

Vascular Trauma: Epidemiology and Natural History

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INTRODUCTION

Vascular injury is a major cause of death and disability in society, with trauma being the principle etiology. Despite the establishment of mature trauma systems to improve delivery of prompt and effective treatment, as well as innumerable technological advances with improved clinical outcomes and expanded application of data collection systems, the burden of traumatic injuries continues to increase in society. In the US, trauma is the number one cause of death for those between the ages of 1 and 46 and is the third highest overall cause of mortality across all age groups.¹ As of 2014, traumatic injury by any mechanism was the number one cause of years of

potential life lost at 31.7% and accounted for nearly 200,000 deaths overall.² Certainly mortality from trauma is complex, but hemorrhage is overwhelmingly the agent of death in most instances. From a military perspective, vascular injury with associated hemorrhage remains the leading cause of potentially preventable wartime mortality, despite rapid transport, point-of-injury hemorrhage control and early operative intervention.^{3,4} Long-term disability from limb loss, chronic pain and post-traumatic stress disorder impact many victims, adding to the societal burden incurred from vascular trauma.⁵

While our ability to diagnose and treat vascular injuries has improved substantially over time, our overall impact on this incidence and prevalence has been lacking. This dismal reality

underscores the complex nature of traumatic injury and its myriad contributing factors. An epidemiologic approach to the characterization and management of traumatic vascular injury has not been extensively explored. According to the World Health Organization, epidemiology is defined as the study of the distribution and determinants of health-related states or events in specified populations, and the application of this study to the control of health problems. While traditionally applied to the study of communicable and non-communicable disease processes, epidemiologic principles can be applied to the study of any process that impacts the health of a population including vascular injury.

An epidemiologic approach to vascular injury has several potential benefits, particularly as it relates to civilian trauma. At a national level, elucidating the various socioeconomic, geopolitical and cultural factors influencing trauma can serve as a basis for broad public health initiatives, policy change and other efforts aimed at mitigating the impact of trauma on at-risk populations. Application of epidemiologic principles to vascular injury aims to classify and risk-stratify various injury patterns through improved data gathering and scoring methodologies, enabling identification of local and regional differences more accurately. Through recognition of regional differences, these metrics can serve as a vehicle for change at the institution, trauma system and regional legislative levels.

The purpose of this chapter is to provide a framework for understanding the numerous factors that influence vascular injury in society from an epidemiologic standpoint. Providing a historical context, with perspective from the transformative influence of military trauma over the past century, will serve to highlight changes over time affecting the broader delivery of trauma care. Additionally, contemporary efforts employed to characterize the natural history of vascular injury and track outcomes, particularly specific injury patterns, will be explored.

Evolution Through Military Conflict

The incidence, nature and management of vascular injury are often described within the framework of major periods of conflict or war. The sheer volume and severity of trauma associated with combat provides unique opportunity to study vascular injury patterns, create novel surgical techniques, and implement change aimed at mitigation of risk. However, the changing nature of warfare over time inherently influences the distribution of vascular injury patterns, requiring ongoing evolution of our techniques and practices.

A classic paper by DeBakey et al. characterizes the vascular injury burden during World War I and II, noting an incidence of vascular injury during both wars of approximately 1%. Of the 2471 arterial injuries documented during World War II, only 81 underwent attempted repair, with an amputation rate of 36%. In 40 patients, attempt was made to repair larger arterial disruptions using vein interposition grafts, unfortunately the amputation rate was 50%. There was a routine posture towards ligation of vessels in most patients. Although this practice was understood to be less than ideal, it was deemed necessary given resource constraints of this era.⁶

The Korean War experience beginning in 1952 resulted in an increased incidence of vascular injuries at approximately 2% of all casualties relative to previous conflicts. This may reflect improved evacuation methods during this conflict, increasing the number of patients with vascular injuries surviving to definitive surgical therapy. There was a paradigm shift in the management of vascular injuries witnessed during this era, with 88% undergoing an attempt at primary repair/anastomosis (60%) or interposition graft (27%).^{7,8} While early reconstruction strategies employed cadaveric femoral artery as an interposition graft conduit,⁹ this was largely abandoned after some time due to an increased failure and amputation rate (33%) compared with vein grafts (12%).⁸ Several more comprehensive reports on successful arterial repair performed during the Korean War followed, including classic papers from Colonel Carl Hughes, demonstrating an impressive reduction in the amputation rate among 269 repairs, from 40% in World War II to 13% during the Korean War.¹⁰⁻¹² While improvements in casualty evacuation during the Korean War were achieved, significant time delays and resuscitation requirements remained hindrances to successful vascular injury management.¹⁰ Despite these improvements, the burden of vascular injury and its effect on mortality remained obscure.

The Vietnam War ushered in several significant advances in vascular injury management, particularly the creation of the Vietnam Vascular Registry. This provided the ability to characterize injury patterns and outcomes following intervention. Rich et al.¹³ published landmark reports of the first 500 and 1000 patients, describing increased repair rates (93%) with improved patency and lower amputation rates. In total, this registry captures nearly 10,000 vascular injuries in over 7500 injured warfighters.⁷ There was also an increased emphasis on routine repair of venous injury as a vital component of limb salvage strategies, albeit controversial.^{14,15}

For decades to follow, the vascular injury experience of these previous wars was thought to be unapproachable with regard to the duration of conflict and the number of injuries. With more than 10,000 deaths (US military and civilian contractors) and over 60,000 combat-related injuries in more than a decade, the Global War on Terror (GWOT) has proved to be a formidable and sustained military campaign. During this conflict, modern advances have allowed a concerted effort to reduce deaths from potentially survivable vascular injuries and to improve the quality of functional extremity salvage (i.e., saving life *and* limb).¹⁶

The development of the Joint Theater Trauma System has improved surgical care and reduced mortality by implementing clinical practice guidelines and performing outcomes research emerging from the Joint Theater Trauma Registry (JTTR). The GWOT Vascular Initiative is a comprehensive registry designed to study patterns of vessel injury and methods of vascular repair and to provide more complete long-term analysis of patient outcomes.

At the beginning of the GWOT, the Department of Defense implemented a testing, training, and fielding program for battlefield tourniquets.¹⁷⁻¹⁹ Although widespread tourniquet use began with trepidation, the forward deployment of surgical capabilities has provided for limited tourniquet duration, thus increasing the effectiveness of tourniquets and reducing



Figure 179.1 Optimal hemorrhage control for military trauma is achieved with early placement of pre-hospital tourniquets for extremity wounds and timely resuscitation with blood products.

the rate of associated complications. The effectiveness of early tourniquet application observed in Iraq and Afghanistan has led to doctrinal changes that have produced a surge of patients with vascular injuries who, in the past, would not have reached a field hospital alive (see Fig. 179.1).^{20,21} The application of tourniquets for extremity vascular injury is now routine and has been formalized in the Tactical Combat Casualty Care (TCCC) guidelines.²² Of interest, the 12% incidence of vascular injuries based on data from the JTTR is the highest ever reported during wartime. Certainly the widespread application of tourniquets contributes to this statistic, however improved detailed data collection as well as a shift in injury mechanism from conventional weaponry to the improvised explosive devices (IEDs) likely impacts this as well.²³ Additionally, the widespread use of body armor in conjunction with increased exposure to IEDs has led to an epidemiologic shift in injury patterns, with a proportionally higher rate of extremity vascular injury (53%) and decreased rate of major truncal vascular injury (15%). Not surprisingly, there has been an increased incidence of vascular ligation (35%), underscoring the devastating nature of many extremity wounds in modern warfare, negating attempts at limb salvage.²³

Other modern advances include application of surgical adjuncts, such as temporary vascular shunts to facilitate delayed definitive vascular repair and the routine performance of fasciotomies to minimize the incidence of undiagnosed compartment syndrome.^{24–29} Progress in the management of increasing complex vascular injury patterns and the associated management of complex soft tissue wounds through closed negative pressure wound therapy has been impressive.³⁰ Last, the application of endovascular technologies for the diagnosis and treatment of certain types of vascular, pelvic, and solid organ war-related injuries has become more widespread and generally accepted as a mainstay of surgical care.^{31–33}

Despite these tremendous achievements, it is challenging to draw definitive comparisons between modern and historical conflicts with regard to killed in action, died of wounds and other outcomes measures given the heterogeneity of not only the technological advancements but also due to the changing tactical

environment and subsequent injury patterns. Nonetheless, many warfighters continue to succumb to potentially survivable injuries on the battlefield. In several contemporary studies evaluating combat-related mortalities from Operation Enduring Freedom and Operation Iraqi Freedom, approximately 15%–25% of casualties were deemed to have potentially survivable injury patterns at the time of autopsy, of which 80%–87% are attributed to hemorrhage.^{3,16,34,35} This not only underscores the lethality of vascular injury and hemorrhage, but alludes to the fact that viable strategies for more timely intervention remain elusive.

Data Repositories and Registries

One of the most important factors in understanding the epidemiology and patterns of vascular injuries is the collection of thorough and reliable data, which was traditionally done by single centers performing chart review analyses or by analysis of wartime experience with limited follow-up data. Computerized databases, automated data collection, and establishment of trauma systems with dedicated registrars have facilitated the collection and analysis of large, multicenter trauma data. An early example and model for future development was the establishment of the Vietnam Vascular Registry, with subsequent landmark reports on the epidemiology and outcomes of a wide variety of vascular injuries.¹³ The recognized importance of the prospective collection of data from wartime experiences led to the establishment of the Joint Theater Trauma System and the Joint Theater Trauma Registry, which collects critical information from combat operations in Iraq and Afghanistan. Additional examples include the establishment of the National Trauma Data Bank (NTDB) by the American College of Surgeons, which is the largest trauma registry ever assembled. The NTDB data is subject to extensive auditing to ensure data quality and accuracy and enables uniform data collection across institutions. The NTDB does possess certain limitations that influence its ability to accurately characterize the epidemiology of traumatic injury, particularly certain vascular injury patterns. One limitation is that non-admitted patients are uniformly not captured in this database. This is important in that patients who die without transport to a treatment facility are not captured. Thus, major vascular injuries where exsanguination results in rapid demise are likely underrepresented, creating an inherent selection bias for more stable, less severely injured patients, confounding analyses aimed at characterizing certain injury patterns, particularly truncal injury patterns. The NTDB is also not a true population-based dataset, as the data reflects only participating institutions. Hence, the experience of non-participating facilities, likely smaller community and rural hospitals, will not be reflected in the NTDB data set. Also, the database does not fully characterize the outcomes following vascular injury and is limited to in-hospital outcomes and complications.

The PROspective Vascular Injury Treatment (PROOVIT) registry, supported by the American Association for the Surgery of Trauma, has been established to address key limitations of the NTDB. The initial publication of this data set included 542 patients from 14 centers from March 2013 to February 2014, in whom 484 sustained arterial injuries and 71 experienced isolated venous injuries. Additional strengths of this database include the

capture of key elements of pre-hospital care such as the application of tourniquets, seen in 20% of patients within this series.³⁶

Vascular Injury Classification and Scoring

The ideal vascular injury classification and scoring system has yet to be developed. Standard injury scoring systems such as the Injury Severity Score (ISS), Revised Trauma Score (RTS) and Trauma and Injury Severity Score (TRISS) fail to accurately and reliably capture the impact of major vascular injury on morbidity and mortality.^{37,38} Beyond this, the myriad factors that influence mortality independent of the injury itself cannot be effectively captured using ISS, specifically. In a study by Markov, mortality rates for patients with ISS >15 following civilian vs. military-related vascular trauma with similar injury patterns was 40% and 10%, respectively, further underscoring the limitations of ISS to characterize vascular injury outcomes.

The American Association for the Surgery of Trauma Organ Injury Scaling (AAST-OIS) system is the most widely used grading system for traumatic injuries and is well validated for predicting outcomes and need for intervention in solid organ injuries. This grading system is organized primarily around the exact identity of the vessel rather than the severity of the vascular injury or the degree of hemorrhage or ischemia and thus provides little additional information about treatment or outcomes. For major extremity fractures and soft tissue injury, several scoring systems have been developed to characterize the “mangled extremity,” including the Mangled Extremity Severity Score (MESS), the Mangled Extremity Syndrome Index, the Predictive Salvage Index, and the Limb Salvage Index.³⁹ Although they contain different components, the presence of vascular injury and limb ischemia is a universal key variable. In addition to providing an objective classification system for epidemiologic purposes, these scores have been studied for their ability to predict the need for extremity amputation in both civilian and military settings.^{39,40} Although they have been correlated with the need for amputation, prospective trials have found that they lack adequate predictive ability to be used for individual patients and did not correlate with limb salvage when arterial reconstruction was performed.⁴⁰ This is likely due to improved techniques in the management, including endovascular therapies, tourniquets and shunting, resulting in improved limb salvage rates even in the face of severe injury scores.⁴¹

Contemporary Civilian Vascular Injury

Epidemiologic Patterns and Trends

The exact incidence and distribution of vascular injury mechanisms may vary widely between centers, depending on the setting (urban versus rural) and population served. According to a recent NTDB analysis of all trauma admissions, the incidence of vascular trauma is 1.6% for adults and 0.6% for pediatric patients, which is significantly lower than the 6% to 12% incidence among combat casualties.^{23,28} However, the reported incidence of major vascular injury is likely to be an underestimate as mentioned previously and does not include patients who die at the trauma scene. One analysis of autopsy reports of 552 trauma deaths identified

penetrating injury as the reported mechanism in 42% of patients, with approximately 80% dying from hemorrhage and isolated vascular injury in 10%. Of the patients who had vital signs in the field, 26% were identified as having major vessel disruption.⁴² The majority of prehospital or immediate deaths from vessel disruption were due to aortic injury (55%), and most (78%) were associated with death within 15 minutes of injury.

While blunt trauma accounts for approximately half of trauma deaths, vascular injury due to blunt trauma is relatively uncommon, with death from blunt vascular injury being relatively rare.⁴² The most lethal blunt vascular injury pattern involves laceration or transection of the thoracic aorta, accounting for approximately 10% of all trauma-related pre-hospital deaths.⁴³ While many patient will succumb to this injury pattern immediately, those who survive to undergo definitive care fare well, with an injury-specific mortality rate of 12%.⁴⁴

Major vascular disruption or bleeding continues to be associated with approximately 25% of early trauma deaths. The average age of all trauma patients and those with vascular injury is steadily increasing, with a 10-year increase in the average age of trauma patients between 1996 and 2004.⁴⁵ The classically described “young and healthy” trauma patient is being replaced with more elderly patients who have a higher incidence of pre-existing vascular disease that may increase the risk for vessel injury and alter treatment options.

Factors Influencing the Natural History of Vascular Injury

Most vascular injury patterns mandate prompt treatment to optimize outcomes. That treatment may come in the form of definitive treatment or temporizing measures aimed at facilitating a safe delay in care. The most significant improvements in vascular injury management over the past century have addressed these key factors. The development of a robust network of emergency medical services that can provide rapid transport and basic or advanced life support measures are an essential component of modern trauma care. One study from the UK demonstrated a decreased ratio of pre-hospital to in-hospital death over an 8-year period (1996–2004) from 1.5 to 0.75, underscoring improved efficiency of pre-hospital EMS.⁴⁵ Gunst et al. reported consistent findings where decreased transport times and advances in pre-hospital care resulted in more critically injured patients surviving until arrival at a hospital, particularly those with non-survivable injury patterns. This led to a temporal shift in early in-hospital trauma deaths towards an earlier time point. When these early deaths were scrutinized, 76% were deemed non-survivable.⁴⁶ While these statistics do not support the claim that decreased transport times lead to increased survival, it does at a minimum confirm the ability of pre-hospital EMS to provide for potential salvage of critically injured patients that would have otherwise died in the pre-hospital setting.

For severely injured patients, damage control techniques including abbreviated surgery, application of endovascular techniques, balanced resuscitation, and temporary intravascular shunts (both arterial and venous) have been associated with major reductions in both mortality and limb loss.^{47–49} These innovations have altered the classic trimodal distribution of trauma-related mortality

towards more of a bimodal distribution, with a significantly decreased incidence of late deaths beyond 24 hours.⁴⁶

Arguably the most significant development in modern vascular surgery is the emergence of endovascular techniques for managing vascular disease, and these techniques are now being extended to traumatic injuries. Although initially applied to injuries for which open repair was highly morbid (thoracic aorta) or provided limited exposure (distal carotid, subclavian artery), endovascular techniques for temporizing acute control of hemorrhage or as definitive management can be applied to a wide array of arterial and venous injury patterns.^{33,50–52} A national analysis demonstrated a 27-fold increase in the use of endovascular therapy, and this was associated with a decrease in morbidity, hospital stay, and mortality.⁴⁹ Endovascular techniques have even now been extended to the combat setting as well, with high technical success rates (90% to 100%).⁵³

Impact of Age and Gender on Vascular Injury

One of the largest determinants of risk for traumatic injury is age, with the overwhelming burden impacting young adults. According the 2015 NTDB report, traumatic injury increases progressively beginning at age 14, peaking at approximately age 21, and progressively declining thereafter. The majority of overall traumatic injuries are due to motorized vehicle collisions between ages 14 and 49, with falls as the dominant injury mechanism in all other age groups. While penetrating injury due to firearms accounts for less than one third of injuries in the peak age demographic, the case fatality rate is nearly four times greater at approximately 15%. Greater than 70% of traumatic injuries in the peak age group occur in males, with up to 90% for penetrating extremity wounds.⁵⁴

The overall incidence of vascular injury in the pediatric population (age <16) is 0.6%, compared to 1.6% in adults based on a recent NTDB analysis.⁵⁵ Injuries to vessels in the thorax were approximately seven times lower in children as compared to adults. Penetrating injury patterns were less common in children (41.8%) as compared to adults (51.2%).⁵⁵ Unfortunately, firearm and stab wounds accounted for the majority of penetrating injuries in children, with a mortality rate of 20% for those injured by a gun.⁵⁵ The upper extremity was the most common location of pediatric vascular injury (37.9%), with most injuries involving either the radial or ulnar arteries (22%). The upper extremity is also the most common site of penetrating vascular injury for both adults and children. With regard to blunt injury patterns, the upper extremity (33%) and chest (33%) are the most common sites of blunt vascular injury in children and adults, respectively.⁵⁵ The amputation rates for both children and adults were similar.

The incidence of vascular injury in the geriatric population (mean age 70 years) is similar to that of the pediatric population, at 0.7%. There is still a gender bias towards injury in males at 60%, albeit less pronounced than in the younger adult population. Blunt mechanisms (MVC and falls) account for the overwhelming majority of injuries (84%), with blunt thoracic aortic injury being most frequent (39%). Mortality rates were higher in the geriatric population compared to younger adults (44% vs. 22%), with injury to the thorax carrying a high mortality rate of

66%. For penetrating injuries, the forearm vessels are most commonly injured (31.5%). Amputation rates following extremity vascular injury were similar in both populations.⁵⁶

Ethnicity and Socioeconomic Factors

Disparity in healthcare outcomes has been extensively documented for multiple disease processes, with traumatic injury being no exception. Beyond outcomes, there is also a disparate distribution in the burden of traumatic injury within society, based on race and socioeconomic status. The effect of race and insurance status has been explored in multiple studies, with most indicating pronounced effects.^{54,57–61} However, there appears to be a lack of uniformity across various injury patterns.

In a large review of the NTDB from the years 2001–2005, Haider et al. demonstrated marked differences in the incidence of penetrating trauma as a function of both race and insurance status. The overall incidence of penetrating injury in this sample was 8.7%. For insured patients the incidence by race was as follows: white = 3.1%, black = 18.3%, and Hispanic = 11.7%. For the uninsured patients, a similar distribution was seen: white = 7.4%, black 31%, and Hispanic = 21.8%. Unadjusted mortality rates were statistically different for white, black and Hispanic patients at 5.7%, 8.2%, and 9.1%, respectively. When stratified by insurance status these findings persisted, with mortality rates for uninsured white, black, and Hispanic patients at 7.9%, 11.4%, and 11.3%, respectively. These findings continued to persist despite adjustment for injury severity and demographic variables.⁵⁷

Several authors point to heterogeneity within the trauma population as a potential confounding factor in explaining outcomes based on race and insurance status. To control for this, Crandall et al. analyzed the outcomes in a more homogeneous populations involving patients who sustained isolated lower extremity vascular injury. Mortality rates were higher for uninsured patients (31.7% vs. 21.5%), however when stratified by mechanism only penetrating injury remained significant. In a risk-adjusted model, race remained a predictor of increased mortality following penetrating lower extremity vascular injury in Black Americans (OR 1.45, $P = 0.03$).⁵⁴ In another study evaluating outcomes of pedestrians struck by motor vehicles, Maybury demonstrated that Black Americans (OR 1.22) and Hispanics (OR 1.33) had a significantly higher mortality rate than Whites utilizing a multivariate risk-adjusted model, with uninsured status also an independent predictor of increased mortality.⁶¹ Explanation for these outcome disparities prove challenging and may include variables that are not appropriately captured within the NTDB. Nonetheless, these findings highlight the need for further research and outreach programs aimed at minimizing the impact of traumatic injury in at-risk populations.

Mechanisms of Injury, Ballistics, and Biomechanics

The clinical presentation, pattern of associated injuries, need for intervention, and outcomes after traumatic vascular injury will be highly dependent on the mechanism of injury and the specific characteristics of that mechanism. For blunt injury,

this mainly involves the velocity or forces of impact, the use of restraints or protective devices, and the primary anatomic areas that sustain the brunt of the kinetic forces. In addition, patients sustaining blunt trauma may suffer penetrating-type vascular injury from impalements, glass or sharp debris, and puncture by bone fragments. The most common blunt mechanism associated with major vascular injury is motor vehicle collision, resulting in injury from stretching and shearing forces or from vascular avulsion (usually due to rapid deceleration).⁶² Several additional etiologic factors have been proposed, including the generation of a sudden spike in intravascular pressure as well as forward inertial deceleration of blood impacting the anterior vessel wall (“water-hammer” effect).^{62,63}

For penetrating trauma, the wounds can be primarily classified as due to stab/puncture or from missiles/projectiles. Stab and puncture wounds result in direct vascular injury without significant transmission of kinetic energy or damage to surrounding tissues. Missiles may injure vascular structures by direct laceration or by transfer of energy due to proximity, with an impact kinetic energy (iKE) related to the mass (M) and velocity (V) of the projectile ($iKE = \frac{1}{2} MV^2$). Terms such as “high velocity” are often used but are frequently poorly defined or misunderstood. There are multiple projectile characteristics, such as shape, deformity, fragmentation, pitch, and yaw, that are as important, if not more so, than velocity.⁶⁴ Additional injury to surrounding tissue has been attributed to both the “sonic wave” and the stretching of tissue due to the pressure wave (“temporary cavity”).⁶⁴ While histologic evidence of vessel injury can be seen in grossly normal-appearing vessels, this does not correlate with outcome following repair, thus excessive debridement of grossly normal vessel or surrounding tissues is not required.¹¹ Finally, mention should be made of shotgun injuries, which are classified as low velocity, yet result in diffuse devascularization from multiple pellets that can be greater than high-velocity gunshots.^{65,66} Although less common, they are associated with vascular injury in up to 25% of wounds, creating vessel micropunctures that are difficult to diagnose.^{65,67}

Blast injury, such as that seen with improvised explosive devices in the wars in Iraq and Afghanistan, has unique wounding

patterns and mechanisms. These injuries include a combination of blunt force, penetrating fragment injuries, and thermal injury. Blast injuries in modern combat series now outnumber standard gunshot wounds and account for 73% of vascular injuries among wounded soldiers. Blast effects are classified as primary (direct blast pressure), secondary (penetrating fragments), tertiary (collision with objects or vehicles), and quaternary (thermal injury). The relative distribution of each varies by type of explosive, enclosed versus outdoors, and presence of protective equipment. Most vascular and other injuries are due to secondary and tertiary blast effect (81%). Blast-induced extremity vascular injury with coexisting fracture is associated with a 50% amputation rate with attempts at limb salvage and a 77% amputation rate overall.⁶⁸ Blast injuries may also occur with civilian incidents such as terrorist bombings, and vascular injuries have been identified in up to 10% of victims.

EPIDEMIOLOGY AND NATURAL HISTORY OF SPECIFIC VASCULAR INJURIES

The epidemiology of major traumatic vascular injuries will vary widely by injury location, mechanism, and specific blood vessel involved, as will the natural history and outcomes following injury and repair. As many of these injuries are rapidly fatal or are immediately managed by either ligation or repair, the evolution and long-term outcome of untreated vascular injury is inherently poorly understood. Clinical data on their natural history mainly come from the experience with the management of nonoperative or asymptomatic injuries.^{69,70} Table 179.1 shows a summary of the known natural history and potential complications that may develop after either blunt or penetrating major vascular trauma. Occult injuries such as vasospasm, small intimal flap or pseudoaneurysm, and minor arteriovenous fistulae may resolve without operation in up to 86% of cases, and only 2% overall require operative intervention.^{66,70} More significant injuries will frequently progress, representing a threat to life and/or limb.

TABLE 179.1 Natural History of Various Types of Vascular Injuries and Potential Future Complications

Injury Type	Natural History*	Complications
Penetrating or Iatrogenic		
Laceration	Pseudoaneurysm or thrombosis	Ischemia, rupture, embolization
Contusion	Stenosis, thrombosis	Ischemia, embolization
Arteriovenous fistula	Increase in size and flow	“Steal” syndromes, heart failure
Blunt		
Intimal dissection or thrombosis (<25%)	Spontaneous resolution	None unless progression
Intimal dissection or thrombosis (>25%)	Pseudoaneurysm, thrombosis	Rupture, ischemia
Pseudoaneurysm	Increase in size	Rupture, embolization
Thrombosis	Occlusion, recanalization	Ischemia, stenosis
Arteriovenous fistula	Increase in size and flow	“Steal” syndromes, pseudoaneurysm
Transection	Thrombosis, pseudoaneurysm	Ischemia, compartment syndrome

*Natural history without intervention and assuming initial hemorrhage control.

Neck

The incidence of vascular injury in the neck is 20% with penetrating and 1% with blunt injury in civilian series, with the majority of injuries involving the carotid artery.^{71,72} Yet there is marked difference in the epidemiology and treatment paradigms of blunt versus penetrating carotid artery injuries. The overall incidence of diagnosed carotid artery injury is 0.2%, with 41% due to penetrating trauma versus 59% from blunt mechanisms.⁷³ Interestingly, a series examining missed arterial injuries identified the carotid as the most common site (39%).⁷⁴ Based on a review of the NTDB, the majority of carotid artery injuries are located in the internal carotid (37%) and less commonly the common carotid (20%), with mixed injuries (10%) least common. However, unspecified injuries accounted for 33% of documented carotid injuries.⁷³

The majority of blunt carotid artery injuries are due to hyperflexion/extension and are commonly located in the distal internal carotid artery.^{72,73} Bilateral carotid artery injury or associated vertebral artery injury is present in 20% to 50% of cases. This injury pattern is usually associated with motor vehicle crashes and multisystem injury (79%), including a 50% incidence of severe brain injury.^{73,75} The increased utilization of CT angiography for trauma has resulted in a surge of blunt carotid/vertebral artery injury diagnoses.⁷¹ The primary predictors of outcome are the grade of injury, the degree of associated injuries, and the development of neurologic symptoms. Mortality of up to 50% has been reported, but the injury-associated mortality is 15% and the incidence of severe neurologic deficits among survivors is 16%.^{72,75,76}

Vertebral artery injury is relatively uncommon, accounting for 1%–7% of penetrating neck injuries and 1% of blunt injuries.^{71,77,78} The majority of vertebral artery injuries (78%) have an associated cervical spine fracture, and the incidence of vertebral artery injury is up to 40% when a cervical spine fracture is present.^{71,75} The incidence of posterior circulation stroke with blunt vertebral artery injury has been reported to be as high as 24%, with an 8% attributed mortality rate. However, aggressive screening with early diagnosis by CT angiography was associated with a reduction in the stroke risk from 14% to 0%.⁷⁹

Truncal Injury

Noncompressible Truncal Hemorrhage

Injuries to named vessels of the thorax or abdomen from either blunt or penetrating mechanisms are highly morbid, with most patients dying before they reach a hospital. A study of 1203 battlefield casualties in Iraq and Afghanistan found no survivors among those with injury to a named abdominal or thoracic vessel.⁸⁰ Even in pediatric patients who are typically more tolerant to injury and hemorrhage, mortality approaches 100% for thoracic vascular injuries with hemodynamic instability at presentation. However, exsanguinating hemorrhage may not necessarily involve injury to named vascular structures. NCTH irrespective of injury pattern carries a high associated mortality, accounting for 60% to 70% of potentially preventable mortality in the civilian setting.⁸¹ From experience during Operation

Enduring and Iraqi Freedom, 24% of deaths were deemed survivable, of which 42% were due to NCTH, accounting for 10% of all combat-related deaths.^{16,34} Unlike peripheral or junctional injuries, these are not typically amenable to direct compression or inflow control in the prehospital or emergency department phase and thus have been a focus of great interest and innovation. Critical concepts or interventions for patients with NCTH are: (1) minimizing delays in transfer from the emergency department to the operating room; (2) permissive hypotension until vascular control is obtained; (3) balanced or “hemostatic” resuscitation with early use of plasma; (4) procoagulant adjuncts such as tranexamic acid; and (5) use of damage control surgery techniques and intravascular shunts when indicated.^{47,82}

Novel strategies aimed at the pre-hospital phase of care have been developed, including Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA).^{33,50} This intervention is minimally invasive, potentially obviating the need for open aortic cross-clamping.^{83,84} Experience with this technique is increasing, particularly utilizing partial occlusion strategies and purpose-built devices meant to increase the duration of intervention and secondary ischemic injury, thus allowing for increased pre-hospital utilization of this lifesaving therapy.^{85,86}

Thorax

Thoracic aorta

While most cardiothoracic vascular injury (90%) is due to penetrating trauma, only 28% of penetrating aortic injuries involve the thoracic portion.⁸⁷ In a landmark study of 5760 vascular injuries, Mattox et al. found that 14% involved the thoracic aorta, of which 86% were due to penetrating mechanisms.⁸⁸ Thoracic aortic injuries can be classified into several groups, including intrapericardial/ascending (8%–27%), aortic arch (8%–18%), and descending thoracic (47%–75%).⁸⁹

Blunt thoracic aortic injury (BTAI) is highly lethal and is seen in one-third of patients who die from blunt mechanisms.⁹⁰ For patients who die following blunt trauma, 80% with a concomitant BTAI will die at the scene vs. 60% without BTAI, further underscoring its lethality.⁹⁰ BTAI involving the descending thoracic aorta is the most frequently diagnosed and encountered blunt thoracic vascular injury, with greater than 60% of isolated injuries occurring at the isthmus/ligamentum arteriosum (Fig. 179.2).^{90,91} Autopsy studies have also suggested that approximately 15%–20% of blunt trauma victims will sustain multiple aortic injuries.⁹⁰ The most common mechanisms of injury are motor vehicle accidents, falls from a height with impact on a hard surface, and impact from a large mass. Additionally, blunt aortic injury is identified in up to 30% of airplane crash victims.⁹² Injuries are classified as follows: intimal tear (grade I), large intimal flap or intramural hematoma (grade II), pseudoaneurysm (grade III), and rupture (grade IV).^{93,94} There is interest in revisiting this grading scheme to more accurately reflect the natural history of these injuries, as well as to inform the need for and timing of repair. This includes not only changing the naming and number of grades, but also incorporates new morphologic features that

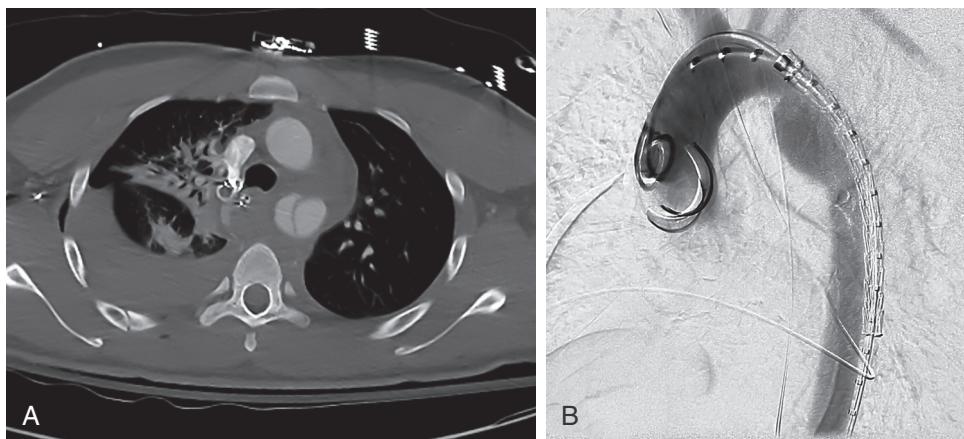


Figure 179.2 Blunt Aortic Injury. (A) CT angiogram demonstrating type III blunt aortic injury with pseudoaneurysm. (B) Conventional angiography demonstrating an aortic pseudoaneurysm immediately prior to TEVAR.

more strongly correlate with the severity of injury, namely external aortic contour. This new three tier system is as follows: Minimal (no external contour abnormality or intimal tear <10 mm) – no repair; Moderate (external contour abnormality or intimal tear >10 mm) – semi-elective repair; and Severe (active extravasation) – immediate repair.⁹⁵

Blunt trauma to the ascending aorta occurs from sudden displacement of the intact sternum or displacement of the fractured or dislocated sternum onto the anterior surface of the ascending aorta. Several injuries can occur: a subadventitial hematoma, a subintimal hematoma or tear, or a complete tear with pseudoaneurysm formation or free rupture.⁴³ Another injury pattern is a torsion tear caused by sudden rotation of the heart apex posteriorly with a spiral tear starting above the aortic valve. Most (98%) of these die at the scene from pericardial tamponade or exsanguination and thus rarely survive to hospital diagnosis or repair.^{43,96}

In contemporary practice, blunt descending thoracic aortic injuries are managed primarily with thoracic endovascular aortic repair (TEVAR). A landmark multicenter study by Fabian et al. (AAST-1) in 1997 characterized the traditional approach to blunt thoracic aortic injuries, with 35% undergoing clamp and sew repair and 65% having repair with cardiopulmonary bypass.⁷² Mortality was 31%, and the paraplegia rate was 9%. In a recent multi-center experience, TEVAR was utilized in 76% of cases, with overall and aortic-related mortality rates of 8.6% and 2.5%, respectively, and paraplegia in a mere 0.5%.⁹⁷ Additionally, the literature supports delayed repair of grade II–III injuries when significant associated injuries exist.^{44,97} Endovascular repair has been extended to patients with acute penetrating thoracic injury, pseudoaneurysm or arteriovenous fistula.⁵³ Finally, nonoperative management of select patients with low-risk injuries (grade I/II) or with prohibitive surgical risk is becoming more widely accepted.^{44,97,98}

Great vessels

Injury to the great vessels (innominate, brachiocephalic, proximal subclavian) is frequently fatal (90% occur within 30 minutes) due to hemorrhage or associated injury to the airway, heart, or aortic arch.⁸⁹ Additionally, mortality rates of 70% to 80% have been reported for isolated subclavian artery injuries.^{52,99,100} The incidence of great vessel injury is approximately

5% with gunshot wounds and 2% with stab wounds; it is extremely uncommon (<1%) in blunt trauma.^{56,89} In a large autopsy study of trauma deaths, 12.7% of deaths were due to injury to the aorta or great vessels, and no patient with a penetrating wound survived to hospital arrival compared with 5% of blunt injuries.⁴³ Many patients will have multiple-vessel injury (65%), and the majority (77%) present with clear signs of hemorrhage or a large and expanding hematoma.¹⁰¹ Operative approaches are dependent on individual surgeon and institution preferences but typically involve a median sternotomy or left thoracotomy. High patency rates (>90%) have been described with both vein and prosthetic repairs, with no significant incidence of prosthetic graft infection reported.^{99,101} Ligation of the subclavian arteries may be performed in select cases and is frequently well tolerated because of collateral circulation.¹⁰⁰ TEVAR for blunt thoracic aortic injury involving routine coverage of the left subclavian artery supports this, with only a 4% to 5% incidence of left arm ischemic symptoms. Long-term deficits among survivors are attributed to central nervous or brachial plexus injuries more than to the vascular injury.⁹⁹

Axillosubclavian vessels

Although the proximal subclavian vessels are primarily mediastinal structures, the distal subclavian and proximal axillary vessels cross anatomic zones including the chest, neck, and extremity. Proximal subclavian injuries adhere to the principles described before for great vessels, but axillosubclavian injuries often have a different presentation and complicating factors. Among modern combat casualties, the incidence of upper extremity vascular injury is 1.7%, and 23% of these involve the axillary or subclavian vessels.¹⁰² The majority of civilian injuries are due to penetrating trauma (55%–75%) and are most common in shotgun wounds (18%), followed by gunshots (10%) and stab wounds (9%).⁶⁶ Concomitant vein injury is present in one third of patients.¹⁰³ Blunt axillosubclavian injuries are rare and are due to either shear forces or clavicle fracture with nearly universal presence of nerve injury or fracture.¹⁰⁴ Limb function is often severely impaired, but this is primarily due to the high incidence (40%–100%) of associated brachial plexus injury.¹⁰³ Amputation is ultimately required in 3% to 15% and mortality is up to 25%, with worse outcomes among blunt injuries patterns.¹⁰⁴

There is increasing interest and success in endovascular management of these injuries, particularly for pseudoaneurysms and arteriovenous fistulae. A series of 57 patients with long-term follow-up demonstrated stenosis in 20% and occlusion in 12%; all were managed percutaneously with no limb ischemia or loss.⁵² Additionally, these techniques have been shown to decrease hospital length of stay and have a high technical success approaching 100%.⁵² These techniques may also be extended to so-called unstable patients and in fact may achieve faster hemorrhage control in systems that are prepared for emergent endovascular intervention.⁵³

Abdomen

In a large series of 5760 vascular injuries, 34% involved the abdominal vasculature and 24% involved multiple vessels.⁸⁸ The majority of abdominal vascular injuries are due to penetrating mechanisms (89%), with 36% of patients having multiple vessels injured.^{105,106} Hemodynamic instability or complete arrest requiring emergency department thoracotomy has been reported in up to 15% of patients.¹⁰⁷ There appears to be a relatively equal distribution of the type of vessel injured, with vein injuries in 53% versus arterial injuries in 47%.¹⁰⁷ Approximately half (53%) of patients will have two or more abdominal vessels injured, with mortality increasing from 45% with one vessel to 60% and 73% with two and three vessels injured.¹⁰⁷ The most commonly injured major vessels are the inferior vena cava (IVC) and branches (52%), the aorta and branches (35%), the superior mesenteric artery (SMA) and branches (45%), and the portal or hepatic venous system (22%).^{105,106} The reported high mortality rates of 30% to 60% highlight the need for early diagnosis and intervention, with 70% to 89% of deaths attributed to bleeding.¹⁰⁵ Hypotension will be present in 25% of patients and is one of the strongest independent predictors of death (odds ratio, 4.5 to 18).¹⁰⁵

The major abdominal vasculature is located in the retroperitoneum, and four-zone anatomic classification for retroperitoneal hematomas based on the vasculature is commonly used: zone 1 (central) contains the aorta and vena cava, zone 2 (lateral) contains the renal vessels, zone 3 (pelvic) contains the iliac vessels, and zone 4 (hepatoportal) contains the portal vein and retrohepatic vena cava. Zone 3 predominates in blunt trauma (70%), whereas zone 2 is the most commonly seen in penetrating injury (50%).¹⁰⁸ Retroperitoneal hematomas are associated with morbidity of up to 60% and mortality from 13% to 40%.¹⁰⁸

Abdominal aorta (Zone 1)

Blunt injury to the abdominal aorta is rarely seen, and most of these injuries probably result in prehospital death from bleeding or severe associated injuries. Injury to the abdominal aorta represents only 5% of aortic injuries and was identified in 9% of all fatal aortic injuries from vehicular trauma.¹⁰⁹ More than half (56%) of fatalities involve transection of the aorta and have been associated with the deceleration and hyperflexion with lap belt restraints.¹⁰⁹ Only 25% of patients will have abdominal wall bruising or injury, and the most common early signs are arterial insufficiency (81%), acute abdomen (55%),

and weakness or paralysis (47%).¹⁰⁹ These injuries are virtually always in the infrarenal aorta, most commonly at the junction with the inferior mesenteric artery or the iliac bifurcation.^{62,109} An intimal flap with distal dissection is the most common pathologic lesion (57%), followed by thrombosis (40%) and pseudoaneurysm (15%).¹⁰⁹ Mortality is 20% to 40% and increases to 83% with advanced age (>65 years).^{107,109}

Penetrating abdominal aortic injury is most frequently due to gunshot wounds (78%) and carries an overall survival of less than 50%.¹⁰⁷ Approximately 30% will present in extremis with no measurable blood pressure, and up to 21% may require emergency department thoracotomy.⁸⁷ The location of injury is most commonly infrarenal (45%), followed by suprarenal (37%) and subdiaphragmatic (18%).¹¹⁰ Injury to the suprarenal aorta is particularly difficult and highly morbid, although Mattox et al. reported survival in 10 of 28 (36%) patients.¹¹¹ Overall mortality rates of 33% to 81% have been reported.^{87,111} Although modern trauma management principles have been found to have no impact on the high mortality of these injuries, a study from San Diego found that mortality decreased from 78% to 40% when the patient was brought directly to the operating room (bypassed the emergency department) for resuscitation.^{87,110}

Inferior vena cava (Zone 1)

Although the IVC is part of the lower pressure venous system, injuries cause rapid exsanguination unless they are contained or immediately controlled. Excluding mesenteric vessels, the IVC is the most commonly injured major abdominal vessel, representing 6.7% of all penetrating vascular injuries and 3.4% of blunt injuries.⁵⁶ The majority of injuries (>90%) are due to penetrating mechanisms and are located in the infrarenal cava (85%). Approximately half of patients will present with hypotension.¹¹² Associated visceral injuries are present in 90% of patients and include injury to the duodenum (31%), liver (29%), and pancreas (26%).¹¹²

Mortality with isolated IVC injury is reportedly as high as 70% and increases to 78% in combination with another venous injury.¹⁰⁷ However, survival of up to 96% is reported among patients with an isolated infrarenal IVC injury and no hemodynamic instability.¹¹² Data from combat injuries of the IVC in Vietnam found that the majority underwent surgical repair (72%) versus ligation (19%), with a 23% mortality rate. IVC injury in the wars in Iraq and Afghanistan accounts for 1.4% of all vascular injuries, with approximately 50% undergoing ligation versus shunting with delayed repair or immediate repair in the remainder.²³

Celiac and mesenteric vessels (Zone 1)

Traumatic injuries to the celiac trunk, SMA, or superior mesenteric vein are extremely uncommon and represent only 0.01% to 0.1% of all vascular injuries.¹¹³ Similarly low incidences have been reported from recent large series of battlefield injuries (0.19% celiac, 0.83% SMA).²³ A much higher incidence of 6.3% was demonstrated in a large NTDB series, but this probably includes branch vessels and more distal mesenteric injuries.⁵⁶ The majority of visceral artery injuries are due to

penetrating trauma, representing 90% to 95% of celiac artery and 52% to 77% of SMA injuries.^{113,114} Associated injuries are the rule, with a mean of 4.2 injuries per patient and with 35% of patients having a coexisting superior mesenteric vein injury.¹¹³ The clinical presentation is typically either hemodynamic instability or peritonitis, with a mean estimated blood loss of 8.5 liters.¹¹⁴ Mortality is 20% to 40%, with most deaths due to intraoperative or early postoperative bleeding (71%) and later postoperative complications (29%).^{105,113} Mortality is directly correlated to both the injury severity and the number of coexisting vessel injuries.^{87,105}

Renal vessels (Zone 2)

Although renal injuries are common with both blunt and penetrating trauma, true renovascular injury is much less so. For blunt mechanisms (incidence of 0.08%), a distinction should be made between renal parenchymal injury involving the segmental vessels or renal avulsion (AAST-OIS grade 4 to 5 injuries) and primary renovascular injury that is usually due to stretching and subsequent dissection or thrombosis. The injury mechanism is equally distributed between blunt (49%) and penetrating (51%).¹¹⁵ Associated abdominal injuries are present in 77%. Nephrectomy was required in 51% of penetrating injury, with a mortality of 30%.¹¹⁶

Iliac vessels (Zone 3)

Iliac vascular injury is uncommon, with an overall incidence of less than 1% and representing 14% of civilian penetrating arterial injuries and 2% of combat vascular injuries.^{117,118} Iliac artery and vein injuries are a particular challenge because of difficulties of exposure and obtaining distal control in the deep pelvis. Injuries to the main vessels are predominantly due to gunshot wounds (86%–95%), and 56% will have multiple iliac injuries.¹¹⁸ The majority (68%) of injuries that survive to hospital admission are to the common or external iliac vein, and 32% involve the iliac arteries. Mortality with iliac vessel injury is 28% to 49% and appears to be decreased with the use of damage control techniques including intravascular shunts.⁴⁷ Injuries to branch vessels or the internal iliac vessels are most commonly due to blunt trauma with an associated pelvic fracture. Injury to the main iliac or femoral vessels with pelvic fracture is exceedingly rare (0 of 429 patients in one series). Several series have identified control of pelvic hemorrhage as the most frequent preventable cause of death from bleeding, and surgical intervention with packing or iliac artery ligation may be required.

