femoris, vastus lateralis, vastus intermedius, vastus medialis, and sartorius muscles. The posterior (shaded in green) including the biceps femoris and semimembranosus and semitendinosus. These compartments are decompressed through a lateral incision. The medial compartment includes the adductor longus, magnus, and gracilis and are decompressed through a separate medial incision when necessary

lateral thigh is used and extended to the fascia lata. Incision of the fascia lata decompresses the anterior thigh. The fascia over the vastus lateralis is then opened to decompress the compartment. The vastus lateralis can be retracted anteriorly to enter the posterior compartment dividing the posterior intermuscular septum [16]. The medial compartment is rarely subject to compartment syndrome, and decompression is generally not specifically performed. However, if needed, a separate medial incision should be included.

Leg

The leg has four compartments. They include the following:

- Anterior compartment contains the anterior tibial, extensor digitorum longus, extensor hallucis longus, and peroneus tertius muscles and the anterior tibial artery and vein.
- The lateral compartment contains the peroneus longus and brevis muscles and the superficial peroneal nerve.
- The superficial posterior compartment contains the gastrocnemius and the soleus muscles and the posterior saphenous vein and the sural nerve.
- The deep posterior compartment contains the tibialis posterior, the flexor hallucis longus and the flexor digitorum longus. The posterior tibial artery, vein, and nerve are also in this compartment.

The most common surgical approach to lower extremity fasciotomy is through two incisions to decompress the four compartments. Position the patient supine. A triangle placed behind the knee supporting the leg is useful to improve visualization. Important landmarks to identify include the patella, the tibial spine medial and lateral malleolus, the medial border of the tibia, and the head of the fibula. The anterolateral incision, which will decompress the anterior and lateral compartments, is made in a longitudinal line 1 fingerbreadth or about 2 cm anterior to the edge of the fibula. This may not be easily palpable and may be identified by drawing a line from the fibular head to the lateral malleolus, marking the course of the fibula. When the fascia is identified, try to find the intermuscular septum. Perforating vessels mark the location. Palpate the tibial spine; the anterior compartment will be directly adjacent to it. A horizontal incision is made across the septum. Then insert scissors with closed tips facing away from the septum. This avoids injury to the peroneal nerve. Insert the scissors through the other limb of the "H" to complete the decompression. Be particularly cautious in opening the fascia over the distal third of the lateral compartment. The superficial peroneal nerve exits the fascia and continues subcutaneously. Injury to the superficial peroneal nerve can

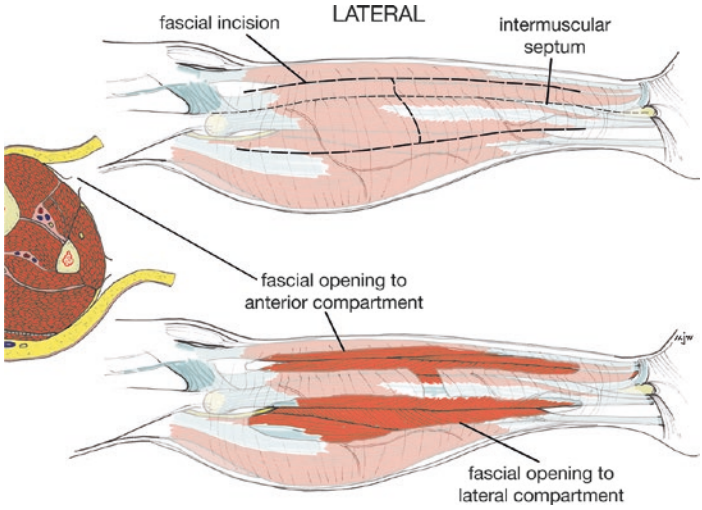

FIGURE 21.5 Decompression of the anterior and lateral compartments. The intermuscular septum separates the anterior and lateral compartments. An H-shaped incision is made to assure decompression of both compartments. The superficial peroneal nerve runs deep to the intermuscular septum and should be avoided when incising the fascia

lead to foot drop [19]. Figure 21.5 illustrates the lateral incision and decompression of the anterior and lateral compartments.

The medial longitudinal skin incision is made 2 fingerbreadths below the tibial plateau, to 2 cm proximal to the medial malleolus, and the tibial spine is a midpoint reference between the lateral and medial skin incisions. There should be 7 cm between the medial and lateral incisions anteriorly [18]. This avoids skin compromise that could expose the tibia. Try to preserve the saphenous vein and accompanying nerve when opening the skin and subcutaneous tissues [20]. Perforating veins should be ligated, as they can cause profuse bleeding. The fascia over the superficial posterior compartment is opened 2 cm posterior to the edge of the tibia for the length of the skin incision. To open the deep compartment, detach the soleus from the tibia. Visualization of the

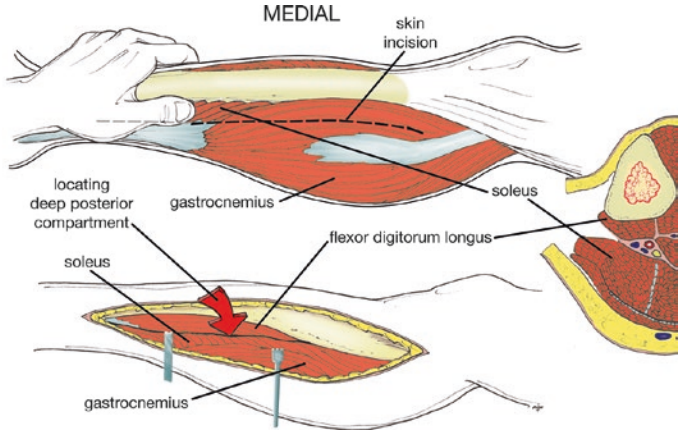

FIGURE 21.6 Medial incision lower leg fasciotomy. The fascia overlying the gastrocnemius and soleus is incised to release the superficial compartment. The soleus fibers are separated from the tibia to access the deep compartment. Visualizing the posterior tibial neurovascular bundle assures the deep compartment has been decompressed

posterior tibial neurovascular bundle assures the deep posterior compartment has been reached [21]. Figure 21.6 illustrates the medial fasciotomy incision to decompress the superficial and deep compartments. The plane between the soleus and gastrocnemius is sometimes mistaken for deep compartment. There is a high risk of compartmental hypertension in this area, which can affect the viability and function of the foot significantly as it contains major structures: the tibial nerve and the posterior tibial and peroneal arteries [22]. The use of generous incisions is important, as the skin envelope can be a limiting factor [23].

Foot

Foot compartment syndrome (FCS) is uncommon and accounts for <5% of limb compartment syndromes, and isolated foot injuries result in compartment syndrome only 2%

of the time. The most common cause of FCS is high-energy trauma producing fracture of the calcaneus. FCS occurs in 10% of these cases [24].

The foot contains nine compartments. Three run longitudinally the length of the foot: the medial, lateral, and superficial. The four interosseous and adductor compartments are within the forefoot, and the deep or calcaneal compartment is in the hindfoot and communicates with the deep posterior compartment of the leg. This is important as blood or infection can readily spread through these compartments. Because of this communication, tibial fracture can produce foot compartment syndrome [1]. The deep compartment contains the posterior tibial nerve, artery, and vein. It also contains the lateral plantar nerve artery and vein and the quadratus plantae muscle. There are four interosseous compartments that contain the plantar and dorsal interosseous muscles. The adductor compartment contains the adductor muscles. The medial compartment contains the flexor and abductor hallucis muscles. The lateral compartment contains abductor digiti minimi and the flexor digiti minimi brevis muscles. The superficial compartment contains the flexor digitorum brevis muscle and the flexor digitorum longus tendons [24].

Foot pain is a common complaint with foot ACS and generally expected based on the mechanism of injury. The pain is not improved with anticipated doses of analgesics. Dorsiflexion of the toes leads to stretch of the intrinsic muscles. One series found 86% of patients with ACS had this finding. Serial sensory examinations are considered to be more sensitive than a single examination [1]. The ability to discriminate two points on the plantar aspect of the foot is more sensitive than pinprick assessment. Motor deficits in the foot are difficult to assess and found late in the course of ACS, and as previously mentioned, pulselessness is a very late finding [1].

Pressure measurements are helpful in detecting ACS that is not clinically obvious. The deep (calcaneal) compartment has higher ICP than other foot compartments. Thresholds

based on leg and forearm pressures are used to diagnose ACS. Pressures are measured in the superficial compartment by inserting the needle in the arch of foot entering the flexor digitorum brevis. The medial and deep compartments are measured through an approach 4 cm inferior to the medial malleolus. The lateral compartment is reached through a needle inserted below the 5th metatarsal, and the interosseous compartments are accessed dorsally between the 1st and 2nd metatarsals, and the needles are deepened to enter the adductor compartment [25].

Orthopedic specialists most often perform foot fasciotomy. The landmarks for decompression are as follows:

1. Make dorsal incisions to approach the interossei and adductor compartments medial to the 2nd metatarsal and lateral to the 4th metatarsal (MT). Dissection of the muscle medially from the 2nd MT exposes the adductor fascia that is incised. The incision lateral to the 4th MT allows access to the interossei on either side [26]. Figure 21.7 illustrates the incisions and routes to the various compartments.

2. Extend the medial plantar incision for 6 cm beginning 4 cm from the posterior aspect of the heel and 3 cm superior to the plantar surface paralleling the plantar surface. The abductor hallucis is retracted superiorly, and the intermuscular septum is incised bluntly medially avoiding injury to the lateral plantar neurovascular bundle to enter the deep compartment. The fascia is opened throughout the length of skin incision taking care to avoid injury to the medial plantar nerve at the distal extent of the opening. The quadratus should bulge through the incision indicating decompression of the deep compartment. Entry into the superficial and lateral compartments requires retraction of the medial compartment superiorly, and the superficial compartment is opened. The flexor digitorum brevis is released and retracted inferiorly to reach the lateral compartment [23]. The septum is opened posterior to anterior with sharp dissection; visualization of the muscles of the compartment assures decompression [1].