Hepatoportal vessels (Zone 4)

Injuries to the hepatoportal system are highly morbid because of common factors including massive hemorrhage, associated pancreaticoduodenal injury, difficult surgical exposure, and surgeon inexperience with these uncommon injuries. Injury to the hepatic veins or retrohepatic vena cava occurred in 9% and portal vein in 5% of abdominal vascular injuries, and 94% are due to penetrating trauma.¹⁰⁵ Among patients with injury to the IVC, there is a 19% incidence of combined injury with the portal vein.¹¹² Portal vein injuries are 92% fatal in association

with other portal triad injuries and 100% fatal with hepatic artery injury. Injuries of the retrohepatic vena cava have an associated mortality of 70% to 100% even with various shunt or exclusion techniques.¹¹² Injury of the retrohepatic vena cava (88%) and injury of the portal vein (69%) represent two of the top three causes of death from abdominal vascular trauma.¹⁰⁵

Extremity

Extremity trauma is extremely common in all settings from both blunt and penetrating mechanisms, accounting for approximately 1% to 2% of all civilian trauma.⁵⁶ Vascular injury is more common in the lower extremities (66%) versus upper (34%).²⁸ In contrast, approximately 50% of modern combat injuries involve the extremities, with 75% due to blast mechanisms.¹¹⁹ Although blunt mechanisms account for the large majority of overall extremity injuries, penetrating trauma mechanisms cause most (60%–80%) extremity vascular injuries.¹²⁰

Pre-hospital management of extremity vascular injury in military conflict involves widespread use of tourniquets and hemostatic dressings. Tourniquet use has resulted in a staggering reduction in pre-hospital death from 23.3 deaths per year to 3.5 deaths per year, for an overall reduction in potentially survivable death of 85%.⁷⁶ Currently, extremity vascular injury accounts for 13.5% of potentially survivable vascular injuries in modern conflicts.⁷⁶

Isolated civilian extremity trauma with vascular injury carries a 10% risk of mortality or limb loss, with higher rates for penetrating and more proximal vessel injuries.¹²¹ Blunt extremity vascular injury is associated with an 18% amputation rate and a 10% mortality rate.¹²⁰ With adjustment for other variables, lower extremity vascular injury is independently associated with an increased amputation rate (odds ratio, 4.3) and higher mortality (odds ratio, 2.2).¹²²

Temporary intravascular shunt use is increasingly applied in the context of polytrauma, with contemporary series reporting its use in 9% of civilian vascular injuries and up to 24% of combat extremity vascular injuries (Fig. 179.3).^{28,123} In the largest published combat series of temporary intravascular shunts, patency varied widely from 86% for proximal injuries to 12% for distal vessels.²⁸ Despite the varied patency rates, early limb salvage in this population was 88% for distal shunts and 95% for proximal shunt placement ($P = \text{NS}$) and is comparable to reported limb salvage rates of 75% to 100% in civilian series.¹²³

Upper Extremity

A recent analysis of the NTDB found that the upper extremities were the site of 27% of all civilian vascular injuries. Approximately 25% of blunt extremity vascular injuries are in the arm, with 50% located in the brachial artery (Fig. 179.4). Among patients who required upper extremity amputation, the most commonly injured vessel was the brachial artery (12% of patients).⁵⁶ Compartment syndrome is present or may develop in 21% and is associated with multiple vessel injuries and open fractures. Mangled extremity predictive scoring systems have

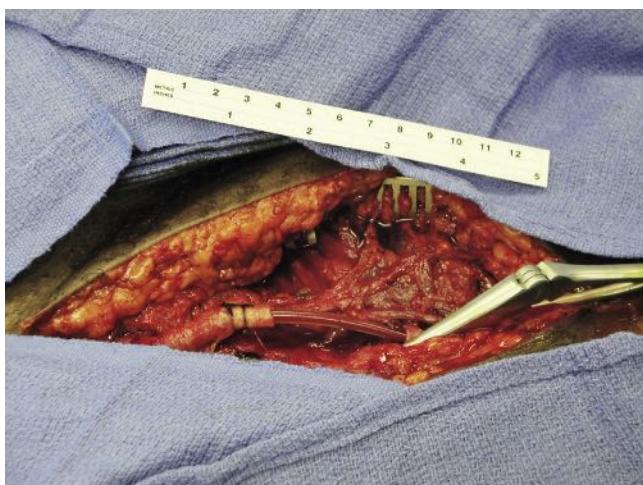


Figure 179.3 Temporary Vascular Shunt. Injury to the superficial femoral artery from a gunshot wound. Due to concomitant injury, temporary shunting was performed using an Argyle shunt to maintain limb viability while more life-threatening injuries were managed.

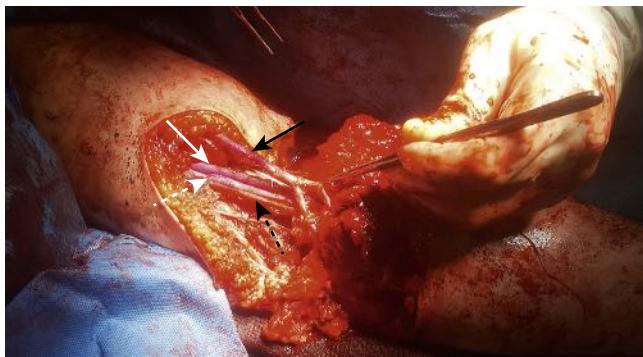


Figure 179.4 Blunt Upper Extremity Injury. All-terrain vehicle accident resulting in near-complete avulsion of the left arm just proximal to the elbow joint, with transection of all named vascular structures. *Solid black arrow* highlights the reconstructed cephalic vein. *Solid white arrow* highlights the reconstructed brachial vein. *Dashed white arrow* highlights the reconstructed brachial artery. *Dashed black arrow* highlights the median nerve. All reconstructions were performed with saphenous interposition grafts.

been found to be less predictive of outcomes for upper extremity injury, and limb salvage has been demonstrated in 90% of patients. Blunt injury is associated with a significantly higher amputation rate (20%) and mortality compared with penetrating mechanisms.¹²⁰

Contemporary military experience reveals an overall incidence of upper extremity arterial injury in 30%–34% of patients, with 11% proximal (brachial artery) and 19% distal (radial or ulnar).^{23,102} Associated injuries to the ulnar or median nerve are present in up to 50%, and functional outcomes are mainly related to the nerve injuries rather than the vascular trauma.¹²⁴ Associated injuries to the ulnar or median nerve are present in up to 50%, and functional outcomes are mainly related to the associated nerve injuries rather than to the vascular trauma.¹²⁴

Most upper extremity vascular injuries in both civilian and military trauma are to the forearm vessels, including the radial and ulnar arteries.^{56,102,122} Penetrating trauma is the cause of up

to 81% of injuries, but stab wounds are more common than gunshot injury (opposite of lower extremity).¹²² Blunt radial or ulnar injury is almost always seen with a coexisting fracture of the forearm or elbow dislocation (95%) and is associated with higher mortality and limb loss.¹²⁰ Injury to forearm nerves, bone or soft tissue is the major determinant of functional outcomes.¹²⁴

Lower Extremity

Femoral and popliteal vessels

The majority of penetrating extremity wounds are to the lower extremity (71%) and have a 10% incidence of vascular injury versus 1% for blunt trauma (Fig. 179.5).^{66,69,120} Most femoral artery injuries are due to penetrating trauma, but blunt trauma is now the predominant cause (61%) of popliteal injury.¹²⁵ Knee dislocations are particularly high risk, with up to a 30% incidence of popliteal artery injury.¹²⁶ Femoral artery injuries represent 14% of all lower extremity vascular injuries from blunt trauma and 42% from penetrating.^{56,120} An NTDB analysis of 651 patients demonstrated injury to the common femoral artery (CFA) in 18%, the superficial femoral artery (SFA) in 28%, and the popliteal in 36%.¹²¹ The most feared complications include hemorrhage for more proximal injuries (CFA and SFA) and limb loss from ischemia for popliteal injuries. Up to 46% of CFA and SFA injuries have an associated injury to the femoral vein, and 40% to 50% of popliteal injuries are combined.¹²⁵ Between 7% and 25% of patients will have an associated nerve injury, and long-term function is related to the nerve and soft tissue injuries more than to the vascular trauma. An analysis of almost 30,000 patients with vascular injury from the NTDB found that among patients who required lower extremity amputation, the popliteal artery was the most commonly injured vessel (28% of patients).⁵⁶ The majority of deaths due to extremity hemorrhage in both the civilian and military population are from injuries to the femoral vessels. After adjustment for confounding factors, femoral or popliteal vascular injury is associated with increased mortality (odds ratio, 2.2) and limb loss (odds ratio, 4.3).¹²²

Tibioperoneal vessels

The true incidence of tibioperoneal vessel injury is unknown as the majority are likely to be clinically silent. Tibioperoneal vessels represent the majority (63%) of blunt lower extremity vascular injuries, 10% of penetrating leg trauma, and 44% among combat casualties.^{23,56,120} Among patients with isolated lower extremity trauma with vascular injury, the posterior tibial artery is injured in 13% and the anterior tibial artery is injured in 8.6% (combined injury in 1.1%).¹²¹ Whereas observation or ligation for isolated single-vessel injury is universally well tolerated, up to 50% of multivessel injuries develop symptoms of limb ischemia and are associated with an odds ratio of 5.2 for amputation.^{121,127} Associated injuries include tibial or fibular fractures in 64%, severe soft tissue injury in 32%, and nerve injury in 36%.¹²⁷ Amputation is required in approximately 10% of patients and is twice as frequent with blunt trauma as with penetrating. Overall mortality is less than 5% and is three times less common than with more proximal arterial injuries.¹²⁷

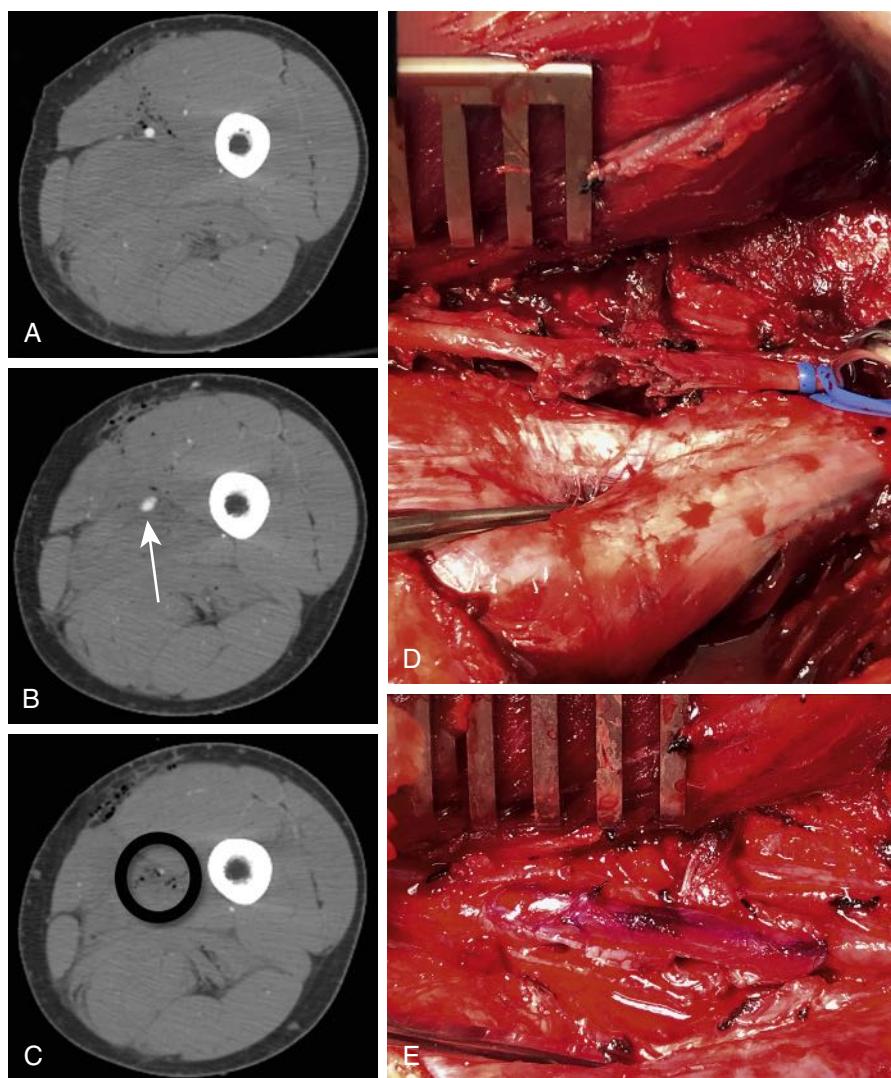


Figure 179.5 Superficial Femoral Artery (SFA) Injury from Gunshot Wound. (A) Computed tomography angiography proximal to the level of injury, demonstrating normal caliber of the SFA and air within the soft tissues. (B) White arrow highlights a pseudoaneurysm of the SFA at the site of injury. (C) Black circle highlights vasospasm of the SFA distal to the site of injury. (D) Operative exploration revealed near-transection of the vessel with thrombus visualized within the lumen. (E) Reconstruction with saphenous vein graft.

SUMMARY

Vascular injury remains a common source of morbidity and mortality in both military and civilian settings. While significant progress is evident, the overall blight of traumatic injury remains a scourge on society. The greatest opportunity to influence outcomes does not stem from improvements in pre-hospital care, innovation in technique and therapeutics, or surgical capabilities, but rather from efforts targeting injury prevention. An epidemiologic approach to trauma and vascular injury serves to identify the complex factors that influence the incidence and prevalence in society. This establishes the foundation for public health and legislative initiatives aimed at mitigating the impact on at-risk populations. Improved data gathering through registries and databases, using standardized metrics and outcomes measures, is important to accurately characterize injury across the spectrum of care. Overall, a more systematic and comprehensive approach is warranted to minimize impact on the individual, society, and our healthcare system as a whole.

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A complete reference list can be found online at www.expertconsult.com.

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Vascular Trauma: Head and Neck

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Cervical vascular injuries are notoriously difficult to evaluate and to manage, mostly secondary to complex anatomy confined to a relatively narrow and layered anatomic space. The initial evaluation of these patients is often obscured by associated injuries to the head, chest, or abdomen. In addition, signs of cerebral ischemia, cranial nerve deficits, or cervical nerve compression may not be present on initial evaluation. The evaluation and appropriate management of these injury patterns have been controversial and continue to evolve. Advances in noninvasive imaging (primarily computed tomography)

have revolutionized the evaluation of stable patients with cervical vascular injuries, aerodigestive injuries, and associated fractures. In addition, endovascular surgery has added another facet to the care of these trauma patients. Injuries to the distal internal carotid artery, proximal common carotid artery, subclavian artery, or vertebral arteries are now amenable to endovascular methods to arrest hemorrhage, to exclude dissections and pseudoaneurysms, or to assist with open repair. This chapter addresses the presentation, evaluation, and treatment of cervical vascular injuries.

CAROTID ARTERIES

Penetrating Injury

After penetrating cervical trauma, cervical blood vessels are the most commonly injured structures in the neck and account for a 7% to 27% stroke rate and a 7% to 50% mortality.¹ In this population, 80% of deaths are stroke related.

Clinical Presentation

The neck has classically been divided into three zones that dictate diagnostic evaluation and treatment² (Fig. 180.1):

- Zone I: below the cricoid cartilage – proximal control obtained in the chest.
- Zone II: between the cricoid cartilage and the angle of the mandible – proximal and distal control obtained in the neck.
- Zone III: above the angle of the mandible – distal control difficult to obtain.

Zone II is the most commonly injured (47%), followed by zone III (19%) and zone I (18%). It is not uncommon for the injury to traverse two zones of the neck.³ In addition to location, the physical examination triages patients on the basis of “hard signs” of vascular injury (mandating exploration) and “soft signs” of vascular injury (observation vs. further diagnostic evaluation). Hard signs include shock, refractory hypotension, pulsatile bleeding, bruit, enlarging hematoma, and loss of pulse with stable or evolving neurologic deficit. Soft signs include history of bleeding at the scene of injury, stable hematoma, nerve injury, proximity of injury track, and unequal upper extremity blood pressure measurements. Ninety-seven percent of patients with hard signs have a vascular injury as opposed to only 3% with soft signs.³

On the basis of mechanism of injury, gunshot wounds are more likely to cause a large neck hematoma and vascular injury (27%) compared with stab wounds (15%).³ Shotgun wounds, blast injuries, and transcervical (crossing midline) gunshot wounds have a higher rate of vascular injury and should be approached with a high index of suspicion. Associated injuries to the tracheobronchial tree, esophagus, and spinal cord are present in 1% to 7% of patients.³ In addition to hard signs of a vascular injury, patients may present with hard signs of a tracheobronchial injury (respiratory distress or air bubbling from the wound), mandating operative exploration. Other soft signs of cervical neck injury include painful swallowing, subcutaneous emphysema, hematemesis, and signs of nerve injury (cranial nerves IX, X, XI, and XII) or brachial plexus injury (axillary, musculocutaneous, radial, median, and ulnar nerves). A focused and detailed clinical evaluation reliably identifies patients with vascular injuries that require treatment. A physical examination with normal findings has a negative predictive value of 90% to 100% for vascular injuries.⁴

Special consideration should be given to patients who present with coma, a dense hemispheric stroke, or documented carotid thrombosis. The treatment of this specific injury pattern has come full circle from revascularization in the 1950s, to routine ligation in the 1970s, followed by revascularization as the

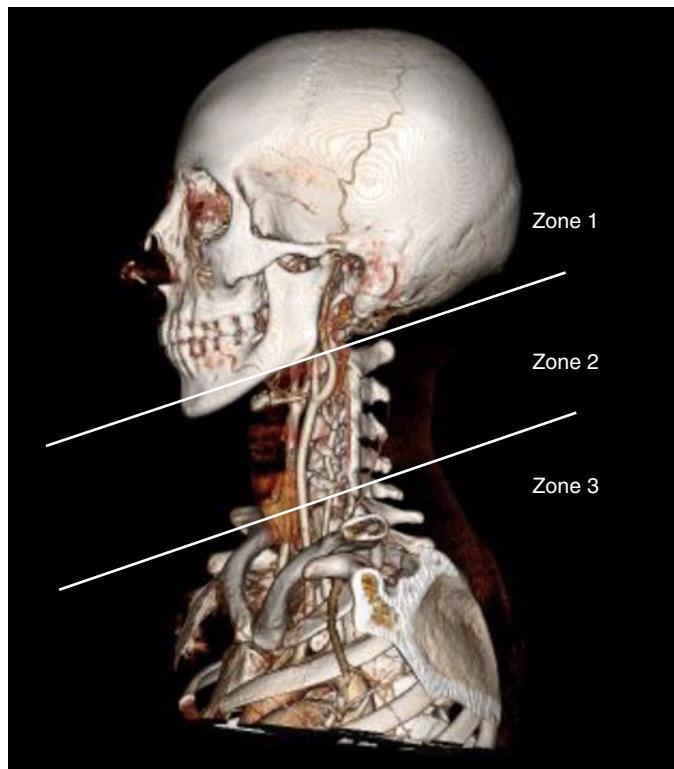


Figure 180.1 Anatomic Zones of the Neck for Penetrating Neck Injuries.

current mainstay of treatment. In the 1970s, authors reported only a few patients with dense hemispheric stroke who developed hemorrhagic stroke after revascularization, leading to the recommendation of internal carotid artery ligation distal to the thrombus.^{5–7} Follow-up studies demonstrated that the extent of anoxic brain injury (not hemorrhagic conversion of the injury), development of reperfusion injury, cerebral edema, and resultant uncal herniation accounted for patients with worsening neurologic status and death.^{8,9} However, to date, there is no preoperative marker other than time (>24 hours from time of injury) that predicts those patients unlikely to benefit from revascularization. Early revascularization has consistently demonstrated improvement or stabilization of neurologic symptoms in patients with dense hemispheric strokes (100%), even in patients who present obtunded (50%).^{1,10}

Diagnostic Evaluation

Patients with hard signs of a vascular injury should proceed to the operative suite. All patients should have plain radiographs of the neck and chest to determine the track of the injury and to diagnose occult hemothoraces or pneumothoraces. There have been several advances in the treatment of penetrating neck injuries, and data are now sufficient to support selective exploration in hemodynamically stable patients who do not have hard signs of a vascular or tracheobronchial injury. Exploration of cervical injuries based on platysma muscle penetration carries an unacceptably high negative exploration rate of 50% to 90%.¹¹

Computed tomography is the modern workhorse for trauma evaluation and should be the initial diagnostic step in

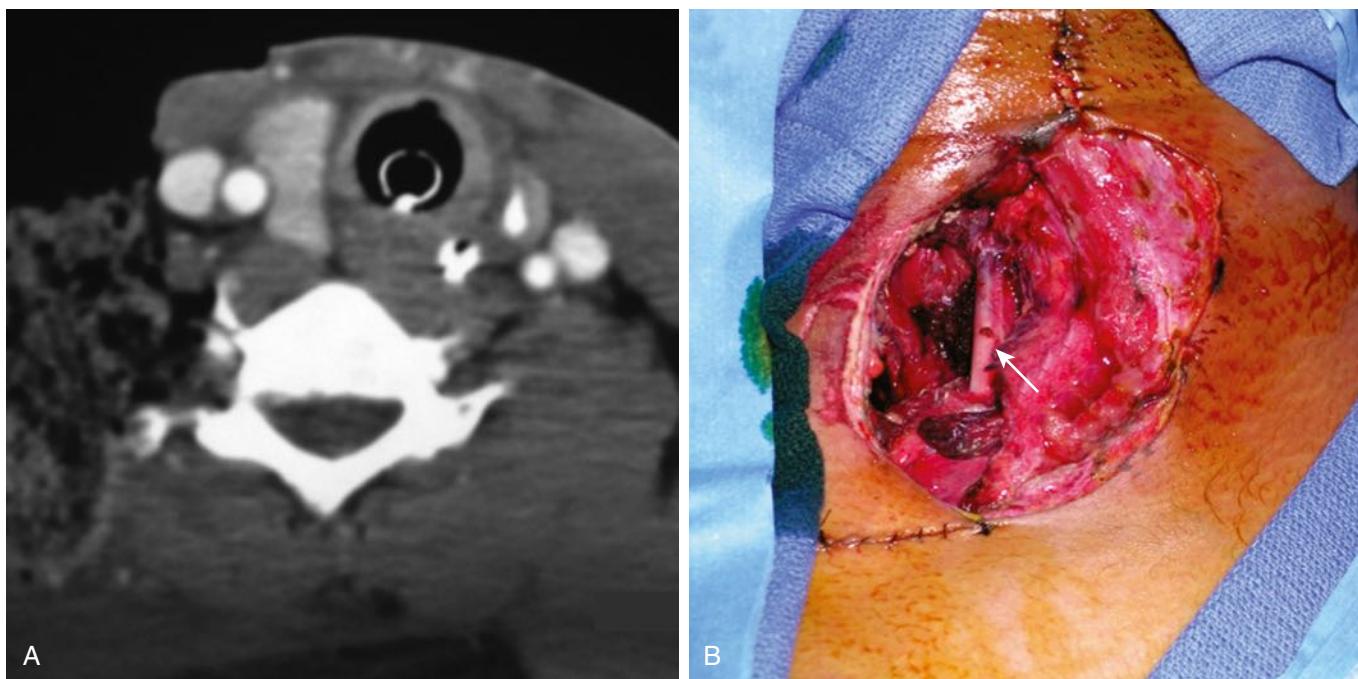


Figure 180.2 This patient sustained a high-velocity gunshot wound to zone I of the neck. On initial evaluation, he did not have hard signs of a vascular injury. (A) CTA demonstrates no injury to the internal jugular vein or common carotid artery. In addition, there is no injury to the aerodigestive tract. (B) The patient's wound was debrided in the operative suite; the arrow marks the cords of the brachial plexus.

evaluating patients with penetrating neck injuries who do not have hard signs of vascular or aerodigestive injury. Contrasted axial imaging with reformatting software allows exact determination of the injury track, vascular injuries, proximity to the esophagus and trachea, spinal fractures and cord involvement, and extension into the head or chest (Fig. 180.2). In the setting of penetrating cervical injuries, computed tomographic angiography (CTA) has a 90% sensitivity and 100% specificity for vascular injuries that require treatment.^{12,13} CTA may be limited secondary to missile fragments (especially shotgun injuries) or bone fragments obscuring the cervical vasculature; arteriography should be used for these patients as a confirmatory study. Ultrasonography has been used for penetrating neck trauma, but its utility is limited to zone II neck injuries.¹⁴ In addition, subcutaneous air, fragments, and hematomas make ultrasound less reliable.

Medical Treatment (Nonoperative Management)

Occult injuries (intimal flaps, dissections, and pseudoaneurysms) identified during evaluation for penetrating cervical injury should be managed just as those caused by blunt trauma (detailed later). Isolated intimal flaps are rare in penetrating trauma, and dissections occur in only 2% of cases. Pseudoaneurysms are the most common occult injury identified. Large pseudoaneurysms should be considered for early intervention, whereas small pseudoaneurysms should be treated with antithrombotic therapy and early follow-up imaging. The natural history of these lesions is not known; however, patients should be closely monitored for development of embolic symptoms.

Endovascular Treatment

An endoluminal approach to neck injuries may avoid the morbidity of median sternotomy, a high thoracic incision, or difficult dissection at the base of the skull. Another benefit is that endoluminal therapy can be performed under local anesthesia, allowing the provider direct assessment of the patient's neurologic status. For zone I and zone III injuries, endovascular exclusion of a pseudoaneurysm, partial transection, or arteriovenous fistula remains a viable option based on the location of injury and the patient's clinical status. Self-expanding covered stents can be safely delivered to these locations with limited morbidity.^{15–18} Zone II injuries should be approached with operative repair.

Surgical Treatment

Proximal and distal control in the neck

Obtaining control of the injury in each zone presents unique challenges. All patients should have their proximal thighs (potential vein conduit) and chest (potential proximal control) prepared into the operative field. Zone I injuries that are manifested with hard signs may be approached through a cervical incision, but a median sternotomy or high anterolateral thoracotomy will be required to obtain proximal control. If the patient is in shock, endovascular attempts at proximal control should not delay performing a median sternotomy. Depending on the patient's hemodynamics and the location of injury, proximal control of the great vessels may be performed from a femoral approach in the operative suite with balloon occlusion (a large 33-mm compliant balloon catheter). Alternatively, if

the proximal vessel can be visualized from a cervical approach but not secured with a vascular clamp, a compliant balloon or Fogarty catheter can be passed retrograde for temporary proximal control. Once the vessel is properly exposed, the balloon can be replaced with a vascular clamp.

An overt injury in zone II can be readily approached through a cervical incision and repair performed under direct visualization. The most common vessel injured by penetrating mechanisms is the internal jugular vein followed by the common carotid artery. The operative feasibility, ability to examine the aerodigestive tract, and relatively low risk to exploration in this region favor open exploration over endovascular techniques in emergent situations.

Hemorrhage from a zone III injury can be devastating, and an immediate operative exploration through a cervical incision can be used first to control inflow and to assess the injury pattern. Even after subluxation of the mandible and division of the posterior belly of the digastric muscle, the distal extent of the injury may not be visualized. If the vessel is transected with inadequate length for clamp application, distal control can be obtained by placing a Fogarty balloon (No. 3–4) within the vessel lumen. If the vessel is lacerated, a sheath can be placed in the common carotid artery and a Fogarty catheter can be passed antegrade through the injury to control back-bleeding. Once the Fogarty balloon is inflated, arteriography can be performed through the side arm of the sheath to delineate the injury with respect to the skull base and further guide operative exposure. Once hemorrhage is arrested, the surgeon must decide whether to proceed with operative repair, embolization of the carotid artery, endoluminal stenting, or temporary shunting or to return the patient to the intensive care unit for resuscitation, imaging of the brain, and delayed repair. If a damage control approach is used, the patient should have serial imaging to evaluate evolving cerebral edema, and cerebral perfusion pressures should guide further resuscitative maneuvers.

Surgical repair of cervical vessels

Once the injury has been delineated and controlled, the surgeon must decide whether to ligate, repair, or temporarily shunt the vessel. The internal jugular vein and external carotid artery may be ligated with limited morbidity. Ligation of the internal carotid artery results in a 45% mortality,¹ and it should be reserved only for injuries at the base of the skull that are not amenable to reconstruction. Clean-based lacerations caused by stab wounds may be repaired primarily; however, gunshot wounds, fragmentation wounds, and shotgun injuries typically require reconstruction of the common carotid or internal carotid artery. Shunts should be used in patients who are already at risk of cerebral hypoperfusion secondary to shock and to all injuries of the internal carotid artery. If the distal clamp can be placed below the carotid bulb, the internal carotid artery will receive adequate back-bleeding through the external carotid artery. Heparin (50 units/kg) should be given before clamps are placed.

The greater saphenous vein has good size match with the internal carotid artery and when used as an interposition graft has demonstrated excellent patency and limited infectious risk. The external carotid artery can also be transposed to the

internal carotid artery for injuries in the proximal internal carotid. In addition, superficial femoral artery can be used in the common or internal carotid artery but requires an additional reconstruction in the lower extremity with polytetrafluoroethylene (PTFE).¹⁹ PTFE typically has a better size match than the greater saphenous vein in the common carotid artery, and in this location, there is no difference in patency rates between the two conduits. In the setting of associated aerodigestive injuries, autogenous conduits should be used, the esophageal repair should be drained away from the vascular repair, and a muscle pedicle (cervical strap muscles, omohyoid muscle, digastric muscle, or sternal head of the sternocleidomastoid) should be placed between the two repairs.

After repair of the vascular injury, all patients must be monitored for signs of cerebral edema and intracranial hypertension. If a clinical neurologic examination cannot be performed, direct intracerebral pressure monitoring or serial head imaging should be obtained.

Blunt Cerebrovascular Injuries

The overall incidence of blunt cerebrovascular injury (BCVI) has been universally reported as less than 1% of all trauma admissions for blunt trauma, but this relatively small population of patients has stroke rates ranging from 25% to 58% and mortality rates of 31% to 59%.^{20–22} The variability in incidence of BCVI is 0.19% to 0.67% for unscreened populations compared with 0.6% to 1.07% for screened populations.²⁰

Clinical Presentation

The recognition and treatment of BCVI have dramatically evolved during the past 2 decades. As imaging technology has improved with respect to both image quality and acquisition times, its use has become a fundamental diagnostic tool in blunt trauma evaluation. Paralleling advances in noninvasive imaging, a heightened awareness of BCVI has emerged. Through aggressive screening, these injuries have increasingly been recognized before devastating neurologic ischemia results.

Mechanism of blunt cerebrovascular injury

Three basic mechanisms of injury are encountered: (1) extreme hyperextension and rotation; (2) a direct blow to the vessel; and (3) vessel laceration by adjacent bone fractures.²³ The most common mechanism causing blunt carotid injury is hyperextension of the carotid vessels over the lateral articular processes of C1–3 at the base of the skull, which is typically a result of high-speed automobile crashes. There are also scattered case reports of chiropractic manipulation²⁴ and rapid head turning with exercise causing BCVI.²⁵ A direct blow to the artery typically occurs in the setting of a misplaced seat belt across the neck during a motor vehicle crash or in the setting of hanging. This injury pattern typically occurs in the proximal internal carotid artery as opposed to the distal aspect. Basilar skull fractures involving the petrous or sphenoid portions of the carotid canal can injure the vessel at this location.

Common mechanisms of injury associated with BCVI include motor vehicle crash (41%–70%), direct cervical blow

TABLE 180.1

Screening Criteria for Blunt Cerebrovascular Injury

	Denver Criteria [†]	Memphis Criteria	Modified Criteria (odds ratio)
Signs and Symptoms	Arterial hemorrhage or expanding hematoma	Neurologic examination findings not explained by brain imaging	GCS score <6 (1.98)
	Cervical bruit	Horner syndrome	
	Neurologic examination findings inconsistent with head CT findings	Neck soft tissue injury (seat belt sign, hanging, or hematoma)	
	Stroke on follow-up head CT		
	Focal neurologic deficit		
Risk Factors	Le Fort II or III fracture pattern	Le Fort II or III fracture pattern	Le Fort II or III fracture pattern (3.7)
	Basilar skull fracture with involvement of the carotid canal	Basilar skull fracture with involvement of the carotid canal	Petrosus fracture (2.64)
	Diffuse axonal injury with GCS score <6	Cervical spine fracture	Diffuse axonal injury (3.09)
	Cervical spine fracture		
	Near-hanging with anoxic brain injury		

CT, computed tomography scan; GCS, Glasgow Coma Scale.

*Modified from Biffl WL, et al. The unrecognized epidemic of blunt carotid arterial injuries: early diagnosis improves neurologic outcome. *Ann Surg*. 1998;228:462–470.

[†]Modified from Miller PR, et al. Prospective screening for blunt cerebrovascular injuries: analysis of diagnostic modalities and outcomes. *Ann Surg*. 2002;236:386–393; discussion 393–395.

[‡]Modified from Biffl WL, et al. Optimizing screening for blunt cerebrovascular injuries. *Am J Surg* 1999;178:517–522.

(10%–20%), automobile versus pedestrian (12%–18%), fall from height (5%–15%), and hanging events (5%).^{20,22} Most common associated injuries at the time of diagnosis include closed head injuries (50%–65%), facial fractures (60%), cervical spine fractures (50%), and thoracic injuries (40%–51%).^{20,22}

Signs and symptoms of BCVI

Case reports, as early as 1967, described BCVI with recognized symptoms of cerebral ischemia, and all patients were symptomatic at the time of diagnosis.²⁶ Carotid injuries typically are manifested with a contralateral sensory or motor deficit, decreased mental status, or neurologic deficits not explained by closed head injury. A carotid-cavernous fistula may be manifested with orbital pain, proptosis, hyperemia, cerebral swelling, or seizure. Depending on whether the vessel is occluded or whether the resultant injury is a nidus for embolic events, the symptoms may be variable. Patients typically have coexisting traumatic brain injuries that may mask signs and symptoms of BCVI.

Patients may present to the trauma center with obvious signs of BCVI; however, many patients are initially asymptomatic and subsequently develop symptoms after a latent period. Several authors have reported times from 1 hour to several weeks after injury before the development of symptoms.^{27–30} Evaluating an unscreened trauma population, Berne et al. found a median time to diagnosis of 12.5 hours for survivors of BCVI and 19.5 hours for nonsurvivors, suggesting a sufficient window of opportunity for diagnosis and treatment.²² Neither admission Glasgow Coma Scale score nor baseline neurologic examination correlates with subsequent development of symptoms attributed to BCVI.

Screening for blunt cerebrovascular injury

Although there is no consensus on the ideal screening protocol, several authors have found associations with signs, symptoms, and risk factors identified on admission. The first and most comprehensive screening protocol was initiated at the Denver Health Medical Center. The criteria are listed in Table 180.1.^{20,31} With this screening protocol, the authors reported an overall BCVI incidence of 0.86%. Exactly 4.8% of all trauma patients were screened on the basis of defined risk criteria, and 18% of screened patients were found to have an injury. Fifty-two percent of these screened patients were asymptomatic. Neurologic morbidity was 16%, and BCVI-associated mortality was 15%.²⁰ Using the Memphis criteria (see Table 180.1), they found an incidence of 1.03%; 3.5% of all blunt trauma patients were screened, and 29% of screened patients were found to have an injury.³² Both screening regimens mandated four-vessel cerebral angiography if the patient met at least one of the screening criteria.

Several authors have evaluated a more restricted screening protocol in an effort to reduce the cost of screening and to limit the number of examinations with normal findings. A cervical seat belt sign has been evaluated in several prospective studies and has not been found to be predictive of BCVI.^{20,33} Biffl et al. performed a multivariate analysis on a prospectively screened population and found four independent risk factors for BCVI listed in Table 180.1.³⁴ Patients with one factor had a 41% risk of BCVI; two factors, 56% to 74%; three factors, 80% to 88%; and all four factors, 93%. However, 20% of patients with BCVI did not have any of the four risk factors. The bulk of the available literature supports an appropriate screening protocol for BCVI, and all major trauma centers should have predetermined screening criteria for BCVI.

TABLE 180.2 Blunt Cerebrovascular Injury Grading Scale

Injury Grade	Angiographic Findings	Stroke Risk (%)	Mortality (%)
I	Luminal irregularity or dissection; intramural hematoma with <25% luminal narrowing	3	11
II	Dissection or intramural hematoma ≥25% of the lumen	11	11
III	Pseudoaneurysm	33	11
IV	Vessel occlusion	44	22
V	Vessel transection	100	100

Modified from Biffl WL, et al: Blunt carotid arterial injuries: implications of a new grading scale. *J Trauma*. 1999;47:845–853.

Borsetto D, et al. Penetrating neck trauma: radiological predictors of vascular injury. *Eur Arch Otorhinolaryngol*. 2019;276(9):2541–2547.

Diagnostic Evaluation

Duplex ultrasound

Duplex scanning has been evaluated in multiple trauma centers for diagnosis of BCVI. In the evaluation of carotid artery stenosis, duplex ultrasound is limited when lesions of less than 60% stenosis are evaluated; likewise, duplex ultrasound will not often identify small intimal tears or nonocclusive dissections. It is often difficult to obtain adequate visualization of the internal carotid artery at the base of the skull, where the majority of these injuries occur. The sensitivity of duplex ultrasound for detection of BCVI ranges from 38% to 86%^{29,35}; therefore, it should not be used as a screening modality.

Digital subtraction angiography

Selective digital subtraction angiography (DSA) has, in the past, been considered the diagnostic “gold standard” for screening patients with suspected BCVI. The Denver group proposed an angiographic grading system for BCVI (Table 180.2), which has become the standard for reporting BCVI.³⁶ Most important, the grading scale held prognostic value for patients’ future risk of subsequent stroke.

There are several limitations of DSA that make it a difficult diagnostic tool. First and foremost, it is an invasive procedure with technical limitations and a complication profile that carries a risk of stroke (<1.0%).²⁰ Performing screening DSA on all patients at risk of BCVI may impose a large economic and workload burden on the angiography suite; some institutions cannot support this type of demand.

Computed tomographic angiography

Helical CTA offers several potential advantages over conventional DSA and should now be considered the “gold standard” diagnostic modality. It is a noninvasive study that can be obtained in less than 5 minutes, and as opposed to cerebral angiography, CTA obtains three-dimensional images of the vessel wall (Fig. 180.3). In addition, the workup of blunt trauma



Figure 180.3 This three-dimensional CTA reconstruction with bone subtraction demonstrates occlusion of the right internal carotid artery. This patient presented with a zone II neck hematoma and a seat belt mark across the right side of the neck after an automobile crash. The arrow marks the occlusion at the origin of the internal carotid artery.

patients will inevitably involve CT imaging of the head, chest, abdomen, or all of these. CTA of the carotid–vertebral circulation can easily be obtained during this examination, sacrificing little in time (60 seconds per scan), contrast burden (approximately 100 mL), or radiation exposure.

The ability to use CTA for screening depends on the quality of the scanner at the host institution. Using early-generation single-slice and four-slice CT scanners, two prospective comparative studies (performed by the Denver and Memphis groups) found CTA to have a sensitivity of 47% to 68% and specificity of 67% to 99% compared with DSA.^{32,37} CTA missed 55% of grade I, 14% of grade II, and 13% of grade III injuries.³⁷ CTA technology rapidly improved during the ensuing years such that the number of detectors progressively increased and post-imaging processing became readily available. Bub et al. prospectively compared multidetector CTA (four- and eight-slice scanners) with DSA and found a sensitivity of 83% to 92% and a specificity of 88% to 98% from three different radiologists.³⁸ The interobserver reliability was also higher for CTA than for DSA.

In 2005, Biffl et al. reported their experience using 16-slice CTA for the diagnosis of BCVI, and contrary to the prior study in Denver disqualifying early-generation CT scanners, DSA was not used as the gold standard.³⁹ During an 11-month period, 331 patients were screened, and 5.4% were diagnosed with BCVI. In this final study, CTA scans with normal findings were followed with clinical observation, and no patients developed neurologic symptoms consistent with delayed presentation of missed BCVI. All abnormal examination findings were

confirmed with DSA. Of the 18 injuries identified by CTA, 17 were correctly graded, whereas one patient was upstaged to grade III on DSA (a small pseudoaneurysm was not identified on CTA; false-positive rate of 1.2%).³⁹ In 2006, a prospective comparative study validated 16-slice CTA as a primary screening modality for BCVI.⁴⁰ In this report, CTA was performed in addition to DSA in 162 consecutive patients; 20 carotid and 26 vertebral injuries were diagnosed. In that population, this resulted in an incidence of 1.25% and a screening yield of 28%, which is comparable to historic controls. CTA and DSA were 100% concordant for blunt carotid injuries, resulting in a sensitivity of 100% and a specificity of 100%.⁴⁰ This is the only study comparing conventional 16-slice CTA directly with DSA and demonstrating equivalence. On the basis of these findings, a minimum of 16-slice CTA should be considered the primary screening modality for BCVI. Many authorities, however, still remain passionate that DSA should be the primary screening test of choice for patients with BCVI.⁴¹

In 2009, the initiation of a CTA-based screening and diagnostic program at one institution, along with interdisciplinary standardized treatment guidelines, reduced the time to diagnosis of BCVI 12-fold and the institutional stroke rate due to BCVI 4-fold. The authors concluded that this effect may be due to earlier diagnosis and initiation of definitive therapy.⁴²

Magnetic resonance angiography

Magnetic resonance angiography (MRA) is an attractive non-invasive modality because of the resolution of images obtained in this anatomic region, the infinite number of projections of the vessel, and the ability to assess the intracranial architecture for signs of stroke.^{43,44} Limitations include availability and the time required for image acquisition. This modality is thus impractical in a trauma patient with multiple competing injuries. In comparative studies of DSA versus MRA, MRA performs poorly, with sensitivities of 50% to 95%.^{32,45} The latest report is from 2002, and improved technology may therefore change the role of MRA in BCVI.

Medical Treatment

The mainstay for treatment for BCVI is antithrombotic therapy; however, there are no randomized controlled trials to support this recommendation. Fabian et al. reported the first prospective observational study demonstrating improved neurologic outcome associated with early use of antithrombotic therapy.²¹ Their analysis revealed the benefit of heparin therapy for decreasing the rate of neurologic deterioration after symptoms developed and decreasing the rate of new neurologic events. Heparin therapy was associated with a dramatic reduction in neurologic morbidity (29%) compared with no treatment (73%). Biffl et al. confirmed that patients benefited from early anticoagulation, documenting the greatest benefit to patients who were asymptomatic at the time of heparinization.²⁰ In the asymptomatic group, only one patient developed subsequent stroke. On analysis of the symptomatic cohort, 93% of patients had improvement in their neurologic deficits with anticoagulation compared with only 67% without

anticoagulation. On the basis of these two studies, anticoagulation became the first line of treatment of BCVI.

Complications associated with anticoagulation range from 25% to 54% in the trauma population.²⁰ Most concerning is intracranial hemorrhage, but more common are gastrointestinal bleeds, retroperitoneal hemorrhage, blunt solid organ injury with hemorrhage, and rebleeding from surgical wounds. Eacheppati et al. noted that few patients were able to receive heparin therapy at the time of BCVI diagnosis (14%), and they found a complication rate of 16% among those who received heparin therapy.⁴⁶ Because of the complication profile of full anticoagulation therapy, several authors have focused on antiplatelet therapy as an alternative to traditional anticoagulation.

A prospective comparison of antiplatelet therapy with anticoagulation for BCVI does not exist. There is one study by Biffl et al. that reported anticoagulation (heparin with transition to coumadin) to be superior to antiplatelet therapy (aspirin or clopidogrel), with stroke rates of 1% versus 9%.²⁰ Several follow-up studies have failed to confirm this result. Miller et al. found that resultant stroke rates after BCVI treated with heparin therapy and antiplatelet therapy (5% and 3%, respectively) were similar.⁴⁷ Follow-up studies further demonstrated no difference in stroke rates for those patients treated with heparin (5%–8%) compared with those treated with antiplatelet therapy (3%–7%).^{31,32,48}

Either heparin or antiplatelet therapy can be used with similar results. If the patient has no contraindications to anticoagulation, a prudent protocol would be heparin therapy (goal activated partial thromboplastin time of 50 to 60 seconds) and transition to coumadin (goal international normalized ratio of 2.0) for 3 months. Antiplatelet therapy should be used for the same time period.

All patients who are medically treated should undergo serial CTA or DSA at 1-week and 3-month follow-up. At 3-month follow-up, one can expect 72% of grade I injuries to completely heal.⁴⁹ Grade II injuries are fairly evenly distributed: 33% improve, 33% are stable, and 33% progress to pseudoaneurysms.⁴⁹ Grade III injuries tend either to remain unchanged (50%) or to enlarge (40%). Grade IV injuries universally did not improve and probably do not warrant follow-up imaging after discharge.⁴⁹

Endovascular Treatment

Endovascular therapy has primarily been reserved for evolving dissections that are surgically inaccessible, pseudoaneurysms that persist or enlarge after antithrombotic treatment, or patients with worsening neurologic symptoms on medical therapy. When patients develop a symptomatic injury, a pseudoaneurysm, or a chronic dissection, endoluminal treatment with either a bare or covered stent is an alternative to open repair.^{50,51} Balloon-expandable and self-expanding stents have been used in this location, and in all cases, apposition of the dissection to the wall was achieved with no neurologic events reported.^{51,52}

Follow-up imaging of BCVI treated with antithrombotic therapy is imperative. Pseudoaneurysms are unlikely to resolve with medical management,⁵³ and 33% of acute nonocclusive

dissections treated with anticoagulation develop pseudoaneurysms on follow-up arteriography.²¹ These lesions have a low risk of rupture, but they tend to be the source of chronic embolic events or thrombosis.^{54,55}

Because of the potential for embolic stroke and their failure to resolve with antithrombotic therapy, pseudoaneurysms that fail to resolve, enlarge, or result in ischemic complications should be excluded from the cerebral circulation. Based on the location of these injuries in the distal internal carotid artery, endovascular therapy offers several advantages over open repair. Self-expanding covered stents can be safely delivered to these locations with limited morbidity.^{15–18}

Initial reports from Parodi et al. relied on balloon-expandable bare Palmaz stents (Cordis Johnson & Johnson, Miami Lakes, FL) to cover the orifice of the pseudoaneurysm.⁵⁶ Covering the orifice will typically promote thrombosis of the pseudoaneurysm, but if the sac fails to thrombose, an option has been to coil embolize the sac through the interstices of the bare stent.⁵⁷ In these series, the mean follow-up was 3.5 years without neurologic sequelae, but thrombosis and embolic potential as well as the potential for restenosis remain a concern after endovascular placement of devices in the carotid artery.⁵⁸ Whereas thromboembolic complications are most common immediately after stent placement, patients should be committed to lifelong follow-up for unforeseen complications, such as stent fracture.⁵⁹ Post-stent therapy is variable, but on extrapolation of data from carotid artery stenting for atherosclerotic disease, a regimen of dual antiplatelet therapy (aspirin and clopidogrel) appears adequate to prevent stent thrombosis and embolic ischemic events.⁶⁰ If antiplatelet therapy is discontinued, stent thrombosis and resultant stroke are inherent risks.¹⁶

The enthusiasm for carotid stenting for BCVI paralleled advances in carotid stenting platforms utilized for carotid artery stenosis. There are several positive case reports followed by sporadic case series in the literature. However, Cothren et al. provided a sobering report highlighting the risk of stent thrombosis. They evaluated blunt carotid pseudoaneurysms (grade III) persisting 7–10 days after injury in 46 patients, 92% were asymptomatic. Twenty-three patients were excluded with carotid Wallstents, and 23 patients were treated with either anti-platelet or anti-coagulation therapy.⁶¹ Three (13%) periprocedural ischemic complications occurred. Eight (45%) in the carotid stent group experienced carotid artery occlusion during follow-up and only 1 (4%) in the medical treatment group experienced carotid artery occlusion.⁶¹ This is the highest rate of carotid stent occlusion published, and it demonstrates the challenges of stenting a dissected artery at the skull base.

As opposed to the experience of Cothren et al., Edwards et al. placed 22 carotid stents for BCVI; 18 patients had pseudoaneurysms, and four patients were treated for extensive dissections. They experienced no periprocedural complications. Twelve patients were treated with postprocedural antiplatelet therapy, and eight received anticoagulation. With a mean angiographic follow-up of 7 months, none were occluded (100% patency).⁴⁹ The variance in outcomes can be explained by the challenges of treating this injury pattern.

Follow-up for this patient cohort is imperative, and further prospective studies with long-term follow-up are needed to determine the risks and efficacy of carotid stents for BCVI. Compliance with medications and follow-up surveillance should be considered in planning appropriate therapy for trauma patients.

Surgical Treatment

The indications for surgical intervention parallel those for endovascular intervention.^{21,62} Patients with evolving dissections, pseudoaneurysms that persist or enlarge after antithrombotic treatment, or patients with worsening neurologic symptoms should undergo repair. Whether an open repair or endovascular repair is used should be based on the patient's associated injuries, the location of the injury, the ability to comply with the antiplatelet regimen, and the ability to achieve long-term follow-up.

Schievink et al. have addressed blunt carotid pseudoaneurysms with operative repair in 22 patients.⁶³ To exclude the lesions, five patients required carotid ligation, 13 underwent resection with reconstruction, and four required cervical to intracranial carotid bypass. In their series, two patients experienced ischemic stroke, and the most common complication was cranial nerve neurapraxia secondary to high surgical exposure. This series illustrates the difficulty of treating these lesions at the base of the skull and the resultant morbidity.

If the injury is located at the base of the skull, the only option for treatment may be endovascular exclusion. When the lesion is located in the proximal internal carotid or common carotid artery, the vessel should be approached by an anterior exposure. The vessel may be repaired primarily or, more commonly, by patch angioplasty with either greater saphenous vein or prosthetic material.

VERTEBRAL ARTERIES

Vertebral artery injuries are rare occurrences with an incidence of 0.20% to 0.77% of all trauma admissions.^{20,21} Whereas injuries to the first portion (V1) of the vertebral artery are readily accessible, those to the second portion (V2, within the bony foramen of the cervical canal), the third portion (V3, as the vessel exits the bony foramen and enters the base of the skull), and the fourth portion (V4, intracranial segment to the basilar artery) can be extremely difficult to control.

Clinical Presentation

Penetrating injuries are most commonly due to gunshot wounds and stab wounds.⁶⁴ Life-threatening hemorrhage is rare from an isolated vertebral injury (mortality, 4%); however, penetrating injuries frequently involve the common carotid artery, subclavian artery, internal jugular vein, and subclavian vein, which may be life-threatening. Patients typically are asymptomatic or complain of associated neurologic injury secondary to either compression from hematoma or direct injury.⁶⁴

The most common mechanism for blunt vertebral artery injury is fracture of the transverse foramen through which

the vessel courses (vertebrae C2–6).⁶⁵ The vertebral vessels are relatively fixed throughout the vertebral canal, making the V2 segment susceptible to hyperextension and stretch injuries. Because of the rich collateral circulation in the neck, unilateral vertebral artery injuries are often asymptomatic in 80% of cases. With regard to dissections, patients may complain of subtle neck pain or posterior headache. Vertebrobasilar ischemia may be manifested with protean symptoms, including dizziness, vertigo, nausea, tinnitus, dysarthria, dysphagia, ataxia, visual deficits, and hoarseness. The degree of ischemia is determined by the extent of distal propagation into the basilar or posterior inferior cerebellar arteries. Patients with bilateral vertebral artery injuries may present with more severe symptoms: coma, fixed pupils, and loss of respiratory drive. Approximately 25% of patients will have bilateral blunt vertebral artery injuries, and 33% will have an associated blunt carotid injury.⁶⁶

Diagnostic Evaluation

Vertebral artery injury typically is manifested with penetrating neck injury. If the patient presents with refractory shock, with hard signs of a vascular injury, or with hard signs of a tracheal injury (continuous air bubbling from the wound), the management is straightforward, and the injury should be diagnosed in the operating room. Plain films in the emergency department are useful for determining the track of the missile and any foreign bodies within the wound. Hemodynamically stable patients with a normal Glasgow Coma Scale score who do not have hard signs of a vascular injury should undergo CTA of the neck. The provider should also include the head and chest in the evaluation of the injury to fully evaluate associated injuries and to identify the track of the missile or knife. This mechanism of injury should also prompt a high suspicion for aerodigestive injuries, which may require additional evaluation (bronchoscopy, rigid esophagoscopy, or upper gastrointestinal swallow studies).

Blunt vertebral artery injuries were originally diagnosed by DSA, and the same grading criteria (see Table 180.2) were applied to the vertebral artery as for the carotid artery. In contrast to blunt carotid injuries, grade of blunt vertebral artery injuries does not correlate with increasing risk of stroke. The stroke risk of blunt vertebral artery injuries is 20% irrespective of grade.⁶⁶ The same controversy about screening and imaging surrounds the evaluation of blunt vertebral artery injuries. The same screening criteria used for blunt carotid trauma (see Table 180.1) are applied to blunt vertebral artery injuries; however, cervical spine fracture is the only factor that is independently associated with blunt vertebral artery injuries (odds ratio, 14.5).³⁴

All imaging modalities are less accurate for diagnosis of blunt vertebral artery injuries compared with carotid injuries. Duplex evaluation of the vertebral vessels is extremely limited; however, with color-flow imaging, the operator can assess for vessel patency. MRA has the previously stated limitations in the setting of trauma. In the evaluation of blunt vertebral artery injuries, CTA has a sensitivity and specificity of 40% to 60% and 90% to 97%, respectively.³⁸ If there is a high index of suspicion

based on cervical fracture, a confirmatory study should be obtained. MRA is an acceptable option when it is available; however, if concern persists, DSA should be performed.

Medical Treatment

Medical management has no role for penetrating injuries, but it does have a role for blunt injuries identified during the diagnostic evaluation. The posterior circulation stroke rate attributed to blunt vertebral artery injury is 24% with an associated mortality of 8%.⁶⁶ Symptomatic patients should be treated with heparin and monitored for hemorrhagic conversion with serial neurologic examinations. Heparin therapy was first evaluated in asymptomatic patients and found to reduce neurologic events in the posterior circulation from 20% to 35% (no anticoagulation) to 0% to 14% (heparin therapy).^{47,66} In patients who could not tolerate anticoagulation, the efficacy of heparin was similar to that of aspirin. Follow-up studies increasingly used aspirin secondary to the bleeding complications associated with heparin therapy. Miller et al. treated 43 vertebral artery injuries; 32 patients received aspirin or clopidogrel, and only eight patients received heparin therapy.⁴⁷ None of the patients developed stroke. Symptomatic patients or those patients without contraindications to anticoagulation should be treated with 3 to 6 months of anticoagulation with radiographic follow-up. Asymptomatic patients should be treated with either 3 to 6 months of anticoagulation or dual antiplatelet therapy; data are insufficient for one treatment to be recommended at this time.

Endovascular Treatment

Endovascular treatment of the vertebral artery is used for uncontrolled hemorrhage not controlled with surgery, backbleeding from the V3 segment, pseudoaneurysm, and symptomatic patients who cannot tolerate anticoagulation (Fig. 180.4). The endovascular technique of crossing the vertebral artery confluence at the basilar artery is technically challenging, but when possible, both proximal and distal ends of the transected or lacerated vessel should be treated with embolization. If the vessel is intact, the injury may be crossed from an antegrade approach, allowing embolization of both the outflow and inflow (trap-door technique). In nearly half of vertebral artery injuries evaluated with endovascular techniques, the vessel was thrombosed and required no treatment at all.⁶⁷ Preservation of the vertebral artery may be a concern when other extracranial blood supply has also been compromised. In these complex cases, endovascular stenting of the vertebral artery has been performed as a salvage procedure.⁴⁷ There are no data to support routine stenting for blunt vertebral artery injuries.

Surgical Treatment

Operative management is reserved for patients with active bleeding from the vertebral artery at the time of neck exploration. In a review of the largest series of penetrating vertebral artery injuries, 50% of patients who underwent open exploration required postoperative endoluminal embolization to arrest

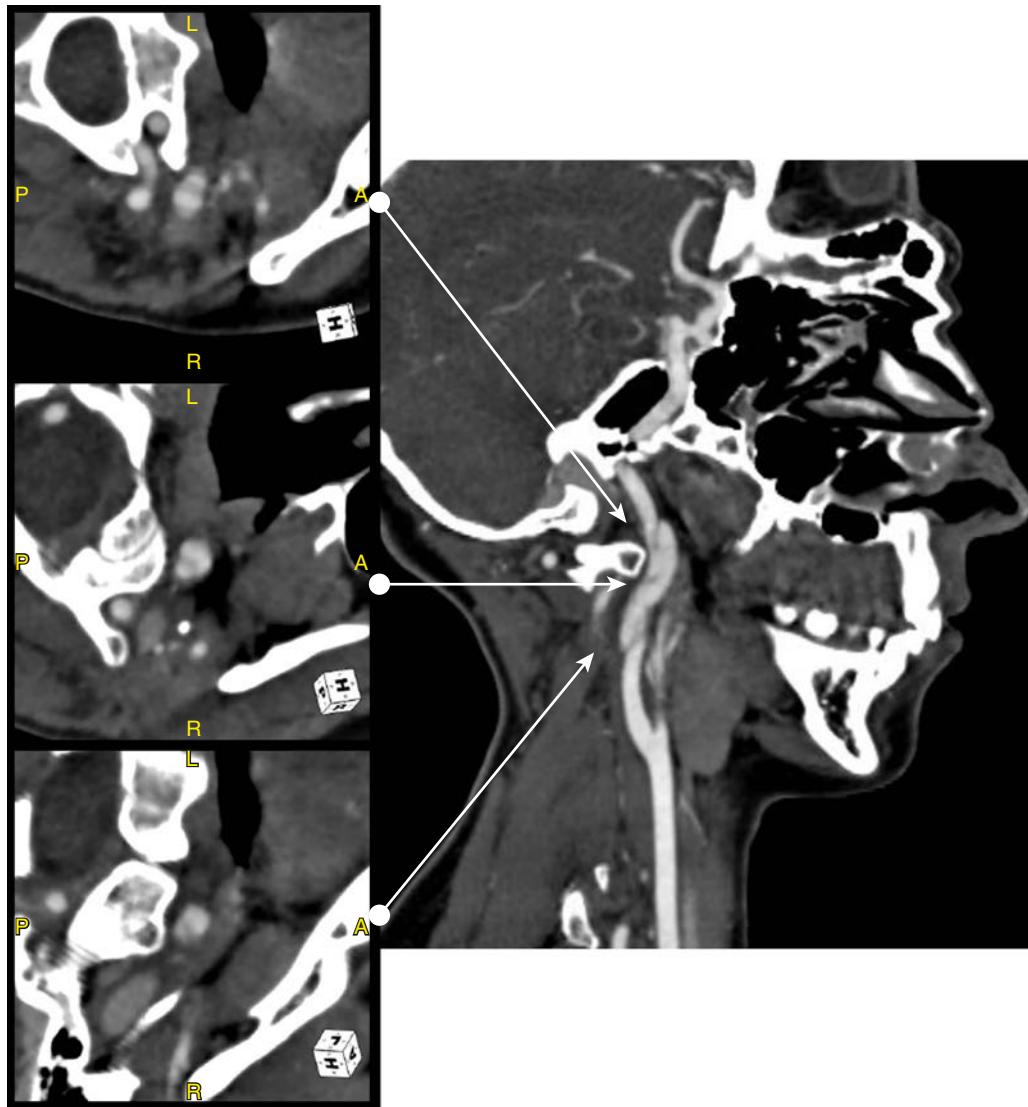


Figure 180.4 Penetrating vertebral artery injury secondary to a stab wound at the base of the left side of the neck. CT angiography demonstrates serial slices showing both a pseudoaneurysm as well as a dissection.

bleeding or to control arteriovenous fistulae.⁶⁷ Controlling the vertebral artery can be challenging for even the most experienced trauma or vascular surgeon. Unilateral surgical ligation results in a stroke rate of 3% to 5%.⁶⁸

The approach is the same as that described previously for exploration of the carotid artery. Once the sternocleidomastoid muscle has been retracted laterally, the V1 segment can be visualized by transecting the sternocleidomastoid muscle from the sternum and clavicle. Next, the internal jugular vein is identified, and laterally, the scalene fat pad is divided vertically, with care taken to identify and protect the phrenic nerve coursing lateral to medial across the anterior scalene muscle. Division of the anterior scalene muscle from the first rib allows visualization of the first portion of the subclavian artery and the origin of the vertebral artery, thyrocervical trunk, and internal mammary artery. On the right side, the recurrent laryngeal nerve should be protected, and care should be taken on the left side to avoid injury to the thoracic duct. Partial manubrium and clavicle resection can gain exposure in this location.

The V2 segment is more challenging to control, and most surgeons should simply ligate the proximal V1 segment as repair of the vertebral artery is futile and exposure of the V2 segment is fraught with venous bleeding. Exposure of V2 involves retracting the sternocleidomastoid muscle laterally, dividing the omohyoid muscle, and retracting the carotid sheath medially. Identification of the cervical spinal bodies and the associated prevertebral fascia is mandatory. The sympathetic chain ganglia should be protected as they course through this region. Longitudinal opening of the anterior longitudinal ligament and paraspinous muscles allows identification of the transverse process of the spinal bodies. Removal of the anterior aspect of the transverse processes by use of a handheld rongeur allows exposure of the vertebral artery. Posterior to the vessel lie the cervical nerve roots. Hemorrhage in this location should be controlled with proximal ligation of V1. Bone wax can be packed into the bony canal, and postoperative embolization of the distal vertebral artery may be performed if needed.

Most vascular and trauma surgeons are unfamiliar with the V3 segment at the base of the skull. Rather than a timely

dissection, the patient may be better served by proximal ligation of the V1 segment, packing of the wound (with bone wax, gauze, or balloon occlusion), and then endoluminal embolization. To expose the V3 segment for ligation, the incision is extended posteriorly behind the ear onto the mastoid process.⁶⁹ The sternocleidomastoid muscle is retracted medially and may be divided from the skull base. During this step, the spinal accessory nerve should be identified and preserved. The transverse process of C1 is identified anterior and below the mastoid process. The muscle attachments to C1 and C2 are cleared with a periosteal elevator, and then, after resection of the lateral process of C1, the vertebral artery is exposed.

SUBCLAVIAN ARTERY

Injuries to the thoracic outlet are extremely lethal. Prehospital mortality is 50% to 80%, and of those who survive transport, 15% die during treatment.⁷⁰

Clinical Presentation

Blunt injuries to the subclavian vessels are extremely rare, but when they occur, patients typically have associated clavicular fractures, mediastinal injuries, and pulmonary contusions. Penetrating injuries account for the majority of trauma to the subclavian vessels; in one U.S. trauma center, gunshot wounds accounted for 74% and stab wounds accounted for 26% of injuries.⁷¹ These patients often present to the emergency department in cardiac arrest, and consideration for resuscitative thoracotomy must be based on time from injury and signs of life. Those patients who survive transport may present with hard signs of vascular injury as described earlier, which mandate immediate operative exploration. All other patients with soft signs of a vascular injury should have further diagnostic evaluation. A normal radial pulse does not reliably exclude an upper extremity vascular injury. Concomitant cervical and thoracic injuries are common in 70% of patients.

The upper extremities rarely suffer from ischemia because of the intense collateral circulation; the long-term morbidity of these injuries is secondary to brachial plexus injuries. Brachial plexus injuries can be caused by penetrating injuries that directly transect nerve roots; blunt injuries that result in shear or traction forces; and operative exposure, which can result in iatrogenic injury. Blunt injury to the brachial plexus is typically secondary to stretch injuries to the upper extremity or bone fractures.

Diagnostic Evaluation

In the unstable patient, there are few options other than immediate exploration, but because of the fibrous attachments surrounding the subclavian vessels, injuries will frequently result in a contained extrapleural hematoma that may extend into the supraclavicular fossa. If time allows, CTA can be invaluable to identify the location of injury and to evaluate the mediastinum. Hemodynamically stable patients with asymmetrical pulses or neurologic deficits should undergo evaluation with

CTA, including the chest, neck, and upper extremity. Injury to the axillary vessels can range from intimal disruption to pseudoaneurysms, dissection, and even thrombosis. In addition, axial imaging will appropriately evaluate the mediastinum, cervical vasculature, and associated bone fractures.

Medical Treatment

Intimal disruptions and dissections that are not flow limiting should be treated with clinical observation. Anticoagulation or antiplatelet therapy may be added at the discretion of the surgeon. If the patient develops embolic symptoms, antithrombotic therapy should be instituted with arteriography of the injury.

Endovascular Treatment

Endovascular treatment in this area can obviate the need for an extensive dissection at the base of the neck. Covered stent grafts have been increasingly used in this location, with several authors reporting immediate technical success for treatment of pseudoaneurysms, lacerations, arteriovenous fistulae, and even complete transections (Fig. 180.5).⁷²⁻⁷⁴ Approximately 42% to 50% of patients are candidates for endovascular treatment.

These injuries may be approached from a transfemoral, transbrachial, or combined technique. Acute thromboses in patients with malperfusion symptoms can be treated through a retrograde brachial approach, and after flow is restored, a covered stent can be used to treat the injury. Most transected vessels can be crossed with a hydrophilic wire. Undoubtedly, endovascular techniques reduce the morbidity of operative exposure and potential nerve injury in a blood-stained field. There are relatively few contraindications to this approach. The patient must be hemodynamically stable. Some consider a large supraclavicular hematoma with brachial plexus compression a relative contraindication; however, the injury may be treated with a covered stent followed by hematoma evacuation in a controlled field.

The mobility and compression between the first rib and clavicle raise concern about long-term patency in the young trauma population; it is imperative to observe these patients for late sequelae. In addition, endovascular repair does not preclude stent explantation with formal open repair; this approach would ideally reduce the potential for iatrogenic nerve injury in the acute setting. In one of the largest reported series, three early stent thromboses were encountered in 56 patients.⁷² All three were opened with a secondary intervention, and no patient experienced upper extremity ischemia.⁷² Endovascular therapy in the thoracic outlet offers a less invasive, rapid treatment and has the added benefit of avoiding injury to the brachial plexus, which has long-term implications for a functional recovery.

Surgical Treatment

Injuries to the thoracic outlet can be difficult to expose in an acute setting; it is important to prepare the neck, chest, and

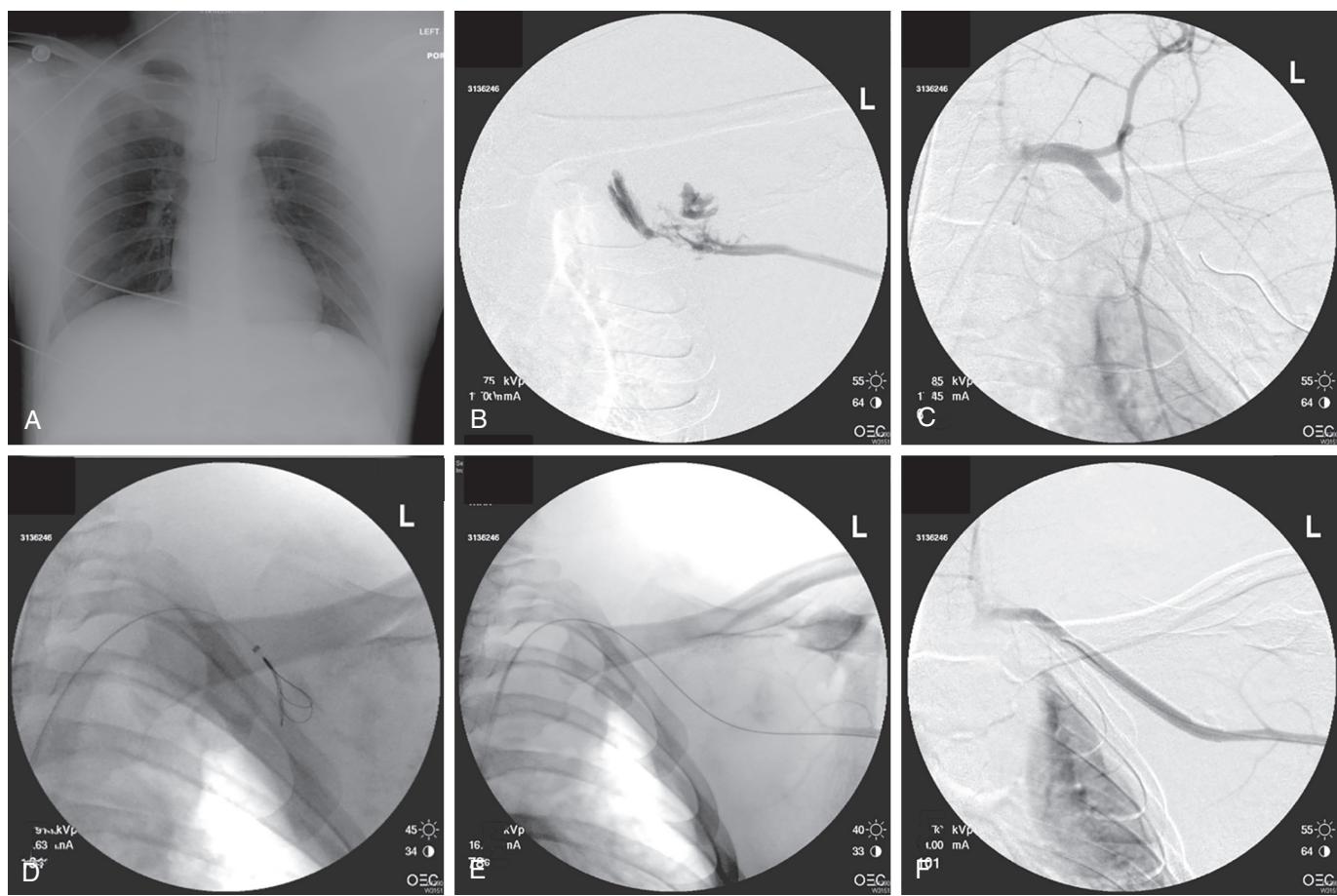


Figure 180.5 Blunt subclavian artery transection. (A) The patient presented with a large stable hematoma in the suprACLAVICULAR fossa extending into the axilla after a high-speed motorcycle crash. The chest radiograph demonstrates the soft tissue density over the left clavicle. (B) From the left brachial approach, there is extravasation at the level of the subclavian to axillary artery junction. (C) From the right groin, left subclavian artery selective angiography was performed. (D) A curved Glidewire was used to cross the transection. (E) Once the transection was crossed, the brachial wire was snared within the hematoma, obtaining femoral–brachial wire access. The transection was treated with a covered stent. (F) Completion angiogram demonstrates complete exclusion of the transected segment and no residual extravasation.

groin into the surgical field. Proximal control requires either a median sternotomy for the innominate and right subclavian arteries or a high left anterolateral thoracotomy with potential clavicular resection for the left subclavian artery. Rather than direct exploration of the hematoma, a remote occlusion balloon can be placed either from the groin or retrograde from the brachial artery. In doing so, the surgeon can perform arteriography, and with proximal control, the operative tempo changes from emergent to semiurgent. Either an endovascular treatment can ensue or the surgeon can proceed with a meticulous dissection at the base of the neck and avoid either a sternotomy or thoracic incision.

If the patient is in extremis, subclavian artery ligation rarely results in upper extremity ischemia. When the vessel is repaired, the injury typically requires an interposition graft. PTFE works well in this location and has excellent patency.^{75,76} If the field is grossly contaminated, saphenous vein, internal jugular vein, and superficial femoral artery have all been used in this location. Nearly 50% of penetrating subclavian injuries will have an associated subclavian vein injury in addition to the arterial injury.⁷⁵

CERVICAL VENOUS INJURIES

Venous injuries to the neck and thoracic outlet are invariably due to penetrating injuries. Blunt venous injuries are extremely rare and should be considered with sternal and clavicle fractures. Venous injuries to the internal jugular vein occur in 20% of penetrating neck trauma, and subclavian vein injuries occur in 50% of penetrating subclavian injuries.^{76,77}

Clinical Presentation

When patients present in extremis, the venous injury is typically identified in the operative suite. Isolated venous injuries are manifested with hard or soft signs of a vascular injury, but patients are rarely hypotensive.⁷⁸

Diagnostic Evaluation

The diagnostic evaluation should parallel the evaluation described before for each arterial anatomic region. If the patient

undergoes CTA for a suspected vascular injury, delayed acquisition (ranging from 30 seconds to 3 minutes) will provide improved imaging of the venous anatomy once the contrast material has cleared the associated arterial anatomy.

Endovascular Treatment

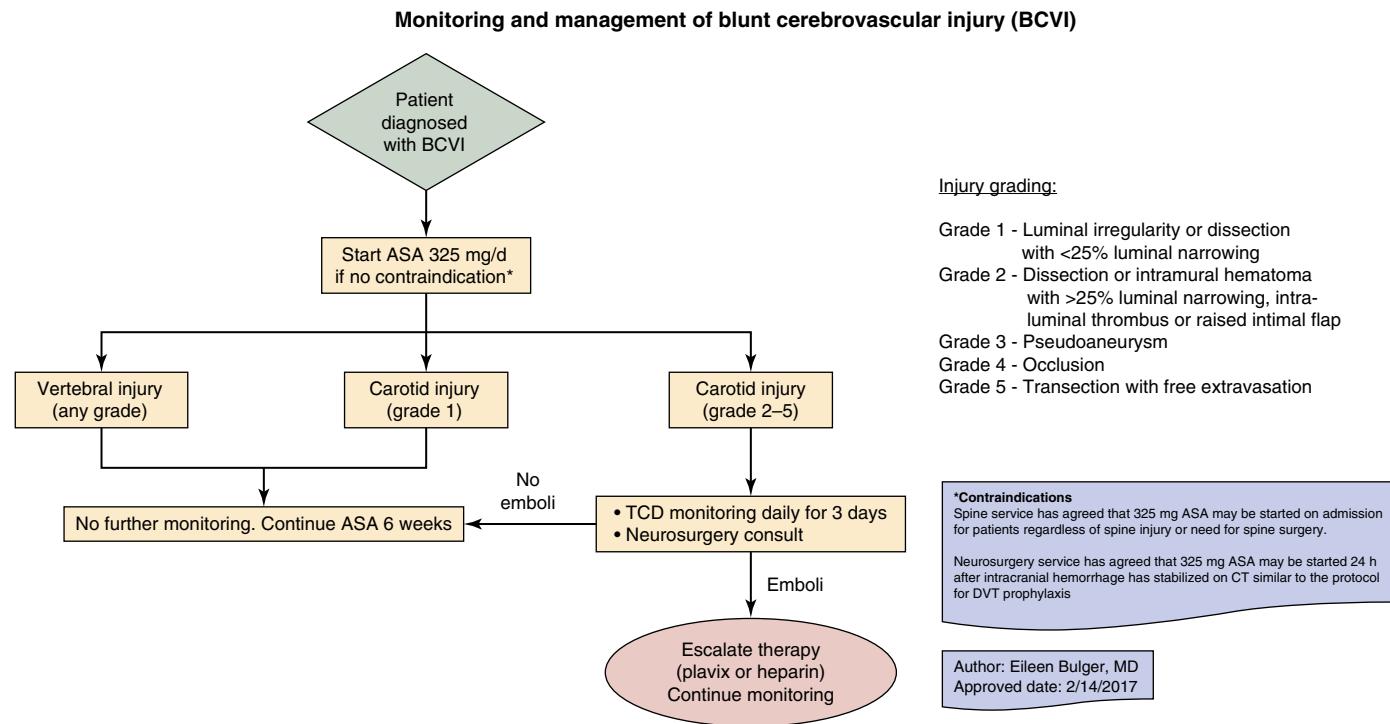
Endovascular stents have increasingly been used in the central venous system for subclavian and superior vena cava chronic thromboses. The efficacy of covered stents for venous trauma has been described only in case reports, with good technical results.^{79,80} This technology will be increasingly used for isolated venous injuries in hemodynamically stable patients.

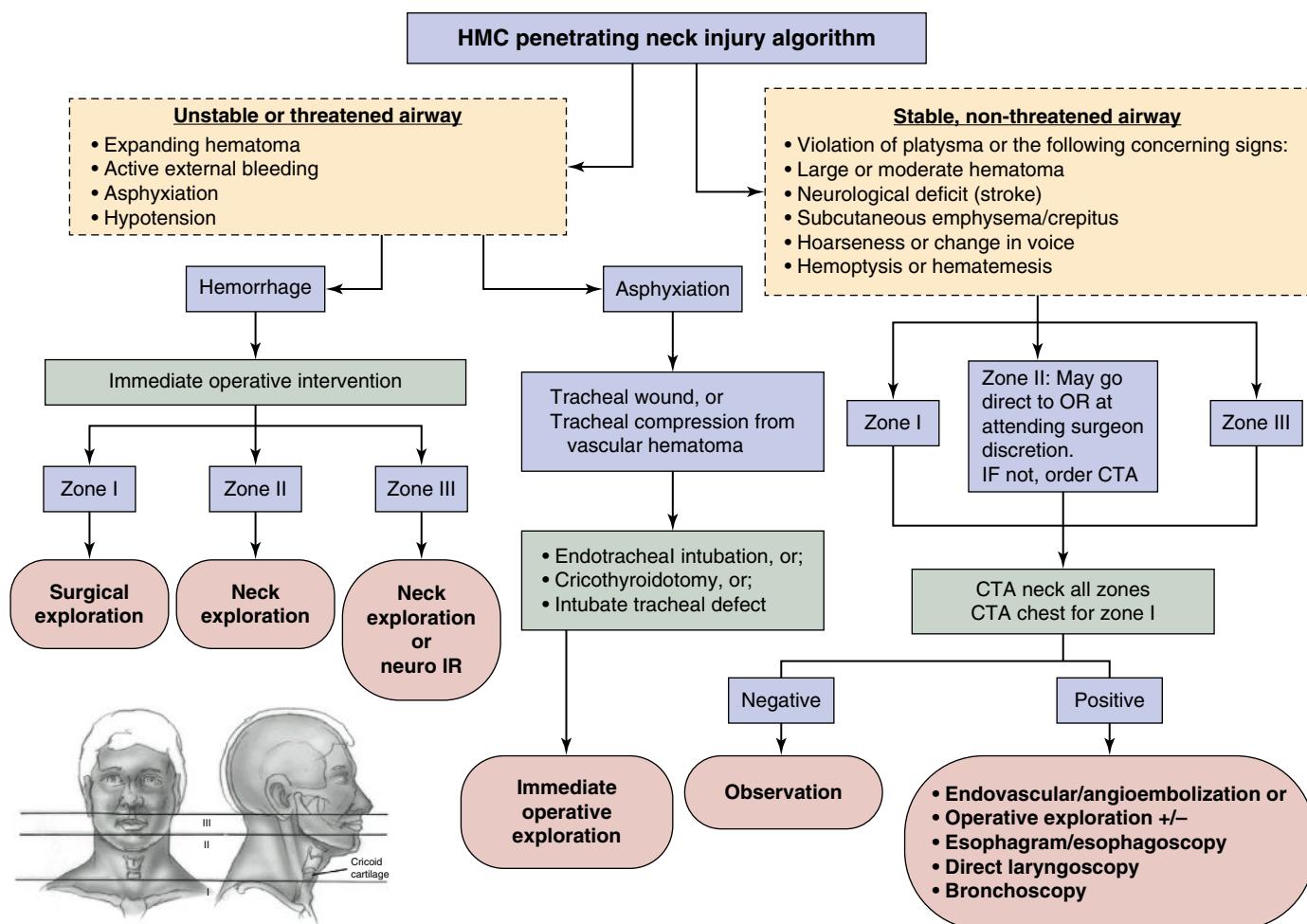
Surgical Treatment

The surgical approach to each vein is identical to the arterial exposure described earlier. If the patient has hard signs of a vascular

injury and is in extremis, the neck and subclavian veins can be ligated with limited morbidity. If the internal jugular vein is ligated, the patient should be monitored for cerebral edema, but this is a rare occurrence even with bilateral internal jugular vein ligation. Internal jugular vein reconstructions (with either spiral vein grafts or externally reinforced PTFE) in the setting of bilateral neck dissections have an 18-month patency of 64%; elevated stump pressures (>30 mm Hg) may improve patency.⁸¹ When the subclavian vein is ligated, the upper extremity should be elevated and placed in compression; transient edema typically resolves during the course of 7 to 10 days, and long-term venous stasis is rare.⁷⁷ Simple lacerations of the vein can be repaired with lateral venorrhaphy if less than 50% of the wall is involved.⁷⁷ All repairs are at risk of eventual thrombosis; mechanical and chemical thromboprophylaxis should be considered. Extensive vein repairs (end-to-end, venous interposition, or spiral/panel grafts) typically do not have a role in penetrating neck injuries (as opposed to lower extremity injuries) when time is critical.

CHAPTER ALGORITHM





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Cothren CC, Moore EE, Biffl WL, et al. Anticoagulation is the gold standard therapy for blunt carotid injuries to reduce stroke rate. *Arch Surg.* 2004;139:540–545; discussion 545–546.

Prospective, nonrandomized, noncontrolled, observational study that demonstrated reduced neurologic events in patients with blunt cerebrovascular injury treated with one of the following: systemic heparin, low-molecular-weight heparin, or antiplatelet agents.

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Thoracic Vascular Trauma

CASSRA N. ARBABI and ALI AZIZADEH

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INTRODUCTION

Thoracic injuries are common sequelae of both blunt and penetrating trauma, with blunt thoracic injuries proving responsible for approximately 8% of all trauma admissions in the United States, with motor vehicle crashes being the most common (>70%) mechanism.^{1–4} Penetrating injuries are also commonly encountered, with one report from a high-volume, urban trauma center³ identifying penetrating chest trauma among 7% of all trauma admissions and 16% of all penetrating trauma admissions overall.

Despite the prevalence of thoracic injury following trauma, the majority of patients presenting with these injuries will not require thoracic operative intervention. Appropriate utilization of tube thoracostomy and comprehensive inpatient management will prove definitive treatment for the majority of patients with these injuries. Even among penetrating mechanisms, only 14% of stab wounds and 15% to 20% of gunshot wounds to the chest require operative intervention for thoracic injury.³

It is important to note, however, that vascular injury occurring within the thoracic region continues to be associated with high mortality,^{1–4} with many patients likely not surviving to

reach a facility capable of providing care.^{5–7} Among those who do present to trauma centers, however, the continued evolution of imaging capabilities and improved approaches for nonoperative and operative management continue to improve the care of these patients.^{5–8} Significant progress has been made over the last two decades in the diagnosis and treatment of thoracic vascular injuries. This paradigm shift includes the widespread use of computed tomography angiography (CTA) for diagnosis, aggressive blood pressure control, delayed repair for stable patients, adoption of endovascular repair as the treatment modality of choice for anatomically suitable candidates, availability of later generation conformable, flexible and lower-profile devices and medical management of minimal aortic injuries. These advances have significantly improved the outcomes of patients with thoracic vascular trauma. Nevertheless, the effective treatment of these injuries requires careful consideration of treatment dilemmas, thoughtful decision making, a comprehensive appreciation of anatomic relationships within the thoracic cavity, and prompt operative intervention when indicated. This chapter summarizes the current diagnosis and management of thoracic vascular injuries.

Presentation and Evaluation

Any patient with significant blunt chest trauma or penetrating injury around the thoracic cavity is at risk for vascular injury. The patient may provide useful history, but often this information is provided by prehospital personnel. The mechanism of injury, time from injury, vital signs, and neurologic status at the scene, as well as any changes during transport, are critical. With blunt injury specifics, such as prolonged extrication, the location and degree of occupant compartment vehicle deformation, speed and direction of impact, or height of fall may provide useful information. With penetrating trauma, the specific details are typically vague and often unreliable.

Evaluation proceeds according to the Advanced Trauma Life Support (ATLS) guidelines, and potentially life-threatening conditions are immediately treated. As with any trauma patient, thorax trauma may necessitate intubation for airway control. While these patients require evaluation and imaging for potential neurologic, intraabdominal, or extremity trauma, the discussion herein will focus largely on the diagnosis and characterization of thoracic vascular injuries. Physical examination, which should be performed both rapidly and thoroughly, has been shown to be reliable and can often help make the diagnosis. The presence of distended neck veins, tracheal deviation, subcutaneous emphysema, chest wall ecchymosis and/or instability, absent breath sounds, or muffled heart sounds may all provide crucial information. Likewise, the absence of an upper extremity pulse suggests a proximal arterial injury. Vital signs should be frequently monitored with careful observation of the work of breathing and oxygen saturation. Overall, the most common thoracic injuries are hemothorax and/or pneumothorax. However, rapid diagnosis of vascular injury is paramount to success in most instances.

Penetrating thoracic trauma in a hemodynamically unstable patient warrants operative intervention. The decision regarding surgical exposure may be problematic, especially if there is concomitant abdominal injury. Clinical judgment is paramount in this situation. The hemodynamically stable patient may benefit from additional imaging, with CTA as the first-line modality, which provides more detailed and organ-specific information.⁹

IMAGING

Plain radiography of the chest remains the most commonly utilized initial radiographic imaging following thoracic trauma and is an adjunct in the ATLS algorithm. An adequate plain film should make the diagnosis of any large hemothorax or pneumothorax. Since screening chest X-rays (CXR) are usually performed supine, however, hemothorax can be somewhat difficult to adequately diagnose. Haziness of one hemithorax, when compared with the other, may be the only real radiographic sign. If this is of any substance, a chest tube should be placed.

A CXR may also allow the clinician to evaluate the mediastinum for the possibility of a blunt traumatic aortic injury (BTAI; Fig. 181.1). A variety of clinical findings may prove suggestive of BTAI, including a widened mediastinum, left apical capping, depressed left main bronchus, indistinct or abnormal

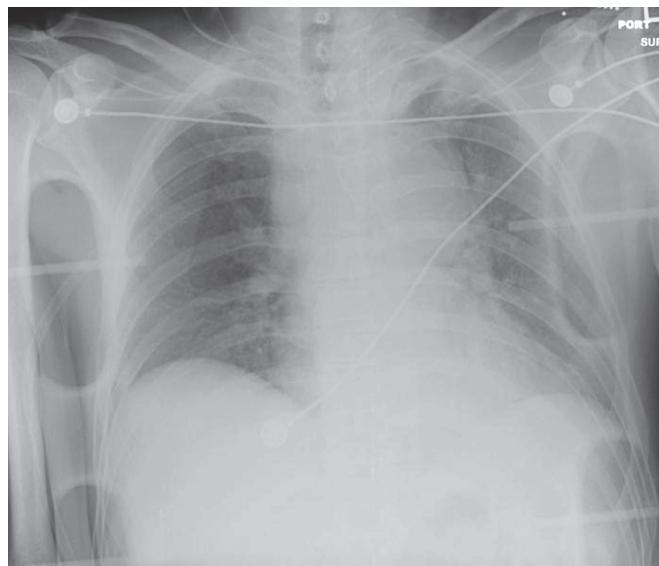


Figure 181.1 Chest radiograph of a patient with blunt thoracic aortic injury showing a widened mediastinum and obliteration of the aortic knob.

aortic contour, tracheal deviation, or large left hemothorax. It is important to note, however, that plain chest radiography in this setting is neither specific nor sensitive for the presence of BTAI. A recent study funded by the Centers for Disease Control and Prevention (CDC) and University of California Center for Health Quality and Innovation (CHQI) found that widened mediastinum on CXR had only a 33% sensitivity for aortic injury.¹⁰ These findings highlight an improved understanding that CXR alone is not a reliable screening modality for BTAI. They also emphasize the need for careful consideration of other factors – including mechanism of injury – in determining those who need subsequent CTA imaging after trauma.

In the modern era, CTA has become a tool of trauma evaluation that is liberally employed. Computed tomography (CT) protocols that utilize angiographic contrast are ubiquitous to initial evaluation protocol of most major trauma centers, and have specific benefits with regard to the diagnosis and characterization of vascular injury. This imaging modality, particularly with the advent of advanced multi-slice imaging capabilities, affords both precise evaluation of the aorta and other vascular structures of the chest. Post-imaging processing can also rapidly and effectively provide good resolution – three-dimensional evaluation of the thoracic vascular structures.^{11–13} CTA of a patient with BTAI is shown in Figure 181.2. Intravascular ultrasound (IVUS) provides real-time, 360-degree imaging of the aorta using a high-frequency (10 MHz), miniature ultrasound probe placed through a femoral arterial sheath (Fig. 181.3).¹⁴ IVUS is a useful tool for evaluation of traumatic aortic injury, especially in cases with equivocal CTA, or in cases of periaortic hematomas in the absence of a direct sign of aortic injury.¹⁵ IVUS does not require contrast or radiation and can be performed concurrently using the same femoral puncture as angiography. The disadvantages of IVUS are additional cost (capital equipment and disposable catheter), larger sheath (8 F), and operating room time.¹⁶

Angiography, once considered the “gold standard” for diagnosis, remains a valuable imaging modality in the management

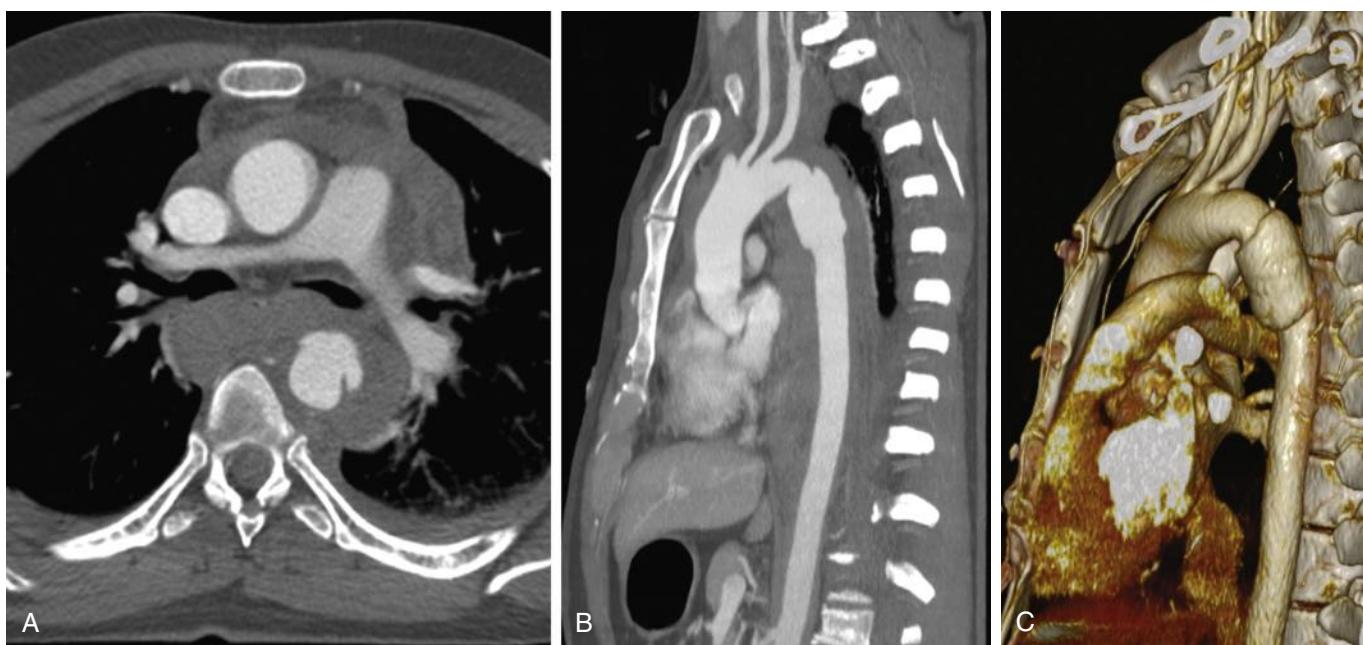


Figure 181.2 Computed tomography angiography of a patient with blunt thoracic aortic injury. (A) Axial view demonstrating the injury and periaortic hematoma. (B) Sagittal view demonstrating the typical location of the injury at the isthmus distal to the left subclavian artery. (C) Three-dimensional computed tomography reconstruction.

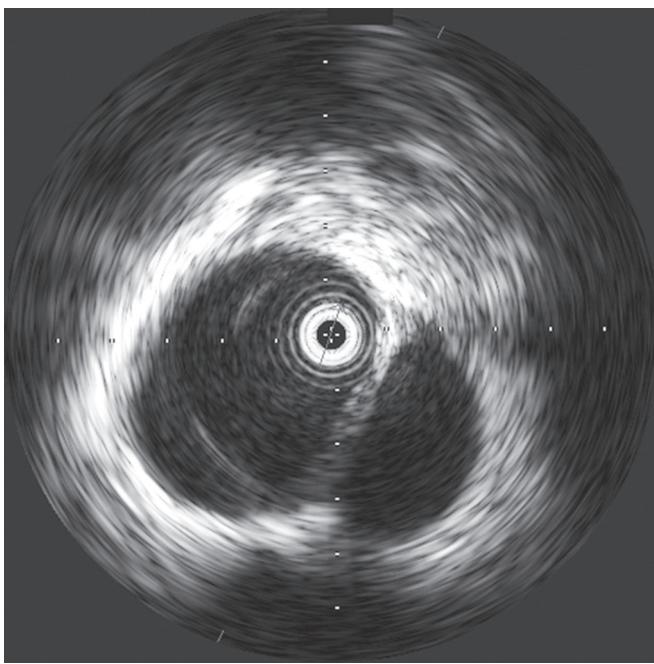


Figure 181.3 Intravascular ultrasound image of a patient with blunt thoracic aortic injury demonstrates a pseudoaneurysm.

of patients with thoracic vascular trauma. However, it has taken on a therapeutic, rather than purely diagnostic, role, given the wide utilization of CTA. Disadvantages include invasive nature, cost, use of intravenous contrast, radiation exposure, access site complications, and the need to transport the patient to an angiography suite or operating room (CTAs are commonly performed in the emergency center). There is a small incidence of false-positives on CTA or conventional angiography from known anatomic variants that can mimic aortic injury. These include a

ductus diverticulum, infundibula of bronchial or intercostal arteries and aberrant brachiocephalic arteries.^{17,18} Differentiation of these abnormalities from BTAI is critical, and can often be determined by presence or absence of radiographic findings indicative of trauma such as mediastinal or periaortic hematoma, adjacent intimal flap or intramural hematoma.¹⁹

Indications for Emergent Operation

While the vast majority of traumatic thoracic injuries can be managed nonoperatively, surgery may be indicated emergently, urgently, or in a delayed fashion. Indications for emergent thoracic exploration include tracheobronchial perforation, endobronchial blood loss, cardiac tamponade, shock with a penetrating chest injury, initial chest tube output of 1500 cc, or persistent chest tube output of 200 cc/h for 2–4 hours. There is likely a linear relationship between the total amount of thoracic hemorrhage and mortality, necessitating a familiarity of the treating surgeon with the most common exposures that will be required in an emergent setting. The patient's overall clinical condition and astute surgical judgment are of paramount importance when deciding to operate and what type of surgical exposure or approach to utilize.