3. Wounds are left open and gauze or NPWT is used.

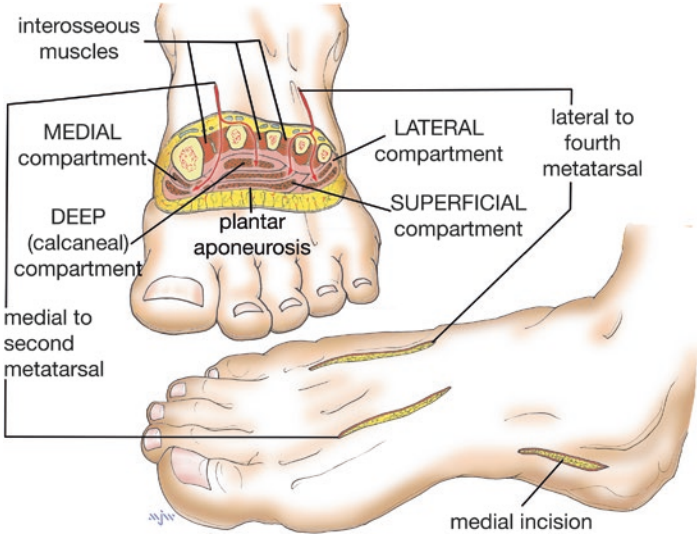

FIGURE 21.7 Foot fasciotomy. Incisions on the dorsum of the foot medial to the 2nd and lateral to the 4th metatarsals are used to decompress the interosseous and adductor compartments. The medial incision is used to decompress the medial, lateral, superficial, and deep (calcaneal) compartments

Upper Extremity

Arm

The upper arm has three compartments. The anterior compartment includes the biceps, the brachialis, and the coracobrachialis muscles. Several nerves including the median, ulnar, radial, medial, and lateral brachial cutaneous nerves pass through this compartment distally. The posterior compartment contains the triceps and anconeus muscles. The posterior antebrachial cutaneous nerve and nerve to the anconeus track through the posterior compartment. The third compartment includes the deltoid muscle [2].

Make a lateral skin incision that extends from the insertion of the deltoid on the humerus to the lateral epicondyle. This

incision can be extended proximally to release the deltoid if it is involved. The incision is deepened to the fascia. Open the fascia over the anterior and posterior compartment using longitudinal incisions. A medial incision can be used when access to the brachial artery is needed. This incision can be easily extended to the forearm when necessary [27].

Forearm

There are three compartments in the forearm. The volar and mobile wad are decompressed through a volar skin incision. The volar compartment contains the wrist and finger flexors. The mobile wad contains the brachioradialis and the extensor carpi radialis longus and brevis muscles. The radial nerve and radial artery are between the mobile wad and the volar compartment. The volar compartment contains the anterior interosseous artery and branches of the radial artery, the median nerve, and branches of the ulnar artery [6]. Begin the volar incision medial to the biceps tendon, and angle it distally across the flexor crease, and then continue onto the forearm extending to the palm to include release of the carpal tunnel. Always cross the flexor crease at the wrist at an angle. Figure 21.8 illustrates the incision. At the elbow, divide the lacertus fibrosus (the bicipital aponneurosis) to prevent constriction of the brachial artery. The superficial muscles of the volar compartment are released individually. Retracting the flexor carpi ulnaris (FCU) and the ulnar neurovascular bundle medially and the flexor digitorum superficialis (FDS) and median nerve laterally exposes the flexor digitorum profundus (FDP). The FDP fascia is then released [28]. The superficial and deep volar muscles are released with these maneuvers. To release the mobile wad, incise the fascia overlying it, and free the three muscles in the compartment. Distally the carpal tunnel is released by dividing the transverse carpal ligament medial to palmaris longus. Look for the median nerve just proximal to the wrist [29].

The dorsal compartment contains the extensor muscles of the wrist and fingers and the posterior interosseous artery.

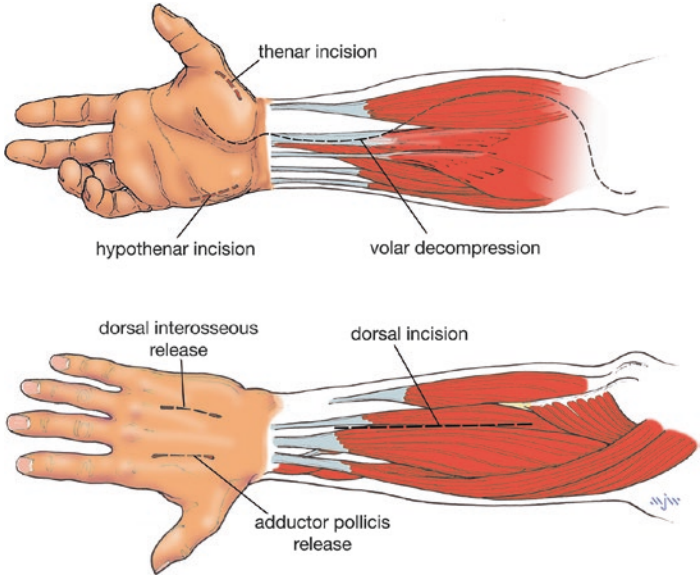

FIGURE 21.8 Forearm fasciotomy. The upper panel illustrates the volar incision. It should extend to the elbow and can be extended proximally should vascular control be necessary. Distally it extends onto the hand to allow for decompression of the carpal tunnel. The lower panel illustrates the dorsal incisions. The incision extends from distal to the lateral condyle to the midline of the wrist. Incision over the hand is placed over the 2nd and 4th metacarpals. Thenar and hypothenar incisions are made at the junction of the palmar and dorsal skin when needed

Extensor muscle release is not always needed because release of the volar compartments may decompress the extensor muscles. If there is an ongoing concern, the dorsal incision and release is added [30]. The dorsal compartment is decompressed through an incision on the dorsal forearm placed between the ulna and radius. The incision begins distal to the lateral epicondyle and extends to the midline of the wrist (Thompson's approach). Incise the fascia longitudinally and free the muscles individually [8]. Figure 21.7 illustrates the volar incision.

When decompression of the hand is required, two dorsal incisions are made. They are positioned over the 2nd and 4th metacarpals (MC). Incise the fascia over the dorsal interosseous muscle. Blunt dissection along the ulnar side of the metacarpal (MC) allows decompression of the volar interosseous muscles and the adductor pollicis. Add incisions over the thenar and hypothenar compartments at the junction of palmar and dorsal skin [31]. Figure 21.8 illustrates the placement of incision to decompress the hand. Figure 21.9 shows a cross section of the forearm and hand with the compartments and muscles labeled.

After freeing the muscles, determine the need for muscle debridement. Obviously nonviable tissue should be removed at once but questionable tissue left and reassessed at the next procedure [1]. Leave the incisions open. NPWT or moist gauze dressings are applied. Reassess the wounds in 24–48 hours.

Wound Management and Closure

Initially all fasciotomy wounds are left open. As mentioned previously, options for wound management are NPWT (negative pressure wound therapy) and moist gauze dressings. There are advantages to each, and circumstances might make one option more desirable than another. Wounds in which hemostasis has been challenging because of coagulopathy or inflammation are better suited to gauze dressings. These dressings are relatively inexpensive and easier to change at the bedside. Dressing changes at the bedside will identify evolution of muscle necrosis or bleeding. Adding a non-adherent contact layer will prevent further tissue trauma with dressing changes. Non-adherent contact dressings are either silicone based or impregnated with petroleum ointment. Examples include Mepitel® (Molnylcke Norcross, GA, USA), open mesh petroleum emulsion impregnated cellulose acetate fiber dressing (Covidien™/Curity™, USA, non-adherent dressing), or Restore® contact layer flex dressing (Hollister, Libertyville, IL, USA). Dressings containing hemostatic agents are useful in patients that have persistent coagulopathy and nonsurgical bleeding. QuickClot®

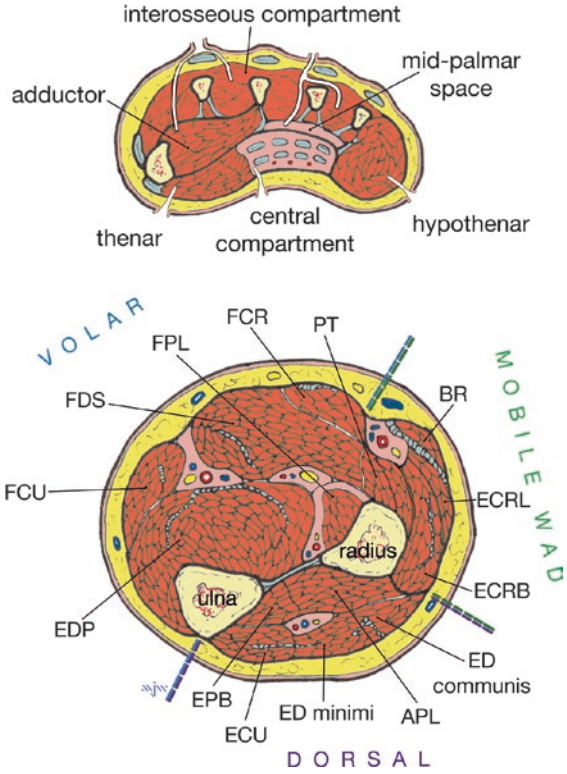

FIGURE 21.9 Cross section of the hand and forearm with the compartments labeled. Top: Cross section of the hand. The interosseous compartment is accessed through incisions over the 2nd and 4th metacarpals (MC). The adductor compartment can be reached through the incision at the 2nd MC. The midpalmar space is reached through the incision at the 4th MC. Thenar and hypothenar incisions are made to decompress those compartments. Bottom: Cross section of the forearm with the three compartments and their contents labeled. The volar compartment includes FCU (flexor carpi ulnaris), FDS (flexor digitorum superficialis), FPL (flexor pollicis longus), FCR (flexor carpi radialis), and (FDP) flexor digitorum profundus. The mobile wad includes BR (brachioradialis), ECRL (extensor carpi radialis longus), and ECRB (extensor carpi radialis brevis). The dorsal compartment includes the EPB (extensor pollicis brevis), ECU (extensor carpi ulnaris), ED minimi (extensor digiti minimi), ED communis (extensor digiti communis), and APL (abductor pollicis longus)

(Z-MEDICA, LLC, Wallingford, CT, USA) and ChitoGauze® Pro (Portland, OR, USA) hemostatic dressings are available in a variety of forms including gauze pads, Z-fold, rolled gauze, and trauma pads.

NPWT uses a porous foam or gauze dressing combined with continuous negative pressure. Fluid is removed and can be quantified. There is a theoretic potential for improved tissue blood flow and removal of harmful cytokines as well. The negative pressure can amplify bleeding when hemostasis is inadequate and can potentially desiccate tissue when the vacuum seal is incomplete.