Open Surgical Exposure/Incisions

There are several surgical approaches to the thorax, each with advantages and disadvantages for emergent trauma applications. The surgeon should be familiar with all of them, and the clinical situation should determine the choice of incision. Hemodynamically unstable patients may not tolerate lateral positioning, as it may exacerbate hypotension. In addition, in the emergent exploration of the unstable trauma patient, the

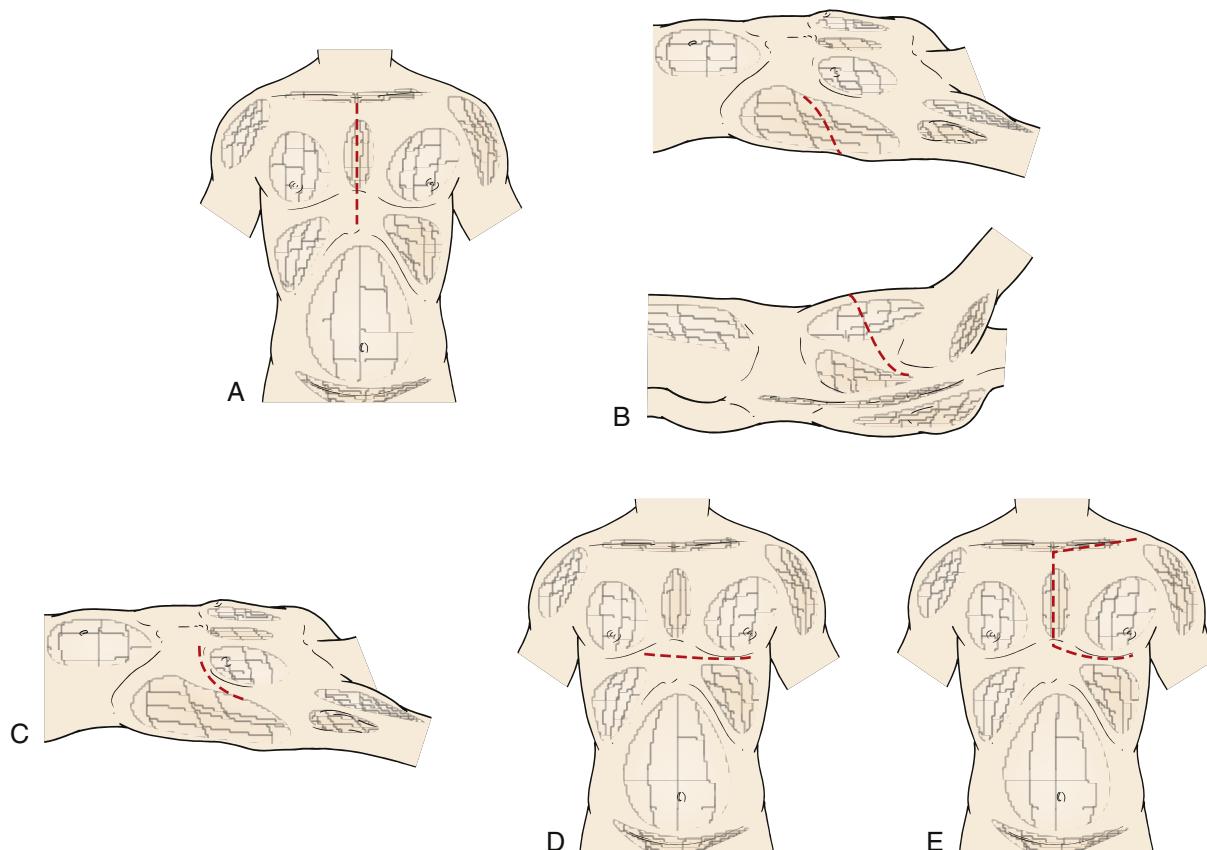


Figure 181.4 Incisions for Thoracic Trauma. (A) Median sternotomy with right or left neck extensions can be used to treat injuries to the heart, proximal aorta, innominate, right and left carotid arteries. (B) Left posterolateral thoracotomy for repair of descending thoracic aortic injuries. (C) Left anterolateral thoracotomy for patients in extremis (ED thoracotomy); also provides access to the left subclavian artery. (D) Extension of the anterolateral thoracotomy to the opposite chest as a “clamshell” incision. (E) “Trapdoor” thoracotomy.

only imaging is likely to be portable chest radiograph. In this scenario, the surgeon will have limited knowledge of potential mediastinal involvement, the projectile’s path, or additional cavitary involvement. With penetrating thoracic trauma, there is the possibility of injury to adjacent body regions, such as the abdomen and neck. Therefore, a thoracic incision must prove versatile in accommodating flexibility in the conduct of operation. In this setting, incision selection requires careful consideration of the appropriate extensions that can be made to best optimize exposure and control options.

In the stable patient, additional imaging data is more likely to have been obtained. Armed with better understanding of the location and nature of injury, a better decision regarding optimal therapy can be formulated. In the modern endovascular age, this information may facilitate the effective utilization of less-invasive adjuncts. However, knowledge of open surgical options and defaults remain paramount. If open exploration or repair is selected, the choice of incision should be guided by the CT findings.²⁰

Commonly employed open operative approaches include anterolateral thoracotomy, posterolateral thoracotomy, bilateral thoracotomy (or “clamshell thoracotomy”) and median sternotomy (Fig. 181.4). The left anterolateral approach is perhaps the most expedient – and extending it across the midline affords excellent exposure to both pleural spaces, the anterior mediastinum

and nearly the full complement of thoracic vascular structures. Likewise, the incision can be continued as a celiotomy for abdominal exploration and is preferred over the posterolateral approach in the patient in shock. The main disadvantage of the left anterolateral approach is exposure of posterior thoracic structures. By extending the ipsilateral arm and placing a bump to elevate the thorax approximately 20 degrees, the incision can be carried to the axilla – a maneuver that will improve posterior exposure. The posterolateral thoracotomy affords optimal exposure of the hemithorax, especially the posterior structures, and is the standard incision for most elective thorax operations. Its lack of versatility limits the usefulness in trauma, but is the preferred approach to repair any injury pattern that also involves intrathoracic tracheoesophageal injuries. Sternotomy provides excellent access to the heart, proximal great vessels, and anterior mediastinum. This particular incision is versatile, and can be extended as an abdominal, periclavicular, or neck incision. The “trap door” incision is rarely used, since left-sided thoracic vessels can be approached via sternotomy with extension.²¹

Operative Techniques

Thoracic vascular injuries after significant trauma rarely occur in isolation, particularly after penetrating mechanisms.

Therefore, it is useful to appreciate a few key nonvascular operative techniques that may be required during emergent operative exploration. Among patients requiring emergent thoracic exploration, between 20% and 30% will need a pulmonary resection, which ranges from wedge resections to major anatomic resections.^{22,23} A large retrospective National Trauma Data Bank study analyzed 3107 adult patients who sustained severe chest trauma and required any type of lung resection found that wedge resection was performed in 54.3%, followed by lobectomy in 38.2% and pneumonectomy in 7.5%, with an overall morbidity and mortality of 32% and 27.5%, respectively.²⁴ Pneumonorrhaphy, wedge resection, tractotomy, and formal anatomic resection are all techniques that can be utilized to manage pulmonary parenchymal injury. The trauma surgeon should be facile with all of these techniques, and the choice of a particular one is driven by the patient's clinical condition and the extent and location of the pulmonary injury. The widespread adoption of a variety of surgical staplers has facilitated tractotomy and pulmonary resections. Pneumonorrhaphy is the simplest technique and is applicable to superficial pulmonary lacerations. These can be closed with an absorbable running simple or mattress suture. Injuries near the lung periphery can be excised by a stapled wedge resection. The lung is mobilized, any adhesions are lysed, and the inferior pulmonary ligament may need to be divided to improve lower lobe exposure. A stapled wedge resection is performed by grasping the parenchyma with a Duval or Foerster lung clamp and firing the stapler to excise the peripheral tissue. Tractotomy is rapid and ideal for through-and-through parenchymal injuries.²⁵ Placing the jaws of the stapler through the tract and firing it will open the tissue. Bleeding vessels or air leaks can then be individually ligated. Tissue thickness will determine the appropriate size staples, and at times, the staple line needs to be oversewn with a running absorbable suture.

More centrally located injuries, especially those close to the hilum, require special consideration. Precise understanding of the pulmonary vasculature and hilar anatomy are necessary to safely perform a stapled resection for centrally located injuries. If a nonanatomic resection is precluded, as with extensively damaged lung parenchyma, a formal lobectomy is necessary. Hilar injuries present a significant challenge. Hemorrhagic shock is invariably present, and the injury necessitates pneumonectomy, which carries a prohibitive mortality. Several techniques can be used to obtain hilar control, including lung torsion, manual compression, and application of a vascular clamp. Manual compression followed by clamping is the most widely used. Very proximal hilar injuries are particularly problematic, and opening the pericardium will occasionally allow vascular control.

MANAGEMENT OF SPECIFIC THORACIC VASCULAR INJURIES

Blunt Thoracic Aortic Injury

BTAI remains the second most frequent cause of mortality after blunt force trauma.^{26,27} An autopsy study conducted by

Teixeira and colleagues at Los Angeles County Hospital identified thoracic aortic injury (TAI) as a contributor in one third of automobile accident deaths. National Vital Statistics data suggest that the majority of deaths (80% to 85%) due to BTAI occur prior to the arrival to a hospital facility.²⁸

Among patients who survive to receive care, continued medical advances have improved the ability to expediently diagnose and effectively treat BTAI. Thoracic endovascular aortic repair (TEVAR) has replaced traditional open repair (OR) as the primary treatment modality utilized for BTAI among anatomically suitable patients.^{13,29–43} Among patients with severe BTAI, the subsequent use of TEVAR has been associated with improved morbidity and mortality compared with traditional OR approaches.^{13,29–32,44} Given these results, and following a 2011 analysis of the accumulated literature, the Clinical Practice Guidelines from the Society for Vascular Surgery (SVS) suggested that "endovascular repair be performed preferentially over open surgical repair or nonoperative management."⁴⁰

Diagnosis

The clinical presentation of BTAI can vary considerably. On arrival to a treating facility, patients may be asymptomatic or complain of pain in the chest or radiating to the neck, back, or shoulder. Normal vital signs do not rule out BTAI, as hemodynamics may range from normotensive to frank shock. In a recent study by the Aortic Trauma Foundation (ATF), hypotension was present on arrival in only 17.2% of patients.⁴⁵

The initial diagnostic test of choice is the plain CXR, which has a number of findings (widened mediastinum, left apical capping, depressed left main bronchus, indistinct or abnormal aortic contour, tracheal deviation, large left hemothorax) that are suggestive – but not confirmatory – of the diagnosis. Various definitions have been utilized to define pathologic widening of the mediastinum on plain radiography. These have included an 8-cm (supine) or 6-cm (upright) wide mediastinum at the level of the aortic knob, or a width at the same level that exceeds 25% of the total chest width.^{46,47} It is imperative, however, that any visualized abnormality undergo advanced imaging, particularly in the context of suspicious mechanisms of injury. It is also important to appreciate that a normal plain chest radiograph does not exclude TAI and – as previously discussed – has a low sensitivity for this purpose.¹⁰

Over the last two decades, advances in CT imaging technology have led to an increased overall sensitivity (96%–100%) and specificity (95%–99.8%), and has made CTA the gold standard for diagnosis of TAI.⁴⁸

Classification/Grading

BTAs represent a spectrum of lesions that range from intimal tears to free rupture. The mechanism often involves rapid deceleration, where the greatest point of strain is at the aortic isthmus (Fig. 181.5). Classification is based on the extent of injury to the anatomic layers of the aortic wall. Based on imaging, BTAI is classified into four grades: intimal tears (grade 1); intramural hematoma (grade 2); pseudoaneurysm (grade 3);

and free rupture (grade 4; Fig. 181.6).^{32,40} Grade 1 injuries do not cause an external aortic contour abnormality and are best visualized on CTA or IVUS. Angiograms in grade 1 patients may be interpreted as normal. Injuries involving the media, such as intramural hematomas or dissections, are considered grade 2. Grade 2 injuries cause an abnormality in the aortic contour and can be visualized on CTA, IVUS, or angiography. Pseudoaneurysms and ruptures (grade 3 and 4) are easily visualized with all imaging modalities. Among patients with BTAI who present to the hospital, approximately 50% are grade 3, followed by 25% grade 1, 15% grade 2 and 10% grade 4.⁴⁵

Additional classification systems based on contemporary imaging have also been proposed.^{32,49–53} Starnes and colleagues⁵² proposed a simplification of the SVS grading criteria into minimal, moderate and severe BTAI based on treatment differences among the three groups. Minimal aortic injuries (SVS grade 1 and 2) are defined as having no external contour abnormality and an intimal tear and/or thrombus <10 mm. The recommended treatment is medical therapy with antiplatelets (aspirin 81 mg) for 4 to 6 weeks and optional follow-up imaging. Moderate aortic injuries (SVS grade 3) are defined as having any external contour abnormality, such as a pseudoaneurysm, or intimal tear >10 mm. After stabilization of concomitant injuries, and only if these other injuries are deemed survivable, patients with moderate aortic injury are treated with antiplatelets and

anti-impulse therapy, followed by TEVAR in the first 24 to 72 hours. Severe aortic injuries (SVS grade 4) are those with active extravasation or a contained rupture with a left subclavian hematoma >15 mm and should be taken for immediate repair.⁵⁴ Accurate diagnosis (grade) of BTAI is critical because it affects management decisions and is an important tool in evaluating outcomes.

Blunt Thoracic Aortic Injury Treatment – Medical Therapy

Immediate management of all BTAI injuries, either as definitive therapy or during preparation for repair, includes anti-impulse therapy, which involves aggressive blood pressure and heart rate control. Effective pharmacologic suppression of aortic pressure fluctuations is utilized to reduce the stress on the injured aortic wall. These efforts have been shown to potentially reduce the risk of aortic rupture after BTAI diagnosis from 12% to 1.5%.⁵⁵ For grade 1 injuries, medical management alone is the mainstay of definitive therapy. For grade 2, 3, and 4 injuries, the SVS guidelines suggest medical management be utilized as a bridge to subsequent repair. However, contemporary evidence suggests that minimal aortic injuries (SVS grade 1 and 2) can be managed safely with medical therapy alone.^{36,37,45,54,56,57}

The optimal blood pressure goal and control regimen has not been well established, with different institutions having proposed various approaches. Data from the ATF international multicenter registry show the majority of institutions (66.3%) target a goal SBP <120 mm Hg, most commonly using a continuous titratable infusion of beta-blockers.⁴⁵ At our institution, we initiate anti-impulse therapy using a continuous esmolol infusion, with a target goal SBP <120 mm Hg and heart rate <60 beats per minute. For patients with known underlying congestive heart failure (CHF) or unable to tolerate large volume infusions, the esmolol solution is concentrated and administered via central venous access.

It is important to consider that specific patterns of associated traumatic injury may affect the ability to employ medical therapy for BTAI. Specifically, patients with traumatic brain injury (TBI) requiring optimization of cerebral perfusion pressure (CPP) may require elevation of blood pressure to optimize

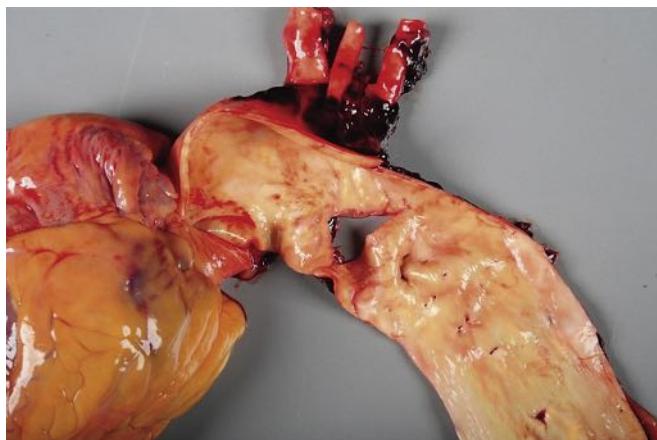


Figure 181.5 Postmortem aorta with injury in the descending aorta at the isthmus.

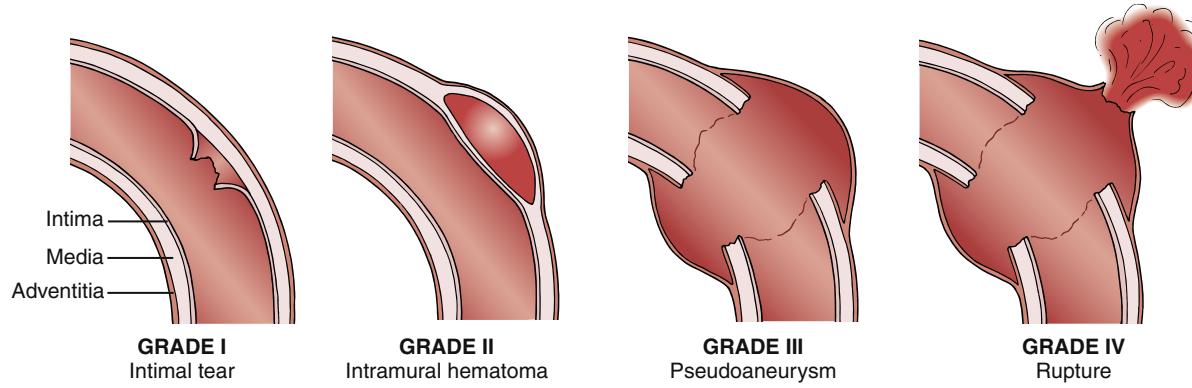


Figure 181.6 Classification of Traumatic Aortic Injury.

neurologic outcome. This required elevation is counter to the medical management of BTAI and may necessitate earlier intervention for aortic injury to facilitate optimal brain injury outcome. Given the common complexity of severely injured trauma patients, it is advisable that all discussions about the appropriateness of medical therapy and timing of potential BTAI repair be undertaken in a collaborative fashion by the key stakeholders in care.

The managing team must also remain vigilant for other indications to abort medical therapy, including obvious signs of malperfusion or possible early progression of BTAI while undergoing medical management. In the largest series to date, the Aortic Trauma Foundation (ATF) international prospective multicenter registry analyzed demographics, injury characteristics, management and outcomes of patients with BTAI in whom medical management was initiated as definitive therapy. A total of 432 patients were evaluated, of which 114 (26.4%) were treated with medical management (MM) as planned definitive therapy. Twelve (10.5%) patients (grade 1, 1; grade 2, 0; grade 3, 10; grade 4, 1) required subsequent intervention after MM, 11 (9.6%) of whom underwent thoracic endovascular aortic repair (TEVAR), while one (0.9%) required open repair. Overall in-hospital mortality for patients selected for MM was 7.9%. There were no aortic-related deaths in patients undergoing MM.^{13,45}

Blunt Thoracic Aortic Injury Repair

While prompt repair of BTAI is preferred, existing data suggest that selective delayed repair may result in optimal outcomes.^{33,58} This approach is commonly required among trauma patients, requiring treatment of more immediately life-threatening injuries, such as laparotomy or emergent craniotomy – a common occurrence among the severely injured cohort.^{5,13} Until definitive intervention for BTAI is undertaken, medical management utilizing anti-impulse therapy is performed in an appropriate intensive care unit. The subsequent timing of definitive repair is then individualized for the specific patient through a multidisciplinary, decision-making process.

Once the optimal timing of intervention for the individual patient has been established, endovascular repair has emerged as the mainstay of subsequent definitive BTAI treatment in the modern era. The evolution in endovascular technologies occurring over the last decade or more has afforded that TEVAR can be safely and effectively performed in the majority of patients with BTAI. In the largest multicenter retrospective BTAI examination to date, conducted by the ATF between 2008 and 2013,¹³ TEVAR was the treatment utilized for 76.4% of the 382 BTAI patients studied, with only 23.6% requiring OR.

Documented improved outcomes following TEVAR for the treatment of BTAI are due both to increased experience with endovascular approaches to treatment and device advances. The first endovascular device approved by the US Food and Drug Administration (FDA) for the treatment of thoracic aortic aneurysms was the Thoracic Aortic Graft (TAG; W.L. Gore & Associates, Flagstaff, AZ) in 2005. This device was initially used for TEVAR in patients with BTAI in an off-label

indication. Subsequent introduction of a variety of on-label FDA-approved devices in 2008 has increased the tools available for definitive treatment of these injuries by effective endovascular means.

The technique utilized for endovascular repair at our institution has been described previously.³⁰ In summary, all endovascular procedures are performed under general anesthesia in a hybrid operating room equipped with fixed imaging equipment (Axiom, Siemens Medical, Malvern, PA). Intraoperatively, the chest, abdomen and bilateral groins are prepped in standard fashion. An arch aortogram is performed through femoral access, and the location of the injury is confirmed. The cerebrovascular anatomy is evaluated based on the arch angiogram, especially if left subclavian artery coverage is planned. IVUS is a useful imaging adjunct that can confirm aortic diameter as well as location of the injury, and can be used selectively based on the discretion of the attending surgeon. The patient is anticoagulated using a weight-based heparin protocol (100 IU/kg) if there are no contraindications. Otherwise, a smaller dose of heparin (3000 to 5000 IU) is administered.

The thoracic device is selected based on CT images according to the manufacturer's sizing recommendations. Measurements are made based on two-dimensional, thin-cut axial CT scans with IV contrast. The device is delivered and deployed using a standard technique without any pharmacologic adjunct; extension pieces may be deployed as indicated. The subclavian artery is covered, as needed, to obtain a proximal landing zone, or gain better apposition with the lesser curvature of the aortic arch. We maintain a policy of selective delayed subclavian artery revascularization for such cases.⁵⁹ However, if the patient has absolute contraindications to left subclavian coverage, such as previous coronary artery bypass graft using the left internal mammary artery, an urgent carotid–subclavian bypass should be performed at the time of TEVAR. Post deployment balloon angioplasty is performed selectively when incomplete apposition of the graft at the proximal landing zone is noted. The heparin is then reversed with protamine. Postoperatively, patients are returned to the surgical-trauma intensive care unit and discharged following stabilization of their other injuries. A diagnostic and completion angiogram of a patient with a grade 3 BTAI treated with a GORE® TAG® Thoracic Branch Endoprosthesis (TBE; W. L. Gore & Associates) is shown (Fig. 181.7). A follow-up CTA shows successful exclusion of the pseudoaneurysm.

Select patients with unsuitable anatomy or other confounding factors may continue to require open surgical repair. In the modern era, OR is reserved for specific scenarios. Among patients showing signs of hemodynamic compromise and the need for emergent repair, not all facilities will have the capability to martial endovascular resources in the required time frame to optimize outcome. While rapid mobilization of endovascular resources is inherent to many modern trauma centers, local capabilities must be considered.

Other considerations in selecting OR include the consideration of patients anatomically unfavorable for TEVAR. The foremost criteria determining the need for OR is the absence of an adequate proximal landing zone to allow for proper “seal”

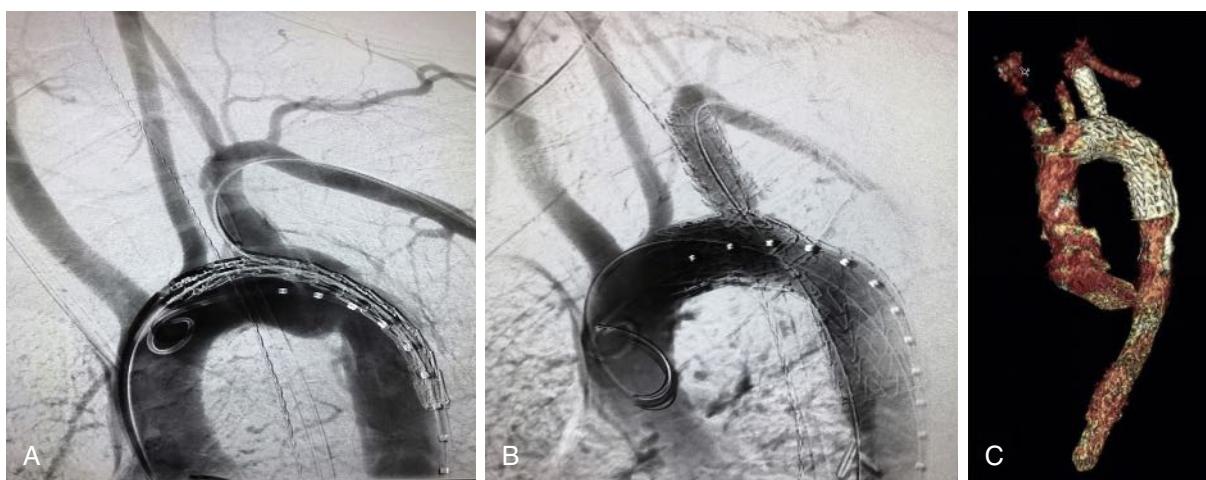


Figure 181.7 (A) Diagnostic and (B) completion angiography of a patient with a grade 3 blunt thoracic aortic injury treated with a GORE® TAG® Thoracic Branch Endoprosthesis (TBE). (C) Follow-up computed tomography angiography shows successful exclusion of the pseudoaneurysm.

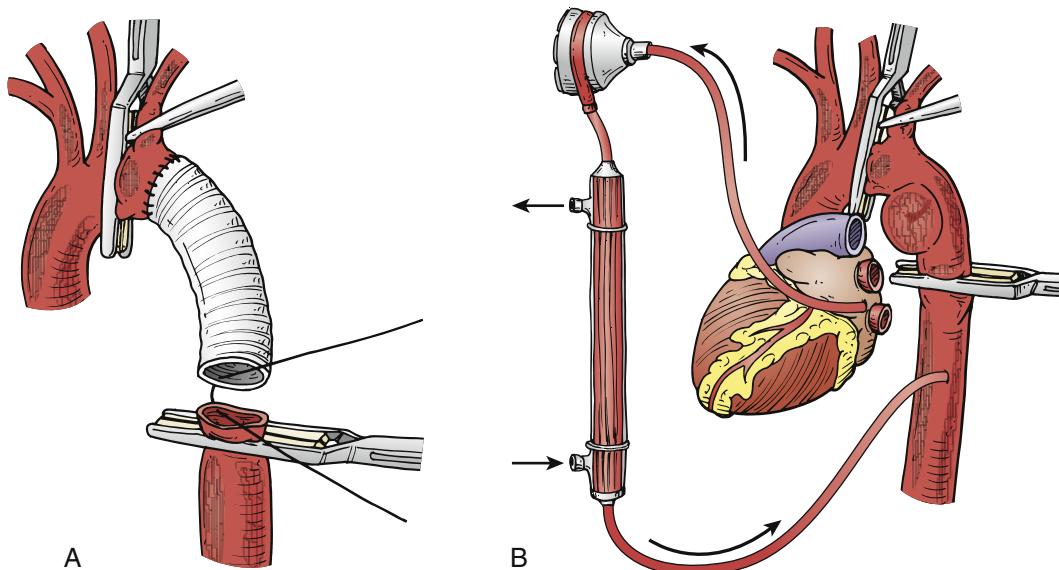


Figure 181.8 Open repair of blunt thoracic aortic injury can be performed using (A) a clamp and sew technique or (B) distal aortic perfusion.

of the site of injury by the device. In approximately 40% of BTAI patients, left subclavian artery coverage is required to achieve this objective.¹³ Available data suggest that the majority of trauma patients requiring left subclavian artery coverage in this setting will have good short- and mid-term outcomes without the need for subsequent bypass.^{13,59} Other anatomical criteria that may preclude the ability to safely conduct TEVAR for BTAI include small or diseased iliofemoral vessels, with diameters of these vessels less than 7 mm being a risk factor for access site complications, including dissection, rupture, and hematoma formation.

When OR is required, a number of approaches can be considered to facilitate BTAI treatment, depending on the site and length of the descending thoracic aorta involved. Given that the most commonly encountered location of BTAI is in the region of the isthmus, a left posterolateral thoracotomy to enter the chest through the fourth space is usually the most

expeditious and optimal incision choice. Once the chest is opened, the initial objective is to acquire proximal and distal control around the area of injured thoracic aorta. The proximal clamp is typically applied between the left common carotid and left subclavian arteries, with care to avoid injury to the recurrent laryngeal nerve, while the distal clamp is placed at some point on the distal descending thoracic aorta distal to the zone of injury (Fig. 181.8A).

Depending on the clinical scenario and patient condition, a distal aortic perfusion strategy should be expediently developed. The most expeditious technique is “left-heart bypass,” which can be performed through cannulation of the left inferior pulmonary vein and distal thoracic aorta (see Fig. 181.8B). Rarely, the proximal clamp positioning is untenable. In this scenario, a clampless proximal anastomosis can be performed under circulatory arrest with full cardiopulmonary bypass (CPB), commonly through a femoral artery and a femoral vein

cannulation. If total circulatory arrest is warranted, then careful venting of the left ventricle during times of cooling and rewarming is crucial. Due to risk of aortoesophageal fistula, the surgeon should avoid using the inclusion technique for the proximal anastomosis and should completely dissect the posterior aorta off the esophagus.

One of the caveats, which is often problematic for the poly-trauma patient, is the need for systemic anticoagulation with the use of bypass. For left-heart bypass, this is achieved through administration of intravenous heparin at a dose of 100 U/kg to achieve an activated clotting time (ACT) of more than 200 seconds. In cases where CPB is required, heparin is used at a dose of 400 U/kg to achieve an ACT of greater than 480 seconds. Once the patient is on bypass and the perfusion circuit is satisfactory, the periaortic hematoma is incised, the extent of injury explored, and the ensuing aortic repair undertaken. It is paramount that the aortic adventitia is incorporated into the subsequent suture lines, as this layer provides the majority of the tensile strength of the aorta. The patient is gradually rewarmed during the latter phase of the anastomosis to facilitate removal of the clamps at a moderate degree of hypothermia (32°C to 34°C). Once the clamps are off, the patient is weaned off the bypass until adequate hemostasis is achieved and the wound is closed in its respective layers.

A significant consideration when undertaking open BTAI repair is spinal cord protection and the importance of distal aortic perfusion in minimizing the risk of postoperative paraplegia.⁶⁰ Several potential strategies designed to mitigate the risk of postoperative paraplegia have previously been described in the literature.^{61–65} Perhaps the most significant contributor to spinal cord ischemia occurs as a result of aortic clamping and subsequent occlusion of critical segmental spinal cord arterial branches. Important factors in determining the incidence of immediate-onset or delayed paraplegia following aortic repair include duration of cross-clamping, level and length of aortic segment excluded by clamping, duration of systemic hypotension, cerebrospinal fluid (CSF) pressure, distal aortic pressure, and the number of intercostals ligated during repair.⁶⁶ Multiple adjuncts have helped lower the incidence of paraplegia following aortic repair for BTAI, including CSF drainage, generalized and localized hypothermia, avoidance of hypotension and hypoxemia, maintaining hemoglobin >10 mg/dL, as well as reattachment of key intercostal arteries during the conduct of repair.^{63–66}

Postoperative Management

The hemodynamic state of the patient is closely monitored following the procedure, and meticulous care is provided for any other trauma they have experienced. Ideal blood pressure parameters are largely dependent on the presence of concomitant injuries. In patients with concomitant TBI or spinal cord injury (SCI), a higher mean arterial pressure is maintained for improved cerebral or spinal cord perfusion pressure. Significant vigilance must be maintained for any of the common complications that can arise following aortic trauma, including bleeding, infection, and neurologic changes. There is no data to

support continuation of prophylactic antibiotics in the postoperative period. Long-term data on durability and major complications is limited to less than 10 years, as widespread adoption of TEVAR did not occur until the mid-2000s. Knowledge of optimal follow-up practice is, subsequently, limited. At present, the follow-up surveillance strategy utilized at our center includes clinical examination and CTA at 1, 6, and 12 months, and yearly thereafter. Patients treated nonoperatively should be advised to avoid heavy lifting, excessive straining and strenuous activity until injury healing is confirmed on follow-up imaging. However, routine exercise at a light to moderate level is still acceptable.

Potential Complications of Thoracic Endovascular Aortic Repair

Current data suggest that in comparison to OR, TEVAR may reduce early death, paraplegia, renal insufficiency, transfusions, reoperation for bleeding, cardiac complications, pneumonia, and length of hospital stay.^{13,32–34} In their landmark 2008 report, Demetriades and colleagues of the AAST BTAI study group³⁴ documented significant improvements in BTAI care associated with the transition from open to TEVAR. In their examination of 193 patients with BTAI, they found that TEVAR was associated with significantly decreased transfusion requirements and lower mortality compared with OR. The more contemporary findings from the ATF study group experience support these findings, with a significantly lower PRBC requirement (mean 5.9 vs. 3.1 units, $P < 0.002$) in the first 24 hours and a lower overall mortality (8.6% vs. 19.7%, $P = 0.021$) and aortic-related mortality (13.1% vs. 2.5%, $P = 0.003$) among TEVAR-treated patients compared with open counterparts.¹³

The common concerns for paralysis and stroke that have been associated with OR appear to also be mitigated with the increasing use of TEVAR. In the aforementioned ATF multi-center study of 382 BTAI patients,¹³ only 1 paralysis following TEVAR was noted, occurring in an 81-year-old male requiring 20-cm device coverage of the thoracic aorta. Likewise, stroke was a very rare occurrence, identified in only two patients, ages 62 and 85, respectively. Both required coverage of the left subclavian artery to facilitate TEVAR. These findings support the safety profile for TEVAR in the setting of BTAI, but suggest that older patients with possible native atherosclerotic burden and BTAI patterns requiring more extensive endograft coverage may increase risk of ischemic complications.

Access- and device-related complications also appear to be rare complications of TEVAR in contemporary practice. This improvement in safety profile has emerged as a benefit of increased experience and improvements in device technology. In the contemporary published experience of the ATF Study Group, which captured the treatment of 382 BTAI patients, a low risk of TEVAR-related complications was documented.¹³ This group noted six malpositions of the endograft at initial TEVAR occurred (3.0%) and a post-TEVAR endoleak rate of 2.5%. Only one delayed stent migration was noted. In addition, just two access site complications (one pseudoaneurysm, one persistent bleeding requiring intervention) were identified.

Among the six defined TEVAR treatment failures encountered in this large series, all underwent subsequent salvage with re-intervention (two repeat TEVAR, four OR). The ATF study group identified only one patient treated with repeat TEVAR who suffered aortic-related mortality. No other mortalities were observed among the TEVAR failures.

Although increasingly rare in the modern era, it is important to note that complications during or after TEVAR remain possible in specific settings. Excessive oversizing or undersizing of endografts can lead to propagation of aortic injury or failure to achieve adequate seal. Patient-specific anatomical issues may also contribute, including the presence of a tight curvature of the aortic arch, native atherosclerotic burden and the aforementioned diameter limitations of iliac access vessels.

Outcomes After Blunt Thoracic Aortic Injury Management

In the 2015 ATF multicenter retrospective study of BTAI outcomes, 382 patients were included from nine American College of Surgeons–verified Level I trauma centers in North America.¹³ This study captured a distribution of BTAI grades of injury (94 grade 1 injuries [24.6%], 68 grade 2 [17.8%], 192 grade 3 [50.3%], and 28 grade 4 [7.3%]). Overall in-hospital mortality for all patients with BTAI was 18.8% (nonoperative management 34.4%, OR 19.7%, TEVAR 8.6%), with an aortic-related mortality of 6.5% (nonoperative management 9.8%, OR 13.1%, TEVAR 2.5%).

Among the ATF BTAI study patients, the majority of aortic-related deaths (18/25) occurred prior to the opportunity for completion of OR or TEVAR. Of the seven patients who died after OR (3) or TEVAR (4), all had an Injury Severity Score (ISS) in excess of 25, consistent with severe polytrauma. Using multivariate logistic regression, the investigators found that the independent predictors of aortic-related mortality included higher overall ISS and higher grade of BTAI injury. On multivariate regression, TEVAR utilization proved the only variable protective against aortic-related mortality.

Remaining Controversies and Future Directions

The current SVS guidelines for BTAI management⁴⁰ provide an evidence-based, consensus-derived grading system and suggested course of treatment. These guidelines represent the most comprehensive effort in BTAI care optimization yet conducted by a major medical organization. The SVS guidelines do, however, have some limitations – including the fact that they are specific to the aortic lesion alone. Potential future improvements include further refinement of injury grading paradigms, standardization of care, and the establishment of effective prospective long-term follow-up mechanisms. A number of initiatives are actively underway to promote these needed advances.

There has been a continued debate over the management of “minimal aortic injuries” (SVS grade 1 and 2), specifically with respect to management of grade 2 BTAI. The SVS clinical practice guidelines currently recommend TEVAR for grade 2 injuries, however recent literature has shown that these injuries

can be safely managed nonoperatively, with very low rates of disease progression and aortic related mortality.^{36,37,56,57,67} The largest-to-date study regarding the natural history of nonoperatively managed grade 2 BTAI demonstrated no evidence of disease progression up to nearly a 5-year follow-up.⁵⁶ This study corroborated the findings of multiple other studies,^{36,37,57,67} including Starnes and colleagues, showing that patients with “minimal aortic injury” managed nonoperatively did not have progression of disease and had no aortic-related deaths.⁵⁴ Recent data from the ATF international registry also demonstrated no failures of medical therapy in grade 2 patients and no aortic-related deaths.⁴⁵ While the existing data suggest that the nonoperative management of SVS grade 2 injuries is safe, further studies with longer term follow-up are required, ideally in the context of a prospective multicenter study.

Another challenge includes consideration of the fact that BTAI patients commonly have significant associated injuries that are pertinent to overall outcome. In some instances, the optimal care of these associated injuries proves counter to optimal BTAI care, a common example being TBI which has been reported in 20% to 50% of BTAI patients.⁵⁸ Hypotension and hypoxia can be detrimental in patients with TBI and lead to increased risk of secondary brain injury. However, the need to maintain an optimal cerebral perfusion pressure among patients with TBI contrasts with the pharmacologic blood pressure impulse control recommended to minimize wall stress in BTAI. In these cases, early TEVAR should be considered to allow for the increased mean arterial pressures required to maintain adequate cerebral perfusion. In patients with intracranial hemorrhage or other hemorrhagic complications of trauma, TEVAR can be performed with minimal or no anticoagulation with low risk for thrombotic complications.⁶⁸

Complex scenarios such as concomitant BTAI/TBI should be managed using a multidisciplinary approach and on an individualized basis, as early TEVAR can have significant risks, including aortic-related morbidity and mortality. Rabin and colleagues retrospectively studied patients with BTAI and concomitant moderate to severe TBI over 12 years, and found that early aortic intervention (<24 h) was independently associated with worsening TBI regardless of repair modality or anticoagulation use. Early aortic repair was also associated with increased aortic morbidity and mortality.⁵⁸ Further investigation is warranted to determine the ideal timing of intervention in patients with concomitant BTAI and TBI. An optimal algorithm (see Chapter Algorithm) should incorporate some consideration of associated injuries into individualized BTAI care.

Ideal timing of BTAI treatment is a remaining issue that requires further study. The results of the American Association for the Surgery of Trauma Aortic Injury Study Group, reported in 2009, suggested that improved outcomes were associated with initial medical management, including blood pressure optimization.³³ This group found that patients treated in a delayed (>24 hours) fashion, after this period of optimization, had improved survival compared with BTAI patients treated operatively within less than 24 hours. This is contrary to the SVS guidelines which recommend urgent (<24 hours) TEVAR. There remains a need, however, to adequately define whether

specific risk factors associated with BTAI represent a higher risk for early aortic rupture and need for more emergent repair.

Finally, the long-term durability of endovascular devices used for BTAI treatment remains to be determined. Lower profile delivery systems, flexible design, controlled deployment and improved conformability to aortic contour are some of the attractive features of the later-generation devices. The ongoing study of branched graft devices may soon obviate the need for coverage of the left subclavian artery during TEVAR entirely. However, this remains an unresolved issue of BTAI care. Optimal graft sizing and graft utilization in pediatric patients – or patients with small aortic diameters – are also inadequately studied issues. These challenges are augmented by the fact that optimal device indications and utilization has primarily been subjected only to industry-funded study. These issues require more objective investigation in a large, multicenter fashion.

VASCULAR INJURIES TO THE AORTIC ARCH VESSELS AND THORACIC OUTLET VASCULATURE

While TAI is perhaps the hallmark thoracic vascular injury, a wide variety of traumatic pathologies at various locations within the chest can be encountered.⁶⁹ The majority of these injuries are initially diagnosed with contrast-enhanced CT, now ubiquitous to initial trauma evaluations. As with TAI, the patient condition and associated injuries remain considerations in defining optimal management of identified vascular injuries. For unstable patients requiring immediate surgical

intervention, particularly after penetrating mechanisms, initial open repair approaches are commonly utilized. The aforementioned issues regarding initial incision and subsequent extensile options should be considered carefully in these instances.

Ascending Aorta and Transverse Arch

Rupture of the ascending and transverse aortic arch is uncommon, but the exact incidence is unknown due to the lethality of these injuries. A widened mediastinum and cardiac tamponade are frequently associated with ascending aortic ruptures. Repair of these injuries requires a median sternotomy, CPB, and systemic heparinization (Fig. 181.9). Management of the aortic tear may be primary or with an interposition graft. Special attention should be paid to the status of the aortic valve. Injuries to the aortic arch may require hypothermic circulatory arrest and associated antegrade and retrograde cerebral perfusion techniques. Survival depends primarily on the severity of associated injuries.^{70–72}

Innominate Artery

Rupture of the innominate artery is the second-most common thoracic arterial injury following blunt trauma. Innominata artery injuries can generally be repaired via a median sternotomy with a right cervical extension when necessary. Blunt injury typically involves the base of the innominate artery, and this is most expeditiously repaired with a bypass from the ascending aorta to the distal innominate artery, followed by oversewing of the innominate stump (Fig. 181.10). Division of the

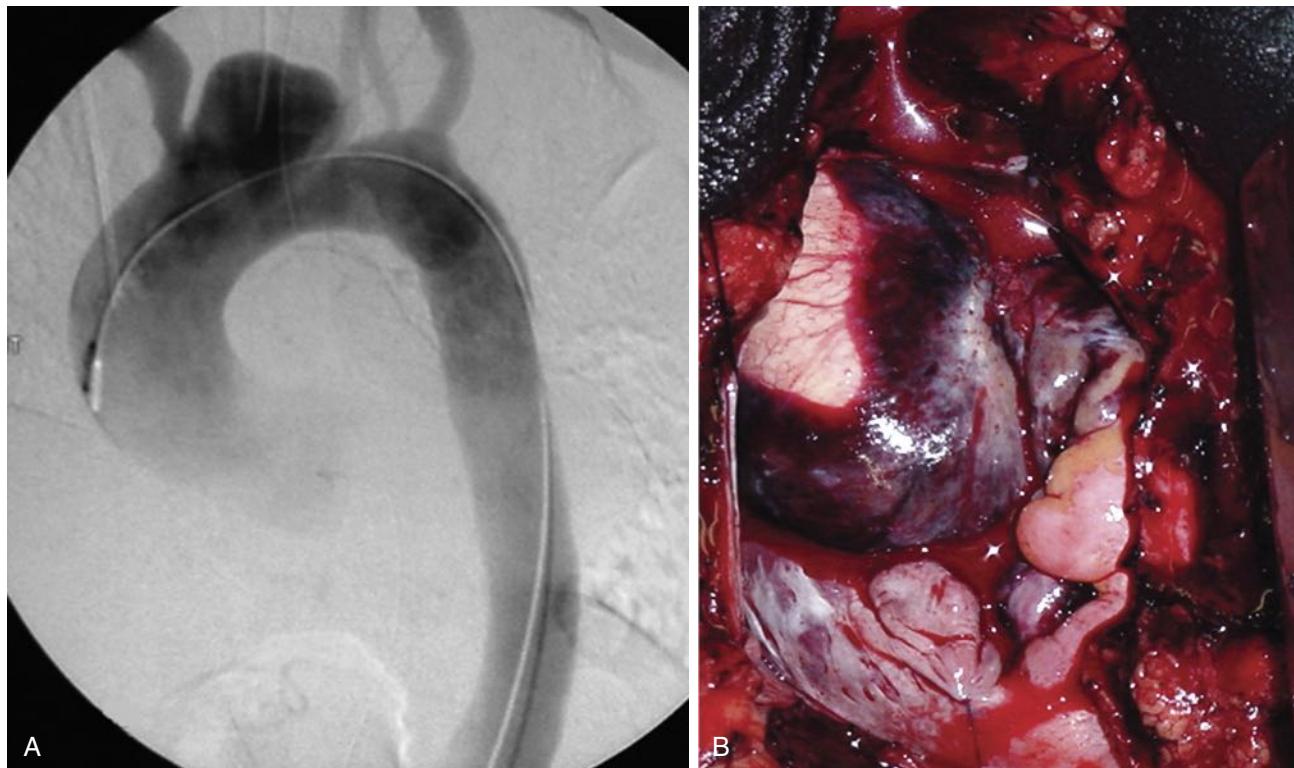


Figure 181.9 Blunt Trauma to the Aortic Arch. (A) Diagnostic arch angiogram shows a large pseudoaneurysm. (B) Intraoperative photograph after median sternotomy. (Courtesy Dr. Anthony L. Estrera.)