The wound is reevaluated in the operating room at 24–48 hours. Any necrotic tissue is removed. The skin edges will retract. Tension applied to the skin edges will prevent extensive retraction that could make primary closure impossible. Homemade or commercial devices are effective for preventing skin retraction. Homemade options include the so-called "Jacob's ladder" or shoelace configuration composed of skin staples and heavy crisscrossing silicon vessel loops knotted at one end and stapled to the skin. Staple the vessel loop to one side of the wound and advance distally, and staple the vessel loop to the other side of the wound. Multiple vessel loops are necessary to completely encompass the wound. The loops can be tightened on return to the operating room or at the bedside until the skin edges are approximated closely enough to allow for delayed primary closure (DPC). Another option is interrupted pulley sutures. These are triple mattress sutures that disperse tension across the wound. Place the far suture 2 cm from the edge, the middle 1 cm from the edge, and near a few millimeters for the edge [32]. These sutures can be progressively placed and/or tightened as the tissue edema resolves. DermaClose® (Synovis, Birmingham, AL, USA) is a commercial device utilizing a similar Jacob's ladder configuration with hooks and heavy suture attached to a spring-loaded device (Fig. 21.10). In either case, too much tension results in skin necrosis and loss. When the wounds have stabilized, there is no further tissue necrosis and the edema has resolved closure is possible. When dual incisions

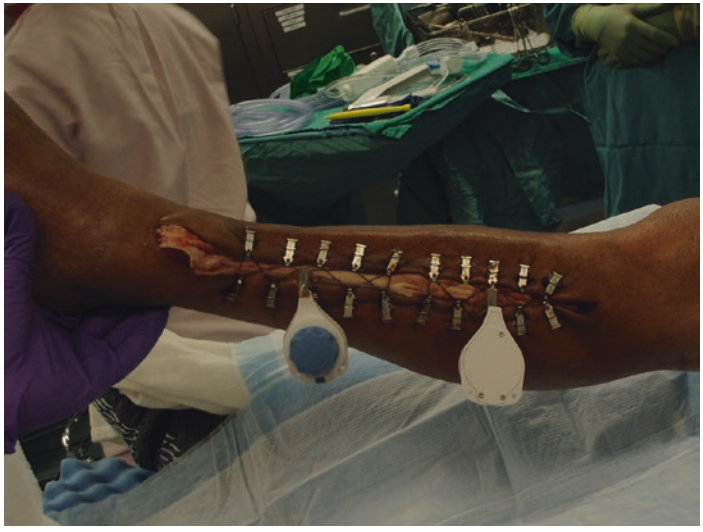

Figure 21.10 DermaClose® (Synovis, Birmingham, AL, USA) was used to apply tension to the skin. This photo is after continuous traction has been applied for 1 week, and the wound edges can now be approximated with sutures

are used and closure of only one incision is possible, choose to close over exposed bone. When primary closure is impossible, split-thickness skin grafting is used. In cases where there has been extensive muscle debridement, flap coverage could be needed. Closure may be possible as soon as 5–10 days following the initial procedure.

Complications

The consequences of failure to perform a necessary fasciotomy include limb loss or dysfunction, acute renal dysfunction, infection, and death. Although fasciotomy is performed to prevent morbidity, the procedure is not risk-free. Early complications include soft tissue infections, pain, and deep venous thrombosis. Long-term wound complications include dry

scaly skin, itching, tethered scars or tendons, excessive scarring, extremity swelling, poor wound healing, osteomyelitis, and recurrent or chronic wounds. Complications and their relative frequency are listed in Table 21.3. Incomplete fasciotomy is also possible and can result in nerve damage and muscle loss [8]. In the lower extremity, the compartments that are most often incompletely decompressed or missed are the anterior and the deep posterior. Placing the lateral leg incision too far posteriorly can result in failing to decompress the anterior compartment. Use the tibial spine to aid proper orientation. The deep posterior compartment is sometimes confused with the superficial. Releasing the soleus from the tibia and visualizing the posterior tibial neurovascular bundle avoids this confusion. In the forearm, the connective tissue surrounding each muscle bundle can cause constriction. Thorough inspection of each muscle and release of the connective tissue surrounding individual muscles will assure adequate decompression. A military review found patients with missed or incomplete compartment release more fre-

Table 21.3 Frequency of fasciotomy-related complications

Symptom	Frequency (%)
Altered wound sensation	77
Dry, scaly skin	40
Itching	33
Wound discoloration	30
Tethered scars	26
Limb swelling	25
Recurrent ulceration	13
Muscle herniation	13
Wound pain	10
Tethered tendons	7
Chronic venous insufficiency	–

Data from: Fitzgerald et al. [34]

quently required muscle excision, were twice as likely to require amputation, and had increased mortality [29]. It is estimated that 80–95% of all patients undergoing fasciotomy experience long-term complications [2].

Outcome

Prognosis depends on several factors that include:

- Extent of injury
- Duration of inadequate perfusion
- Comorbidities and functional status
- Time to fasciotomy

The goal of fasciotomy is to avoid permanent muscle and nerve injury. Amputation is therefore one of the most severe complications. The need is based on damaged caused by prolonged ACS rather than the fasciotomy procedure. Amputation is required in 6–13% of those with extremity ACS. Factors that increase that risk are male gender, vascular injury, and diagnostic delay. Persistent disabilities occur even when amputation is not required. Chronic pain, sensory abnormalities, decreased range of motion, and foot drop are not uncommon following fasciotomy. Pain with activity and unsatisfactory scar appearance affect quality of life. One series reported nearly 70% did not return to work [33].

Summary

Fasciotomy is indicated to treat acute compartment syndrome and to prevent development of compartment syndrome in selected high-risk patients. When ACS is diagnosed, fasciotomy should follow quickly to minimize permanent muscle and nerve injury. Physical examination is important even though the findings are fairly nonspecific. Tense swelling, unexpectedly severe pain, and pain with passive stretch of compartmental muscles are the most reliable signs to look for.

Vigilance is required to assure ACS is diagnosed early. Patients who cannot be evaluated clinically or those with equivocal exams may benefit from ICP measurements. It is therefore important the trauma surgeon is familiar with the anatomy of and approaches to the compartments of the upper and lower extremity. Table 21.2 contains a summary of the compartments and approaches to them. Full incisions with compete compartment release should be undertaken within 6 hours of symptom onset. Well-performed procedures may still result in wound complications but are preferable to the morbidity associated with missed compartment syndrome or incomplete compartment release.

References

1. Fulkerson E, Razi A, Tejwani N. Review: acute compartment syndrome of the foot. Foot Ankle Int. 2003;24(4):180–7.
2. Elliott KG, Johnstone AJ. Diagnosing acute compartment syndrome. J Bone Joint Surg Br. 2003;85(5):625–32.
3. McQueen MM, Gatson P, Court-Brown CM. Acute compartment syndrome: who is at risk. J Bone Joint Surg Br. 2000;82(2):200–3.
4. Zuchelli D, Divaris N, McCormac JE, Huang EC, Chaudhary ND, Vosswinkel JA, et al. Extremity compartment syndrome following blunt trauma: a level I trauma center's 5-year experience. J Surg Res. 2017;217:131–6.
5. Branco BC, Inaba K, Barmparas G, Schnüriger B, Lustenberger T, Talving P, et al. Incidence and predictors for the need for fasciotomy after extremity trauma: a 10 year review in a mature level I trauma center. Injury. 2011;42:1157–63.
6. Whitesides TE Jr, Heckman MM. Acute compartment syndrome: update on diagnosis and treatment. J Am Acad Orthop Surg. 1996;4(4):209–18.
7. Seiler JG III, Womack S, De L'Aune WR, Whitesides TE, Hutton WC. Intracompartmental pressure measurements in the normal forearm. J Orthop Trauma. 1993;7:414–6.
8. Donaldson J, Haddad B, Khan WS. The pathophysiology, diagnosis and current management of acute compartment syndrome. Open Orthop J. 2014;8(Suppl 1:M8):185–93.
9. Bradley EL III. The anterior compartment syndrome. Surg Gynecol Obstet. 1973;136:289–97.

10. Valdez C, Schroeder E, Amdur R, Pasual J, Sarani B. Serum creatinine kinase levels are associated with extremity compartment syndrome. J Trauma. 2013;74(2):441–5.
11. Walls MH, Landing T. Compartment syndrome: an orthopedic emergency. J Emerg Nurs. 2017;43(4):303–7.
12. Boody AR, Wonworawat MD. Accuracy in the measurement of compartment pressures: a comparison of three commonly used devices. J Bone Joint Surg Am. 2005;84(11):2415–22.
13. Heckman MM, Whitesides TE Jr, Grewe SR, Rooks MD. Compartment pressure in association with closed tibial fractures: the relationship between tissue pressure, compartment, and the distance from the site of the fracture. J Bone Joint Surg Am. 1994;76:1285–92.
14. McQueen MM, Court-Brown CM. Compartment monitoring in tibial fractures. The pressure threshold for decompression. J Bone Joint Surg Br. 1996;78(1):99–104.
15. Hayden G, Leung M, Leong J. Gluteal compartment syndrome. ANZ J Surg. 2006;76:668–70.
16. MacLean J, Wustrack R, Kandemir U. Gluteal compartment syndrome. Tech Orthop. 2012;27:43–6.
17. Rocos B, Ward A. Gluteal compartment syndrome with sciatic nerve palsy caused by traumatic rupture of the inferior gluteal artery: a successful surgical treatment. BMJ Case Rep. 2017;2017:bcr2016216709.
18. Hessman MH, Ingelfinger P, Rommens PM. Compartment syndrome of the lower extremity. Eur J Trauma Emerg Surg. 2007;33:589–99.
19. Bowyer M. Lower extremity fasciotomy: indications and technique. Curr Trauma Rep. 2014;1:35–44.
20. Owen C, Cavalcanti A, Molina V, Honore C. Decompressive fasciotomy for acute compartment syndrome of the leg. J Visc Surg. 2016;153:293–6.
21. Injuries to the extremities: compartment syndrome and fasciotomy. ASSET American College of Surgeons Committee on Trauma; 2010. p. 28–40.
22. Burns JB, Frykberg E. Management of extremity compartment syndrome. In: Cameron JL, editor. Current surgical therapy. 10th ed. Philadelphia: Elsevier; 2011. p. 1028–32.
23. Papachristos IV, Giannoudis PV. Acute compartment syndrome of the extremities: an update. Orthop Trauma. 2018;32(4):223–8.
24. Dodd A, Le I. Foot compartment syndrome: diagnosis and management. J Am Acad Ortho Surg. 2013;21:657–64.

25. Towater LJ, Heron S. Foot compartment syndrome: a rare presentation to the emergency department. J Emerg Med. 2013;44(2):e235–8.
26. Yoon P. Compartment syndrome of the foot. Tech Ortho. 2012;27:58–61.
27. Maeckelbergh F, Colen S, Ludwig A. Upper arm compartment syndrome: a case report and review of the literature. Ortho Surg. 2013;5(3):229–32.
28. Ronel DN, Mtui E, Nolan WB. Forearm compartment syndrome : anatomical analysis of surgical approaches to the deep space. Plast Reconstr Surg. 2004;114(3):697–705.
29. Schellenberg M, Chong V, Cone J, Keeley J, Inaba K. Extremity compartment syndrome. Curr Probl Surg. 2018;55:256–73.
30. Burgess AR, Aziz A. Fasciotomy. In: Dua A, Desai SS, Holcomb JB, Burgess AR, Frieschlag JA, editors. Clinical review of vascular trauma. New York: Springer; 2014. p. 65–7.
31. Prasam ML, Ouellette EA. Acute compartment syndrome of the upper extremity. J Am Acad Ortho Surg. 2011;19(1):49–58.
32. Rhee P, Dubose J. Soft tissue wounds and fasciotomy. In: Martin M, Beekley A, editors. Front line surgery: a practical approach. New York: Springer; 2011. p. 239–67.
33. Lolla I, Grabinsky A. Clinical and functional outcomes of acute lower extremity compartment syndrome at a major trauma hospital. Int J Crit Illn Inj Sci. 2015;6:133–42.
34. Fitzgerald AM, Gaston P, Wilson Y, Quaba A, McQueen MM. Long-term sequelae of fasciotomy wounds. Br J Plast Surg. 2000;53(8):690–3.

Chapter 22
Treatment of Pelvic Fractures

Matthew Bradley

Introduction of the Problem

It takes a considerable amount of force to fracture a pelvis. This energy transmitted to the patient can often cause additional injuries and lead to life-threatening hemorrhage. Due to the anatomical relationship, pelvic fractures are most frequently associated with retroperitoneal injuries including vascular and genitourinary trauma. However, in many instances, concomitant traumatic brain, intrathoracic, intra-abdominal, and other musculoskeletal injuries accompany pelvic fractures.

There is no single, straightforward algorithm for managing pelvic fractures. Multiple disciplines have expertise that can be helpful in these patients. Hemodynamics drive most of the important decisions in these dynamic patients. Trauma surgeons must have a keen sense of the patient's physiology and understand local expertise to provide the best care.

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History of Care of Pelvic Fractures

In the nineteenth century, Joseph-Francois Malgaigne, an influential French surgeon and pioneer, published several texts on the diagnosis, fracture pattern, and management of various orthopedic injuries. His writings provided some insight on pelvic fracture management during this time period [1]. As there were no X-rays, physicians had to rely solely on physical exam for diagnosis. To that end, Malgaigne encouraged manual manipulation of the pelvis to adequately assess for a fracture. There were limited treatment options available, and nonoperative management appeared to be the mainstay. According to Malgaigne's principles, treatment consisted of manual reduction of the fracture, traction, and prolonged periods of immobilization [2, 3]. As a result, morbidity and mortality were high, and patients often succumbed to complications from bleeding, wound infections and sepsis, and other associated injuries [1–4].

At the turn of the century, diagnostic accuracy improved with the development of radiographic technology. However, patients continued to have poor outcomes into the early part of the twentieth century as treatment was primarily nonoperative and still involved prolonged periods of mandatory bed rest. In the second half of the twentieth century, management of the pelvic ring injuries evolved with the introduction of external and internal fixation and the recognition of the benefit of earlier mobilization. Timing of and approach to (external vs internal) fracture stabilization became a subject of debate. Initially, external fixation was the primary modality, particularly in the early management of unstable fractures, with internal fixation limited to certain vertical fracture patterns [5, 6]. However, in the later part of the twentieth century, the indications for early internal fixation were expanded to include unstable pelvic ring disruptions [7–9]. It was realized though that while early internal stabilization was advantageous, patients too physiologically deranged would benefit from early external stabilization following the principles of damage control orthopedics (DCO) developed at our institution [10, 11].

In addition to fracture stabilization paradigms, treatment began to focus primarily on minimizing retroperitoneal hemorrhage associated with pelvic injuries. A few of these therapies designed to control pelvic hemorrhage stemmed from military concepts and technology. One such device, the military antishock trousers (MAST), popularized during the Vietnam War, came about from the antigravity suits (G-suits) used in World War II [12]. These suits were worn by pilots to prevent pooling of blood in the lower extremities and thus the loss of consciousness. These were designed to redistribute blood from the lower extremity and pelvis and into the upper torso while also providing a tamponade effect on pelvic bleeding. The MAST was initially introduced in the civilian Emergency Medical System in the 1970s but have since fallen out of favor due to complications such as abdominal compartment syndrome and pulmonary insufficiency resulting from the compressive effects of the trousers [13–15]. Additionally, their application for trauma was not ideal due to the amount of space they occupy in an ambulance and the difficulty associated with deflating and removing the apparatus. Other compressive devices, such as the pelvic binder, have replaced the MAST. They also reduce the fractured pelvis and are more rapidly applied and less bulky, allow access to the lower extremities, and avoid the unintended complications of the trousers. Improvements in medical technology led the way to catheter-directed angiographic embolization, first introduced over 40 years ago, which has since played a crucial role in the management of pelvic fracture bleeding [16].

Fracture Pattern and Classification

The two most commonly used classification systems are the Tile and the Young and Burgess classification. Both have been helpful in predicting mortality, blood transfusion requirements, and other associated injuries [17]. The Tile system is based on the stability of the fracture, in particular, as it

relates to the posterior ring [7, 18]. Type A fractures are considered stable with minimal pelvic ring and no posterior element involvement. Type B fracture are unstable, or partial stable, with partial disruption of the posterior ring. These fractures are the result of rotational forces. Complete disruption of the posterior sacroiliac complex is classified as a Type C fracture, which results from rotational and vertical forces.

We favor the Young and Burgess classification which was first described at our institution. This characterizes the three main vectors of force that can fracture a pelvis: lateral, anterior-posterior, and vertical (Fig. 22.1a–c). Fractures are further subcategorized by the extent of bony involvement, amount of displacement, and degree of ligamentous disruption (Table 22.1) [19]. Lateral compression (LC) fractures often occur from a side-impact motor vehicle collision or when a pedestrian is struck on the side. An anterior-posterior compression (APC) fracture, which can produce the classic "open-book" appearance on plain films, frequently results

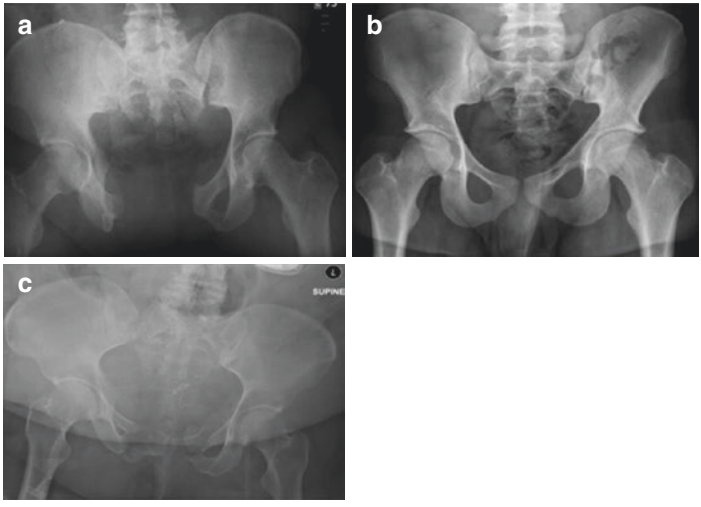

FIGURE 22.1 (**a**–**c**) AP compression fracture (**a**), left lateral compression fracture (**b**), and AP compression fracture (**c**) with vertical shear extension

from a front-impact motor vehicle, motorcycle crash, or other type of crush injury. Vertical shear (VS) injuries commonly develop from a fall from a height onto a straightened lower extremity. In many instances, pelvic fractures are caused by a combination of vectors of force; thus, they may have elements of more than one fracture pattern.

Table 22.1 Young and Burgess classification of pelvic fractures

AP compression	
Type I	Separation of pubic symphysis <2.5 cm and no posterior instability
Type II	Separation of pubic symphysis >2.5 cm and posterior instability of anterior sacroiliac complex
Type III	Complete disruption of sacroiliac joint involving both anterior and posterior complexes
Lateral compression	
Type I	Posterior-lateral directed force with oblique pubic rami fx. No pelvic instability
Type II	Anterior-lateral directed force with pivot point on anterior SI joint causing pubic rami fx, internal rotation of anterior hemipelvis, external rotation of posterior hemipelvis, and rupture of posterior SI ligaments
Type III	Severe ipsilateral internal hemipelvis rotation causing contralateral external rotation resulting in AP compression of contralateral side. Disruption of ipsilateral posterior SI ligaments and contralateral anterior SI, sacrotuberous, and sacrospinous ligaments
Vertical shear	
	Vertical disruptive force on one or both sides of the pelvis lateral to midline Associated with ligamentous disruption and pelvic instability
Complex	
	At least two different vectors of force causing injury pattern

Data from Reference [19]

Diagnosis and Management

Motor vehicle collisions account for the majority of pelvic fractures [20–22]. In addition, motorcycle crashes, fall from a height, pedestrians struck, and crush injuries are other common etiologies [23, 24]. History and physical findings are often relatively nonspecific. Complaints range from pelvic pain to lower abdominal pain, hip pain, lower back pain, and proximal thigh pain. Visual inspection of the pelvis may reveal soft tissue injuries, including abrasions, lacerations, contusions, or hematomas. An important part of the initial physical exam is to assess for pelvic stability. This should be done by exerting gentle manual pressure inward on the anterior superior iliac spines (Fig. 22.2). Any motion should be interpreted as pelvic instability. Rocking the pelvis is not recommended as it will cause the patient significant pain and in the instance of pelvic instability it will almost certainly result in additional blood loss. Palpation along the pubis may be helpful in identifying widening of the symphysis. An inspection of the perineum is also important. If a urethral injury is suspected based on the presence

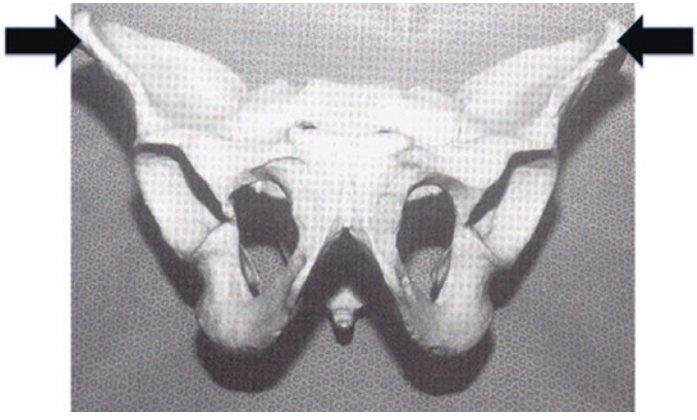

FIGURE 22.2 Direction of gentle manual compression on the anterior superior iliac spines to assess for pelvic instability

of blood at the meatus, a retrograde urethrogram can be performed (Fig. 22.3). Vaginal and rectal exams are likewise important to rule out the possibility of an open pelvic fracture.

In general, an AP film of the pelvis (PXR) should be obtained in all polytrauma patients, regardless of hemodynamic status. A PXR is helpful for predicting associated injuries and outcomes [25, 26]. However, in awake, alert, and reliable patients without a relevant mechanism, a PXR may be omitted if they are without complaints or concerning physical exam findings [27, 28]. Additional imaging is almost always obtained with contrast-enhanced CT scanning, which can accurately identify anatomic locations of bleeding, including both hematoma volume and active hemorrhage [29–34]. In selected cases, inlet, outlet, and Judet views of the pelvis may be helpful.