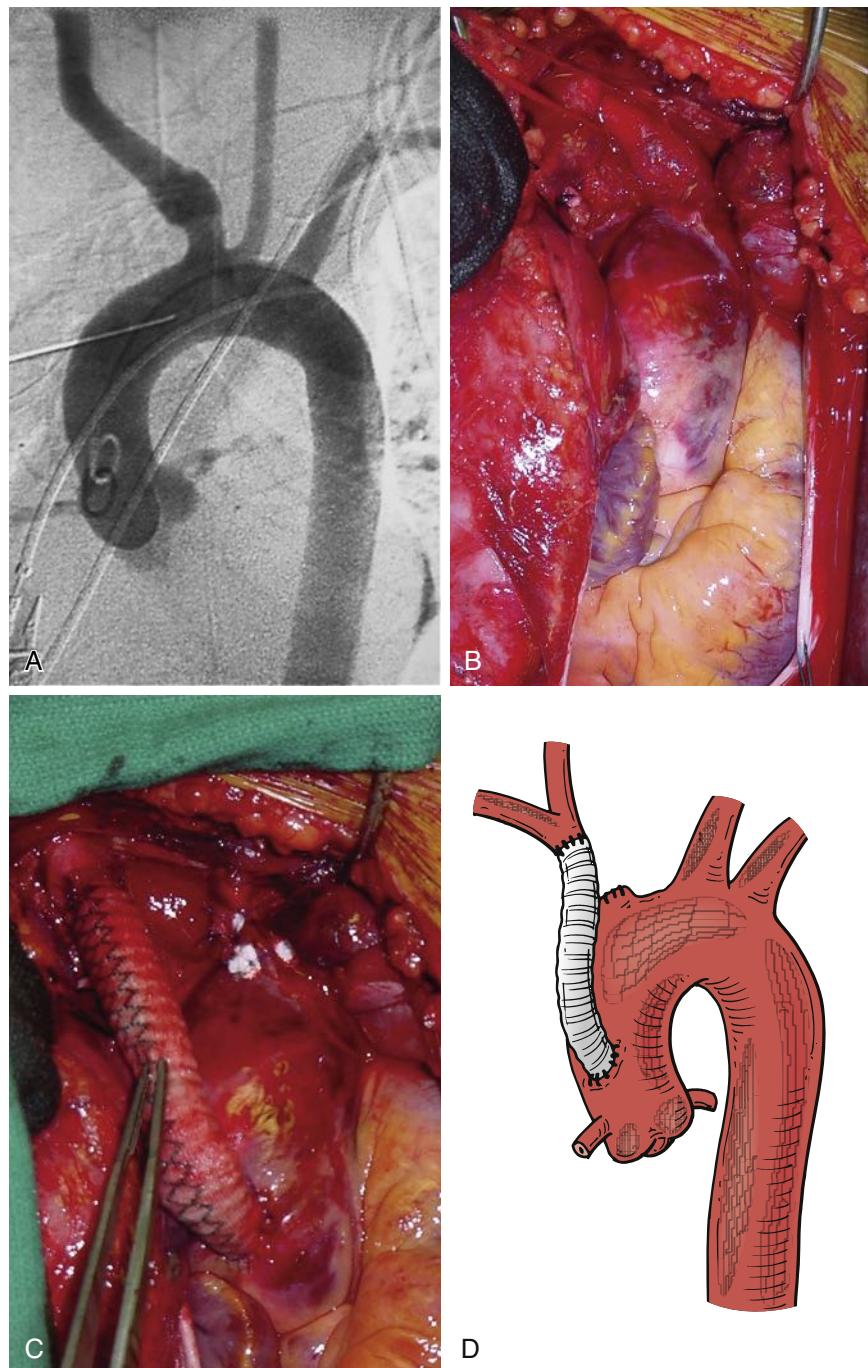


Figure 181.10 Repair of Innominate Artery Injury. (A) Diagnostic angiogram. (B) Intraoperative image showing blunt injury to the proximal innominate artery. (C and D) Placement of an ascending aorta to innominate artery bypass with oversewing of the innominate artery stump. (Courtesy Dr. Anthony L. Estrera.)

innominate vein is occasionally required for exposure, and shunts or CPBs are often not required. Avoidance of the injured area until completion of the bypass leads to a technically easier repair.^{73,74}

Left Common Carotid Artery

The surgical approach to injuries of the proximal left carotid artery mirrors that of the innominate artery – a sternotomy with a left cervical extension, if needed. With injuries of the left carotid origin, bypass graft repair is generally preferred over an end-to-end reanastomosis.⁷⁵ The management of a

carotid injury in the setting of neurologic disturbances is controversial. Generally, if the patient is evaluated soon after injury, revascularization is recommended because hypotension (rather than ischemic infarct) is the most likely cause of morbidity.⁷⁶ Chapter 180 (Vascular Trauma: Head and Neck) contains additional information on carotid injuries. Recently, traumatic carotid lesions have been managed with endovascular techniques. An endovascular approach is especially useful for extensive lesions with involvement near the skull base, where obtaining proximal and distal vascular control may result in increased morbidity. The goal of endovascular therapy is the elimination of a fistula, aneurysm, or stenosis

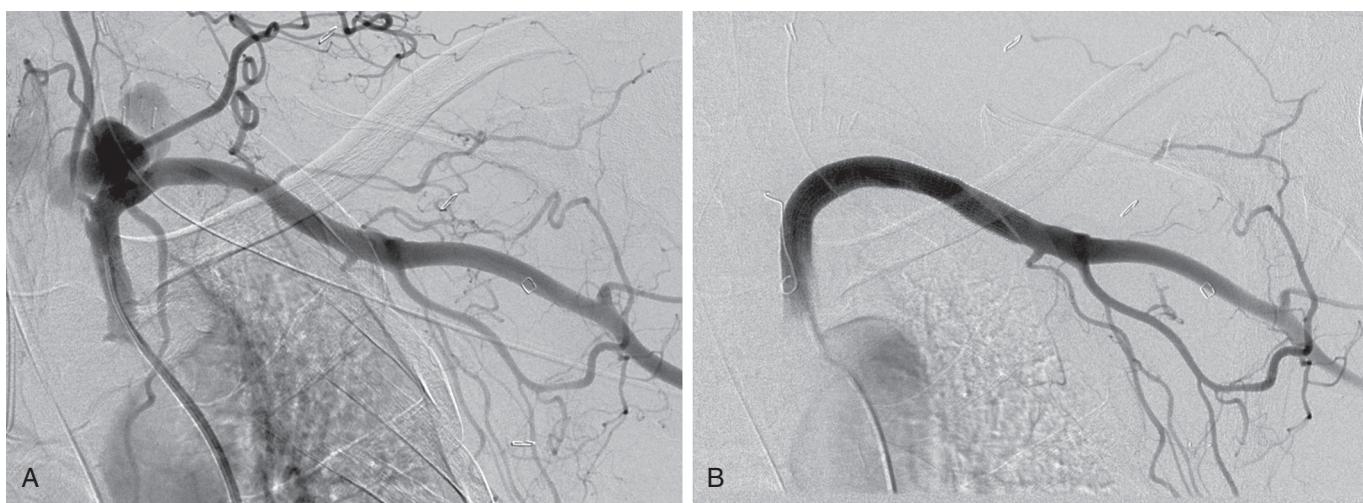


Figure 181.11 (A) Selective left subclavian angiogram of a patient after a stab wound shows a pseudoaneurysm. (B) After coil embolization of the left vertebral artery, endovascular repair of the left subclavian artery injury was performed using a covered stent graft. (Courtesy Dr. Kristofer M. Charlton-Ouw.)

while preserving native flow to the brain. There have been multiple reports on the successful management of traumatic carotid injuries with endovascular techniques.^{72–79} However, the use of stents introduces new considerations, such as the probability of late in-stent thrombosis. The administration of postprocedural medications, such as aspirin and clopidogrel, can minimize the incidence of in-stent thrombosis.^{80,81}

Subclavian Vessels

Injuries to the subclavian vessels are usually caused by penetrating wounds. Subclavian vascular injuries require pre-operative imaging (generally CTA) for appropriate incision planning. Injuries to the right subclavian artery are best addressed via a median sternotomy with a right cervical extension. Proximal control of left-sided subclavian injuries can be obtained via a left anterolateral thoracotomy through the fourth intercostal space. A separate supraclavicular incision can be used for distal control. These two incisions can be connected with a sternotomy to facilitate exposure. This incision should be used sparingly because of reports of post-operative “causalgia” neurologic symptoms.⁷⁵ In addition, the second or third portion of the subclavian artery can usually be exposed without the need for clavicular resection or sternotomy.

Endovascular approaches to innominate, intrathoracic carotid, and subclavian arterial injuries have been described in both blunt and penetrating trauma (Fig. 181.11).^{82–84}

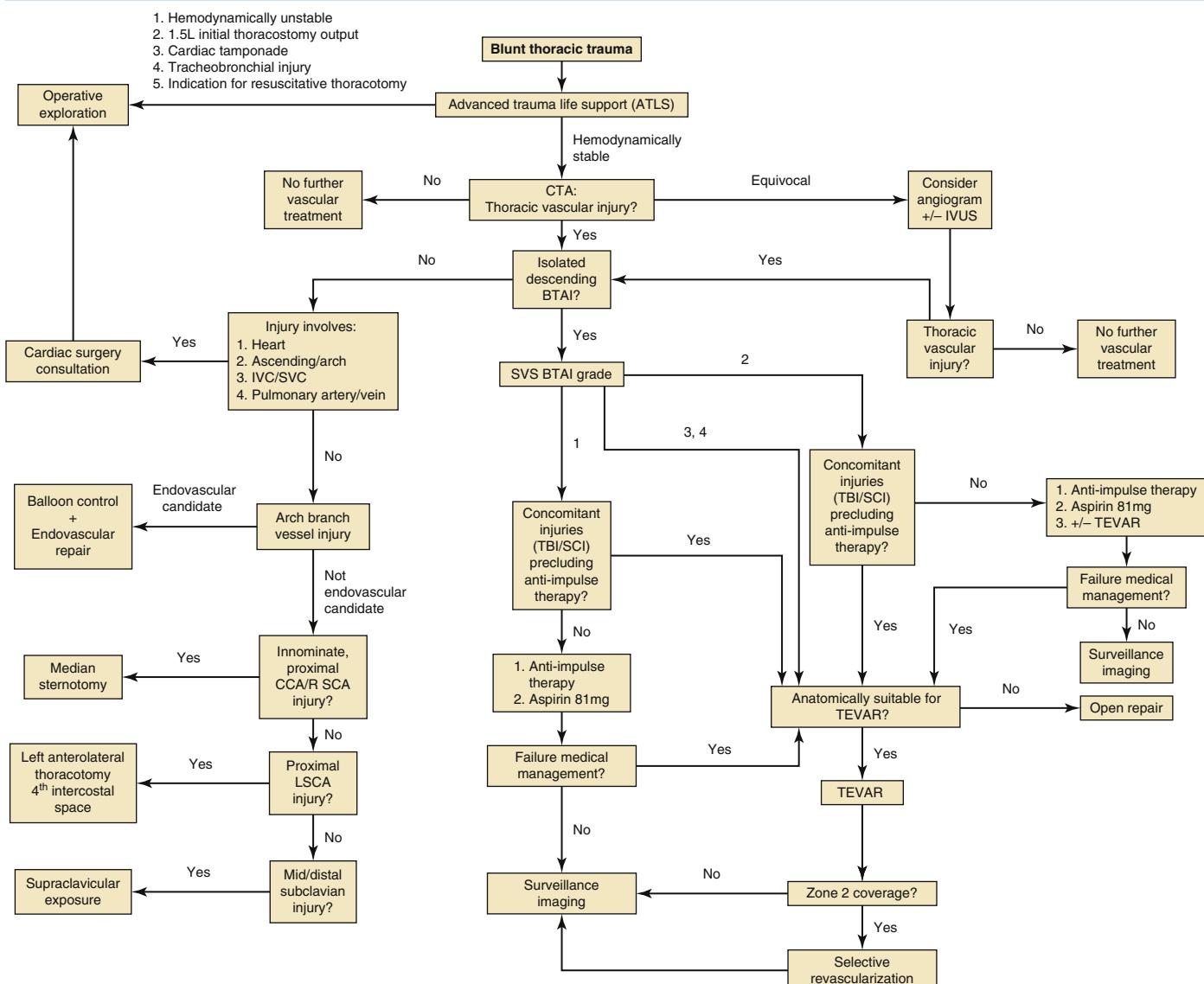
As with TAI, endovascular capabilities are increasingly being utilized to effectively treat vascular injuries to the vessels of the thoracic arch and thoracic inlet.^{66,85–88} Available data suggest that, among appropriately selected patients, endovascular treatment is associated with improved outcome for injuries in these areas – which often represent challenging open exposures for control and repair. In a recent study conducted by Branco and colleagues, investigators found that endovascular treatment of axillo-subclavian injuries was associated

with lower in-hospital mortality and surgical site infections than counterparts undergoing open repair of these injuries.⁸⁵ A subsequent report used a large cohort of patients from the National Trauma Databank to compare matched populations of vascular injured patients undergoing open or endovascular repair.⁸⁶ The study found that outcomes were improved across a wide range of vascular thoracic vascular injuries. Additional literature supports these findings.^{69,87} While this evidence suggests that endovascular repair modalities may have improved outcomes compared to open repair of a variety of thoracic vascular injuries during the initial hospitalization, there remains a significant paucity of long-term outcomes. There remains a need to capture this data in order to better define if endovascular repair is to be considered definitive or simply a temporizing/damage control adjunct over the lifespan of a young trauma patient. To this end, the PROspective Vascular Injury Treatment (PROOVIT) registry, developed under the auspices of the American Association for the Surgery of Trauma, is capturing data in a multicenter, multinational fashion to better define the optimal management and follow-up of these injuries.⁶⁹

CONCLUSION

Thoracic vascular injuries remain significant challenges of modern trauma vascular care. Significant strides have been made with regard to diagnostic and initial management capabilities, facilitated by improvements in contrast-enhanced CT imaging and endovascular treatment modalities. There remains, however, a need to further define optimal treatment algorithms and long-term outcomes after these injuries. Ongoing efforts in this regard, including the Aortic Trauma Foundation Prospective International Multi-Center, TAI registry and the PROOVIT registry, promise to advance additional understanding about the optimal diagnosis, management, and follow-up of these injuries.

CHAPTER ALGORITHM



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A complete reference list can be found online at www.expertconsult.com.

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Vascular Trauma: Abdominal

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Abdominal vascular injuries are the most common cause of death after penetrating abdominal trauma. Accurate diagnosis, rapid surgical exposure and control, and the definitive management of these injuries may challenge the skills and judgment of even the most experienced surgeons. These can be made even more complex by any associated intraabdominal injuries. Rapid transportation to a trauma center, early recognition of the injuries, early surgical intervention, excellent knowledge of the anatomy, and good surgical judgment are critical for optimizing patient survival.

SURGICAL ANATOMY

For vascular trauma purposes, the abdomen is conventionally divided into the intraperitoneal and retroperitoneal areas. The retroperitoneum is further divided into four distinct zones (Fig. 182.1):

- Zone 1, which includes the midline retroperitoneum extending from the aortic hiatus to the sacral promontory. This zone is subdivided into the supramesocolic and inframesocolic areas. The supramesocolic area contains the suprarenal aorta and its major branches (celiac axis, superior mesenteric artery [SMA], and renal arteries), the supramesocolic inferior vena cava (IVC) with its major branches, and the superior mesenteric vein (SMV). The inframesocolic area contains the infrarenal aorta and IVC.
- Zone 2 (left and right), which includes the kidneys, paracolic gutter, and renal vessels.
- Zone 3, which includes the pelvic retroperitoneum and contains the iliac vessels.
- Zone 4, which includes the perihepatic area containing the retrohepatic IVC and hepatic veins.

EPIDEMIOLOGY

Penetrating trauma is responsible for most abdominal vascular injuries and accounts for approximately 90% of cases in urban trauma centers.¹ Low-velocity missiles cause direct injury to the vessel. High-velocity missiles and blast can also cause injury by means of a shock wave and transient cavitation. These injuries may manifest as early or late thrombosis, in addition to hemorrhage.

In patients who undergo exploratory laparotomy for injury, the incidence of vascular trauma is 14.3% for gunshot injuries,² 10% for stab wounds,³ and 3% for blunt injuries.⁴

Blunt abdominal trauma may cause vascular injuries by one of three mechanisms:

1. Rapid deceleration, as occurs in high-speed vehicular collisions or falls from height; this can cause avulsion or intimal tearing and subsequent thrombosis.
2. Direct anteroposterior crushing, from a seat belt or a direct blow to the anterior abdomen.
3. Direct laceration of a major vessel by a bone fragment, for example with severe pelvic fractures.

Abdominal arterial and venous injuries occur with the same incidence. In a review of 302 abdominal vascular injuries from our center, the incidence of arterial injuries was 49% and that

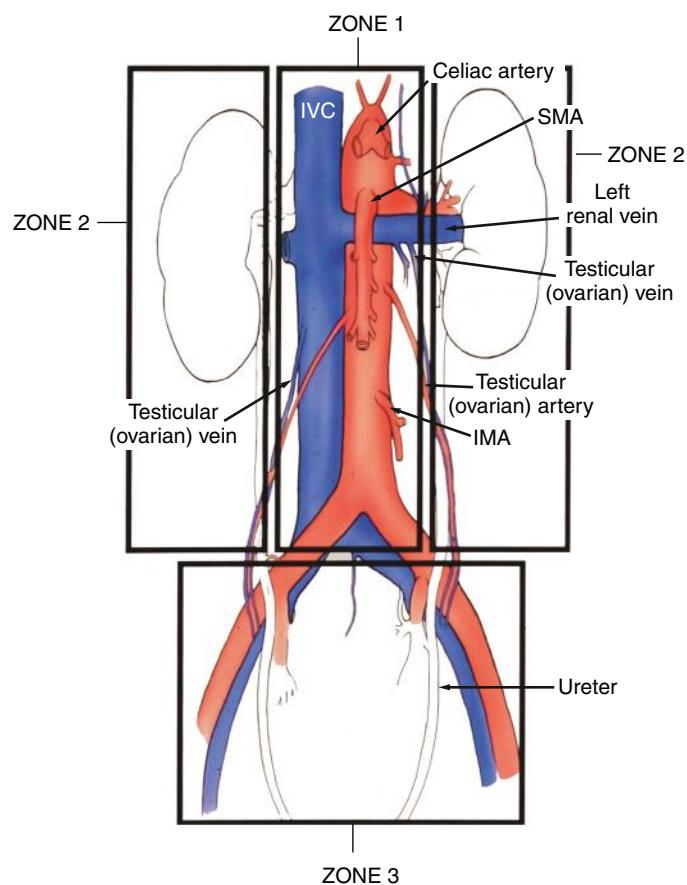


Figure 182.1 Retroperitoneal Vascular Zones. Zone 1 includes the midline vessels from the aortic hiatus to the sacral promontory; zone 2 includes the paracolic gutter and the kidneys; and zone 3 includes the pelvic retroperitoneum. *IMA*, inferior mesenteric artery; *IVC*, inferior vena cava; *SMA*, superior mesenteric artery.

of venous injuries was 51%.¹ The most commonly injured abdominal vessel was the IVC, accounting for 25% of injuries, followed by the aorta (21%), iliac arteries (20%), iliac veins (17%), SMV (11%), and SMA (10%). Overall, patients with penetrating trauma who sustained a vascular injury had an average of 1.7 vascular injuries per patient.¹

CLINICAL PRESENTATION

Many patients with major abdominal vascular injuries die at the scene. Of the patients who are transported to a hospital, approximately 14% lose vital signs en route or on arrival in the emergency department.¹ The clinical presentation depends on the injured vessel, the size and type of injury, the intraperitoneal or retroperitoneal location of injury, the presence of associated injuries, and the time elapsed.

Penetrating injuries to the abdomen associated with hypotension and abdominal distention are highly suggestive of vascular injury. Asymmetric femoral pulses may indicate iliac artery injury. Many patients may be normotensive on admission only to decompensate a few minutes later. Some patients present in a hemodynamically stable condition because of thrombosis of the vessel or containment of the bleeding in the retroperitoneum. In most cases the diagnosis is made intraoperatively.

DIAGNOSTIC EVALUATION

In most patients with abdominal vascular injuries, no investigations are needed because of the altered hemodynamics and obvious need for immediate laparotomy. For penetrating injuries, hemodynamic instability, peritonitis on examination, or an unevaulable patient mandates immediate laparotomy and any vascular injuries will be diagnosed intraoperatively.⁵ Whenever possible, plain radiographs should be obtained (Fig. 182.2) for localizing any bullet fragments. If the patient does not require immediate laparotomy and is to undergo a trial of nonoperative management, CT would be the next diagnostic evaluation and has excellent sensitivity and specificity for vascular injury

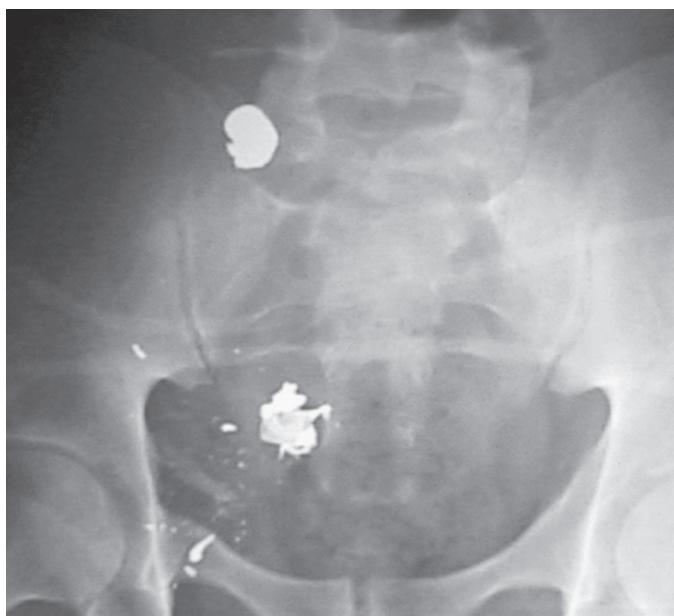


Figure 182.2 The pelvic location of the missile on the abdominal radiograph, combined with hypotension, is highly suggestive of an iliac vascular injury.

(Figs. 182.3 and 182.4). For blunt trauma, peritonitis or hemodynamic instability with a positive abdominal FAST (Focused Assessment with Sonography in Trauma) exam requires immediate laparotomy. For unstable patients with negative FAST, a diagnostic peritoneal aspirate is performed to exclude a false negative FAST and other sources of bleeding are sought. This includes the retroperitoneum, ideally imaged using CT (Fig. 182.5). In selected cases, such as asymmetrical femoral pulses following abdominal trauma in the stable patient, catheter angiography may have diagnostic and therapeutic value (Fig. 182.6).

TREATMENT

Prehospital Treatment

The most important factor for the survival of salvageable patients with vascular injuries is rapid transportation to a trauma center, followed by immediate surgical control of the bleeding. Prehospital advanced life support has no place in penetrating trauma, especially in an urban environment. A policy of “scoop and run” is currently the recommended approach. The role of prehospital intravenous fluid administration is controversial; some studies show improved survival with fluid restriction and others show no effect on survival.^{6–8} Experimental work on abdominal aortic injuries has shown that, in the presence of uncontrolled bleeding, aggressive fluid resuscitation increases mortality and the rate and volume of hemorrhage.^{9,10} However, avoidance of all fluid resuscitation in near-fatal hemorrhage may result in cardiac arrest before bleeding is controlled.¹¹ It seems that some degree of controlled hypotension is beneficial and prevents massive exsanguination while avoiding the risk of cardiac arrest due to massive blood loss and severe hypotension.^{9,12} Experimental work suggests that a systolic pressure of 80 to 90 mm Hg is the optimal pressure in the presence of active vascular bleeding.¹³



Figure 182.3 (A) Computed tomography (CT) scan of a traffic accident victim shows a large pelvic hematoma (circle) due to injury of the right common iliac artery. (B) CT scan with intravenous administration of contrast material in a high-speed traffic accident patient shows poor uptake of the contrast agent in the right kidney because of occlusion of the renal artery (circle).

Emergency Department Treatment

The extent of resuscitation required in the emergency department depends on the clinical condition of the patient. For patients arriving in cardiac arrest, endotracheal intubation and a resuscitative thoracotomy should be performed in the emergency department. A left anterolateral thoracotomy through the fourth to fifth intercostal space is performed, the thoracic aorta is cross-clamped, and the heart is resuscitated. If cardiac activity returns, the operation is completed in the operating room. The survival rate after resuscitative thoracotomy for abdominal vascular injuries is approximately 2%.¹

An attractive alternative to resuscitative thoracotomy for patients with suspected intraabdominal vascular injury, particularly in the pelvis, is endoluminal aortic occlusion with use of a percutaneous balloon inserted through the femoral artery.^{14,15} The balloon is inflated in the distal thoracic aorta. The aortic

occlusion reduces intraabdominal bleeding and facilitates resuscitation. Experimental work comparing endoluminal balloon aortic occlusion to thoracotomy with aortic clamping in a model of hemorrhagic shock showed that aortic balloon occlusion was associated with increased central perfusion pressures with less physiologic disturbance,¹⁶ and clinical experience with this technique is rapidly increasing.

In all other patients with suspected vascular injuries, large-bore intravenous catheters should be placed in the upper extremities or the central veins of the thoracic inlet. Femoral vein catheters should be avoided in case the victim has an injury to the IVC or the iliac veins. As discussed earlier, the concept of controlled hypotension should be borne in mind, and aggressive fluid resuscitation should be avoided. Except for patients in cardiac arrest or at risk for imminent cardiac arrest, endotracheal intubation should be avoided in the emergency department because rapid sequence induction is often associated with cardiovascular decompensation.

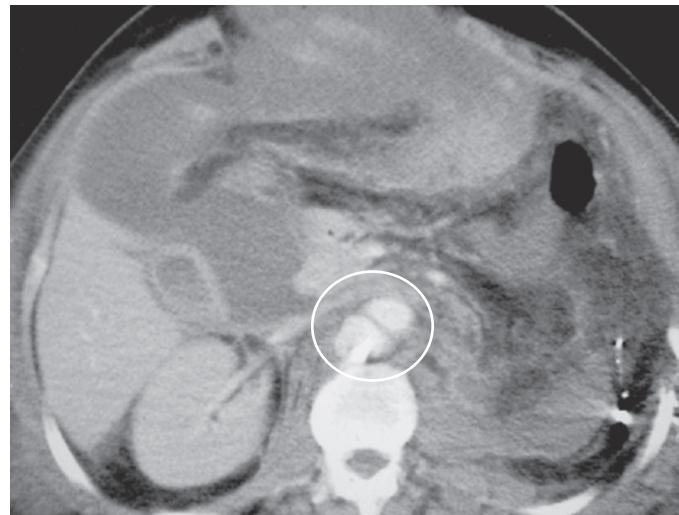


Figure 182.4 Postoperative computed tomography scan of a patient with a gunshot wound shows an abdominal aortic false aneurysm (circle).

Surgical Treatment

General Principles

All possible steps should be taken to mitigate heat loss and hypothermia. All infused fluids should be prewarmed to 40°–42°C, and the patient's extremities should be covered with a warming blanket. Rapid infusion devices should be ready, and the blood bank should be notified. After a vascular injury is identified, the aggressive replacement of shed blood with products in a 1:1:1 ratio directed by a massive transfusion protocol has been demonstrated to improve outcomes. The patient's entire torso, from the neck to the knees, should be prepared and draped. Although the initial incision is a full midline laparotomy, additional thoracotomy or saphenous vein harvesting may become necessary. The surgical team should be ready, and the skin preparation should be performed before induction of anesthesia because it is often associated with rapid hemodynamic decompensation in these patients.

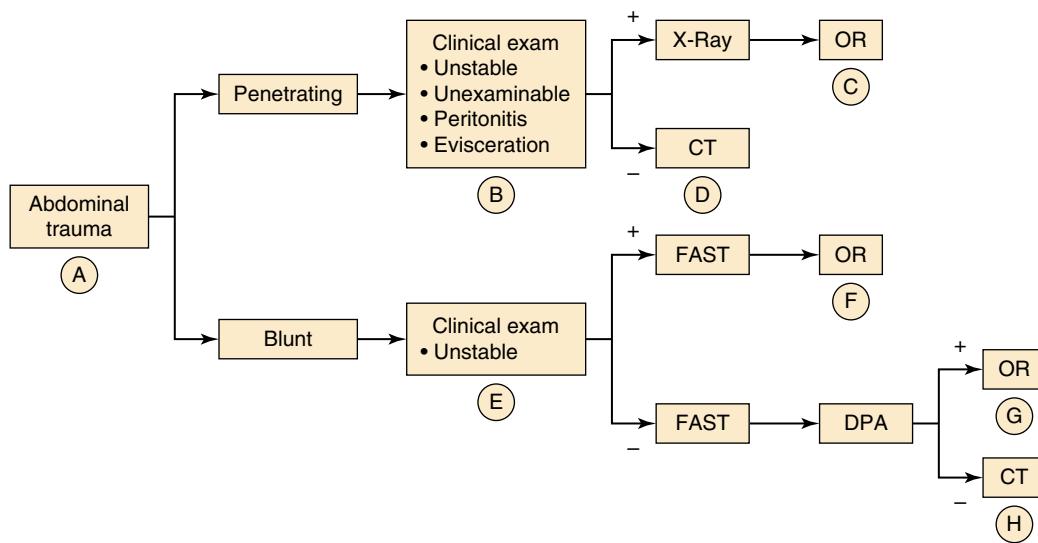


Figure 182.5 Algorithm for the Evaluation of Abdominal Trauma.



Figure 182.6 A 5-year-old child presented with a severe pelvic fracture and absent right femoral pulse. Angiography shows a complete occlusion of the right common iliac artery (arrow).

Some surgeons have advocated a preliminary left thoracotomy and aortic cross-clamping to prevent cardiovascular collapse after anesthesia and laparotomy.^{17,18} The effectiveness of this procedure has been challenged by other authors.¹⁹ We believe that this approach should be considered only for patients at risk of imminent cardiac arrest. A thoracotomy is an additional traumatic insult that may aggravate hypothermia and coagulopathy and has little effect on the control of bleeding from major venous injuries. We advocate an immediate laparotomy, temporary control of bleeding by direct compression, and aortic cross-clamping, if necessary, at the diaphragm. In our experience, this is almost always possible, even in obese patients. To facilitate aortic exposure, division of the left crus of the diaphragm may be necessary. In cases where a retroperitoneal hematoma extends high toward the aortic hiatus, infradiaphragmatic exposure of the aorta is difficult, and a left thoracotomy may be necessary for aortic control.

Endoluminal aortic occlusion, as described before, is promising but still underused. The use of this technique intraoperatively can reduce the need for thoracotomy for proximal vascular control. In addition, it may make the exploration of anatomically difficult upper zone 1 hematomas safer and easier. Training of surgeons in the use of this technique may add a useful tool to the armamentarium available for the management of these complex cases.

Retroperitoneal Hematoma

The management of retroperitoneal hematomas depends on the mechanism of injury. As a general rule, almost all hematomas

due to penetrating trauma should be explored, irrespective of size. Underneath a small hematoma, there is often a vascular or hollow viscus perforation. The only exception to this recommendation is a contained zone 4 retrohepatic hematoma. Surgical exploration of the retrohepatic vena cava or the hepatic veins is extremely challenging and in most cases will be detrimental because these contained venous injuries do very well with nonoperative management.

Retroperitoneal hematomas due to blunt trauma rarely require exploration because of the very low incidence of underlying vascular or hollow viscus injuries requiring surgical repair.²⁰ If the hematoma is in zone 1, it should be explored. However, in patients with zone 2 hematomas, surgical exploration may result in the unnecessary loss of the kidney. Similarly, exploration of a stable zone 3 hematoma due to pelvic fractures may cause severe bleeding that is uncontrollable. Exploration of zone 2 and 3 retroperitoneal hematomas should therefore be limited to patients with expanding, pulsatile, or leaking hematomas. In addition, zone 3 pelvic hematomas associated with an absent ipsilateral femoral pulse should be explored because of the potential for an iliac artery injury. Paroduodenal hematomas also require exploration to exclude an underlying duodenal injury. Finally, hematomas at the root of the mesentery in the presence of ischemic bowel may harbor an injury to the SMA and should be explored. Exploration of these hematomas is technically difficult and potentially dangerous and should not be performed in the absence of ischemic bowel. Unexplored hematomas should be evaluated postoperatively using color-flow Doppler studies (which may be difficult because of bowel gas), CT angiography, or catheter-based angiography. With the advancement of endovascular technology and the availability of hybrid operating rooms with angio-interventional capabilities, there is now the option to perform on-table diagnostic angiography instead of opening a contained retroperitoneal hematoma. Any significant injuries may be managed endovascularly, if amenable, with stenting or embolization. This approach has the additional advantages of keeping the retroperitoneum intact, especially important in the presence of associated hollow viscus injuries.

Once the hematoma is open, in the presence of severe active bleeding, the immediate priority is to control the bleeding by direct compression. After this critical task is achieved, the next step is to identify the bleeding vessel and obtain proximal and distal control. If control is difficult or the patient is severely hypotensive, the abdominal aorta can be compressed digitally or with an aortic compressor at the aortic hiatus. After dissection of the peritoneum over the aorta and, if necessary, division of the left crus of the diaphragm (at the 2-o'clock position to avoid bleeding), the aorta can be cross-clamped. Endoluminal aortic occlusion with a percutaneously placed catheter balloon, as described before, is an attractive but still underused alternative.

The exploration of the area of bleeding or hematoma should proceed systematically. Each anatomic zone requires a different technical maneuver. Zone 1 supramesocolic bleeding or hematoma is the most difficult to approach because of the dense concentration of major vessels (aorta, celiac artery, SMA, renal

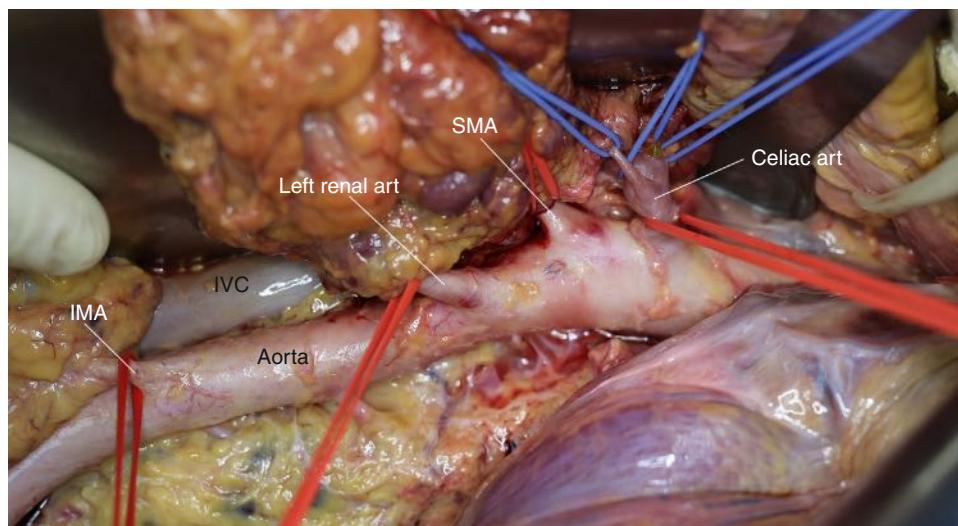


Figure 182.7 Left medial visceral rotation provides good exposure of the supramesocolic aorta and the origin of the celiac axis, superior mesenteric artery (SMA), and left renal vessels. *IMA*, inferior mesenteric artery; *IVC*, inferior vena cava.

vessels, IVC), the difficult exposure of many of these vessels, and the difficult proximal control of the infradiaphragmatic aorta. For some injuries, the only safe way to achieve proximal aortic control is through a left thoracotomy or with endoluminal aortic occlusion. The supramesocolic aorta, along with the origins of its major branches, is best exposed by mobilization and medial rotation of the viscera in the left upper abdomen. The first step of this approach is to divide the peritoneal reflection lateral to the left colon, the splenic flexure of the colon, and the spleen. The fundus of the stomach, spleen, tail of the pancreas, colon, and left kidney are then rotated to the right. This maneuver provides exposure of the aorta, origin of the celiac axis, SMA, and left renal vessels (Fig. 182.7). Some surgeons prefer not to include the left kidney in the medial rotation.²¹ However, for injuries involving the posterior wall of the aorta, inclusion of the left kidney in the visceral rotation improves the exposure. In suspected supramesocolic IVC injuries, zone 1 should be explored through a medial rotation of the right colon and hepatic flexure and Kocher mobilization of the duodenum and head of the pancreas (Fig. 182.8). The inframesocolic zone 1 area can be approached by retracting the transverse colon cephalad and displacing the small bowel to the right. The peritoneum over the aorta and IVC is then incised, and the vessels are exposed. An alternative approach is medial rotation of the right or left colon.

Zone 2 bleeding or hematoma is explored by mobilization and medial rotation of the right colon, duodenum, and head of the pancreas on the right side or the left colon on the left side. The source of bleeding in zone 2 is the renal vessels or the kidneys.

Zone 3 vessels are explored by dissection of the paracolic peritoneum and medial rotation of the right or left colon. In some cases, direct dissection of the peritoneum over the vessels can provide the necessary exposure.

The reconstruction of major vessels with synthetic grafts in the presence of intestinal spillage poses significant risks for graft infection. Copious irrigation before graft placement, use

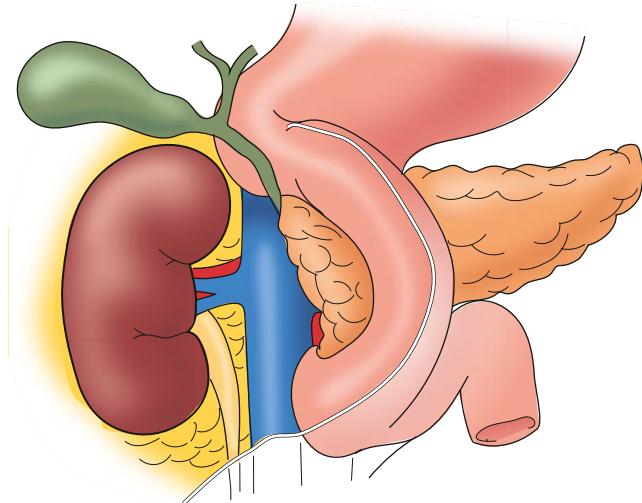


Figure 182.8 Medial visceral rotation of the right colon and hepatic flexure and Kocherization of the duodenum and pancreas provide excellent exposure of the inferior vena cava and the origins of the renal veins. (From Buckman RF, Pathak AS, Badellino MM, Bradley KM. Injuries of the inferior vena cava. *Surg Clin North Am*. 2001;81:1431–1447.)

of autologous graft material whenever possible, and omental wrapping or soft tissue coverage for polytetrafluoroethylene (PTFE) grafts will reduce the risk of infection.

Damage Control Procedures

Many patients with major abdominal vascular injuries require massive blood transfusions, are hypotensive, and become severely hypothermic, acidotic, and coagulopathic intraoperatively. Persistent attempts to definitively reconstruct the injury are ill advised and will result in increased mortality. These patients should undergo early damage control and definitive reconstruction at a later stage when their physiology improves. Earlier reports recommended that damage control procedures be considered in patients *in extremis* who had exhausted their

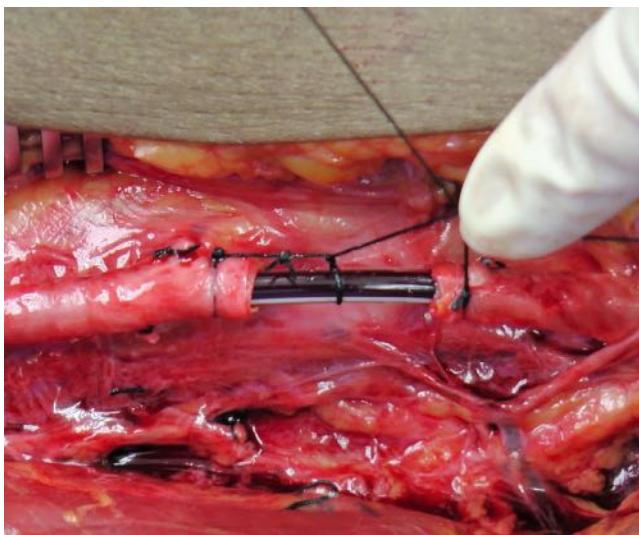


Figure 182.9 Temporary arterial shunt for damage control in a hemodynamically unstable patient with a gunshot wound and complete transection of the iliac artery.

physiologic reserves and were in danger of irreversible shock and death. However, to be effective, damage control should not be a procedure of last resort but should be considered at a much earlier stage, before the patient becomes severely hypotensive and coagulopathic.²² This concept is even more important in patients with major comorbidities, such as older age or chronic medical problems, and in suboptimal environments, such as small community hospitals or combat zones.

With the damage control approach, all complex venous injuries are ligated, arterial injuries are shunted,²³ and any diffuse retroperitoneal or parenchymal bleeding is controlled by tight gauze packing. If commercially produced vascular shunts are not available, temporary intraluminal shunts can be constructed from sterile intravenous or nasogastric tubing. The shunt is secured with proximal and distal ligatures (Fig. 182.9). The abdomen is then closed temporarily with a prosthetic material or vacuum dressing techniques and the patient is transferred to the intensive care unit (ICU) for further resuscitation. The abdomen should never be closed primarily because of the very high incidence of abdominal compartment syndrome. The patient is returned to the operating room after resuscitation and stabilization for definitive vascular repair and abdominal wall closure.

Endovascular Techniques

Endovascular techniques have revolutionized the management of blunt thoracic aorta and other arterial injuries.²⁴ Hybrid operating rooms with angiographic radiology capabilities provide an opportunity to improve the management of abdominal vascular injuries by either temporary control of life-threatening bleeding or definitive care with angioembolization or stenting. In the appropriate cases the combination of endovascular adjuncts and open surgery may be lifesaving.

The role of endovascular techniques in penetrating abdominal vascular trauma, which is almost always associated

with severe active bleeding, is usually limited. However, the use of endoluminal balloons for temporary proximal aortic control can be a useful adjunct during open surgery. In addition, endovascular techniques may be the best therapeutic intervention in most cases with delayed vascular complications such as aneurysms, arteriovenous fistulae, and arterial occlusions.

Abdominal Compartment Syndrome

The normal intraabdominal pressure in the resting supine position is near zero. Elevation of the intraabdominal pressure greater than 12 mm Hg constitutes a diagnosis of intraabdominal hypertension.²⁵ A pressure that exceeds 20 mm Hg in addition to organ dysfunction results in abdominal compartment syndrome. Abdominal compartment syndrome is characterized by a tense abdomen, tachycardia with or without hypotension, respiratory dysfunction with high peak inspiratory and plateau pressures in mechanically ventilated patients, and oliguria. However, significant organ dysfunction may begin long before classic abdominal compartment syndrome is manifested.

All patients with severe abdominal trauma, especially vascular trauma, are at risk for development of abdominal compartment syndrome. Major risk factors include massive blood transfusions, prolonged hypotension, hypothermia, aortic cross-clamping, damage control procedures, and closure of the abdominal wall. After severe trauma, the abdomen should never be closed under tension. Similarly, after damage control procedures, the abdominal wall fascia should not be closed because progressive postoperative bowel edema will frequently result in abdominal compartment syndrome.

The diagnosis of abdominal compartment syndrome is based on clinical examination and intraabdominal pressure measurements. All high-risk patients and those in whom abdominal compartment syndrome is suspected clinically should be monitored closely with serial intraabdominal pressure measurements. The intraabdominal pressure can be measured reliably through the bladder catheter. The catheter is used to empty the bladder. Approximately 20 mL of sterile saline is injected back into the bladder to allow pressure transduction without the false elevation in pressure expected from bladder wall tension in a full bladder. The pressure is then measured through the catheter by either a simple water column or a commercial pressure transduction device. Definitive management requires surgical decompression. In general, pressures higher than 20 to 30 mm Hg are considered strong indicators for surgical decompression of the abdomen. If organ dysfunction is present, the pressure threshold may be lower. The abdomen can be opened in the operating room or even in the ICU if necessary. Temporary abdominal wall closure can be achieved with the use of a commercially available vacuum closure device, or a sterile X-ray cassette cover, laparotomy sponges, closed suction drains, and sticky drapes can be used to accomplish temporary coverage. When the bowel edema improves, usually within 2 to 3 days, the patient is returned to the operating room for definitive abdominal wall closure.

SPECIFIC VASCULAR INJURIES

Abdominal Aorta Injuries

Anatomy

The aorta descends into the retroperitoneum between the two crura of the diaphragm at the T12–L1 level and bifurcates into the common iliac arteries at the L4–L5 level, which approximately corresponds to the level of the umbilicus. The first branches of the abdominal aorta are the phrenic arteries that originate from its anterolateral surface. Immediately below is the celiac trunk that originates from the anterior surface of the aorta, and 1 to 2 cm below that is the SMA, followed by the renal arteries 1 to 1.5 cm below the origin of the SMA and finally, the inferior mesenteric artery (IMA), 2 to 5 cm above the bifurcation.

Mechanism of Injury

Blunt injury to the abdominal aorta is extremely rare, diagnosed in 0.04% of all blunt trauma admissions.²⁶ Fractures of the thoracolumbar spine and seat belt injuries are associated with an increased risk of abdominal aortic injuries.^{26,27} Intimal dissection and thrombosis are the most common lesions in patients reaching the hospital alive. False aneurysms occur less frequently. Patients with free ruptures die at the scene and rarely receive medical care.^{27,28} In a review of 28 cases with blunt abdominal aortic injuries, intimal tears or large intimal flaps were the most common lesion (60%), followed by free rupture (30%) and pseudoaneurysm (10%).²⁹

Penetrating injuries are by far the most common cause of abdominal aortic injuries. In a review of 1218 patients with abdominal gunshot injuries from our center, there were 33 abdominal aortic injuries (2.7%). In 529 knife wounds to the abdomen, the aorta was injured in 1.5%.³⁰ The infrarenal aorta is injured in 50% of patients, the supraceliac aorta in 25%, and the aorta between the celiac trunk and the renal arteries in 25% of patients.³¹

Clinical Presentation

The clinical presentation depends on the mechanism of injury (blunt or penetrating), type of aortic injury, presence of free intraperitoneal bleeding or retroperitoneal hematoma, associated injuries, and time elapsed since the injury. In blunt trauma, two-thirds of patients have acute symptoms of bleeding or visceral or lower extremity ischemia. The diagnosis is made during the initial hospitalization by means of CT or angiography or at laparotomy. In one-third of cases the diagnosis is made many months or even years after the injury.^{29–31}

The clinical presentation of penetrating aortic injuries is usually dramatic. Many victims die at the scene. Of those who are treated, approximately 28% have an unrecordable blood pressure, and approximately 21% require a resuscitative thoracotomy.³⁰ In approximately 18% of cases the bleeding is temporarily contained in the retroperitoneum and the patients are normotensive on admission.³¹ On rare occasions the injury is missed at operation only to manifest at a later date as a false aneurysm or arteriovenous fistula (see Fig. 182.4).

Management

Almost all patients with penetrating aortic injuries require open repair. In blunt trauma the management depends on the location and type of the injury. Many patients with small intimal tears can safely be managed nonoperatively. More severe blunt injuries with free rupture or large intimal flaps require open or endovascular repair.²⁹ In a National Trauma Data Bank analysis of 436 patients with blunt abdominal aortic injuries, 394 patients (90%) were managed nonoperatively and 29 (7%) underwent endovascular repair, with only 13 patients (3%) undergoing open aortic repair or extra-anatomic bypass.³²

Endovascular treatment

Endovascular management has a definitive role in selected cases of infrarenal aortic injury. Patients with limited infrarenal aortic dissection, large intimal flaps, false aneurysms, or aorto-caval fistulae have been treated successfully with angiographically placed stents.^{33,34}

Surgical treatment

The surgical exposure of the vascular structures in zone 1 is achieved by medial visceral rotation, as described in the section on retroperitoneal hematoma. For high supramesocolic injuries, a left thoracotomy may be necessary for cross-clamping of the aorta. Approximately 93% of patients with penetrating trauma have other associated intraabdominal injuries, the most common being injuries to the small bowel (45%), colon (30%), and liver (28%).³⁰ Before any definitive management that requires a prosthetic graft, all enteric spillage should be controlled and the peritoneum should be washed out. Lateral aortorrhaphy is possible in most cases. More complex repairs with prosthetic grafts may be necessary.³⁵ Many authors do not consider the presence of enteric spillage a contraindication to the use of prosthetic material.³⁵

Mortality

The prognosis of abdominal aortic injuries after blunt trauma is significantly better than that of injuries due to penetrating trauma. The reported overall mortality in blunt trauma is approximately 30%.³²

The mortality after penetrating trauma in two large series with 146 patients was 67%.^{30,35} In another series of 57 patients with gunshot wounds, the mortality rate was 85%.³⁶ Suprarenal aortic injuries have a significantly worse outcome than infrarenal injuries.³¹ The mortality in patients undergoing emergency center resuscitative thoracotomy is almost 100%.^{30,31} The prognosis of penetrating abdominal aortic injuries is significantly better than that of injuries to the thoracic aorta, most likely owing to the retroperitoneal containment of bleeding in abdominal injuries. In a comparison of 67 abdominal aortic injuries with 26 thoracic aortic injuries, the mortality rates were 76% and 92%, respectively.³⁰

Celiac Artery Injuries

Anatomy

The celiac artery originates from the anterior wall of the abdominal aorta, immediately below the aortic hiatus, at the level

of T12–L1. The main trunk is 1 to 1.5 cm long, and at the upper border of the pancreas it has three branches (the tripod of Haller): the common hepatic artery, left gastric artery, and splenic artery. Because of the extensive fibrous, ganglionic, and lymphatic tissues that surround the trunk, surgical dissection may be tedious.

Mechanism of Injury

Injuries to the celiac artery are rare and almost always due to penetrating trauma. In a review of 302 abdominal vascular injuries, the celiac artery was involved in 10 cases (3.3%).¹

Surgical Treatment

The surgical exposure can be achieved either by direct dissection over the upper abdominal aorta through the lesser sac or by medial rotation of the upper abdominal viscera, as described previously. The rotation does not need to include the left kidney. The celiac artery can be ligated without ischemic sequelae to the stomach, liver, or spleen because of the rich collateral circulation of these organs. The left gastric and splenic arteries may also be ligated with impunity. Ligation of the common hepatic artery is usually well tolerated because of adequate supply from the portal vein and the gastroduodenal artery.

Mortality

The reported mortality rate of celiac artery injuries ranges from 38% to 75%.³⁷ However, among the 50 collectively reported cases of celiac artery injuries, most patients had other vascular injuries that contributed to the high mortality.

Superior Mesenteric Artery Injuries

Anatomy

The SMA originates from the anterior surface of the aorta, immediately below the celiac artery, behind the pancreas at the L1 level. It then proceeds over the uncinate process of the pancreas and the third part of the duodenum and enters the root of the mesentery. The SMA has the following branches: inferior pancreaticoduodenal artery, middle colic artery, arterial arcade with 12 to 18 intestinal branches, right colic artery, and ileocolic artery. SMA injuries are divided into four zones³⁸:

- Zone 1, between the aortic origin and the inferior pancreaticoduodenal artery.
- Zone 2, between the inferior pancreaticoduodenal artery and the middle colic artery.
- Zone 3, distal to the middle colic artery.
- Zone 4, the segmental intestinal branches.

As a general rule, ligation of the SMA in zones 1 and 2 results in severe ischemia of the small bowel and right colon. Ligation of zones 3 and 4 may result in localized ischemia of the small bowel requiring segmental resection.

Mechanism of Injury

The SMA is second to the renovascular system as the most commonly injured abdominal vessel after blunt trauma. Blunt trauma is responsible for between 10% and 20% of all SMA injuries^{39,40} and can cause thrombosis of the artery by a direct

blow, crushing of the abdomen, or seat belt injuries. Deceleration injuries may cause avulsion of the vessel from its origin in the aorta or intimal tear and subsequent thrombosis. Penetrating injuries are the most common mechanism of injury. Because of the anatomic location of the artery, multiple significant associated injuries are common.⁴¹

Clinical Presentation

The clinical presentation depends on the mechanism of injury, nature of the vascular injury, presence of associated intraabdominal injuries, and time elapsed since injury. Isolated thrombosis of the SMA due to blunt trauma may be missed during the initial evaluation, only to manifest at a later stage with bowel necrosis. Most patients with penetrating trauma present in severe shock. Patients with contained hematomas may be normotensive or mildly hypotensive on admission.

Surgical Treatment

The operative findings may include various degrees of hemothorax, hematoma around the SMA, ischemic bowel, or any combination of these (Fig. 182.10).

After temporary control of the bleeding by direct compression and, if necessary, cross-clamping of the aorta, the SMA should be explored.

Exposure

Exposure of the retropancreatic SMA can be achieved by medial rotation of the left colon, gastric fundus, spleen, and tail of the pancreas, as described earlier. The kidney does not need to be rotated unless injury to the posterior wall of the aorta is suspected. In cases of severe bleeding when immediate exposure is critical, stapled division of the neck of the pancreas may provide fast and direct exposure of the SMA and the portal vein. Exposure of the infrapancreatic SMA can be achieved by cephalad retraction of the inferior border of the pancreas and direct dissection, or it can be achieved through the root of the small bowel mesentery by incising and dissecting the tissues to the left of the ligament of Treitz. An extensive Kocher maneuver may be required to expose this segment of the SMA. More distal sections of the SMA can be approached directly.

Exploration of a hematoma at the root of the mesentery is always a difficult and potentially dangerous task, even in the hands of experienced surgeons. In penetrating trauma or blunt trauma with ischemic bowel, all hematomas around the SMA should be explored. However, it is our practice and recommendation not to explore stable hematomas after blunt trauma in the absence of bowel ischemia. In these patients the SMA is evaluated postoperatively by means of angiography or CT angiography (see Fig. 182.10B).

Operative management

Sharp partial transections of the SMA, such as those inflicted by knife wounds, can be managed by lateral arteriotomy with 6-0 vascular sutures. This approach is possible in approximately 40% of cases.⁴² Because mobilization of the SMA is restricted by the surrounding dense neuroganglionic tissue and its multiple branches, an end-to-end anastomosis is rarely possible.

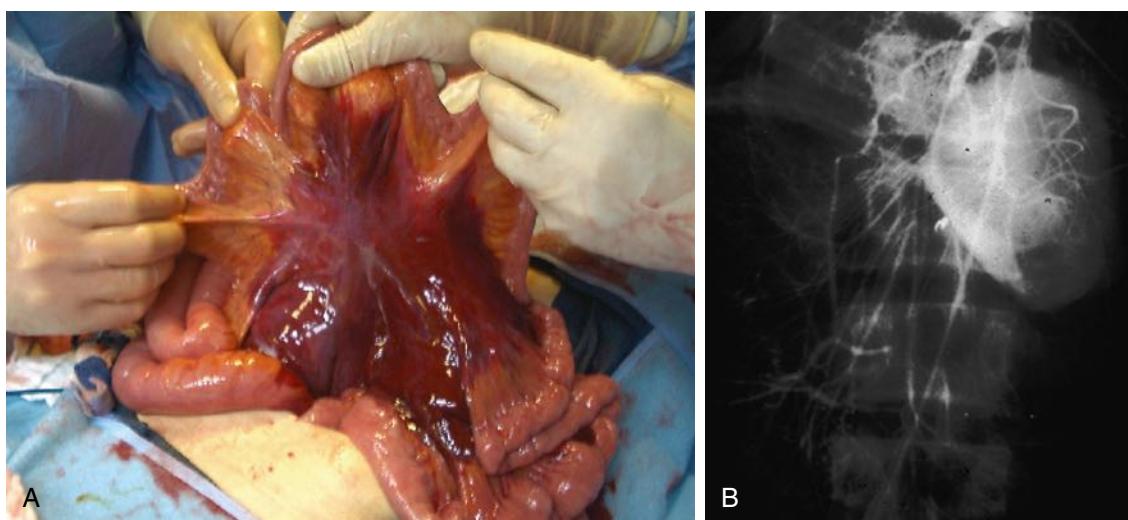


Figure 182.10 (A) Traffic accident victim with a large hematoma at the base of the mesentery found at laparotomy. This is suggestive of a superior mesenteric artery (SMA) injury and needs to be evaluated by angiography, preferably postoperatively. (B) Postoperative angiography shows a large SMA false aneurysm.

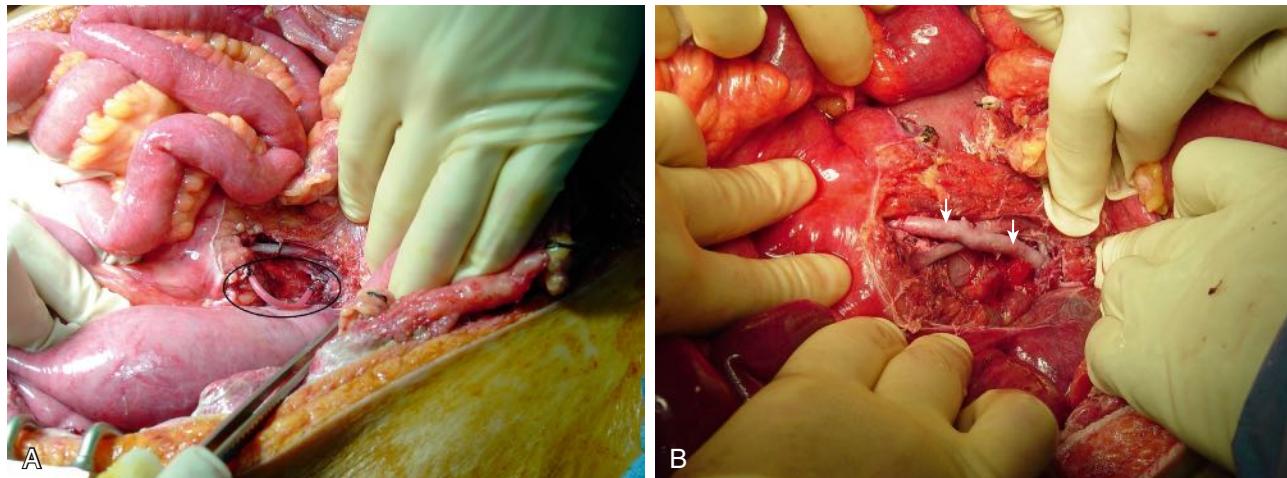


Figure 182.11 (A) Damage control with a temporary endoluminal shunt (circle) of the superior mesenteric artery. (B) Definitive reconstruction with a saphenous vein graft (arrows) 18 hours later.

Depending on the site of SMA injury, the condition of the patient, and the color of the bowel, these injuries can be managed by ligation or an interposition graft. Ligation of the SMA distal to the middle colic artery is associated with a moderate risk of bowel ischemia. Ligation of the proximal SMA results in ischemic necrosis involving the small bowel and the right colon. The first 10 to 20 cm of the jejunum may survive through collaterals from the superior pancreaticoduodenal artery. Ligation of the SMA proximal to the origin of the inferior pancreaticoduodenal artery may preserve critical collateral circulation to the proximal jejunum and is preferable to a more distal ligation. Ligation of the proximal SMA should be performed only in the presence of necrotic bowel. Ligation should be avoided in all other circumstances because of the catastrophic consequences of short bowel syndrome. In patients in critical condition with severe hypothermia, acidosis, and coagulopathy, a damage control procedure with a temporary endoluminal shunt should be considered.⁴³

Definitive reconstruction is performed at a later stage after resuscitation and normalization of the patient's physiologic parameters. The reconstruction can be performed with a saphenous vein or PTFE graft between the distal stump of the SMA and the anterior surface of the aorta. In the presence of an associated pancreatic injury, the vascular anastomosis should be performed away from the pancreas, and every effort should be made to protect it from pancreatic enzymes by use of omentum and surrounding soft tissues (Fig. 182.11).

Postoperative considerations

Postoperatively, the patient should be monitored closely for any signs of bowel ischemia. The threshold for second-look laparotomy within the first 24 hours of operation should be low. Failure to improve postoperatively and the persistence of metabolic acidosis despite adequate fluid resuscitation should prompt re-exploration of the abdomen to rule out bowel ischemia. Some authors practice mandatory second-look laparotomy. Damage

control techniques allow inspection of the bowel through the transparent material used for temporary closure of the abdomen.

Mortality

The mortality directly related to SMA injuries is difficult to assess because most patients have multiple severe injuries, including other major vascular injuries. The reported mortality varies from 33% to 68%.^{39,40,42,44}

Renovascular Injuries

Anatomy

The renal arteries originate from the aorta at the L2 level. The right renal artery emerges at a slightly higher level and is longer than the left and courses under the IVC. Approximately 30% of the population has more than one renal artery, often an accessory one to the lower pole of the kidney. The renal veins lie in front of the renal arteries (Fig. 182.12). The left renal vein

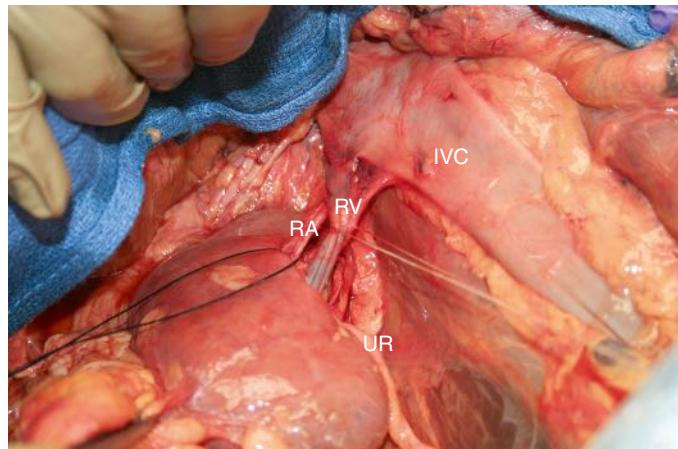


Figure 182.12 Anatomy of the Hilum of the Right Kidney. Note the position of the renal artery (RA, black ligature) behind the renal vein (RV, white ligature) and inferior vena cava (IVC). UR, ureter.

is significantly longer than the right and courses in front of the aorta. It has collateral branches from the left gonadal vein inferiorly, the left adrenal vein superiorly, and a descending lumbar vein posteriorly.

Mechanism of Injury

Renal artery injuries account for approximately 0.05% of all blunt trauma admissions.⁴⁵ The left renal artery is 1.3 to 1.6 times more likely to be injured than the right renal artery.^{45,46} It has been suggested that the right renal artery is protected from deceleration injuries because of its course underneath the IVC.⁴⁷ Rapid deceleration accidents may cause intimal tears and subsequent arterial thrombosis at a later stage. In approximately 50% of cases with blunt renal artery injury, there is thrombosis or an intimal flap (Fig. 182.13). Avulsion of the artery occurs in 12% of cases.⁴⁸ In 9% to 14% of renovascular injuries, the renal artery is involved bilaterally.^{46,48}

Clinical Presentation

The diagnosis of renovascular injury after penetrating trauma is almost always made intraoperatively. However, in blunt trauma, the diagnosis is usually made during routine CT evaluation of the abdomen (Fig. 182.14). The clinical presentation is subtle and nondiagnostic, and the diagnosis is often delayed. In earlier reports, when CT evaluation of the abdomen was not as common, up to 50% of patients did not receive timely treatment because of delayed diagnosis.⁴⁸ Abdominal contrast-enhanced CT is highly sensitive in diagnosing renovascular trauma and should be the first-line investigation (see Fig. 182.14A).⁴⁹ In addition, CT provides useful information about associated injuries.

Endovascular Treatment

Endovascular treatment may play an important role in selected cases of blunt renovascular trauma, and it should be considered the first-line therapeutic option in patients in a stable condition with intimal tears, acute occlusions, false aneurysms, and arteriovenous fistulae (see Fig. 182.14A–C). The experience

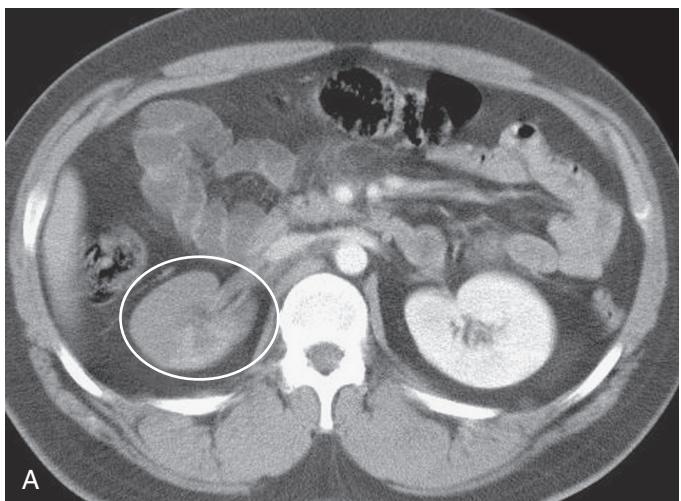


Figure 182.13 (A) CT shows lack of perfusion to the right kidney (circle) due to injury of the right renal artery after a fall from a height. (B) Angiography shows an intimal tear of the right renal artery (circle). Management with an endovascular stent was successful.



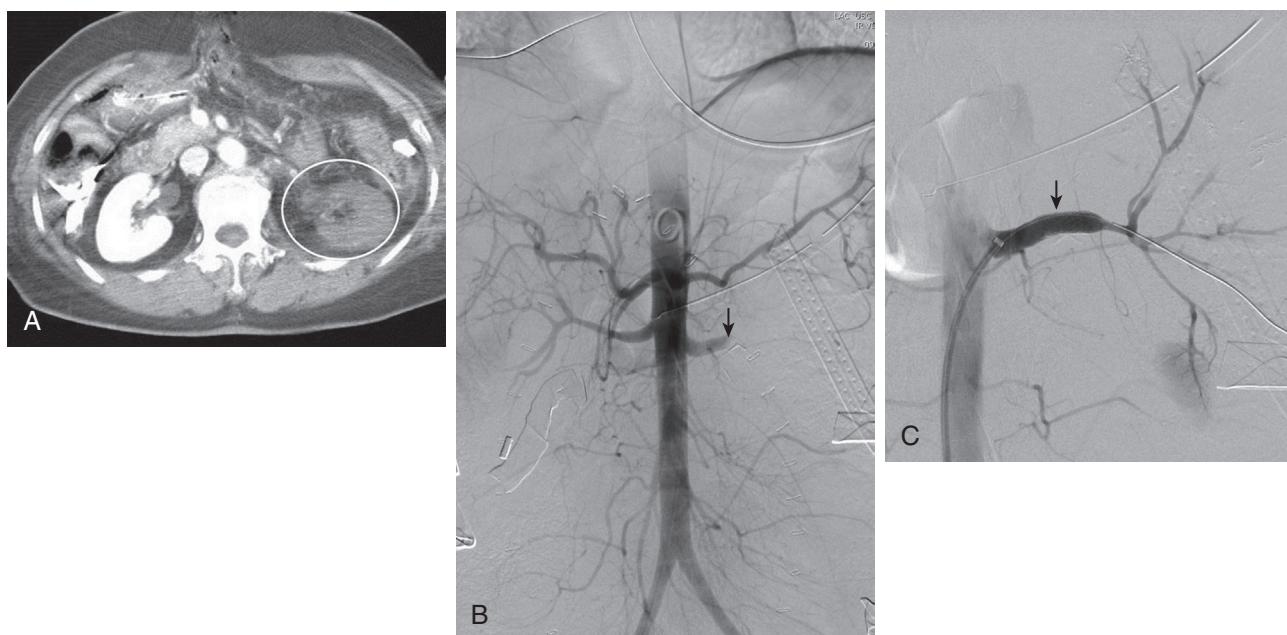


Figure 182.14 (A) Computed tomography scan shows a nonfunctioning left kidney (circle) after a traffic injury. (B) Angiography shows complete thrombosis of the renal artery (arrow). (C) Revascularization with an angiographically placed endovascular stent (arrow).

with this technique is still limited, and the follow-up is short. However, it is very promising and most likely will increase the number of patients undergoing revascularization.^{50–52} In a National Trauma Data Bank study covering the period 1993–2001, none of the 517 blunt renal artery injuries were managed with endovascular techniques.⁴⁵ In a more recent study from the same data bank, covering the period 2004–2009, only 3.2% were managed with endovascular stenting.⁵³ This reluctance to pursue endovascular treatment might be due in part to the frequently delayed diagnosis, which is considered by many surgeons a contraindication for revascularization.

Surgical Treatment

The management of renovascular injuries depends on the mechanism of injury, time of diagnosis, ischemia time, general condition of the patient, and presence of a contralateral normal kidney. In penetrating injuries the diagnosis is almost always made early during exploratory laparotomy, and depending on the extent of the injury and the condition of the victim, reconstruction of the vessels or nephrectomy is performed.

Revascularization

As a general rule, all zone 2 hematomas resulting from penetrating trauma should be explored. The only exception may be a stable perinephric hematoma away from the hilum.⁵⁴ However, in blunt trauma the management of renal artery injuries is complicated by the often-delayed diagnosis and prolonged ischemia of the kidney. Renal function is severely affected after 3 hours of total ischemia and 6 hours of partial ischemia, although with collateral circulation from the renal capsule or surrounding soft tissues, kidney function may be preserved despite prolonged ischemia.⁴⁹

In stable patients diagnosed with renovascular trauma within 4 to 6 hours of injury, the general recommendation is revascularization.^{49,55} However, revascularization is rarely performed, even in patients with no other injuries. In a study based on the National Trauma Data Bank, which is maintained by the Committee on Trauma of the American College of Surgeons, only 9% of the 517 patients with renal artery injury due to blunt trauma underwent revascularization. Of the remaining cases, 73% had no kidney exploration, and 18% underwent immediate nephrectomy. In the group of 87 patients with isolated renal artery injuries, 8% underwent revascularization, 8% had early nephrectomy, and 84% were observed. Multiple regression analysis adjusting for age, Injury Severity Score, and severe associated injuries showed that patients treated with revascularization had a significantly longer ICU and hospital stay than observed patients did.⁴⁵ In patients with bilateral injuries or injury to a solitary kidney, some authors recommend revascularization even up to 20 hours after the injury.⁵⁵ However, most surgeons will avoid revascularization in patients diagnosed more than 6 hours after trauma, unless the injury involves both kidneys or a solitary kidney. The results after revascularization are generally disappointing, and successful surgical revascularization has been reported mainly by surgeons with extensive experience in renovascular surgery. The cumulative success rate of revascularization is 28%.⁴⁶ Even after successful revascularization, subsequent hypertension develops in 12% to 57% of patients, and most require elective nephrectomy.^{46,55,56} The overall poor long-term results have led some authors to suggest that revascularization should be considered only in patients with bilateral renal artery occlusion or those with injury to a solitary kidney.^{46,55}

Nonoperative management is certainly an acceptable option, especially in patients with delayed diagnosis, those with

other major extraabdominal injuries or significant hemodynamic instability, and those with a contralateral normally functioning kidney. In a National Trauma Data Bank review of 778 cases with blunt renal artery injuries, most patients (62%) were managed without any intervention. Of patients managed nonoperatively, 32% to 40% develop renovascular hypertension.^{46,48,53,55} In most patients, the hypertension develops within 1 year of the injury, with a mean of approximately 3 months.⁴⁶

The advancement of endovascular techniques has opened new horizons in the management of these injuries. We recommend liberal revascularization with endovascular techniques because of the safety of this approach and the potential for renal salvage even after prolonged ischemia. Even if late hypertension develops, medical management and nephrectomy are effective ways to address this problem. If stenting is not possible, we advocate observation and long-term monitoring for hypertension. Surgical revascularization should be considered only in renovascular injuries diagnosed intraoperatively, provided the patient's condition is stable enough to tolerate the procedure, or in the rare case of bilateral injuries or injury to a solitary kidney.

Surgical reconstruction

Surgical reconstruction of a renal artery injury can be achieved by simple anteriorrhaphy, vein patch, resection and anastomosis, and interposition grafting. For complex, time-consuming arterial reconstructions, the kidney should be perfused intermittently with iced heparinized lactated Ringer solution or University of Wisconsin solution. Postrevascularization administration of mannitol may improve the parenchymal blood flow and alleviate the reperfusion injury.

Venous injuries

Renal vein injuries can be managed by lateral venorrhaphy if feasible. Extensive injuries should be managed by ligation. Complex reconstruction, especially in a hemodynamically unstable patient, should be avoided. Ligation of the left renal vein near the IVC is well tolerated because of venous drainage through the left gonadal vein, left adrenal vein, and lumbar veins.

Mortality

The true mortality rate of renovascular injuries is difficult to estimate because there are other major injuries in most cases. The reported mortality rate varies from 0% to 57%.⁴⁹ The mortality in renovascular injuries due to blunt trauma is low because of the occlusive nature of most arterial injuries.⁴⁵

Inferior Mesenteric Artery Injuries

Anatomy

The IMA originates from the anterior surface of the aorta 3 to 4 cm above the aortic bifurcation. It provides blood supply to the left colon, sigmoid, and upper part of the rectum. It communicates with the SMA through the marginal artery of Drummond.

Mechanism of Injury

Injuries of the IMA are rare. They are almost always due to penetrating trauma and account for 1% of all abdominal vascular injuries.¹

Surgical Treatment

The diagnosis of IMA injury is made intraoperatively. Ligation is well tolerated, and no cases of colorectal ischemia have been reported in trauma cases.

Iliac Vascular Injuries

Anatomy

The abdominal aorta bifurcates into the two common iliac arteries at the L4–L5 level. The common iliac arteries divide into the external and internal iliac arteries over the sacroiliac joint. The ureter crosses over the bifurcation of the common iliac artery.

The common iliac veins join at the L5 level, below the level of the aortic bifurcation and underneath the right common iliac artery, to form the IVC (Fig. 182.15).

Mechanism of Injury

Penetrating trauma is by far the most common cause of iliac vascular injuries. Approximately 10% of patients who undergo laparotomy for gunshot wounds and 2% of patients with laparotomies due to stab wounds have iliac vascular injuries.⁵⁷ Injury to the common or external iliac artery due to blunt trauma is not common, although there are many case reports. Direct laceration of the iliac vessels from a pelvic fracture or stretching of the iliac artery over the pelvic wall, resulting in intimal tear and subsequent thrombosis, is the usual mechanism of injury after blunt trauma (see Fig. 182.6).⁵⁸ Approximately 26% of patients with iliac vascular injuries have combined arterial and venous injuries.⁵⁷ Penetrating injuries usually involve

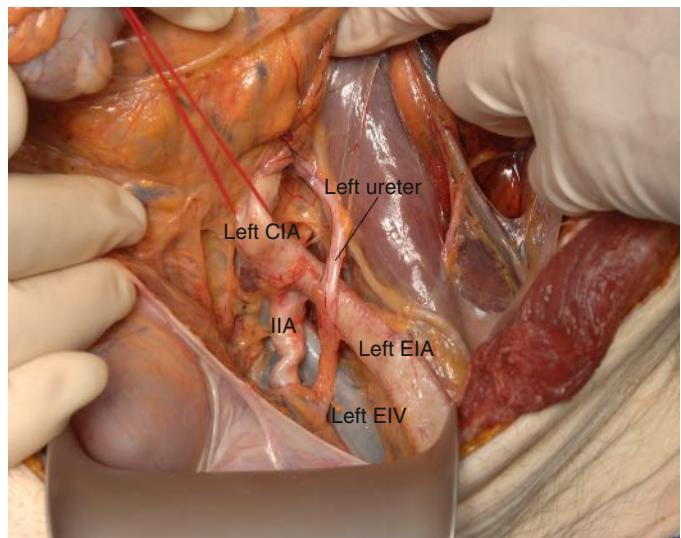


Figure 182.15 Anatomy of the Iliac Vessels. The left ureter crosses over the iliac vessels at the bifurcation of the common iliac artery (CIA) into the external iliac artery (EIA) and the internal iliac artery (IIA). The external iliac vein (EIV) runs medial to the artery.



Figure 182.16 (A) Left common iliac artery thrombosis in a 25-year old man, involved in a motor vehicle crash.
(B) Successful endovascular stent placement.

the common iliac vessels, whereas blunt trauma usually affects branches of the internal iliac artery.

Clinical Presentation

The presence of a penetrating injury in the lower abdomen associated with severe hypotension and abdominal distention is highly suggestive of iliac vascular injuries. An absent or diminished femoral pulse in a patient with penetrating abdominal trauma or pelvic fracture is diagnostic of an injury to the common or external iliac artery. In rare cases of blunt trauma, thrombosis of the iliac artery may not be diagnosed during the initial workup because of the subtle clinical symptoms. In blunt trauma the diagnosis modality of choice is CT angiography.

Endovascular Treatment

Endovascular techniques may play an important role in selected cases of iliac artery injury, especially after blunt trauma. Patients with false aneurysms, arteriovenous fistulae, or large intimal tears with or without thrombosis may benefit from angiographically placed endovascular stents (Fig. 182.16).^{59,60} Because of its safety and low complication rate, this should be the first line therapeutic option in elective cases in patients with subacute or chronic traumatic lesions of the common or external iliac arteries.

Surgical Treatment

Exposure

The operative findings may include free intraperitoneal bleeding, a zone 3 pelvic hematoma, or a combination of the two (Fig. 182.17). Zone 3 hematomas due to blunt trauma should be explored only if there is associated intraperitoneal leak, if

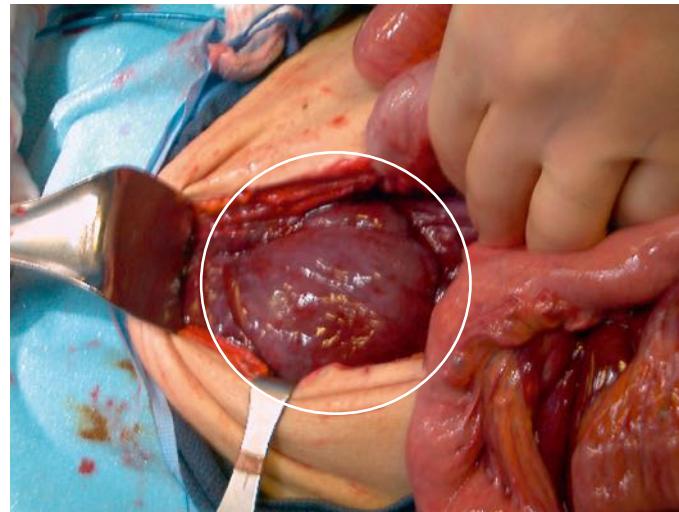


Figure 182.17 Large hematoma in the pelvis (*circle*) after a gunshot wound. This is highly suggestive of an iliac vascular injury, and proximal control should be obtained as soon as possible.

they are expanding rapidly, or if there is an absent or diminished femoral pulse. In penetrating trauma, all hematomas should be explored. Any active bleeding is initially exposed by direct dissection of the overlying peritoneum, although medial rotation of the right or left colon may provide better exposure. The ureter, which crosses over the bifurcation of the common iliac artery, should be identified and protected during the dissection. In addition, care should be taken to avoid iatrogenic injury to the iliac veins that lie directly under the arteries. Isolation and control of the internal iliac artery are essential in arterial injuries because bleeding may persist despite proximal and

distal control. If exposure of the distal iliac vessels is difficult, especially in male patients with a narrow pelvis, extension of the midline incision by adding a transverse lower abdominal incision or longitudinal incision through the inguinal ligament may be necessary.⁵⁷

Arterial injuries

Small arterial injuries can be repaired with 4-0 or 5-0 vascular sutures, with care taken to avoid significant stenosis of the vessel. If necessary, a vein or PTFE patch can be used to avoid stenosis. In most gunshot injuries and in all patients with blunt trauma, reconstruction by an end-to-end anastomosis or with a prosthetic graft (6 to 8 mm) is usually necessary. Local heparin solution should be administered to prevent thrombosis during the repair. The need for systemic anticoagulation intraoperatively remains debated; however, it should not be used if there are concomitant injuries such as a traumatic brain injury. A balloon-tipped catheter should always be passed proximally and distally to remove any clots. Complex arterial reconstructions with an extra-anatomic bypass or with a mobilized internal iliac artery have little or no role in the acute management of trauma. Extra-anatomic bypass should be considered only in late cases with severe purulent peritonitis or infected grafts. There is evidence that the presence of enteric contamination is not a contraindication to synthetic graft use.⁶¹ However, many vascular surgeons still recommend that an extra-anatomic bypass be considered in the presence of significant enteric contamination. If prosthetic material is used, it is important that any enteric spillage be controlled and that the peritoneal cavity be meticulously cleaned before prosthetic graft repair.

It is the authors' opinion that ligation of the common or external iliac arteries should never be performed, even for damage control in patients in critical condition. Ligation is poorly tolerated by most patients and is associated with a high incidence of limb loss, and subsequent attempts to revascularize the leg may cause severe reperfusion injury and organ failure or death. In patients who need damage control, flow can be restored with a temporary intraluminal shunt (see Fig. 182.9); definitive reconstruction of the vessel is performed at a later stage when the patient's condition has stabilized.

Venous injuries

Iliac venous injuries can be technically more challenging than arterial injuries because of the difficult exposure caused by their location behind the arteries (see Fig. 182.15). This problem is even more difficult on the right side because of the location of the right common iliac artery and the confluence of the two common iliac veins behind the right common iliac artery. Despite these difficulties, the recommendation to transect the iliac artery to access the underlying vein⁶² is extreme and should not be performed. In most cases, careful mobilization and retraction of the artery provides satisfactory exposure of the vein. Ligation and division of the internal iliac artery may be helpful in providing better exposure. Repair of the iliac veins by means of lateral venorrhaphy should be considered only if it can be performed without producing major stenosis. Ligation is generally preferable to a repair that produces severe stenosis

because of the risk of thrombosis and pulmonary embolism.⁵⁷ Complex reconstruction with spiral grafts or prosthetic materials, especially in a critically ill patient, is not recommended. Ligation is usually well tolerated, although patients may develop transient leg edema. On rare occasions, ligation results in massive edema of the leg and compartment syndrome that requires fasciotomy. The management of iliac venous injuries in the presence of associated iliac artery injuries is even more controversial. We do not recommend complex venous reconstruction because patients with combined arterial and venous injuries are invariably in extremely critical condition, and any procedures that prolong the operation or increase blood loss should be avoided. However, many authors recommend venous reconstruction with patch venoplasty or PTFE grafts, although there is no evidence of improved outcome with this approach.

Compartment syndrome

Many patients with iliac vascular injuries develop extremity compartment syndrome. In this case, fasciotomy should be performed without delay, often before arterial reconstruction. However, the role of prophylactic fasciotomy is controversial and has been challenged by many authors. Prophylactic fasciotomy is a major procedure that is often associated with increased bleeding due to coagulopathy and increased venous pressures.^{63,64} If fasciotomy is not performed, the patient should be monitored closely with frequent clinical examinations and compartment pressure measurements. Fasciotomy should be performed at the first signs of compartment syndrome.⁵⁷ Perioperative administration of mannitol may play a beneficial role in reducing the effects of reperfusion injury and inhibiting the development of compartment syndrome and the need for fasciotomy.^{64,65}

Mortality

The reported overall mortality varies from 30% to 50% in arterial injuries and 25% to 40% in venous injuries. In isolated iliac vascular injuries, the mortality is approximately 20% for arterial injuries and approximately 10% for venous injuries.⁵⁷

Inferior Vena Cava Injuries

Anatomy

The IVC is formed by the confluence of the two common iliac veins in front of the L5 vertebra and underneath the right common iliac artery. It ascends over the spine to the right of the aorta; at the level of the renal veins it deviates farther to the right, courses behind the liver, crosses the diaphragm, and, after a short course of 2 to 3 cm in the chest, drains into the right atrium of the heart. In its course, the IVC receives four or five pairs of lumbar veins, the right gonadal vein, the renal veins, the right adrenal vein, the hepatic veins, and the phrenic veins. All lumbar veins are below the renal veins, and except for the right adrenal vein, there are no other venous branches between the renal veins and hepatic veins. Besides the three major hepatic veins, there are six to eight accessory veins inferiorly. Some of the accessory veins are large and may bleed profusely in the case of injury or iatrogenic avulsion.

Mechanism of Injury

The IVC is the most commonly injured abdominal vessel and accounts for approximately 25% of abdominal vascular injuries.¹ Blunt trauma is responsible for approximately 10% of IVC injuries, and it usually involves the retrohepatic segment of the vein.⁶⁶ In approximately 18% of patients with penetrating IVC injuries, there is an associated aortic injury.⁶⁷

Clinical Presentation

More than half the patients with IVC injuries who reach the hospital alive are hypotensive, and approximately 18% require emergency thoracotomy.^{67,68} Many patients with contained hematomas may be hemodynamically stable on admission. The diagnosis is almost always made intraoperatively.

Surgical Exposure

Many injuries to the IVC, especially those involving the infrarenal IVC, present with stable hematomas because of the low existing pressures within the IVC. As a rule, all hematomas due to penetrating trauma should be explored. An exception to this approach is the stable retrohepatic hematoma. Exploration of these hematomas is extremely difficult and may lead to uncontrollable hemorrhage and death. The infrarenal and juxtarenal IVC are best exposed by mobilization and medial rotation of the right colon, the hepatic flexure of the colon, and the duodenum (see Fig. 182.8). Exposure of the retrohepatic IVC is technically challenging and usually requires extensive mobilization of the liver by dividing its ligaments and extending the incision to include a right subcostal incision, right thoracotomy, or sternotomy. In our experience, addition of a subcostal incision provides excellent exposure and is the preferred initial step. If this is insufficient to allow access to the injury, a median sternotomy is performed. This provides excellent exposure to the injured structures, and if total hepatic vascular occlusion or an atricaval shunt is required, it allows access to the right atrium as well as the suprahepatic vena cava. Alternatively, if the patient already has a right thoracotomy, connecting this incision to the laparotomy and incising the diaphragm circumferentially, leaving sufficient cuff for reconstruction, allows excellent exposure to all of these structures. These additional incisions should be considered only if perihepatic packing is not effective in controlling the hemorrhage. In cases in which packing is not effective and the additional incisions are not sufficient for adequate visualization and repair of the IVC or hepatic veins, more radical maneuvers including hepatic vascular isolation, an atricaval shunt, or division of the liver should be considered.

Hepatic vascular isolation

Hepatic vascular isolation involves cross-clamping of the infradiaphragmatic aorta, suprahepatic IVC, infrahepatic IVC above the renal veins, and portal triad. Failure to clamp the aorta as a first step may result in severe hypotension and possible cardiac arrest due to the reduced venous return after occlusion of the IVC. The suprarenal IVC can be cross-clamped in the space between the superior surface of the liver and the diaphragm. However, with a juxtahepatic venous injury, this maneuver may worsen the injury if it is done incorrectly. As

discussed previously, accessing the suprahepatic IVC through a sternotomy or right thoracotomy⁶⁸ may be preferable because this will also improve exposure of the injury, facilitating what is under even the best of conditions a challenging repair. Even with hepatic vascular isolation, some back-bleeding can be anticipated.

Atricaval shunt

The atricaval shunt requires placing a tube through a purse-string suture in the appendage of the right atrium and directing it into the IVC, distal to the caval injury. Tourniquets should be applied around the intraperitoneal IVC and the suprarenal IVC.⁶⁹ A large-bore thoracostomy tube or a large endotracheal tube with clamp occlusion of the proximal end and one or two holes created to correspond to the endoatrial part of the tube can be used as a shunt. If an endotracheal tube is used, the inflated balloon can replace a tourniquet around the suprarenal IVC (Fig. 182.18). This is preferable to dissection around the IVC for tourniquet placement. Although numerous reports have highlighted the extremely poor results with atricaval shunts, in our experience, patients have survived when the shunt was used early. This approach entails a major escalation of the operation, but in appropriate cases, it should be used early in the operation.

Division of the liver

Another option is to divide the liver along the gallbladder-IVC plane to provide direct exposure to the IVC. This approach increases bleeding, especially in an already coagulopathic patient, and should be avoided except when the liver is already severely injured and the IVC can be exposed with minimal dissection.

Surgical Options

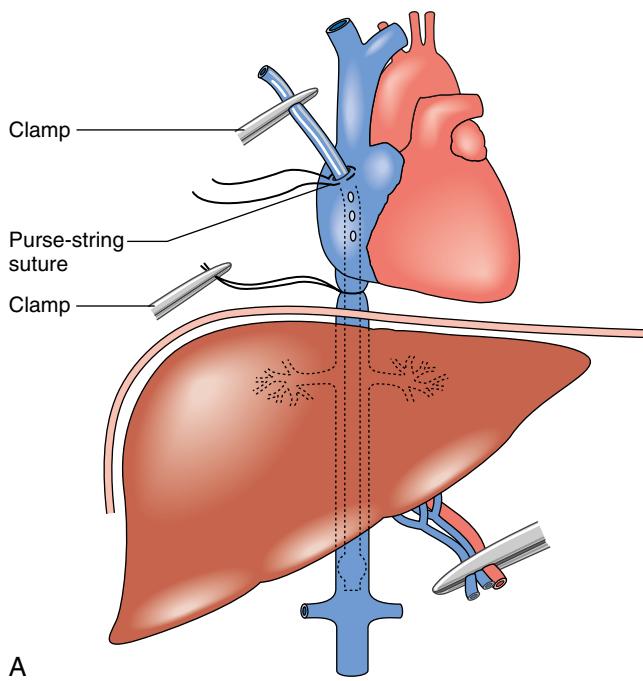
In most patients the IVC can be repaired by lateral venorrhaphy with 3-0 or 4-0 vascular suture material (Fig. 182.19). Most posterior caval wounds can be exposed and repaired by rotating the IVC. In some patients with anterior and posterior caval injuries, the posterior wound can be exposed and repaired from within the vein by extending the anterior wound. Ligation of the vein should be considered in hemodynamically unstable patients with severe infrarenal injuries or when repair produces major stenosis. Ligation of the suprarenal IVC is not an acceptable option because it results in renal failure. In unstable patients an intravascular shunt should be used to temporize.

Postoperatively, patients with IVC ligation should have their lower extremities wrapped with firm elastic bandages and elevated. Most patients develop temporary edema that subsides within a few weeks. However, some patients develop extremity compartment syndrome and require fasciotomy.

In patients with severe IVC stenosis after lateral venorrhaphy, there is a risk of thrombosis and pulmonary embolism. If this cannot be avoided, a caval filter or clips should be deployed above the site of stenosis.

Mortality

Approximately half the patients with IVC injuries die before reaching medical care, and among those who arrive at the



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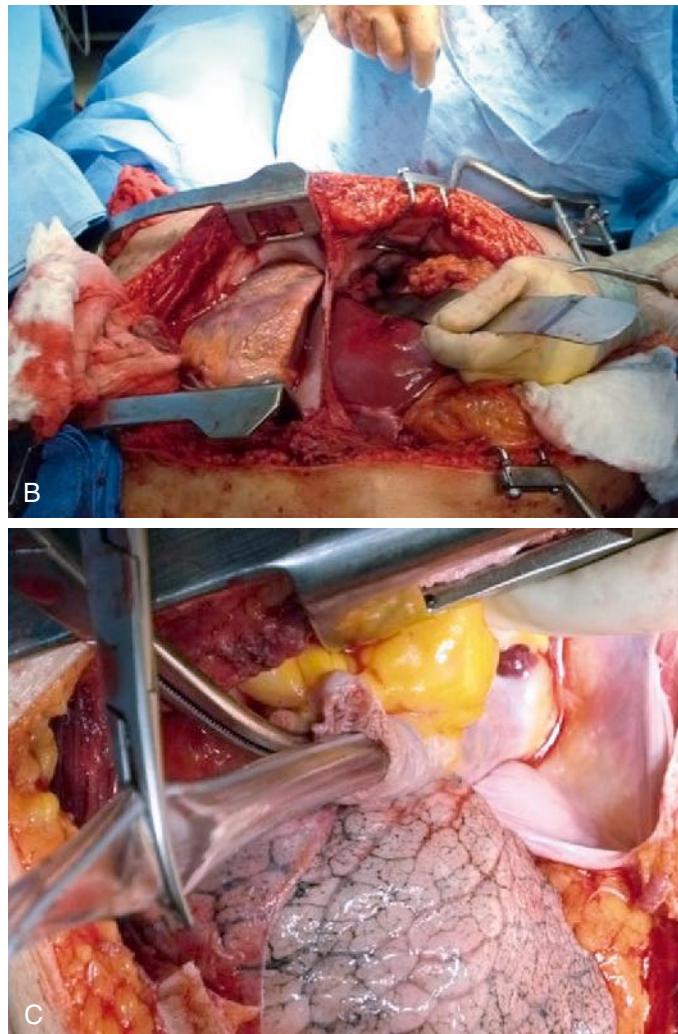


Figure 182.18 Atriocaval Shunt for Severe Retrohepatic Inferior Vena Cava (IVC) Injuries. (A) An endotracheal tube is placed through a purse-string suture in the right atrial appendage, and the cuff is inflated above the renal veins. A tourniquet is applied in the intraperitoneal IVC. Note the extra holes in the endoatrial part of the tube. This technique should be considered when liver packing does not control hemorrhage. (B) Median sternotomy with laparotomy in preparation for atriocaval shunt placement. (C) Insertion of the shunt through the right atrium.

hospital with signs of life, the mortality ranges between 20% and 57%.⁶⁶ In a study of 136 cases with IVC injuries, Kuene et al.⁶⁶ reported an overall mortality of 52%. In patients reaching the operating room alive, the mortality was 35%. The mortality was significantly higher in suprarenal injuries.