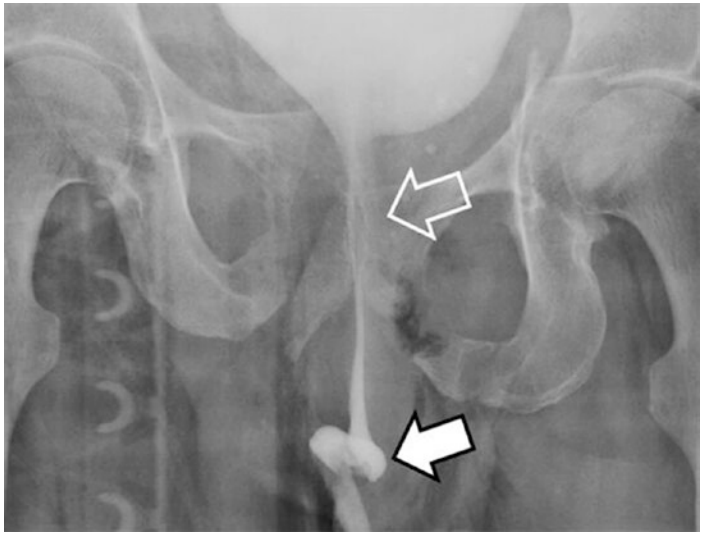

FIGURE 22.3 Retrograde urethrogram demonstrating contrast extravasation from an irregular discontinuous anterior urethra (solid arrow) and stretch injury of the membranous and prostatic urethra (open arrow)

Management of Pelvic Fracture Hemorrhage

Patients who present with pelvic fractures and are hemodynamically unstable can present the greatest challenge. Hemorrhage may be retroperitoneal from the pelvic fracture, from other sites, or from both. Rapid assessment is important to identify all body cavities in which the patient may be losing blood. While covered elsewhere in more detail, a FAST or an eFAST may be very helpful in identifying intra-abdominal or intrathoracic hemorrhage, it will also help identify pneumothorax and/or hemopericardium, both of which can contribute to hemodynamic instability. A chest X-ray may also be helpful. Hemorrhage into muscle compartment is usually identified on physical examination, and external hemorrhage is identified by history.

Patients who have a pelvic fracture and significant hemorrhage, either into the thorax or abdomen, pose a special problem. While patients with pelvic fractures are certainly at risk for retroperitoneal hemorrhage, not all of them are bleeding into their retroperitoneum. In general, patients with intra-abdominal hemorrhage, particularly if large volume on FAST, are best served with diagnostic laparotomy. The size of the pelvic hematoma can then be ascertained by direct inspection.

Fracture pattern may be helpful in identifying patients more likely to be bleeding from their abdomen. A large retrospective study found that 85% of hypotensive patients with hemoperitoneum and a stable pelvic fracture categorized as a lateral compression (LC1 or APCI) had intra-abdominal bleeding as the source of their hypotension. Conversely, 59% of the patients with hemoperitoneum and an unstable pelvic fracture (LC2, LC3, APC2, APC3, or vertical shear) had large pelvic volume bleeding. Thus, laparotomy first seems wise in hypotensive patients with a stable fracture but consideration given to angiography before laparotomy in the patient with hemoperitoneum in an unstable pelvic fracture [35].

Reducing the fractured pelvis is the most important first step to control pelvic bleeding. This reduces pelvic volume, producing tamponade, and helps stop bony bleeding. It likely

does little to control arterial bleeding. This is usually accomplished by external compression which can be accomplished in many ways. In a resource-poor environment, simply placing a sheet around the patient can help. It is ideal if the bedsheet is placed on the stretcher, the patient then placed on top of the bedsheet, and the sheet then crisscrossed across the patient's pelvis. This should be tied down as tightly as possible to reduce the bony elements (Fig. 22.4).

FIGURE 22.4 Application of a bedsheet as an external binder with the tails of the sheet crisscrossed across the pelvis

In the past, emergency external fixation was often used to achieve pelvic bony stability (Fig. 22.5). While this was placed in the emergency department in some institutions, more commonly, it was placed in the operating room. Thus, its use as an emergency therapy was, in some ways, negated. In certain institutions, this remains a possibility. In patients with grossly unstable pelvic fractures, external fixation may reduce the anterior elements but widen the posterior elements, increasing hemorrhage.

In Europe, placement of a C-clamp is common (Fig. 22.6). This is not used very often in the United States. In those institutions that employ it with some frequency, the C-clamp is placed blindly in the emergency department. Traditionally, the C-clamp has been used for fixation of vertical shear injuries with posterior ring disruption [36]. However, a major limitation to the use of a C-clamp is the inability to clearly identify the extent of the posterior ring injury on plain X-rays [37]. The potential for visceral injury and superior gluteal artery hemorrhage from poor pin placement or urological and neurological injuries from over-compression remains

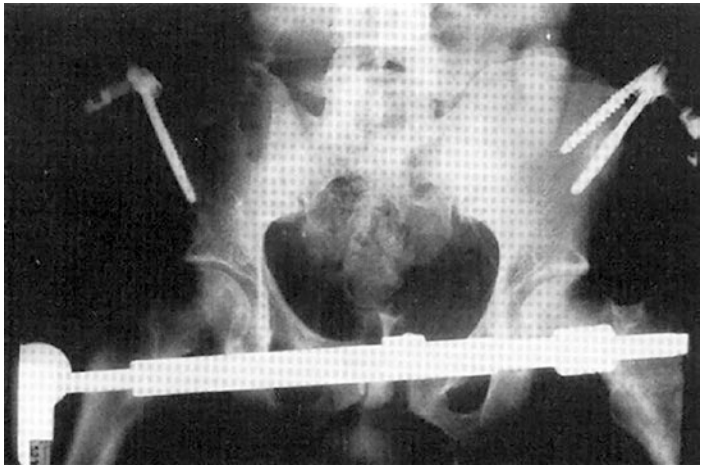

FIGURE 22.5 Radiographic image of pelvic external fixator with pelvic reduction and stabilization

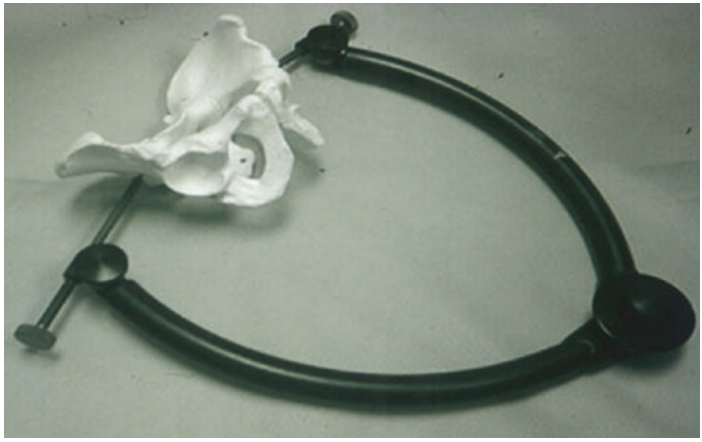

FIGURE 22.6 Image of a C-clamp. Note the pins required for securing the device

high, so application of the clamp should be reserved for experienced surgeons [37]. Similar to the external fixator, if this device is to be placed in the operating room under fluoroscopic control, its use as an emergency hemostatic maneuver is negated.

In the United States, virtually all of these hemostatic techniques have been replaced with the use of the pelvic binder [38–40]. The pelvic binder is a Velcro-based device which is placed around the patient's pelvis. Binders stabilize fractured segments and reduce pelvic volume, thereby producing a tamponade effect. It is important to place the device low enough over the greater trochanters of the femurs (Fig. 22.7a–d). The binder should not be placed over the abdomen or too low over the femurs. Placing the device too high may actually worsen fracture displacement, increase pelvic volume, and ultimately promote bleeding. Once the binder is properly positioned, it can then be laced up and even pressure applied to reduce the bony elements of the fractured pelvis. Unlike the C-clamp or external fixator, the binder is easily placed in the resuscitation bay, often, while other resuscitative maneuvers are ongoing. In addition, binders reduce all portions of

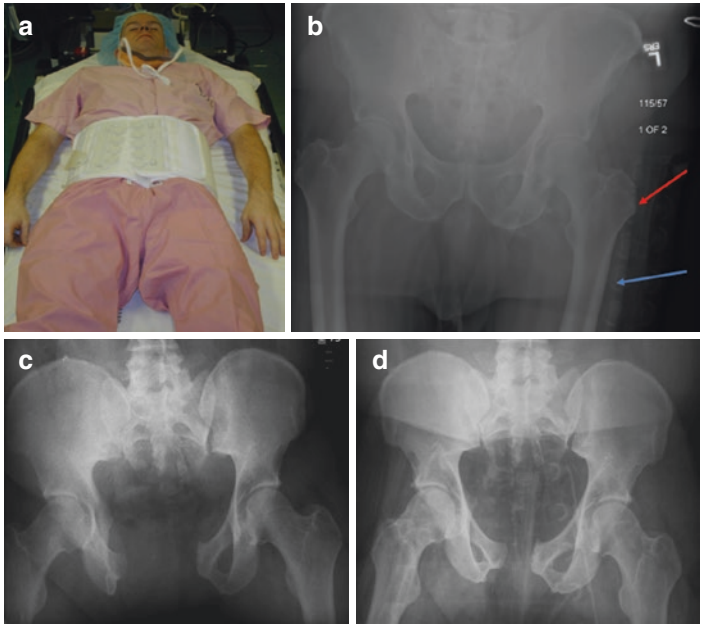

FIGURE 22.7 (**a**–**d**) Pelvic binder application (**a**, **b**). Note the improperly placed binder too high (**a**) over the abdomen instead of the greater trochanters. Pelvic binder positioned too low in **b** with the red arrow identifying greater trochanter and blue arrow locating binder over the femur. "Open-book" AP compression fracture (**c**), with fracture reduction following proper placement of pelvic binder over the greater trochanters (**d**)

the pelvis equally preventing posterior elements from being displaced. The pressure exerted by the binder can be increased or reduced as needed and at the clinician's discretion. Too much pressure can lead to overreduction displacing the fracture and increasing bleeding (Fig. 22.8). Physicians also have to be cognizant of the length of time that the binders have been in place, as these devices have the potential of pressure ulceration, so the skin should be routinely inspected if applied for any length of time [41].

In patients with badly displaced AP compression fractures (open-book fractures), applying the binder can be highly efficacious and even lifesaving. The same may not be true in

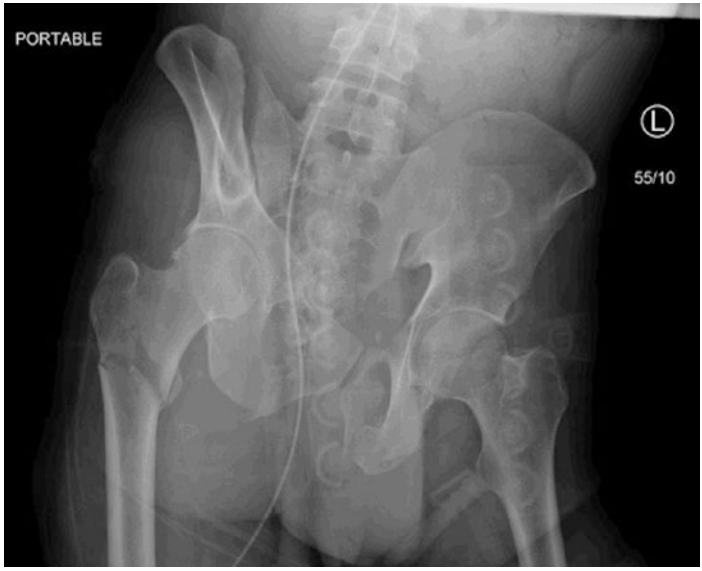

FIGURE 22.8 Overreduction of complex pelvic fracture by pelvic binder (bilateral superior and inferior pubic rami fractures, medial left iliac wing fracture, right sacrum vertical fracture, and diastases of bilateral anterior and posterior sacroiliac joints)

patients with lower-grade lateral compression or vertical shear fracture pattern. However, binders still may help. Patients with pelvic fractures commonly require transport within and between facilities. Keeping the fracture fragments stabilized with the binder may prevent additional bleeding when patients are transferred to and from the stretcher for various diagnostic tests and/or therapeutic maneuvers.

Angiographic Embolization

For over 40 years, angiography has been used to treat pelvic fracture hemorrhage. Angiography can be both diagnostic and therapeutic when embolization techniques are employed (Fig. 22.9a, b). Patients with evidence of significant pelvic hemorrhage, particularly those who are hypotensive following pelvic stabilization or have no other identifiable source

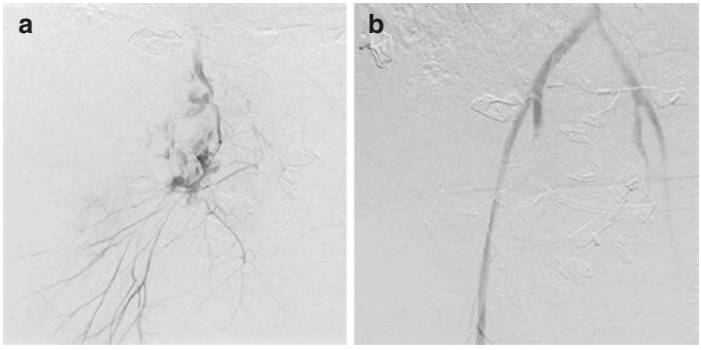

FIGURE 22.9 (**a**, **b**) Contrast extravasation for right internal iliac artery (**a**) and post-angiographic embolization (**b**) with resolution of extravasation

for their hemodynamic instability, should undergo immediate angiography [42, 43]. Transcatheter embolization in the unstable patient has a reported success rate of 95% in obtaining hemorrhage control [44]. Patients who have continued transfusion requirements or unexplained hypotension may benefit from repeat angiography due to persistent pelvic bleeding [45].

In the hemodynamically stable patient, pelvic angiography and embolization may still have a role. Some recommend that all patients with pelvic fractures with contrast extravagation identified on CT scan undergo angiography to prevent hemorrhage [42, 46]. Other clinicians advocate a more selective approach to minimize nontherapeutic angiography in these stable patients [32, 47–49]. One clear indication for angiography should be a large pelvic hematoma (>500 ccs) because of the associated risk for pelvic bleeding [50]. However, a recent CT prediction model from our institution suggested a hematoma volume of 433 mL was highly predictive of arterial hemorrhage requiring angioembolization [34].

It is important that the surgeon caring for the patient have full understanding of the angiographic capability and availability. If access is limited, timely transfer of a stable patient to a center with angiographic capabilities is prudent, as delays are associated with increased mortality [51]. Even in our busy

Level I trauma center with 24-hour, 7 days-a-week access to angiography, we have demonstrated that it can take up to 4 hours to achieve hemostasis via angiographic embolization [52]. Using hybrid ORs may reduce time to hemostasis by reducing setup time for an angiography suite.

In our institution, angiography is the primary means of obtaining hemostasis in patients with pelvic fracture bleeding, including those that are hemodynamically unstable. In addition, all hemodynamically stable patients with a large hematoma or large-volume contrast extravasation undergo diagnostic angiography. Patients with evidence of concomitant solid organ (liver and/or spleen) bleeding can also undergo visceral angiography and embolization at the same time obtaining hemostasis in all vascular beds.

Preperitoneal Packing and REBOA

Preperitoneal packing as temporary, or potentially definitive, hemostasis can be lifesaving in the hemodynamically compromised patient. This technique was originally described in Europe almost 20 years ago [53]. Recently this approach was modified to directly packing the anterior preperitoneal space [54]. To gain entrance to the space of Retzius, either a lower midline incision or a Pfannenstiel incision may be used. The fascia is incised, longitudinally of possible. Occasionally, a portion of the rectus muscle must be divided to get sufficient exposure. The retroperitoneal space is bluntly dissected out laterally and posteriorly as far as possible (Fig. 22.10). While performing this maneuver, it is important not to breach the peritoneum and release its tamponade effects. This dissection is often created by the hematoma making it easy to gain access to the retroperitoneum. Large clot is then evacuated, and laparotomy pads are placed laterally. Some place three laparotomy pads on either side; however, we place as many as needed to achieve tamponade.

Several studies have shown that preperitoneal packing directly resulted in a reduction in blood transfusion requirements and a reduction in the need for angioembolization [55,

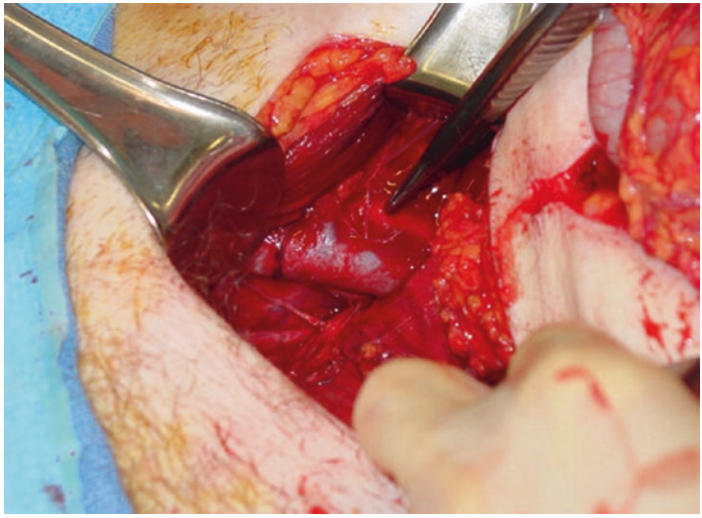

Figure 22.10 Intraoperative exposure of the retroperitoneum. Note the identification of the iliac vessels

56]. Preperitoneal packing can be combined with laparotomy in those unstable patients with both intra-abdominal and retroperitoneal hemorrhage. In these cases, the midline laparotomy incision is stopped midway between the umbilicus and pubic symphysis. Preperitoneal packing is then done through a separate Pfannenstiel incision (Fig. 22.11).

An alternative for temporary hemostasis in the hemodynamically unstable patient with intra-abdominal and/or retroperitoneal hemorrhage is to deploy a percutaneous retrograde endovascular aortic occlusion balloon, otherwise known as a REBOA catheter, at the bedside in the emergency department. This technique discussed elsewhere in this manual involves placement of a common femoral arterial sheath for aortic access and radiographic confirmation for proper balloon placement. We have been successful in achieving hemodynamic stability for both intra-abdominal and retroperitoneal hemorrhage with this device, and in the case of pelvic hemorrhage, it has been valuable as a bridge to embolization [57, 58]. There are risks with the use of this instrument, such as lower

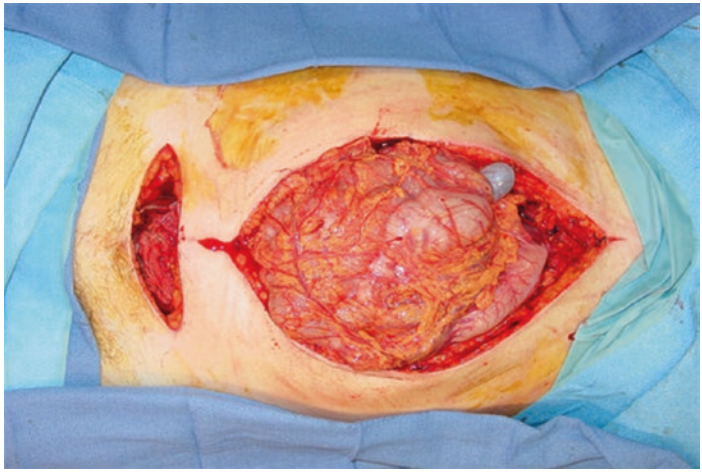

FIGURE 22.11 Combined midline laparotomy for abdominal exploration and Pfannenstiel incision for preperitoneal packing

extremity ischemia, and it may provide a survival advantage over a resuscitative thoracotomy [59, 60].

The hemodynamically unstable blunt trauma victim with a pelvic fracture poses a significant dilemma. Thus, a multidisciplinary team of trauma surgeons, orthopedic surgeons, and interventional radiologists working in concert can determine the best treatment course to maximize physiologic stability and prevent further physiologic derangements (Figs. 22.12 and 22.13) [61].

Open Pelvic Fractures

Open pelvic fractures deserve special mentioning as these injuries are typically severe and associated with a high mortality [62]. Hemorrhage control can be challenging due to external blood loss through open wounds. Packing in combination with binder placement is an important first step for hemostasis. Open wounds should only be formally explored in the operating room and not in the trauma bay. Preventing

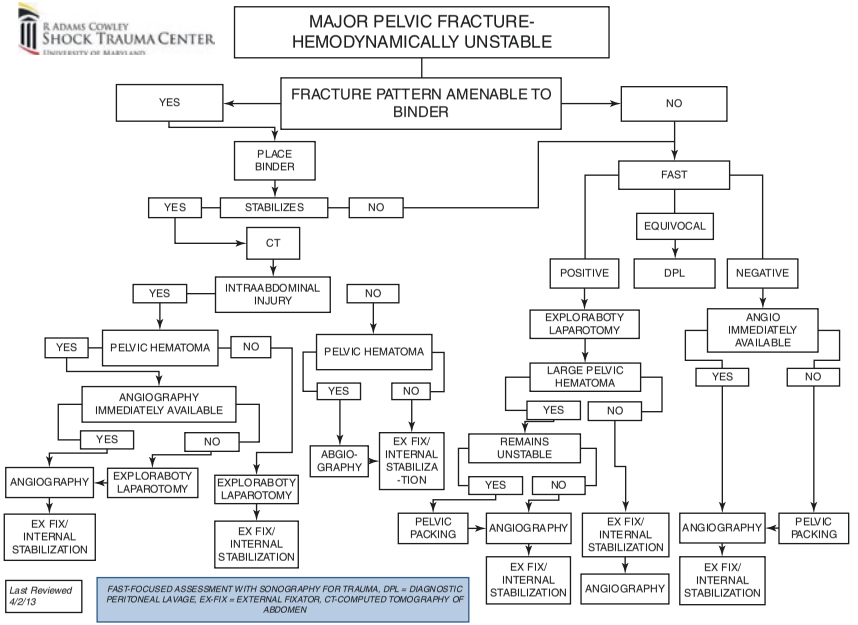

FIGURE 22.12 R Adams Cowley Shock Trauma Center (University of Maryland) algorithm for the hemodynamically unstable pelvic fracture