Portal Venous System Injuries

Anatomy

The portal vein is 6 to 10 cm long and is formed by the confluence of the SMV and the splenic vein behind the neck of the pancreas, to the right of L2, and to the left of the IVC. It passes behind the first part of the duodenum and enters the hepatoduodenal ligament. In the hepatoduodenal ligament, it courses between and behind the common bile duct to the right and the hepatic artery to the left. In the hilum of the liver, it splits into right and left branches. The portal vein has no valves and provides approximately 80% of the hepatic blood flow. The SMV trunk crosses over the third part of the duodenum and

the uncinate process of the pancreas. It then passes behind the neck of the pancreas, where it joins the slightly smaller splenic vein to form the portal vein. The splenic vein courses along the superior border of the pancreas and drains the inferior mesenteric vein, just before the confluence with the SMV.

Mechanism of Injury

Injury to the portal vein trunk is relatively rare and is found in approximately 1% of patients undergoing laparotomy for trauma.⁷⁰ More than 90% of portal vein injuries are due to penetrating trauma. Because of its proximity to other major vessels, the incidence of associated vascular injuries is high, ranging from 70% to 90% of cases.⁷¹

Clinical Presentation

Most patients with penetrating injuries to the portal venous system present with signs of hemorrhagic shock and require emergency laparotomy. In blunt trauma the injury usually involves the SMV and is due to a direct blow to the abdomen or

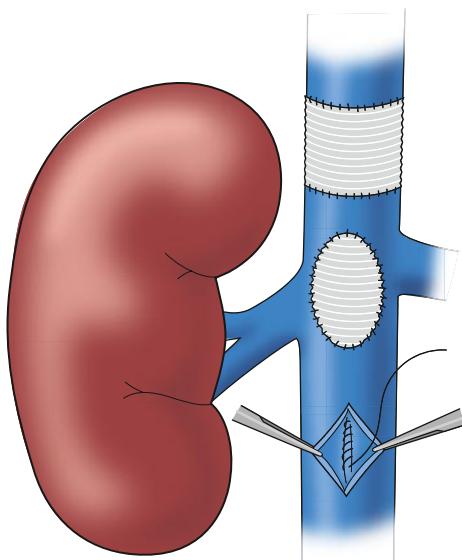


Figure 182.19 Injuries to the inferior vena cava can be managed with lateral venorrhaphy, patch repair or interposition graft. Perirenal and suprarenal injuries should not be ligated. (From: Lam, et al. Inferior vena cava. In: Demetriades, et al., eds. *Atlas of Surgical Techniques in Trauma*. 2nd ed. Cambridge University Press; 2019:291–310).

deceleration forces. These mechanisms often result in thrombosis of the vessels and occasionally avulsion and bleeding. In cases with thrombosis, the diagnosis is delayed and is often made on CT.

Surgical Treatment

The operative findings usually include a combination of local hematoma and various degrees of hemorrhage. Exposure of the retropancreatic portal vein and its major branches can be achieved by mobilization and medial rotation of the right colon and hepatic flexure of the colon and extensive Kocher mobilization of the duodenum. However, this approach often does not provide satisfactory exposure, especially in patients with associated injuries to the SMA. In these cases, stapled division of the neck of the pancreas provides excellent exposure and should be considered early (Fig. 182.20). The suprapancreatic portal vein can be exposed by a combination of mobilization and medial rotation of the right colon and hepatic flexure and a Kocher maneuver.

The portal vein and SMV should be repaired if this can be achieved with lateral venorrhaphy. Complex reconstructive procedures, such as interposition grafts, are rarely feasible or advisable because of the patient's poor condition. Approximately 80% of patients have other vascular injuries that contribute to blood loss and coagulopathy. Complex reconstruction should be undertaken only in patients with associated hepatic artery injury that cannot be repaired. Ligation of both the portal vein and the hepatic artery is not compatible with life. In these cases, reconstruction of the portal vein with a saphenous vein graft should be considered.⁷¹

Ligation of the portal vein with a patent hepatic artery is compatible with life, and the survival ranges from 55% to 85%.^{72,73} After ligation of the portal vein or SMV, the bowel becomes massively edematous, and patients can develop patchy

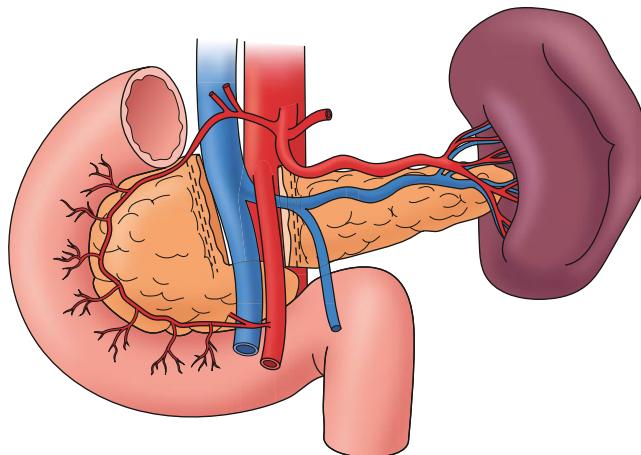


Figure 182.20 Stapled division of the neck of the pancreas provides good exposure of the retropancreatic portal vein and the superior mesenteric vessels.

bowel wall necrosis. The abdomen should never be closed primarily because, without exception, all patients develop abdominal compartment syndrome. Temporary abdominal wall closure should be performed. Second-look laparotomy should be performed in 24 to 72 hours to check the viability of the bowel.

Postoperatively, massive fluid replacement is required because of sequestration in the splanchnic bed. During the next few days, there is significant improvement of bowel edema due to enlargement of the collateral circulation, and abdominal wall closure may be possible. Experience with the long-term effects of portal ligation is limited, but there is evidence that most survivors do not develop portal hypertension.^{70,72,73}

Mortality

The mortality in portal vein injuries is high and ranges between 50% and 72%.^{1,72,73} Most patients have other major injuries, making it difficult to assess the mortality directly related to isolated portal vein injuries.

ADVANCES IN THE MANAGEMENT OF ABDOMINAL VASCULAR INJURIES

There have been some significant advances in the management of abdominal vascular injuries in the past decade. The policy of "scoop and run" and early surgical control of bleeding have now become the standard of care and have improved survival in patients with vascular injuries. The concept of damage control has gained popularity and acceptance, and as a result, many patients with vascular injuries have been saved. The recognition of abdominal compartment syndrome and the use of temporary abdominal wall closure are also important steps in improving outcomes. Endovascular technology has revolutionized the management of selected patients with specific vascular occlusions, arteriovenous fistulae, and false aneurysms. Hybrid operating rooms with angiographic radiology capabilities provide an opportunity to improve the management of abdominal vascular injuries by either temporary control of life-threatening

bleeding with endovascular balloon tamponade or definitive care with angioembolization or stenting. The development of intraoperatively placed sutureless, expandable stent grafts during open surgery is promising and may allow quick and safe vascular reconstruction. Finally, research into powerful new hemostatic agents is promising, and these agents may have a major impact on the management of abdominal vascular injuries.

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A complete reference list can be found online at www.expertconsult.com.

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Vascular Trauma: Extremity

DAVID S. KAUVAR and LARRY W. KRAISS

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GENERAL CONSIDERATIONS

Injury Characteristics

Although vascular injuries are present in a small percentage of injured patients, they are responsible for an outsized share of morbidity, mortality, and resource utilization.^{1,2} The major vessels of the extremities account for 20% to 50% of all vascular injuries^{1–5} (Table 183.1). Extremity arterial injuries result from blunt and penetrating mechanisms with nearly equal frequency although blunt mechanisms are more frequently accompanied by nonvascular extremity injuries as well as concomitant non-extremity injuries.^{6–9} Arterial injuries occur with nearly equal frequency in the upper and lower extremities.^{4,5,10,11} In the upper extremity, regardless of mechanism, the most frequently

injured arterial level is that of the forearm vessels. In the lower extremity, however, mechanism influences the arterial injury pattern, with the popliteal artery most frequently injured in blunt trauma and the superficial femoral artery (SFA) most frequently injured in penetrating trauma (Fig. 183.1). A blunt mechanism is seen more frequently in lower extremity than in upper extremity arterial injuries, and lower extremity injuries have accordingly higher incidences of associated tissue injuries, complications, and mortality.^{11,12}

Concomitant Extremity Injuries

In severe limb trauma, arterial injuries can coexist with fractures, peripheral nerve injuries, and significant muscle/soft tissue disruption; all of which can complicate management.^{7,8,12–16}

TABLE 183.1 Overview of Extremity Arterial Injury Epidemiology from Large Retrospective Studies

Author	Year	Total N	Arterial Injury	Extremity Arterial Injury	Penetrating	Upper Extremity	Lower Extremity	Comment
Mattox et al. ³	1989	Not reported	5,760	1961 (34%)	87%	859 (44%)	1102 (56%)	Vascular injuries only
Barmparas et al. ⁵	2010	1,380,563	22,089 (1.6%)	9937 (45%)	5068 (51%)	5855 (59%)	4082 (41%)	National Trauma Data Bank
Loh et al. ¹	2011	2157	50 (2.3)	14 (28%)	20 (40%)	6 (43%)	8 (57%)	
Perkins et al. ²	2012	5823	256 (4.4%)	87 (34%)	46 (53%)	41 (47%)	46 (53%)	
DuBose et al. ⁴	2015	Not reported	483	240 (50%)	179 (37%)	100 (21%)	240 (59%)	PROOVIT (AAST) Registry

PROOVIT, PROspective Observational Vascular Injury Treatment.

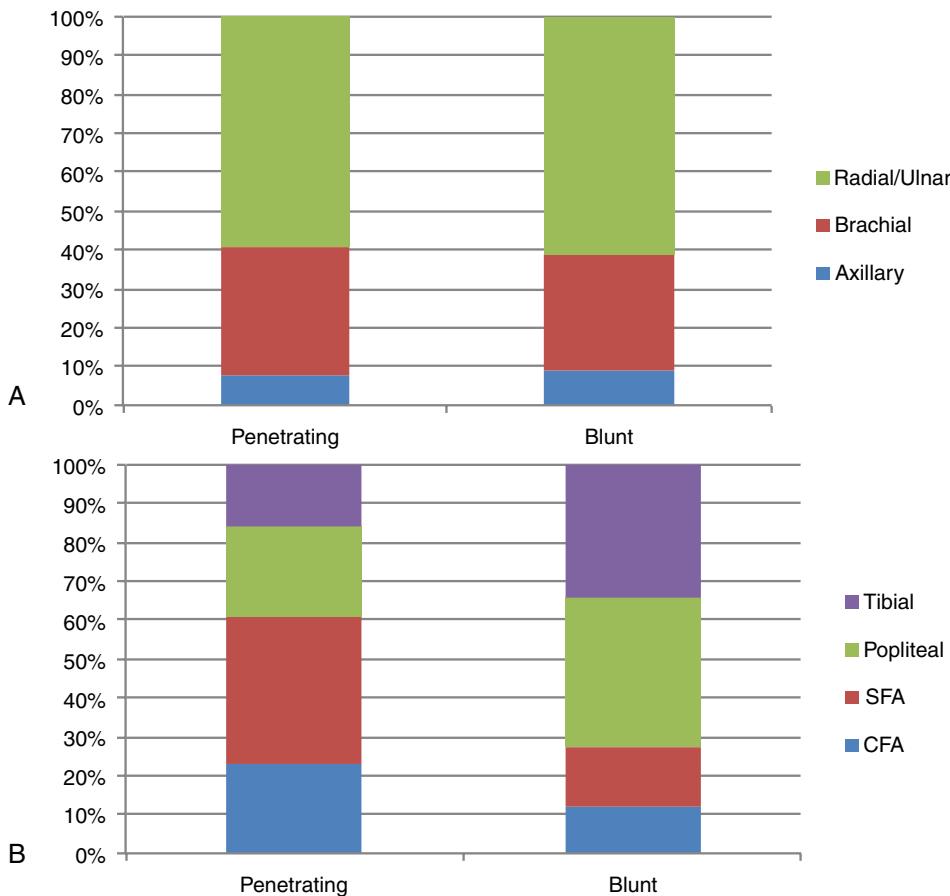


Figure 183.1 Relative frequency of injury to upper extremity (A) and lower extremity (B) arteries resulting from blunt and penetrating trauma. CFA, common femoral artery; SFA, superficial femoral artery.

Fractures are seen with especially high frequency in blunt trauma, with rates as high as 80% to 100% in some series, while in penetrating mechanisms, fractures are reported in only 15% to 40% of limbs with an arterial injury. Regardless of mechanism, fractures (especially comminuted and open fractures) represent a significant risk factor for amputation when combined with extremity vascular injury.¹⁷ Peripheral nerve injuries are difficult to detect in the acute setting and thus they may be underreported given the generally short follow-up and lack of limb functional outcomes in the current literature. In the lower extremity, most studies report rates of around 10% with upper

extremity vascular trauma associated with a rates of 40% to 50%. In general, the presence of a nerve injury does not seem to predispose to limb loss, though neurologic dysfunction may contribute to late amputation decision making if function does not improve over time.^{17–19} Significant soft tissue disruption often accompanies extremity vascular injuries, especially following blunt trauma. As with nerve injuries, the incidence appears to be higher in the upper extremity, reported as 40% to 70%, than in the lower extremity, for which rates are typically reported to be around 30%. The presence of a significant soft tissue deficit does appear to correlate with early amputation in

lower extremity arterial trauma and can add significant complexity to initial surgical efforts.¹⁷

Concomitant and Isolated Vein Injuries

The incidence of named venous injury in the setting of extremity arterial disruption ranges between 15% and 35%, though many (especially upper extremity and infrapopliteal) injuries likely go unreported. Combined major artery and vein injury implies a more severe limb injury complex but is a generally unreliable indicator of poor limb salvage prognosis, regardless of whether the vein is reconstructed or ligated.^{7,8,16,17} Major extremity venous injury in the absence of arterial injury is sparsely reported and is not associated with high rates of limb loss.^{20–23}

There is no consensus as to whether limb outcomes are improved by repair (versus ligation) of extremity venous injuries, though the redundant venous anatomy of the upper extremity distal to the axillary vein and in the calf suggests that vein ligation in these areas is safe in all but the most severely injured (i.e., mangled) extremities where all major venous outflow is disrupted. In the lower extremity, ligation carries the theoretical risk of precipitating venous hypertension potentially leading to compartment syndrome acutely and symptomatic venous insufficiency chronically. These risks remain theoretical, however, having not been consistently demonstrated.^{21,23} Fasciotomies are performed in a high percentage of lower extremities with major venous injuries regardless of the management of the vein, and such a practice seems prudent.^{20,24} Especially in the setting of an arterial injury, presence of a major venous injury is an argument in favor of prophylactic compartment decompression. Extremity venous injury is associated with a 30% to 50% risk of venous thromboembolism (VTE), but VTE risk does not seem to be related to whether the vein underwent repair or ligation, and recent studies have observed higher VTE rates in patients with repaired than ligated extremity venous injuries.^{23,25} The development of limb edema is expected following ligation of a major lower extremity venous injury and is more common with ligation at the popliteal than at the femoral level, with transient swelling seen in up to 90% of ligated popliteal and 50% of ligated femoral injuries. Repair reduces the transient edema by about half, to 50% and 29%, respectively.²⁶ Nearly all patients, regardless of the status of the injured vein, experience significant resolution of edema by the time of or shortly after discharge from the hospital.^{26–28} Significant edema is far less common after ligation of major upper extremity veins due to robust venous collaterals.

Extremity venous reconstruction following severe limb trauma can be technically demanding and should only be considered in patients who can physiologically tolerate the procedure. Reconstruction in the setting of severely compromised limb outflow resulting in early arterial reconstruction thrombosis (or early thrombosis of an arterial shunt) may help to preserve perfusion. This is most likely to be necessary in mangled extremities with venous disruption at the axillary, femoral confluence, and popliteal levels. Vein reconstructions should be preceded by distal thrombectomy performed with an

Esmarch tourniquet and may consist of simple suture repair, lateral venorrhaphy, patch, or interposition graft. The short-term (days to weeks) patency of lower extremity femoral and popliteal venous reconstructions is reported to be around 70%, but large samples of longer-term follow-up of venous repairs are not available.^{22,29,30} Infrapopliteal venous repairs have almost universally poor outcomes and are not recommended.³¹ In general, more complex repairs (spiral, panel, and interposition grafts) have poorer short-term patency rates than simpler repairs; the use of polytetrafluoroethylene (PTFE) grafts in larger vessels has not demonstrated inferior short-term patency compared to autologous vein.^{29,30,32}

Outcomes

Extremity vascular injuries present potential threats to life, limb, and function. Severe injuries carry the acute risks of exsanguination and early limb loss, followed later in the patient's course by the threats of late amputation or functional extremity impairment.^{8,11,33–36} Successful management of extremity vascular trauma requires hemorrhage control to prevent mortality followed by reperfusion to avoid limb loss and, hopefully, restore limb function. When considering the outcomes of vascular limb salvage, it is important to bear in mind the short follow-up periods reported in most studies of civilian trauma patients. The majority of studies report outcomes from the period of initial hospitalization or have follow-up periods of less than one year and data on long-term graft patency and limb and patient functional outcomes are infrequent.^{17,36,37}

Mortality

Exsanguination from extremity arterial injuries is rare, occurring in around 2% of cases.^{8,11,12} Penetrating mechanisms predominate cases in which mortality can be attributed to an extremity vascular injury itself.^{8,33,38,39} Injuries to the proximal vessels of the lower extremity are the most likely to result in exsanguination, with decreasing mortality moving distally. Most extremity exsanguination occurs in the prehospital setting and though the military-inspired adoption of field tourniquets in civilian emergency medical services (EMS) may serve to prevent some of these deaths, junctional (axillary and common femoral) injuries remain difficult to control outside of the operating room.⁴⁰ In all cases, vascular limb salvage begins with establishing expeditious hemostasis in the affected extremity.

Amputation

In most cases, the primary goal of vascular limb salvage after arterial injury is restoration of perfusion to prevent early limb loss, provided vascular reconstruction does not threaten the patient's life. Primary amputation – or removal of the injured limb before attempted revascularization – is rarely necessary. Primary amputation is more often dictated by the patient's overall physiologic condition rather than the surgical challenge of vascular reconstruction.

Secondary amputation refers to limb loss following an attempt at vascular limb salvage. Reported early limb loss rates

for upper extremity arterial injuries are around 2% and limb loss is associated with blunt mechanism and multiple tissue injury, proximal arterial injuries, or disruption of both forearm vessels.^{11,12,37,41} In the lower extremity, secondary amputation rates are reported around five percent in most studies. Blunt high-energy mechanisms predominate and significant damage to bone, nerve, and soft tissue typically drives the decision for amputation rather than the vascular injury itself.^{7–9,11,15,18,42} Consistently identified risk factors for amputation in cases of lower extremity arterial injury include: high-energy mechanism, older age, multiple arterial injuries, severe soft tissue injury, fracture, and development of compartment syndrome.¹⁷

Injuries to both forearm vessels and to multiple tibial vessels are associated with particularly high amputation rates. Given the anatomy of the forearm and calf, both injury patterns indicate trans-extremity tissue disruption, likely accounting for high limb loss rates. Most tibial arterial repairs occur in the setting of injury to multiple tibial vessels; this accounts for the high amputation rates reported following tibial vascular reconstruction.^{43,44} Injury to the popliteal artery is associated with amputation rates as high as 30% because it is the single vessel contributing inflow to all the tibial arteries.^{14,45–48} As with other arterial injuries, blunt mechanism is more likely to result in limb loss in popliteal trauma. One particular arterial reconstruction associated with a high rate of amputation is one in which the distal target is infrapopliteal.⁴⁴ Many injuries requiring such a reconstruction have limited runoff and disrupted geniculate collaterals making the limb completely dependent on a high-risk graft.⁴⁹

Functional Outcomes and Quality of Life

Little investigation has been made into limb- and patient-level functional and quality-of-life outcomes following vascular limb salvage for injury, primarily due to low follow-up rates among trauma patients.^{35,36} These outcomes in lower extremity amputees seem to be understood, with broadly equivalent functional outcomes between amputees and those in whom the limb was salvaged.^{42,50} Specific long-term limb salvage and graft dysfunction rates and their implications are not known. Prolonged ischemic time and neuropathy predict poorer limb function in the upper extremity regardless of graft patency.³⁶ The available data suggest that long-term salvage of upper extremities following traumatic arterial injury is expected and that even when offered amputation for severe limb dysfunction, patients generally prefer to keep even a minimally functional arm.

The available data for the lower extremity suggest that, though long-term function is acceptable, advancing age, blunt mechanisms (and associated limb injuries), and prolonged ischemic times predict poorer extremity function.^{34,46} These findings suggest that ischemic neuropathy has longstanding consequences for vascular limb salvage efforts and highlight the importance of expeditious limb revascularization. Information regarding the social impact of limb salvage in older chronic patients is only beginning to emerge; such outcomes data are all but unavailable for vascular trauma patients.^{51,52} Without these data, we cannot truly understand the impact of extremity vascular injury on patients and their communities.

PRESENTATION, DIAGNOSIS, AND WORKUP

Rapid identification and efficient localization of extremity vascular injuries are of great importance in the early evaluation of a trauma patient as decreased time to revascularization has been identified as an important predictor of successful vascular limb salvage.⁵³ Patient and limb characteristics present on patient arrival represent key indicators of arterial injury and can be used to plan early workup and initial intervention maneuvers.⁵⁴ Traditionally, the presence or absence of “hard” and “soft” signs of vascular injury at presentation have been used to guide the workup of severe extremity trauma. Hard signs include absence of distal pulse, active pulsatile bleeding, palpable thrill or audible bruit, and expanding hematoma and are believed to provide definitive evidence of an arterial injury mandating immediate operative exploration. Soft signs are considered to be suggestive of an arterial injury and include diminished distal pulses, reported history of significant bleeding, neurologic deficit, and proximity of a wound to a named vessel. The presence of any of these has been used to suggest additional imaging evaluation, traditionally catheter-based angiography. Hard and soft signs were developed over 30 years ago for the evaluation of patients with penetrating limb trauma and have not been consistently validated in blunt trauma patients, nor with the routine use of computed tomography angiography (CTA), the currently preferred imaging modality.^{55–57} An assessment for hard and soft signs of vascular injury during the initial trauma evaluation may suggest the presence of an extremity arterial injury and inform the decision to investigate further, but whether the signs are “hard” or “soft” does not adequately characterize the limb’s presentation, and is therefore of limited clinical utility.

Hemorrhagic and Ischemic Signs

Limbs with suspected arterial injury generally present in one of two ways; either with significant or ongoing bleeding or with clinical evidence of impaired distal perfusion. The clinical signs that predominate in either of these two presentations can be categorized as “hemorrhagic” or “ischemic” (Table 183.2). This categorization not only helps to identify, but also characterizes the likely nature of the injury and therefore informs not only the diagnosis, but also the early management of a limb with a suspected vascular injury.

Penetrating trauma causing vascular disruption is most likely to result in a predominance of hemorrhagic signs, primarily found on initial physical exam. The presence of hemorrhagic signs indicates potentially life-threatening major arterial disruption requiring urgent measures to obtain hemostasis, especially if the patient exhibits shock. Urgent bedside hemostasis measures represent the first stage of vascular damage control (further discussed later). Temporary hemostasis should be followed by a rapid evaluation of the likely level of arterial injury and the preferred locations for inflow and outflow control. Some initial vascular damage control interventions may alter or preclude useful imaging, but if a patient with hemorrhagic

TABLE 183.2 Hemorrhagic and Ischemic Signs of Extremity Arterial Injury

Hemorrhagic Signs	Ischemic Signs
Active hemorrhage (especially pulsatile) from a limb wound	Diminished or absent distal pulse
History of large volume of limb hemorrhage	Monophasic or absent distal Doppler signal
Systemic hypotension not accounted for by other injuries	Injured extremity–brachial index <1.0
Pulsatile mass in proximity to suspected area of injury	Cool limb distal to suspected injury
Palpable thrill in proximity to suspected area of injury	Pallor distal to suspected injury
Hematoma (especially expanding) or limb circumference discrepancy	Impaired motor or sensory function distal to suspected injury

signs quickly responds to initial resuscitation, a rapidly performed CTA may greatly assist operative planning. If these conditions cannot be met, urgent operative vascular control is mandatory.

Arterial occlusion resulting in ischemic signs of vascular injury is more likely to follow blunt trauma. In traumatized limbs with suspected ischemia, the distal arterial Doppler exam and measurement of the injured extremity index (ratio of the injured extremity distal systolic blood pressure to that of an uninjured extremity) if anatomically possible can provide important clinical information.⁵⁸ Ischemic signs are frequently accompanied by concomitant fractures and other limb tissue injuries, gross identification of which may assist in general localization of the vascular injury. Though timely revascularization in the face of an ischemic limb is important, procedural planning in a limb with ischemic signs benefits from preoperative CTA imaging because reconstruction in these cases can be a complex undertaking requiring understanding of the entire limb injury picture. It is important in cases in which ischemic signs predominate to ascertain the length of time that the limb has been ischemic to best assess the need for temporary reperfusion vascular damage control strategies such as shunting and the benefit of fasciotomy.

Computed Tomographic Angiography

In recent years, CTA has emerged as the diagnostic modality of choice to identify and localize upper and lower extremity arterial injuries resulting from both blunt and penetrating trauma (Figs. 183.2 and 183.3). The modality is readily and rapidly available in virtually all trauma centers and most seriously injured patients undergo contrasted CT scans as part of their evaluation within minutes of arrival.⁵⁹ Though not universally required for identifying arterial injuries, CTA offers additional value in imaging the bone and soft tissues of the injured extremity simultaneously with arterial interrogation and can provide otherwise unavailable information regarding the status of inflow and outflow vessel candidates.^{60,61} In addition, CTA can assist in the diagnosis of major venous injuries if a delayed phase scan is performed. Both the sensitivity and specificity of CTA in detecting even clinically occult arterial injuries approach 100%.^{62–64} Even difficult-to-identify below-knee and distal upper extremity arterial injuries can successfully be identified and characterized using high-resolution CT imaging.^{65,66}

CTA has some limitations in the evaluation of limb trauma with some studies reporting inconsistent contrast opacification of the arterial system if the timing of the intravenous bolus is not optimal. In addition, imaging artifact from metal fragments can limit diagnostic utility. In patients with a large number of fragments, such as those with shotgun injuries, conventional angiography may offer better diagnostic capability than CTA, but modern artifact-reduction imaging protocols may mitigate this limitation.⁶³

MANAGEMENT PRINCIPLES

Vascular Damage Control

Vascular damage control describes a complement of early temporizing maneuvers short of definitive reconstruction performed with the intent of maximizing life and limb salvage in patients with anatomically complex and physiologically significant injuries. These maneuvers present considerations unique to the treatment of patients with vascular trauma. Considerations for operative planning are presented in Table 183.3.

Temporary Hemorrhage Control

Limbs with a predominantly hemorrhagic presentation may harbor life-threatening bleeding and urgent temporary hemostasis measures may influence the workup and initial management of the vascular injury as noted above. Patients may present for vascular evaluation with a tourniquet placed by EMS; in such cases it is reasonable to loosen or remove the tourniquet to perform a vascular assessment if the patient will tolerate it.^{40,67} Additionally, tourniquets may be placed after a brief evaluation in the trauma bay to control hemorrhage while the patient is transported to the operating room.⁶⁸ The presence of a tourniquet can make preparing and draping the limb for surgery challenging and it may be reasonable to transition to direct manual pressure during the prep followed by a sterile operative tourniquet for temporary proximal vascular control. Junctional (proximal axillary and femoral) hemorrhage is not amenable to tourniquet placement and direct manual pressure is typically required until formal control can be established. Image-guided intraoperative endovascular balloon placement within the subclavian or iliac arteries is emerging as a less morbid option to achieve proximal control. Infrarenal resuscitative endovascular balloon occlusion of the aorta (REBOA) has been

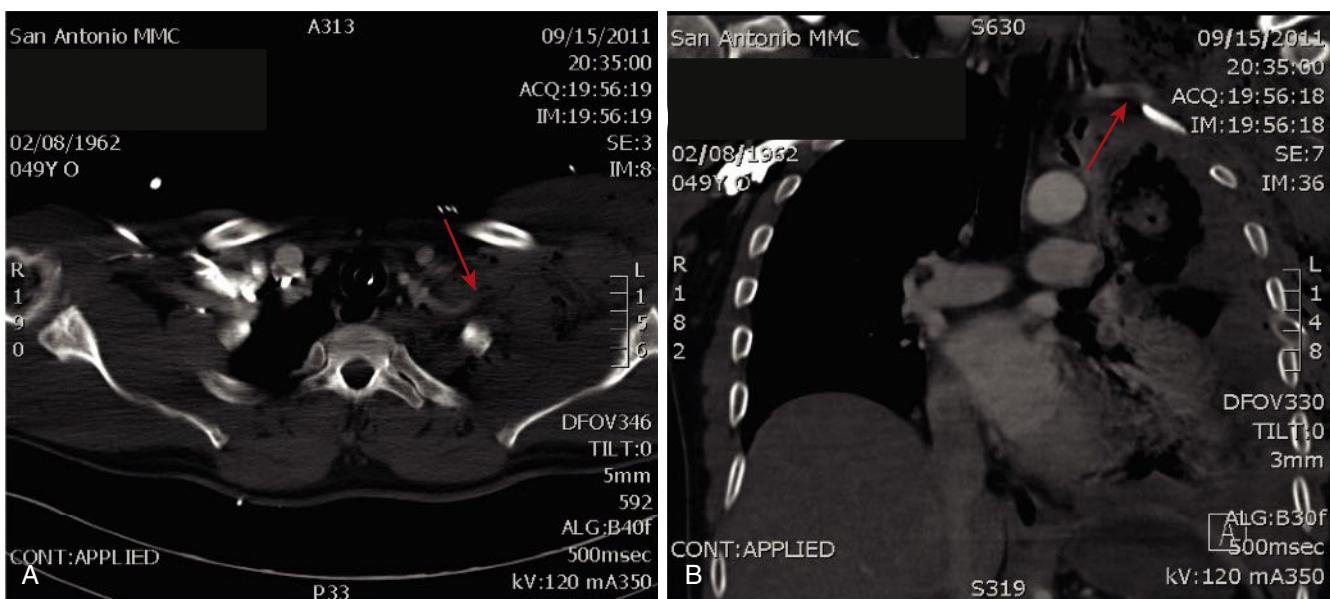


Figure 183.2 Computed tomographic angiography images from a patient sustaining a gunshot wound to the left shoulder with a pulseless left upper extremity. Note abrupt truncation of the left subclavian artery as it enters a large hematoma (A, arrow). Coronal reconstruction images reveal occlusion with intraluminal filling defect at the thoracic outlet (B, arrow).

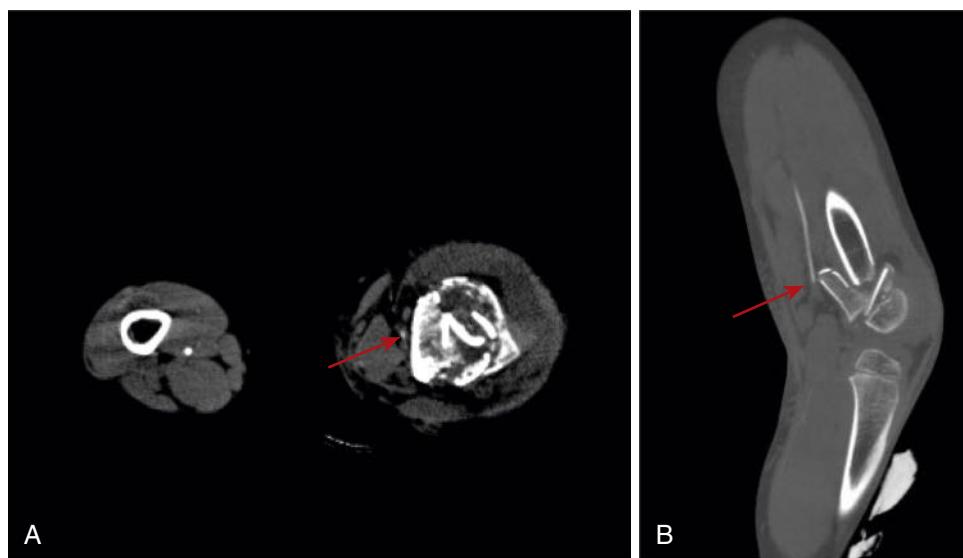


Figure 183.3 Computed tomographic angiography images from a patient sustaining blunt trauma to the left lower extremity with comminuted femur fracture. Note diminutive (likely spasm) left superficial femoral artery at the level of the fracture (A, arrow) just proximal to its abrupt truncation, seen best on reconstructed images (B, arrow).

used in the trauma bay to rapidly occlude arterial inflow in unstable patients with lower extremity junctional hemorrhage.⁶⁹

Temporary Shunting

The placement of temporary vascular shunts has gained wide acceptance as a measure allowing for the restoration of perfusion during orthopedic manipulation or to allow for intensive resuscitation or transfer of a patient prior to formal vascular reconstruction (Fig. 183.4). Shunting is employed more commonly in the lower than the upper extremity and is used primarily in vessels proximal to the elbow and knee. Civilian trauma centers report that 40% to 60% of shunts are placed to allow for resuscitation, with centers reporting dwell times predominantly under six hours.^{70,71} When placed to permit out-of-OR resuscitation

or transport, dwell times are predictably longer.^{72,73} Shunt thrombosis is directly related to dwell time and is more common in distal vessels, with overall thrombosis rates of about 5%.^{70–72} Thrombosis may be mitigated by administering systemic anticoagulation, but this may be contraindicated in multiply injured trauma patients. Dislodgment is an uncommon but possibly devastating complication and careful attention should be given to adequately securing the shunt proximally and distally, especially during limb maneuvers and patient movement.

Any flexible tube with a lumen can be used as an improvised vascular shunt (pediatric feeding tubes are frequently employed). When improvising a shunt, attention should be paid to ensuring adequate luminal diameter to support the intended flow and to providing adequate length to prevent

dislodgement. The most commonly used commercially available shunts are the Argyle and Sundt devices. The Argyle is a relatively rigid simple plastic tube while the Sundt is a flexible, reinforced silicone tube with flared ends to prevent dislodgement. The Argyle is simpler to use, but because it rests entirely within the vessel it risks occluding collateral vessels and it has no protection against dislodgement other than intravascular length. The Sundt is inserted only as far as needed to secure the flared ends of the device and can be looped externally to allow for safe patient and limb movement.

Based on retrospective data suggesting that ischemic time is related to limb complications including amputation, placement of temporary shunts should be considered in any extremity arterial injury case in which revascularization might be delayed for any reason.^{53,74} If there is combined disruption of a proximal limb artery and vein, especially in the face of significant tissue injury (mangled extremity, traumatic amputation), consideration should be given to shunting the venous injury followed by the arterial injury to prevent venous outflow congestion and significant hemorrhage.

TABLE 183.3 Operative Planning Considerations for Extremity Vascular Injury

Sequencing	Consideration of temporary shunting Temporary fracture reduction Vascular reconstruction Fasciotomy
Technique	Hemorrhage control (balloon, tourniquet) Endovascular, open, hybrid Equipment availability and limitations
Inflow and outflow exposures	Incision placement Clamp requirements
Conduit choice/location	Surgical prep and drape Wound category
Tissue coverage	Local flap Negative pressure dressing

Fasciotomy

Development of compartment syndrome is associated with poor vascular limb salvage outcomes in terms of amputation and dysfunction. Limbs undergoing an arterial reconstruction, particularly following a prolonged ischemic interval and in the presence of orthopedic injury, are at high risk for developing this complication and in these cases early (so-called “prophylactic”) fasciotomy should be considered.^{75–77} Despite strong recommendations, fasciotomy is not universally performed, however, with only 40% of patients undergoing lower extremity arterial repair having the procedure.^{54,77} Performance of a fasciotomy concomitant with or soon after lower extremity revascularization is associated with a fourfold reduction in eventual amputation and other limb complications.⁷⁷ Lower extremity fasciotomies are performed more commonly for penetrating than for blunt trauma and are most commonly performed when the popliteal artery is injured, followed closely by the femoral arteries. In the upper extremity, compartment syndrome principally results from arterial injury proximal to the elbow, most frequently to the brachial artery. Approximately 20% of patients with brachial artery injuries will be diagnosed with compartment syndrome, and as with the lower extremity, the syndrome is more common in penetrating trauma. Multiple arterial injuries and fracture predict the development of upper extremity compartment syndrome.⁷⁵

Fasciotomy adds considerable wound burden to an injured limb, and resulting morbidity is relatively common as is the need for multiple (sometimes complex) closure procedures.⁷⁸ If the skin can be brought together, even loosely, without compromising the vascular reconstruction or inducing significant tension, this may help minimize additional wound burden. This may be accomplished with staples, interrupted nonabsorbable sutures, or with silastic vessel loops placed in a “Jacob’s ladder” configuration. The latter method allows for progressive closure at the bedside. Compartment syndrome is best prevented rather than treated, so early calf or forearm fasciotomy should be considered in all patients with restoration of distal perfusion after ischemia resulting from trauma, especially if multiple fractures or arterial injuries are present. Fasciotomy will be of

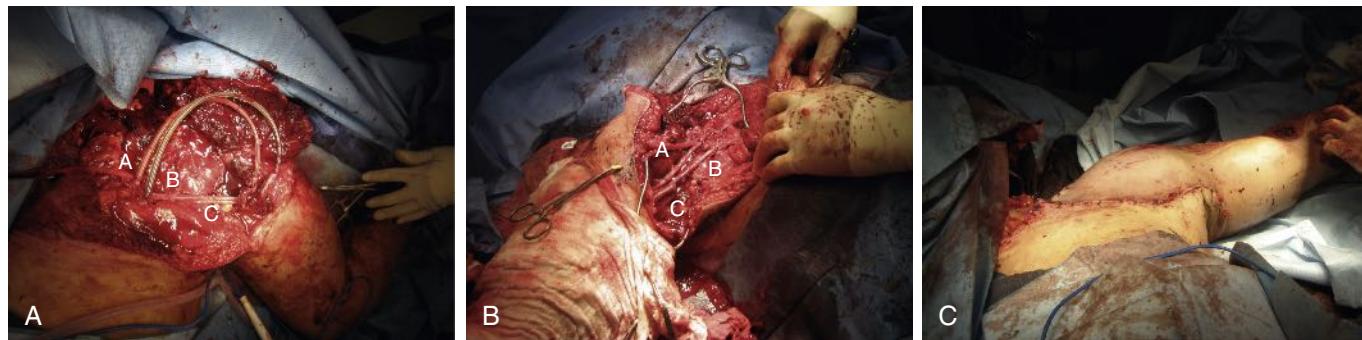


Figure 183.4 (A) Open scapulothoracic dissociation (torso to the left) before return to normal anatomic length with axillary arterial (A) and venous (B) shunts in place. Note brachial plexus component (C) on significant stretch before reduction. (B) After reduction, fixation, and interposition vein grafts to the axillary artery (A) and vein (B), the brachial plexus (C) is returned to its typical anatomic position and tension. (C) Normal-appearing external shoulder anatomy is restored after skin closure.

greater potential benefit with longer ischemic time. Thigh or upper arm fasciotomies should be considered for proximal arterial injuries, especially if there is a proximal venous occlusion and outflow is not restored with venous reconstruction.

Nonoperative Management

Limbs presenting with hemorrhagic or ischemic signs of vascular injury should in almost all circumstances undergo operative or endovascular exploration with the intent to control hemorrhage and/or restore distal perfusion. With the use of CTA imaging in the workup of injured extremities, arterial pathology can be identified without the use of conventional angiography and occult injuries may not require intervention in the majority of cases.^{79–82} Indeed, CTA may have increased the recognition of non-limb-threatening arterial lesions presenting the treating vascular surgeon with a different kind of management challenge.

Nonoperative management of selected arterial injuries is reasonable, such as those that produce no active hemorrhage or distal ischemia, including small intimal tears or flaps, pseudoaneurysms, and arteriovenous fistulas. Close clinical follow-up with serial surveillance using an appropriate imaging modality such as CTA or DUS is recommended along with a high index of suspicion for the development of pseudoaneurysm or ischemic complication. Early operative or endovascular intervention might be more appropriate if prospects for follow-up are uncertain.

Endovascular Therapy

The use of endovascular techniques to treat vascular injuries has increased significantly over the past two decades, with the bulk of the increase seen in the treatment of axial vascular injuries.^{83–86} In the extremities, endovascular therapy is used most frequently in blunt injuries to the legs and appears to have similar or improved outcomes compared with open repair of extremity arterial injuries despite being applied in patients with a significant burden of associated injuries and medical comorbidities.^{83–85,87–89}

Endovascular treatment is most appropriate when the morbidity difference between the open and endovascular procedures is greatest. This is most often the case in the setting of injuries to junctional (subclavian, axillary, and iliac) vessels.⁹⁰ In such cases, the use of endovascular balloon occlusion for proximal vascular control may obviate the need for opening a body cavity. If the vascular lesion can be safely traversed with a guide wire, definitive endovascular treatment is possible in some cases. Balloon angioplasty and bare metal stents have been used successfully to treat occlusive arterial lesions and stent grafts have been successfully deployed to treat injuries manifesting with hemorrhage and occlusion.^{91–93} Catheter-directed embolization with coils and/or glue is an option to treat smaller vessels and seems particularly successful when it is used for the treatment of small pseudoaneurysms and arteriovenous fistulas of the tibial and deep femoral branch arteries^{10,94,95} (Fig. 183.5).

With endovascular equipment and skills in increasingly ready supply, broader application in the treatment of extremity vascular injuries seems inevitable. Enthusiasm should be tempered by the fact that there are minimal long-term data available on the outcomes when these techniques are used “off-label” to treat such injuries.⁹⁶ Except in cases of shock or major concomitant injury, the early beneficial tradeoff of lower procedural morbidity with an endovascular procedure may not be favorable when compared against the likelihood of poorer long-term durability, especially in a relatively young trauma patient. Endovascular outcomes are predictably best in larger vessels, and caution is advised when smaller vessels such as the popliteal and brachial are treated. A period of antiplatelet therapy or therapeutic anticoagulation should be considered in these cases.^{91,96,97} In all situations, the most appropriate treatment should be determined on a case-by-case basis through communication between the trauma surgeon and endovascular provider.

Open Surgical Management Principles

Open surgical vascular control and repair remain the mainstays of management of extremity vascular injuries. If possible, operations should be performed on a table amenable to fluoroscopic imaging to facilitate orthopedic fixation and on-table angiography if needed. The extremity should be prepared and draped widely to facilitate exposure of inflow and outflow vessels, typically circumferentially in the case of extremity injuries. If the need for a vein graft is anticipated, the proposed harvest site should be included in the prepared operative field. Conventional practice is to obtain saphenous vein grafts from uninjured extremities to preserve collateral venous drainage. This dogma is not supported by good-quality data, however, and it may be appropriate to harvest conduit from an injured extremity in the absence of venous injury and if the vessel to be harvested is distant from the site of injury.⁹⁸

The general principles of establishing proximal and distal vascular control and restoring in-line flow to a suitable outflow bed are the same for the treatment of traumatic lesions as for any other vascular defect. Because traumatic vascular injuries frequently present with significant hemorrhage, the ability to expeditiously achieve vascular control is paramount. To ensure adequate exposure for control and repair, longitudinal or extensile anatomic exposures are preferred. Once vascular control is achieved, incisions can be extended as needed to expose the zone of vascular injury. Upper extremity control may require infraclavicular or supraclavicular incisions to expose the subclavian vessels. Retroperitoneal or inguinal ligament-splitting incisions may be required for proximal control in the lower extremity. If feasible, endovascular balloon control may reduce morbidity in such cases. Many trauma patients may be unable to tolerate systemic anticoagulation because of ongoing hemorrhage or associated injuries. In these patients, local anticoagulation with heparinized saline injection directly into the injured vessel proximal and distal to the injury may be employed.⁹⁹

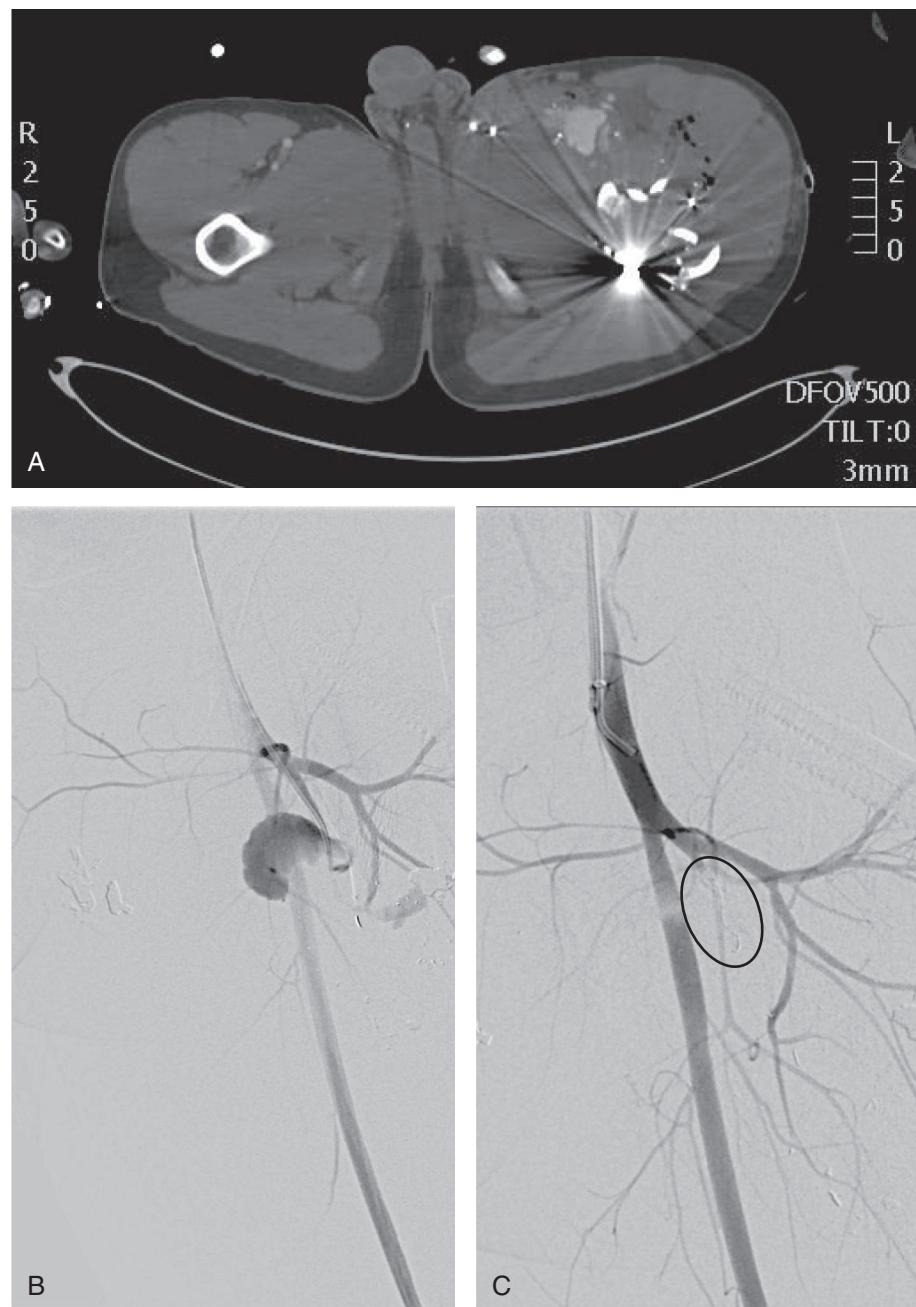


Figure 183.5 (A) Computed tomography angiography image demonstrating pseudoaneurysm of a profunda femoris artery (PFA) branch with thigh hematoma. (B) Femoral angiography revealing the PFA branch disruption. (C) Successful coil embolization of the disrupted PFA branch (*oval*).