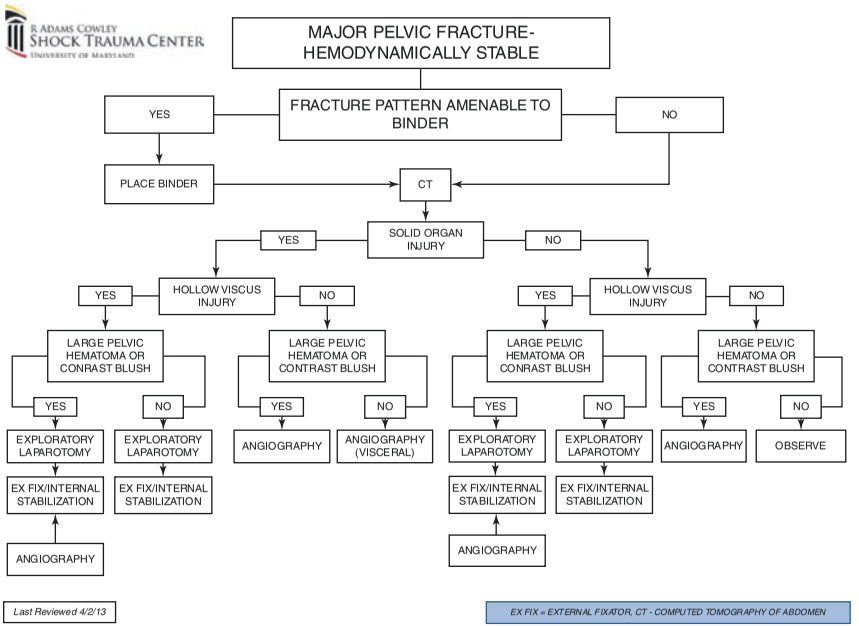

FIGURE 22.13 R Adams Cowley Shock Trauma Center (University of Maryland) algorithm for the hemodynamically stable pelvic fracture

pelvic sepsis is paramount through use of broad-spectrum antibiotics and serial debridements [63]. The temptation for early wound closure must be avoided until all necrotic and nonviable tissue is debrided. Fracture management is further complicated by the frequent requirement for fecal diversion due to the presence of a rectal or perineal injury [64, 65].

Complications and Outcomes

Morbidity associated with pelvic fractures includes increased risk for venous thromboembolism [66, 67]. Early patient mobilization and pharmacologic prophylaxis are the best preventative therapies for VTE. Timing of chemical prophylaxis is important as early, within the first 24 hours after injury, initiation has been shown to decrease the risk of VTE without increasing the risk of hemorrhage [68]. Consideration for IVC filter placement is reasonable in the multi-injured, immobile patient with a pelvic fracture and in patients with a contraindication to anticoagulation [67]. Rare reports of femoral artery injury after angiography have been described [69]. Concern for gluteal necrosis and sexual dysfunction as a complication of angiography even after bilateral internal iliac artery embolization has been refuted [70, 71]. However, sexual dysfunction directly related to the pelvic fracture has been reported and has correlated to the degree of fracture displacement [72, 73]. Nerve injury and chronic pain are frequent complications of pelvic fractures, and functional recovery after nerve injury is worse in this subgroup of patients [74, 75]. Long-term disability is more common with a fractured sacrum or SI joint involvement and is directly related to the adequacy of fracture reduction [8, 76, 77]. Long-term sexual dysfunction, impotence, and dyspareunia also have been described.

Overall mortality associated with pelvic fractures has declined with recent estimates between 5 and 7 percent [78, 79]. However, pelvic fractures remain an independent risk factor for mortality in the blunt trauma patient [80]. In addition, death from pelvic hemorrhage has declined, while the

contribution of sepsis, multiple-organ failure, and associated injuries to mortality has been on the rise [81].

References

1. Stahel PF, Hammerberg EM. History of pelvic fracture management: a review. World J Emerg Surg. 2016;11:18.
2. Peltier LF. Joseph Francois Malgaigne and Malgaigne's fracture. Surgery. 1958;44(4):777–84.
3. Malgaigne JF. Double vertical fractures of the pelvis. 1859. Clin Orthop Relat Res. 2007;458:17–9.
4. Adams GW. Doctors in blue. Baton Rouge: Louisiana State University Press; 1952.
5. Slatis P, Karaharju EO. External fixation of the pelvic girdle with a trapezoid compression frame. Injury. 1975;7(1):53–6.
6. Tile M. Pelvic fractures: operative versus nonoperative treatment. Orthop Clin North Am. 1980;11(3):423–64.
7. Tile M. Pelvic ring fractures: should they be fixed? J Bone Joint Surg Br. 1988;70(1):1–12.
8. Goldstein A, Phillips T, Sclafani SJ, Scalea T, Duncan A, Goldstein J, et al. Early open reduction and internal fixation of the disrupted pelvic ring. J Trauma. 1986;26(4):325–33.
9. Matta JM, Saucedo T. Internal fixation of pelvic ring fractures. Clin Orthop Relat Res. 1989;242:83–97.
10. Brundage SI, McGhan R, Jurkovich GJ, Mack CD, Maier RV. Timing of femur fracture fixation: effect on outcome in patients with thoracic and head injuries. J Trauma. 2002;52(2):299–307.
11. Scalea TM, Boswell SA, Scott JD, Mitchell KA, Kramer ME, Pollak AN. External fixation as a bridge to intramedullary nailing for patients with multiple injuries and with femur fractures: damage control orthopedics. J Trauma. 2000;48(4):613–21; discussion 21–3.
12. Schwab CW, Gore D. MAST: medical antishock trousers. Surg Annu. 1983;15:41–59.
13. Kaplan BC, Civetta JM, Nagel EL, Nussenfeld SR, Hirschman JC. The military anti-shock trouser in civilian pre-hospital emergency care. J Trauma. 1973;13(10):843–8.
14. McCabe JB, Seidel DR, Jagger JA. Antishock trouser inflation and pulmonary vital capacity. Ann Emerg Med. 1983;12(5):290–3.

15. Morse SS, Strauss EB, Sumpio BE. Apparent arterial occlusion due to pneumatic antishock garment: pitfall in trauma angiography (case report). AJR Am J Roentgenol. 1986;147(2):391–2.
16. Margolies MN, Ring EJ, Waltman AC, Kerr WS Jr, Baum S. Arteriography in the management of hemorrhage from pelvic fractures. N Engl J Med. 1972;287(7):317–21.
17. Osterhoff G, Scheyerer MJ, Fritz Y, Bouaicha S, Wanner GA, Simmen HP, et al. Comparing the predictive value of the pelvic ring injury classification systems by Tile and by Young and Burgess. Injury. 2014;45(4):742–7.
18. Tile M. Acute pelvic fractures: I. Causation and classification. J Am Acad Orthop Surg. 1996;4(3):143–51.
19. Young JW, Burgess AR, Brumback RJ, Poka A. Pelvic fractures: value of plain radiography in early assessment and management. Radiology. 1986;160(2):445–51.
20. Poole GV, Ward EF. Causes of mortality in patients with pelvic fractures. Orthopedics. 1994;17(8):691–6.
21. Inaba K, Sharkey PW, Stephen DJ, Redelmeier DA, Brenneman FD. The increasing incidence of severe pelvic injury in motor vehicle collisions. Injury. 2004;35(8):759–65.
22. Dalal SA, Burgess AR, Siegel JH, Young JW, Brumback RJ, Poka A, et al. Pelvic fracture in multiple trauma: classification by mechanism is key to pattern of organ injury, resuscitative requirements, and outcome. J Trauma. 1989;29(7):981–1000; discussion 1000–2.
23. Poole GV, Ward EF, Muakkassa FF, Hsu HS, Griswold JA, Rhodes RS. Pelvic fracture from major blunt trauma. Outcome is determined by associated injuries. Ann Surg. 1991;213(6):532–8; discussion 8–9.
24. Coppola PT, Coppola M. Emergency department evaluation and treatment of pelvic fractures. Emerg Med Clin North Am. 2000;18(1):1–27.. v
25. Burgess AR, Eastridge BJ, Young JW, Ellison TS, Ellison PS Jr, Poka A, et al. Pelvic ring disruptions: effective classification system and treatment protocols. J Trauma. 1990;30(7):848–56.
26. Manson T, O'Toole RV, Whitney A, Duggan B, Sciadini M, Nascone J. Young-Burgess classification of pelvic ring fractures: does it predict mortality, transfusion requirements, and non-orthopaedic injuries? J Orthop Trauma. 2010;24(10):603–9.
27. Salvino CK, Esposito TJ, Smith D, Dries D, Marshall W, Flisak M, et al. Routine pelvic x-ray studies in awake blunt trauma patients: a sensible policy? J Trauma. 1992;33(3):413–6.

28. McCormick JP, Morgan SJ, Smith WR. Clinical effectiveness of the physical examination in diagnosis of posterior pelvic ring injuries. J Orthop Trauma. 2003;17(4):257–61.
29. Killeen KL, DeMeo JH. CT detection of serious internal and skeletal injuries in patients with pelvic fractures. Acad Radiol. 1999;6(4):224–8.
30. Herzog C, Ahle H, Mack MG, Maier B, Schwarz W, Zangos S, et al. Traumatic injuries of the pelvis and thoracic and lumbar spine: does thin-slice multidetector-row CT increase diagnostic accuracy? Eur Radiol. 2004;14(10):1751–60.
31. Cerva DS Jr, Mirvis SE, Shanmuganathan K, Kelly IM, Pais SO. Detection of bleeding in patients with major pelvic fractures: value of contrast-enhanced CT. AJR Am J Roentgenol. 1996;166(1):131–5.
32. Pereira SJ, O'Brien DP, Luchette FA, Choe KA, Lim E, Davis K Jr, et al. Dynamic helical computed tomography scan accurately detects hemorrhage in patients with pelvic fracture. Surgery. 2000;128(4):678–85.
33. Shanmuganathan K, Mirvis SE, Sover ER. Value of contrast-enhanced CT in detecting active hemorrhage in patients with blunt abdominal or pelvic trauma. AJR Am J Roentgenol. 1993;161(1):65–9.
34. Dreizin D, Bodanapally U, Boscak A, Tirada N, Issa G, Nascone JW, et al. CT prediction model for major arterial injury after blunt pelvic ring disruption. Radiology. 2018;287(3):1061–9.
35. Eastridge BJ, Starr A, Minei JP, O'Keefe GE, Scalea TM. The importance of fracture pattern in guiding therapeutic decision-making in patients with hemorrhagic shock and pelvic ring disruptions. J Trauma. 2002;53(3):446–50; discussion 50–1.
36. Heini PF, Witt J, Ganz R. The pelvic C-clamp for the emergency treatment of unstable pelvic ring injuries. A report on clinical experience of 30 cases. Injury. 1996;27 Suppl 1:S-A38–45.
37. Stahel PF, Mauffrey C, Smith WR, McKean J, Hao J, Burlew CC, et al. External fixation for acute pelvic ring injuries: decision making and technical options. J Trauma Acute Care Surg. 2013;75(5):882–7.
38. Bottlang M, Simpson T, Sigg J, Krieg JC, Madey SM, Long WB. Noninvasive reduction of open-book pelvic fractures by circumferential compression. J Orthop Trauma. 2002;16(6):367–73.
39. Krieg JC, Mohr M, Ellis TJ, Simpson TS, Madey SM, Bottlang M. Emergent stabilization of pelvic ring injuries by con-

trolled circumferential compression: a clinical trial. J Trauma. 2005;59(3):659–64.
40. Croce MA, Magnotti LJ, Savage SA, Wood GW 2nd, Fabian TC. Emergent pelvic fixation in patients with exsanguinating pelvic fractures. J Am Coll Surg. 2007;204(5):935–9; discussion 40–2.
41. Jowett AJ, Bowyer GW. Pressure characteristics of pelvic binders. Injury. 2007;38(1):118–21.
42. Miller PR, Moore PS, Mansell E, Meredith JW, Chang MC. External fixation or arteriogram in bleeding pelvic fracture: initial therapy guided by markers of arterial hemorrhage. J Trauma. 2003;54(3):437–43.
43. Cook RE, Keating JF, Gillespie I. The role of angiography in the management of haemorrhage from major fractures of the pelvis. J Bone Joint Surg. 2002;84(2):178–82.
44. Velmahos GC, Toutouzas KG, Vassiliu P, Sarkisyan G, Chan LS, Hanks SH, et al. A prospective study on the safety and efficacy of angiographic embolization for pelvic and visceral injuries. J Trauma. 2002;53(2):303–8.. discussion 8
45. Fang JF, Shih LY, Wong YC, Lin BC, Hsu YP. Repeat transcatheter arterial embolization for the management of pelvic arterial hemorrhage. J Trauma. 2009;66(2):429–35.
46. Ryan MF, Hamilton PA, Chu P, Hanaghan J. Active extravasation of arterial contrast agent on post-traumatic abdominal computed tomography. Can Assoc Radiol. 2004;55(3):160–9.
47. Brasel KJ, Pham K, Yang H, Christensen R, Weigelt JA. Significance of contrast extravasation in patients with pelvic fracture. J Trauma. 2007;62(5):1149–52.
48. Magnussen RA, Tressler MA, Obremskey WT, Kregor PJ. Predicting blood loss in isolated pelvic and acetabular high-energy trauma. J Orthop Trauma. 2007;21(9):603–7.
49. Diamond IR, Hamilton PA, Garber AB, Tien HC, Chughtai T, Rizoli SB, et al. Extravasation of intravenous computed tomography scan contrast in blunt abdominal and pelvic trauma. J Trauma. 2009;66(4):1102–7.
50. Blackmore CC, Jurkovich GJ, Linnau KF, Cummings P, Hoffer EK, Rivara FP. Assessment of volume of hemorrhage and outcome from pelvic fracture. Arch Surg. 2003;138(5):504–8; discussion 8–9.
51. Evers BM, Cryer HM, Miller FB. Pelvic fracture hemorrhage. Priorities in management. Arch Surg. 1989;124(4):422–4.

52. Tesoriero RB, Bruns BR, Narayan M, Dubose J, Guliani SS, Brenner ML, et al. Angiographic embolization for hemorrhage following pelvic fracture: is it "time" for a paradigm shift? J Trauma Acute Care Surg. 2017;82(1):18–26.
53. Pohlmann TGA, Bosch U, Tscherne H. The technique of packing for control of hemorrhage in complex pelvis fractures. Tech Orthop. 1995;9:267–70.
54. Smith WR, Moore EE, Osborn P, Agudelo JF, Morgan SJ, Parekh AA, et al. Retroperitoneal packing as a resuscitation technique for hemodynamically unstable patients with pelvic fractures: report of two representative cases and a description of technique. J Trauma. 2005;59(6):1510–4.
55. Cothren CC, Osborn PM, Moore EE, Morgan SJ, Johnson JL, Smith WR. Preperitoneal pelvic packing for hemodynamically unstable pelvic fractures: a paradigm shift. J Trauma. 2007;62(4):834–9; discussion 9–42.
56. Burlew CC, Moore EE, Smith WR, Johnson JL, Biffl WL, Barnett CC, et al. Preperitoneal pelvic packing/external fixation with secondary angioembolization: optimal care for life-threatening hemorrhage from unstable pelvic fractures. J Am Coll Surg. 2011;212(4):628–35.. discussion 35-7
57. Brenner ML, Moore LJ, Dubose JJ, Tyson GH, McNutt MK, Albarado RP, et al. A clinical series of resuscitative endovascular balloon occlusion of the aorta for hemorrhage control and resuscitation. J Trauma Acute Care Surg. 2013;75(3):506–11.
58. DuBose JJ, Group AS. RE: the AAST AORTA registry and data on REBOA. J Trauma Acute Care Surg. 2016;81(3):617–8.
59. Wasicek PJ, Teeter WA, Yang S, Hu P, Hoehn MR, Stein DM, et al. Life over limb: lower extremity ischemia in the setting of resuscitative endovascular balloon occlusion of the aorta (REBOA). Am Surg. 2018;84(6):971–7.
60. Brenner M, Inaba K, Aiolfi A, DuBose J, Fabian T, Bee T, et al. Resuscitative endovascular balloon occlusion of the aorta and resuscitative thoracotomy in select patients with hemorrhagic shock: early results from the American Association for the Surgery of Trauma's aortic occlusion in resuscitation for trauma and acute care surgery registry. J Am Coll Surg. 2018;226(5):730–40.
61. Biffl WL, Smith WR, Moore EE, Gonzalez RJ, Morgan SJ, Hennessey T, et al. Evolution of a multidisciplinary clinical pathway for the management of unstable patients with pelvic fractures. Ann Surg. 2001;233(6):843–50.

62. Dente CJ, Feliciano DV, Rozycki GS, Wyrzykowski AD, Nicholas JM, Salomone JP, et al. The outcome of open pelvic fractures in the modern era. Am J Surg. 2005;190(6):830–5.
63. Grotz MR, Allami MK, Harwood P, Pape HC, Krettek C, Giannoudis PV. Open pelvic fractures: epidemiology, current concepts of management and outcome. Injury. 2005;36(1):1–13.
64. Jones AL, Powell JN, Kellam JF, McCormack RG, Dust W, Wimmer P. Open pelvic fractures. A multicenter retrospective analysis. Orthop Clin North Am. 1997;28(3):345–50.
65. Pell M, Flynn WJ Jr, Seibel RW. Is colostomy always necessary in the treatment of open pelvic fractures? J Trauma. 1998;45(2):371–3.
66. Britt LD, Zolfaghari D, Kennedy E, Pagel KJ, Minghini A. Incidence and prophylaxis of deep vein thrombosis in a high risk trauma population. Am J Surg. 1996;172(1):13–4.
67. Rogers FB, Shackford SR, Wilson J, Ricci MA, Morris CS. Prophylactic vena cava filter insertion in severely injured trauma patients: indications and preliminary results. J Trauma. 1993;35(4):637–41; discussion 41–2.
68. Jehan F, O'Keeffe T, Khan M, Chi A, Tang A, Kulvatunyou N, et al. Early thromboprophylaxis with low-molecular-weight heparin is safe in patients with pelvic fracture managed nonoperatively. J Surg Res. 2017;219:360–5.
69. Kimbrell BJ, Velmahos GC, Chan LS, Demetriades D. Angiographic embolization for pelvic fractures in older patients. Arch Surg. 2004;139(7):728–32; discussion 32–3.
70. Ramirez JI, Velmahos GC, Best CR, Chan LS, Demetriades D. Male sexual function after bilateral internal iliac artery embolization for pelvic fracture. J Trauma. 2004;56(4):734–9; discussion 9–41.
71. Travis T, Monsky WL, London J, Danielson M, Brock J, Wegelin J, et al. Evaluation of short-term and long-term complications after emergent internal iliac artery embolization in patients with pelvic trauma. J Vasc Interv Radiol JVIR. 2008;19(6):840–7.
72. McCarthy ML, MacKenzie EJ, Bosse MJ, Copeland CE, Hash CS, Burgess AR. Functional status following orthopedic trauma in young women. J Trauma. 1995;39(5):828–36; discussion 36–7.
73. Copeland CE, Bosse MJ, McCarthy ML, MacKenzie EJ, Guzinski GM, Hash CS, et al. Effect of trauma and pelvic fracture on female genitourinary, sexual, and reproductive function. J Orthop Trauma. 1997;11(2):73–81.

74. Patterson FP, Morton KS. The cause of death in fractures of the pelvis: with a note on treatment by ligation of the hypogastric (internal iliac) artery. J Trauma. 1973;13(10):849–56.
75. Tornetta P 3rd, Matta JM. Outcome of operatively treated unstable posterior pelvic ring disruptions. Clin Orthop Relat Res. 1996;(329):186–93.
76. McLaren AC, Rorabeck CH, Halpenny J. Long-term pain and disability in relation to residual deformity after displaced pelvic ring fractures. Can J Surg J. 1990;33(6):492–4.
77. Pohlemann T, Gansslen A, Schellwald O, Culemann U, Tscherne H. Outcome after pelvic ring injuries. Injury. 1996;27(Suppl 2):B31–8.
78. Hauschild O, Strohm PC, Culemann U, Pohlemann T, Suedkamp NP, Koestler W, et al. Mortality in patients with pelvic fractures: results from the German pelvic injury register. J Trauma. 2008;64(2):449–55.
79. Arroyo W, Nelson KJ, Belmont PJ Jr, Bader JO, Schoenfeld AJ. Pelvic trauma: what are the predictors of mortality and cardiac, venous thrombo-embolic and infectious complications following injury? Injury. 2013 Dec;44(12):1745–9.
80. Schulman JE, O'Toole RV, Castillo RC, Manson T, Sciadini MF, Whitney A, et al. Pelvic ring fractures are an independent risk factor for death after blunt trauma. J Trauma. 2010;68(4):930–4.
81. Demetriades D, Karaiskakis M, Toutouzas K, Alo K, Velmahos G, Chan L. Pelvic fractures: epidemiology and predictors of associated abdominal injuries and outcomes. J Am Coll Surg. 2002;195(1):1–10.

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