Wide debridement of contaminated and nonviable tissue within the zone of vascular injury should be performed. During debridement, an assessment of the availability of healthy tissue to cover the vascular repair should be made; as such, coverage is essential to prevent desiccation and possible anastomotic breakdown or graft disruption. Tissue coverage may require coordination with other services operating on the injured extremity. The injured vessel should be circumferentially debrided to healthy vascular tissue before a final reconstruction plan is made. Intimal injury may extend beyond what is appreciated by an injured vessel's outward appearance and a subjective assessment should be made of the bleeding forward and backward from the injured vessel. If not thought to be

adequate, a balloon-catheter thrombectomy and/or a search for additional flow-compromising injuries should be undertaken. In severe limb injuries, especially those with hemorrhagic signs, extensive tissue injury, and venous outflow disruption, distal vessels may not exhibit strong back-bleeding. If this is the case and outflow vessel thrombectomy returns no thrombus, a temporary shunt can be placed for confirmation of distal patency with clinical and continuous-wave Doppler exam or on-table angiography confirming distal patency.

In general, provided the patient's physiologic status will tolerate it, arterial injuries should undergo urgent surgical repair with the goal of restoring inline distal flow. In extreme (and rare) cases, however, acute ligation of an injured extremity

artery may be the most feasible acute option. Sometimes this may be done with a plan for formal revascularization when the patient's overall condition stabilizes. Following an arterial ligation, the extremity should be observed extremely closely for signs of developing ischemia, which should prompt immediate placement of an arterial shunt or more formal revascularization (or amputation if neither of these can be safely performed).^{100,101}

Once the extent of the arterial injury is known, the simplest possible reconstruction should be performed. In general, three options exist for repair: in a limited number of cases, a small arteriotomy can be debrided and a patch angioplasty performed. In straight, nonbranching vessels with focal trauma, a short injured segment can be resected and enough of the vessel freed proximally and distally to perform a spatulated end-to-end anastomosis. If an end-to-end anastomosis is to be performed, the limb should be manipulated to ensure that tension will not be placed on the graft with flexion and extension.¹⁰² If neither of these is feasible, an interposition graft of appropriate length is indicated. Short-term results of vein grafts are good, with 30-day patency rates of over 90%. Most occlusions occur within the first week, suggesting technical problems as the etiology for graft failure.^{36,103,104}

Open traumatic wounds are considered contaminated, and the preferred conduit for repair is autologous saphenous vein. Infections involving prosthetic grafts are notoriously difficult to manage, resulting in high rates of graft explantation and amputation. These issues are exacerbated in severely injured extremities in which there may be inadequate clean, viable soft tissue coverage for the graft.^{100,104} As with reconstructions for chronic arterial disease, the mid- and long-term patency of

prosthetic conduit is likely to be poorer than that of autologous vein, especially in smaller vessels. The temporary placement of prosthetic conduits in extremity vascular reconstruction for trauma may be a viable damage control strategy to avoid the need for vein harvest, but no long-term outcome data are available and it cannot be recommended for formal vascular reconstruction. The use of alternative conduits such as bovine carotid artery and bioengineered vessels have been reported, but there are no series large enough to support a recommendation for their routine use.^{105,106}

SPECIFIC ARTERIAL INJURIES

Axillary Artery

Diagnostic Considerations

Deficiencies in distal pulses and frank ischemic changes are not universally seen in proximal upper extremity arterial injuries, and a deficit is appreciated in only about two-thirds of patients with injury to the axillary artery.^{102,107} A high clinical index of suspicion should be maintained when a patient has an injury in proximity to the course of the axillary artery, especially if there is evidence of a concomitant brachial plexus injury. Doppler indices and vascular imaging with CTA or angiography can be useful in making the diagnosis.

Surgical Considerations

Because the proximal portion of the vessel is relatively fixed and cannot be significantly mobilized longitudinally, injuries to the axillary artery are most commonly primarily repaired or treated with an interposition graft.^{107,108} Owing to the

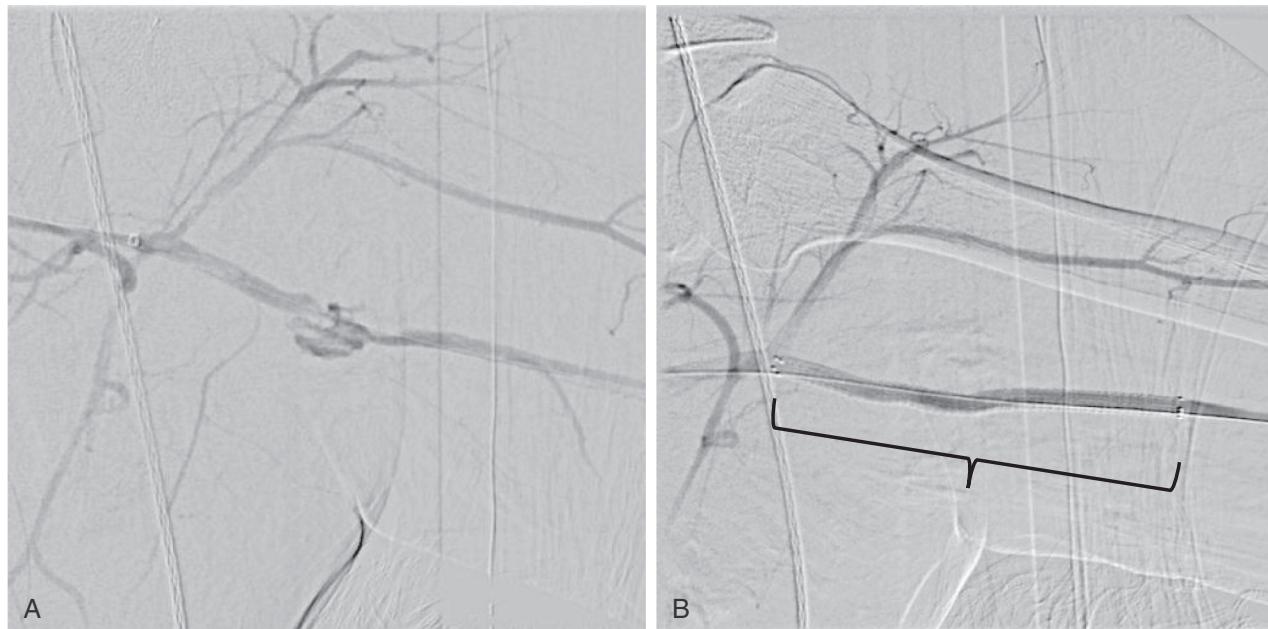


Figure 183.6 (A) Left axillary arteriogram revealing a pseudoaneurysm/disruption of the midportion of the vessel. (B) Successful deployment of a stent graft across the lesion (*bracket*).

proximal location of the axillary artery near the thoracic outlet, certain injuries in hemodynamically stable patients may be amenable to stent-graft treatment if the lesion can be safely traversed with a wire. The endovascular procedure can be performed antegrade by the femoral approach or retrograde by the ipsilateral brachial approach^{89,109} (Fig. 183.6).

Brachial Artery

Diagnostic Considerations

A pulse deficit is appreciated in about three quarters of patients with an injury to the brachial artery.^{102,110} The arterial injury is frequently associated with fractures of the humerus and dislocations of the elbow, so a high clinical index of suspicion is warranted when these skeletal injuries are present.¹¹¹

Surgical Considerations

The brachial artery's course is generally straight, and the vessel can be extensively mobilized and can be repaired with an end-to-end anastomosis in up to 50% of cases; most of the remaining injuries are treated with an interposition graft.^{12,104,110}

Radial and Ulnar Arteries

Diagnostic Considerations

Radial and ulnar artery injuries manifest with corresponding pulse deficits more than 80% of the time and can be associated with significant soft tissue trauma.^{12,102} An essential part of the diagnostic evaluation of a patient with suspected forearm arterial injury is a Doppler-based Allen test to confirm joint radial and ulnar contribution to the anastomoses of the palmar arches.¹⁰²

Surgical Considerations

If only one of the forearm arteries is injured and an Allen test reveals a patent palmar arch, the injured artery may be safely ligated in most cases. If the palmar arch is not patent in the absence of the contribution of the injured artery, the artery should be repaired. In instances in which both the radial and ulnar arteries are injured, preference should be given to repair of the ulnar artery, as it is most commonly the dominant contributor to the perfusion of the hand. The forearm arteries are commonly amenable to mobilization and end-to-end repair.¹²

Femoral Arteries

Diagnostic Considerations

Clinically apparent distal ischemia or pulse deficit is noted in more than 90% of femoral arterial injuries and multiple femoral vessel injuries are seen in up to 50% of cases.¹⁰⁰ An injury to the profunda femoris artery (PFA) may not itself produce diminished distal perfusion, but such injuries can manifest an expanding thigh hematoma or occult pseudoaneurysm. A low



Figure 183.7 Stab wound to the common femoral artery (CFA). In addition to the traumatic anterior CFA laceration (arrow), this vessel also had a smaller, full-thickness, posterior laceration. Note circumferential dissection and loop control of the CFA (A), superficial femoral artery (B), and proximal profunda femoris artery (C). The posterior laceration was primarily repaired with interrupted sutures and the anterior laceration was repaired with a vein patch to prevent loss of luminal diameter.

threshold for the use of CTA should be maintained in cases of penetrating inguinal injury.

Surgical Considerations

The common femoral artery (CFA) is most commonly repaired with a short-segment interposition graft or a vein patch for very focal injuries. Injuries to the CFA should be explored with circumferential dissection and control of the CFA and proximal superficial femoral artery (SFA) and PFA to ensure that concomitant femoral vascular and back-wall injuries are not missed (Fig. 183.7). The PFA may be ligated in an unstable patient, but it should be reconstructed if the patient's physiologic condition will tolerate because of the vessel's role in collateralization (Fig. 183.8). Injuries to branches of the PFA are frequently amenable to endovascular embolization. The SFA is typically repaired with an interposition graft, but if sufficient length can be attained by longitudinal mobilization, end-to-end anastomosis can occasionally be performed.

Popliteal Artery

Diagnostic Considerations

Clinically apparent ischemia is a feature of nearly all blunt and most penetrating popliteal injuries and duration of ischemia consistently predicts amputation. Blunt popliteal artery

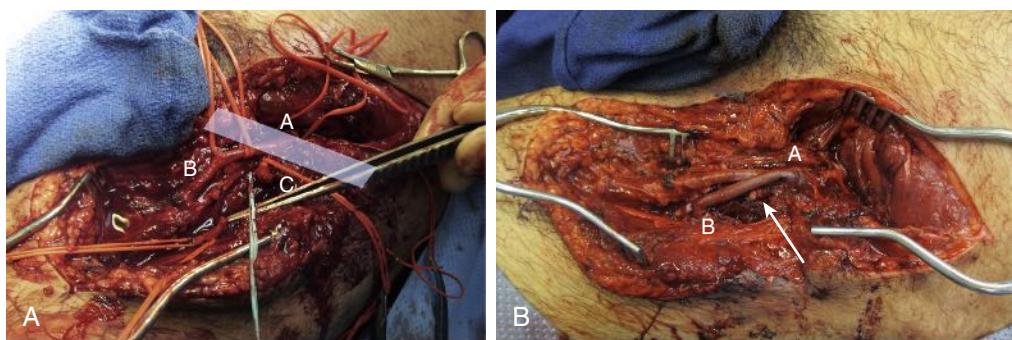


Figure 183.8 (A) Gunshot wound resulting in transection and segmental loss of the proximal profunda femoris artery. Control of the common femoral (*A*), superficial femoral (*B*), and proximal profunda femoris (*C*) arteries has been obtained. The distal profunda is at the forceps tip. The patient had a high femoral bifurcation, necessitating division of the inguinal ligament (*shaded box*) to achieve proximal control. (B) To preserve the most proximal profunda branches, repair was achieved through a common femoral (*A*) to profunda (*B*) interposition graft (*arrow*) with ligation of the proximal profunda stump.

injury is strongly associated with knee dislocations and fractures of the distal femur and tibial plateau as well as with popliteal vein and tibial nerve injuries. These associations and the critical location of the popliteal in the lower extremity arterial system result in reported amputation rates of 20% to 50% and a high incidence of significant long-term functional deficits.^{34,45,46,48,112–114}

Surgical Considerations

Focal above- or below-knee popliteal injuries are uncommon. In nearly all cases, the artery should be exposed via the medial approach with separate medial above- and below-knee incisions. Though the retrogeniculate popliteal artery cannot be directly exposed, this approach allows proximal and distal vascular control and extension to the SFA and tibial arteries if required and permits exclusion of an injury to any portion of the popliteal artery. Primary repair or anastomosis is not typically possible, and the majority of popliteal injuries require an interposition graft, typically tunneled intracondylar from the above- to below-knee popliteal or tibioperoneal trunk with more distal targets having worse outcomes.^{44,45,113} Due to their importance in perfusion to the calf muscles and to providing graft runoff, geniculate vessels should be preserved.⁴⁹

Tibial Arteries

Diagnostic Considerations

Clinically significant ischemia resulting from tibial artery disruption usually occurs in the setting of a severely injured limb because disruption of all three vessels requires a high-energy transtibial mechanism.^{66,115} The majority of tibial artery injuries undergoing reconstruction therefore result from blunt trauma and patients typically have multiple significant injuries.^{43,116}

Surgical Considerations

Very low amputation rates have been reported when single and even double tibial injuries are observed or ligated,

though this has recently been challenged.¹¹⁷ Due to the limb injury setting in which tibial injuries occur, it is difficult to determine the role of ischemia in the decision to amputate in these cases.^{43,116} In cases of multiple arterial injuries or single injuries with clinically ischemic limbs, reconstruction of at least one tibial vessel should be performed. If calf fasciotomies have been or are to be performed, the exposures of the anterior and deep posterior compartments can be used to access the vessels. Tibial–tibial bypasses are rare and the inflow source for bypass to a tibial target is frequently the popliteal artery. Therefore, the status of geniculate perfusion of the (usually severely injured) muscles of the calf and the potential for “interval ischemia” should be considered in the vascular limb salvage decision making process.⁴⁹ Isolated tibial artery injuries can be occult and late pseudoaneurysms and arteriovenous fistulas may develop. These can frequently be treated by endovascular embolization.⁹⁵

OTHER CONSIDERATIONS

Vascular Injury in the Mangled Extremity

Injured extremities with severe trauma to multiple tissue types (some combination of skin, muscle, nerve, and bone) are a difficult clinical problem. Such “mangled” extremities frequently present with one or more arterial injuries and clinically apparent ischemia. Numerous factors influence the decision to perform primary amputation or to attempt vascular limb salvage and there is no clear consensus about which are most appropriate. The Mangled Extremity Severity Score (MESS) was developed over 30 years ago as a decision aid in such cases and may also predict poor vascular bypass outcomes^{118–121} (Box 183.1).

A MESS of 7 or lower has historically been reported as correlating well with limb salvage of the upper or lower extremity, but advances in limb salvage have resulted in the suggestion

BOX 183.1**The Mangled Extremity Severity Score****Skeletal/Soft Tissue Injury**

Low energy (stab; simple fracture; pistol gunshot wound)	1
Medium energy (open or multiple fractures, dislocation)	2
High energy (high-speed motor vehicle crash or rifle gunshot wound)	3
Very high energy (high-speed trauma + gross contamination)	4

Limb Ischemia^a

Pulse reduced or absent but perfusion normal	1
Pulseless; paresthesias, diminished capillary refill	2
Cool, paralyzed, insensate, numb	3

Shock

Systolic blood pressure always >90 mmHg	0
Hypotensive transiently	1
Persistent hypotension	2

Age

<30 years	0
30–50 years	1
>50 years	2

^aScore doubled for ischemia >6 h.

Modified from Johansen K, et al. Objective criteria accurately predict amputation following lower extremity trauma. *J Trauma*. 1990;30:568–572.¹¹⁹

that a higher cutoff value may be more appropriate.^{41,119–121} Ischemia is a component of the MESS, but the degree of bone loss and soft tissue disruption has greater bearing on the decision to perform primary amputation.¹⁸ Neither the MESS nor any other extremity injury score accurately predicts functional outcomes in severe limb trauma.^{42,122} In addition, long-term functional status does not appear to be affected by amputation in a patient with severe lower extremity injury, although this conclusion is likely susceptible to significant selection bias.^{50,123,124} It is unclear whether or not this is also the case with mangled upper extremities.

Patients with mangled extremities and arterial injuries should undergo initial multidisciplinary consultation with the inclusion of a vascular surgeon. If an initial attempt at limb salvage is planned, vascular reconstruction should be performed, provided the graft or repair can be covered with viable tissue. In general, an attempt at initial limb salvage and early revascularization is appropriate. The need for reconstruction of arterial injuries should not be considered an indication for primary amputation, except in select instances, including cases of multiple-level or very long arterial injuries, requiring extensive

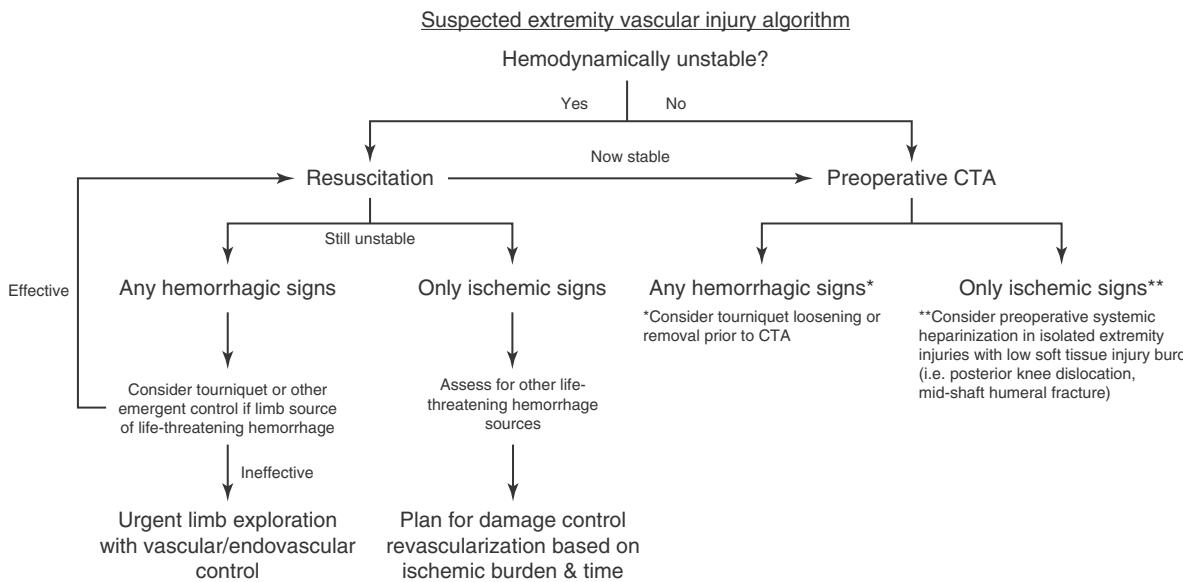
reconstruction or physiologic compromise precluding the reconstructive procedure. The general principle that a patient's life should not be sacrificed for the sake of an injured limb holds true in all cases.

Extremity Frostbite

The distal tissue necrosis seen in severe cold exposure injuries results from the combination of direct cellular destruction by freezing and thawing and severe peripheral vasospasm with post-rewarming small-vessel thrombosis. In combination, these pathologies frequently result in necrosis necessitating partial digital amputation. Thrombosis within the digital arteries, capillaries, and venules is not amenable to surgical treatment, but catheter-directed intra-arterial thrombolysis has been used as an adjunct to the standard treatment of extremities with severe frostbite.¹²⁵ The data supporting catheter-directed thrombolysis for the treatment of frostbite is principally in the form of case series demonstrating treatment safety and improvement in the angiographic appearance of the treated limb.^{126,127} Mixed results on the influence of thrombolysis on the rate and level of digital amputation using historical controls have been reported.^{128,129} Improved results are generally seen when treatment is initiated early in the hospital course in limbs demonstrating angiographic vascular occlusion.

In cases of acute frostbite, diagnostic angiography of distal extremities with clinically apparent ischemia after initial rewarming should be considered. If distal perfusion defects are present that are not solely due to vasospasm (persistent after vasodilator challenge), intra-arterial catheter-directed thrombolysis with recombinant tissue plasminogen activator may be performed. Treatment is initiated through an infusion catheter placed in a proximal extremity vessel (typically the brachial, superficial femoral, or popliteal artery) and can be continued for up to 48 hours, with repeated diagnostic angiography performed at 8- to 12-hour intervals and catheter-directed thrombolysis discontinued when angiography reveals no residual perfusion defects or improvement plateaus. Multiple extremities can be treated simultaneously through multiple arterial access points with the cumulative dose of tissue plasminogen activator administered (0.5 to 1.0 mg/h) divided between the affected extremities. Unfractionated heparin is administered at 500 units/h through each arterial sheath for the duration of treatment.

CHAPTER ALGORITHM



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Conditions Arising from Repetitive Trauma and Occupational Vascular Problems

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Upper extremity work-related injuries are a major societal problem, resulting in significant disability, cost, and loss of productivity. Occupational injuries affecting the shoulders, arms, and hands have been recognized for hundreds of years and are generally categorized into injuries caused either from accidents, or from injuries resulting from long-standing repetitive tasks.¹ Injuries in the latter category are collectively known as cumulative trauma disorders, which are the result of gradual additive tissue damage sustained from repetitive motion. According to 2015 data released by the US Bureau of Labor Statistics, injuries sustained from repetitive motion often result in longer duration of time away from work compared with other classes of injury.² Although most of these injuries affect the musculoskeletal system, injuries to arteries and veins are known to occur.³ Work-related vascular injuries develop from excessive or exaggerated activity involving the shoulders, arms, or hands. Box 184.1 lists arterial disorders associated with occupational trauma secondary to manual labor, occupational exposures, and athletic activity.

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MANUAL LABOR INJURIES

Hand–Arm Vibration Syndrome

The first cases of this injury were reported by Loriga in 1911 among Italian miners presenting with “dead finger.”⁴ Hamilton in 1918 made the connection between cold-induced blanching and numbness of the hands and use of pneumatic drills by stonemasons in Indiana.⁵ Subsequent work by Taylor and Pelmeir⁶ and Ashe et al.⁷ firmly established hand–arm vibration syndrome (HAVS) as a discrete clinical entity.

The disease has been referenced by many names: dead finger, Raynaud of occupational origin, traumatic vasospastic disease, and vibration-induced white finger. The current designation of HAVS was introduced to reflect that the extent of involvement of the upper extremity involves more than just the digit. Regardless, the most common initial symptoms are those of Raynaud phenomenon secondary to prolonged use of vibrating mechanical tools.

BOX 184.1	Arterial Disorders Associated with Occupational or Recreational Activities
Manual Labor	
Hand-arm vibration syndrome Hypothenar hammer syndrome	Chronic hand ischemia Quadrilateral space syndrome Humeral head compression of the axillary artery Thoracic outlet syndrome
Exposure	
Acro-osteolysis Electrical burns Extreme thermal injuries	

Clinical Findings and Risk Factors

The Stockholm Workshop Scale staging system is presented in Table 184.1.⁸ This scale is a modification of the original Taylor–Pelmear scale.⁶ Significant changes include a simplified classification scheme, with removal of level of functional disability, seasonal and subjective neurological criteria from the original scale. Classically, the distal tips of one or more fingers experience attacks of cold-induced, vasospastic blanching. With continued progression, the affected area increases in size, and the blanching extends proximally to eventually span the entire digit. Attacks of white finger typically last approximately 1 hour and terminate with reactive hyperemia (red flush) and considerable pain. Prolonged cold exposure may induce bluish black cyanosis in the affected fingers. Only approximately 1% of cases progress to ulceration or gangrene.⁹

Vibrating handheld machines (e.g., pneumatic hammers and drills, grinders, and chain saws) have been implicated in HAVS. Such injury potential is not restricted to a few types of tools but applies to a variety of situations in which workers' hands are subjected to transmission of vibration energy.¹⁰ There appears to be a linear relationship between the acceleration exposure dose (amount of acceleration and years of exposure) and the onset and HAVS severity.¹¹

The exact mechanism of injury is unknown. The frequency and intensity of vibration affect the extent of damage to the endothelium.¹² Local platelet adhesion seems to be an important factor in arterial occlusion. It has been shown that sympathetic hyperactivity, in combination with local factors such as vibration-induced hyperresponsiveness of the digital vessels to cold, may be responsible for the finger-blanching attacks.¹³ In those with exposure to vibration injury, appearance of symptoms has also been correlated to smoking status.¹⁴ Lower levels of serotonin, as well as polymorphism variants of the HTR1B, have also been shown to increase susceptibility to symptoms.¹⁵ Studies have shown that vibrational energy releases endothelin-1, a potent vasoconstrictor, impairing nitric oxide release from the damaged endothelium.¹⁶

Diagnosis

Key features in the history include use of vibrating tools and symptoms of Raynaud phenomenon. For a vasospastic condition, the most promising single objective test is cold provocation and recording of the time until digital temperature recovers

TABLE 184.1 Stockholm Workshop Scale for the Classification of Hand–Arm Vibration Syndrome

Stage	Grade	Description
0		No attacks
1	Mild	Occasional attacks affecting only the tips of one or more fingers
2	Moderate	Occasional attacks affecting distal and middle (rarely also proximal) phalanges of most fingers
3	Severe	Frequent attacks affecting all phalanges of most fingers
4	Very Severe	As in Stage 3, with trophic skin changes in the finger tips

Modified from Gemne G, Pyyo I, eds. The Stockholm Workshop scale for the classification of cold-induced Raynaud's phenomenon in the hand-arm vibration syndrome. *Scand J Work Environ Health*. 1987;13(4):275–278.

(see Ch. 20, Clinical Evaluation of the Venous and Lymphatic Systems). Digital artery occlusion is best detected by recording the systolic pressure of the affected fingers with transcutaneous Doppler ultrasound^{17–20} or duplex scanning.²⁰ In advanced disease, arteriography is helpful. Barker and Hines²¹ documented arterial occlusion by brachial arteriography in workers who complained of hand blanching and attacks of numbness. Other authors have reported on the use of arteriography in this injury.^{22–24} Use of magnetic resonance imaging is described by Poole and Cleveland to differentiate HAVS from hypothenar hammer syndrome.²⁵

Arteriographic changes can include multiple segmental occlusions of the digits and a corkscrew configuration of vessels in the hands.²⁴ The extent of digital artery occlusion appears dependent on the frequency and duration of exposure.²⁶ Of 80 workers (chippers) with HAVS investigated at the Blood Flow Laboratory at Northwestern University, 25 (31%) exhibited a significant reduction in systolic pressure in one or more digits.²⁷ In 6 of the 25 workers, arteriography showed digital artery occlusion (Fig. 184.1). Incompleteness of the palmar arch was seen not only in the symptomatic hand but also in the contralateral, asymptomatic one. Raynaud phenomenon was present in 73 of 80 workers (91%). An abnormal cold response was observed in 53% of these workers.

Treatment and Prevention

With onset of neurovascular symptoms that interfere with social or work activities, the initial treatment is discontinuation of use of vibratory tools.¹¹ Other preventable causes of vascular injury, such as smoking, should also be discontinued. In advanced cases, calcium channel blockers such as nifedipine (30–120 mg daily) may be useful. Calcium antagonists inhibit the response of arterial smooth muscle to norepinephrine and have been reported to be effective.²⁸ Intravenous infusion of a prostaglandin (prostaglandin E1, prostacyclin, or iloprost) is usually reserved for patients with digital gangrene.²⁹ Surgical treatment, such as cervical sympathectomy or digital sympathectomy, is rarely needed.



Figure 184.1 Arteriogram of the Hand in a Vibratory Tool Worker. Occlusion of the digital arteries (arrow) is apparent.

Prevention is likely to be more effective than treatment. Personal protective equipment, such as anti-vibration gloves that limit exposure to cold and dampen transmission of vibration, should be used. Standards that limit the dose and duration of exposure to vibration have been set in place for factories and workplaces in the United States and internationally.¹¹

Hypothenar Hammer Syndrome

Etiology and Incidence

The anatomy of the ulnar artery at the hypothenar eminence makes it vulnerable to injury with repetitive use of the palm of the hand in activities that involve pushing, pounding, or twisting. The ulnar artery and nerve travel in a tunnel known as the Guyon canal that is bound by the pisiform and hamate bones. In this region, the ulnar artery is superficial and covered only by skin, subcutaneous tissue, and the palmaris brevis muscle (Fig. 184.2). Use of the palm as a hammer compresses the artery against the hook of the hamate bone, which acts as an anvil.

von Rosen (1934)³⁰ and Guttani (1772)³¹ published the original reports of the disease, but it was Conn et al.³² who first recognized the anatomic mechanism of injury and coined the term *hypothenar hammer syndrome (HHS)*. Mechanics, factory workers, carpenters, masons, and any laborers who habitually use their hands as a hammer are at risk for the disease.^{33–35}

The incidence of this rare entity is not precisely known because it is likely unrecognized. In Little and Ferguson's study of 79 mechanics, 14% had evidence of ulnar artery occlusion but none had symptoms severe enough to seek medical attention.³³

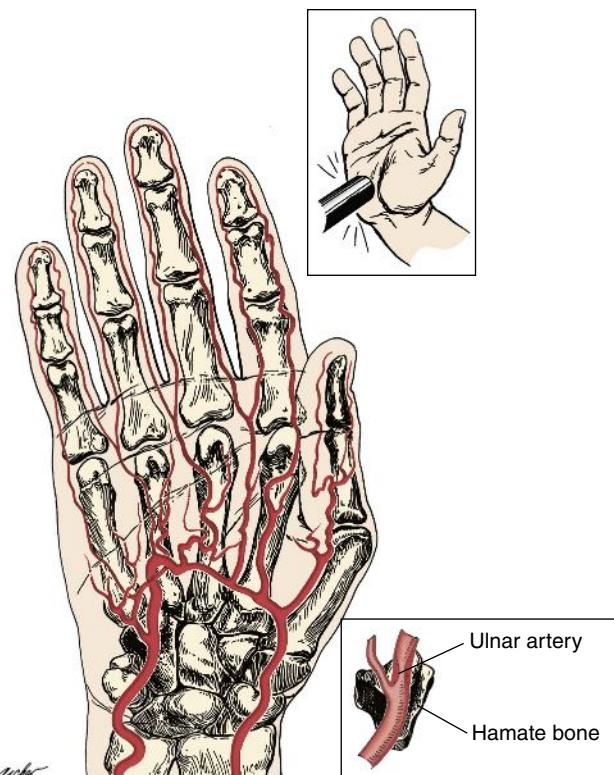


Figure 184.2 Mechanism of ulnar artery injury (upper inset) in a patient with hypothenar hammer syndrome. The terminal branch of the ulnar artery is vulnerable to injury because of its proximity to the hamate bone (lower inset).

Studies focusing on cases of HHS among referrals to tertiary centers for hand ischemia have found an incidence between 1.1% and 1.6%.^{35,36}

The type of arterial abnormality observed often depends on the nature of the vessel injury but includes thrombotic occlusion, aneurysm formation, or both. Vasospasm and damage to the intima can result in platelet aggregation and thrombus formation.³⁷ Damage to the media can lead to aneurysmal degeneration of the artery (Fig. 184.3), although this occurs less frequently. Thrombus formation can also occur within the aneurysm itself and lead to distal embolization.

Research by Ferris et al.³⁶ demonstrated that, in addition to vascular damage from repetitive trauma, underlying vessel abnormalities may also be responsible. Histologic examination of 19 resected ulnar arteries showed hyperplastic proliferation of the intima or media and disruption of the internal elastic lamina suggestive of fibromuscular dysplasia. In 13 patients for whom bilateral angiograms were available, 12 of 13 (92%) showed an abnormality in the contralateral asymptomatic ulnar artery. On the basis of this evidence, they proposed that the etiology of HHS depends on the presence of underlying ulnar artery fibromuscular dysplasia with superimposed repetitive palmar trauma.

Clinical Findings and Diagnosis

Patients typically present with Raynaud phenomenon. However, several key features distinguish HHS from similar syndromes. There is a preponderance of male smokers with a concomitant history of repetitive trauma to the hand.³⁵ The distribution is asymmetrical, often involving only the dominant



Figure 184.3 Open surgical repair of an ulnar artery aneurysm from hypotenar hammer syndrome. Note the aneurysm of the ulnar artery caused by repetitive trauma.

upper extremity. The cyanosis and pallor phases can occur, but the hyperemic redness is usually lacking.³⁴ The digits affected tend to be the ulnar three fingers, with a distinct lack of involvement of the thumb.^{34,36}

Physical examination may reveal cool or mottled digits; severe cases present with ischemic ulcers. A callus may be present over the hypotenar eminence. The result of the Allen test is often positive, indicating ulnar artery occlusion. On occasion, an aneurysm is observed as a pulsatile mass in the hypotenar eminence.

Noninvasive imaging studies such as duplex ultrasound and contrast-enhanced computed tomography or magnetic resonance imaging have been used to make the diagnosis of hypotenar hammer syndrome.^{38–41} However, the “gold standard” imaging modality in hypotenar hammer syndrome remains invasive arteriography. Arteriography is extremely useful in diagnosis and planning of surgical treatment because it defines the type of vascular lesion (spasm, aneurysm, and occlusion), shows the site and extent, and identifies the anatomy of the palmar arch and significant collateral vessels. A corkscrew pattern is typically observed in affected vessels.³⁸ Regardless of which imaging modality is used, proximal arterial segments should be included in the examination to rule out upstream sources of embolization.

Treatment

Given the overall rarity of this disease, the optimal treatment options remain controversial. Based on case series data, it appears that the majority of patients with HHS improve with

nonsurgical interventions.³⁵ These conservative measures include smoking cessation, hand protection involving avoidance of exposure to cold environments, and avoidance of further trauma. Medical therapy consists of calcium channel blockers and antiplatelet drugs. Anticoagulation has been used in cases of digital necrosis.

Surgical therapy is reserved for severe cases of digital ischemia or the presence of an aneurysm. Simple ligation of an aneurysm can be performed if there is adequate collateral circulation. More often, surgical intervention requires resection with primary reconstruction or vein graft interposition. One study suggests the use of arterial interposition graft as well.⁴² Several large case series report that most patients have improvement in symptoms after reconstruction and that long-term results are satisfactory.^{35,42–45}

EXPOSURE INJURIES

Occupational Acro-Osteolysis

Wilson et al.⁴⁶ first described this disease in workers exposed to polyvinyl chloride, leading to resorption of the distal bony phalanges. Many of these workers experienced ischemic symptoms in the hand, with resorption of the distal phalangeal tufts, similar to scleroderma. The dominant initial symptoms are Raynaud-like vasospasm. A few reports of angiography in this syndrome document damage to the digital arteries.^{47,48} Findings include multiple stenoses and occlusions of the digital arteries along with nonspecific hypervascularity adjacent to the areas of bone resorption. The reason for the hypervascularity is not clear, but it may be related to stasis of contrast medium in digital pulp arteries secondary to shortening and retraction of the fingers. Some of these digits were clubbed, a finding that has also been associated with hypervascularity in the fingertips. Treatment is supportive.

Electrical Burns

Electrical energy inflicts tissue destruction in relation to the voltage applied. Currents less than 1000 V cause injuries limited to the immediate underlying skin and soft tissue. High voltage (>1000 V) usually causes extensive damage as the current travels from the point of entry to the point of exit. No tissue is immune to the devastating effects of high-voltage injury, and arterial injury may occur. The upper extremity, especially the hand, is involved more often than are other parts of the body due to its grasping function. The arterial injury from electrical energy often manifests as arterial necrosis with thrombus or bleeding, potentially leading to gangrene and limb loss.

Bookstein⁴⁹ described the angiographic changes in the upper extremity after electrical injury, including extensive occlusion of the ulnar and digital arteries and thrombosis of the radial artery. Arterial spasm may also be present. Later, damage to the media may cause aneurysm formation, which can lead to subsequent rupture (Fig. 184.4). Like other forms of trauma, treatment and salvage of the upper extremity depend on the concomitant electrical injury to the bone and soft tissue.⁵⁰



Figure 184.4 Aneurysm of the brachial artery in an electrician who had sustained a high-voltage electrical burn 9 months previously.

Reconstruction with free flap due to local vascular damage has been described.⁵¹ Occlusion of a major artery documented by arteriography requires bypass grafting, and good limb-salvage results have been reported.⁵²

Extreme Thermal Injuries

Vasomotor disturbances in the hands of individuals exposed to extreme chronic thermal trauma are typically manifested as Raynaud syndrome. Workers at highest risk for thermal injuries are those in a profession in which their hands are subjected to chronic exposure to cold, such as slaughterhouses, canning factories, and fisheries.^{53,54} Epidemiologic studies examining this dilemma are limited. Swedish military trainees exposed to extreme cold for up to 14 months were found to have reduced sensation to temperature changes as well as vibrotactile stimulus; however, there were no alterations in the finger systolic blood pressure.⁵⁵ These symptoms were documented to be present by sensory testing at 4 months; however, symptoms such as cold intolerance and white fingers were present 4 years after initial injury.⁵⁶ The action of alternating ice-cold and hot exposure, use of plastic gloves in cold exposure, and long-term exposure to cold seem to be identifiable risk factors. Treatment is supportive.

ATHLETIC INJURIES

Athletes, particularly professionals who engage in repetitive strenuous or exaggerated hand or shoulder activity, are susceptible to arterial injury, resulting in upper extremity ischemia. Arterial injury can occur anywhere along the course of the upper extremity from the subclavian artery to the digital vessels. Recognized syndromes occur anatomically at the thoracic



Figure 184.5 Occlusion of the palmar arch in a Frisbee player (arrow). Because of this injury, there is poor filling of contrast medium in the second, third, fourth, and fifth fingers.

outlet, the quadrilateral space, the humeral head and in the hand. The exact incidences of these syndromes are unknown. The majority of reported arterial injuries in the elite athlete involve baseball players; however, vascular injury has been reported in such sports as karate, volleyball, handball, Frisbee, lacrosse, golf, weightlifting, and swimming.^{57,58}

Hand Ischemia

Clinical Findings and Risk Factors

The mechanisms of hand ischemia fall into two main categories: direct digital artery injury and embolization from a proximal source in the upper extremity. Hand activity in any sport can cause blunt-force injury to the arteries⁵⁸ (Fig. 184.5); however, direct digital arterial trauma is encountered more often in handball players, baseball catchers, and practitioners of the martial arts. It has been suggested that handball players with more than 200 hours of accumulated playing time are at greater risk for symptomatic alterations in perfusion.^{59,60} In a study of Basque handball players, 57% of those who play, 77% of federated players in the senior category, and 87% of professionals sustain injury to the arteries of the hand and fingers.⁶¹ In a study of beach volleyball players, both time spent playing and female gender were risk factors for developing hand ischemia symptoms.⁶²

Baseball players, particularly catchers, who experience tremendous, repetitive impact, are predisposed to the development of chronic hand ischemia. Many catchers exhibit

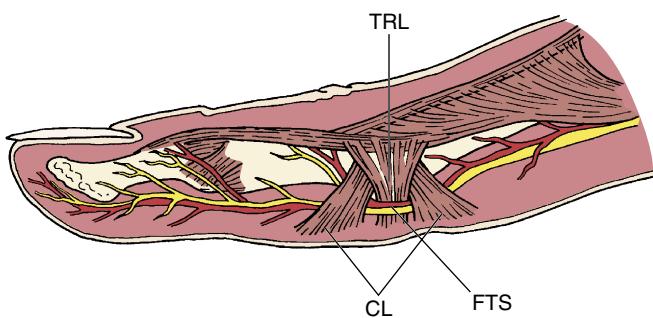


Figure 184.6 With the proximal interphalangeal joint in hyperextension, Cleland ligament may compress and occlude the vascular supply to the fingertip. CL, Cleland ligament; FTS, flexor tendon sheath; TRL, transverse retinacular ligament.

Raynaud's symptoms, especially in the off-season when they engage in outdoor activity in cool autumn or winter weather. Lowrey⁶³ reported decreased digital perfusion to the index finger of the glove hand in 13 of 22 baseball catchers examined by Doppler flow and the Allen test. Of 10 professional catchers studied in that author's laboratory, 40% had evidence of digital artery occlusion.⁶³

Another form of hand ischemia occasionally observed in baseball pitchers is compression of the digital artery by Cleland ligament. These ligamentous structures are found on the palmar surface of the digits and span from the phalanx to the subcutaneous tissue (Fig. 184.6). The proposed mechanism is compression of the digital vessels with hyperextension of the proximal interphalangeal joints.

Treatment

Treatment of hand ischemia depends on the clinical findings. With acute injury, a conservative approach involving intravenous infusion of dextran and pain control is in order. Medical therapy for digital ischemia includes purine derivatives such as pentoxyphylline, calcium channel blockers, and analgesics for pain control.⁶⁰ Botulinum toxin (Botox) injection has emerged as an alternative to surgical intervention for digital ischemia due to vasospasm. In case series, Botox injection into the neuromuscular bundle of the fingers results in 75% to 100% improvement of pain and healing of digital ulceration in 48% of patients.⁶⁴ Surgical intervention is indicated for severe symptoms not alleviated by conservative management. Digital periarterial sympathectomy was shown to be successful in a cohort of handball players with severe symptoms.⁶⁴ Release of Cleland ligament has been successful in relieving digital artery compression in baseball pitchers as well.⁶⁵ Preventing injury is important and can be accomplished by the use of gloves with padding and other protective devices.⁶³

Quadrilateral Space Syndrome

The quadrilateral space is defined as the area bordered by the teres minor superiorly, the humeral shaft laterally, the teres major inferiorly, and the long head of the triceps muscle medially.⁶⁶ Found within this space are the posterior humeral circumflex artery and the axillary nerve.

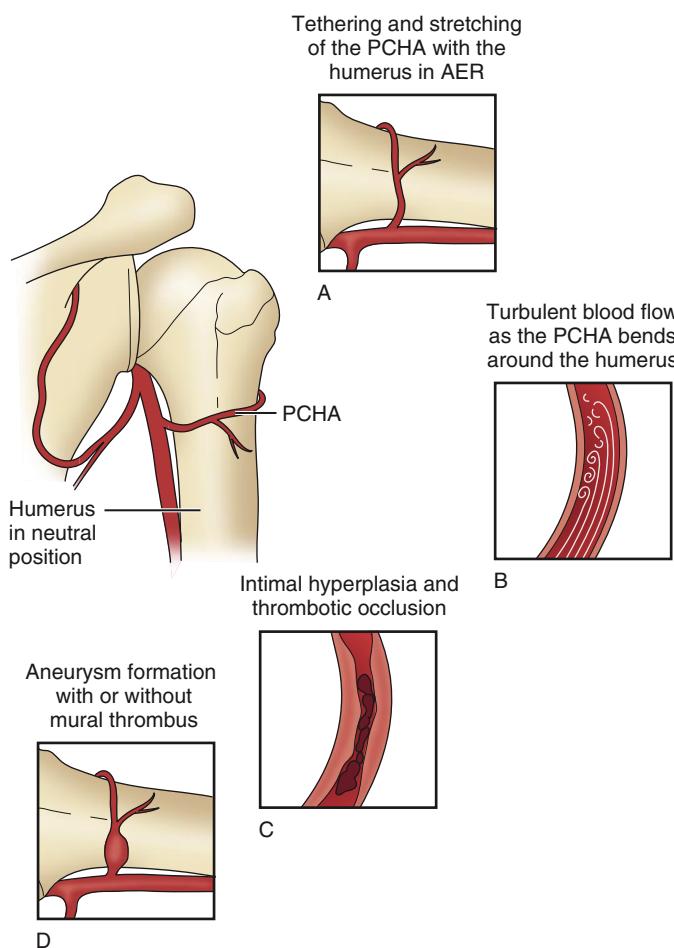


Figure 184.7 Proposed Mechanism for Posterior Circumflex Humeral Artery Degeneration and Thrombosis. (A) The posterior circumflex humeral artery (PCHA) stretches around the neck of the humerus similar to a stretched rubber band leading to intimal injury and weakening of the vessel wall. (B) The repetitive pulley movement likely leads to turbulent blood flow within the bend around the humeral neck. (C) These changes lead to intimal hyperplasias, thrombotic occlusion, and (D) aneurysmal formation. AER, abduction and external rotation. (From Brown SAN, Doolittle DA, Bohanon CJ, et al. Quadrilateral space syndrome: the Mayo Clinic experience with a new classification system and case series. *Mayo Clin Proc*. 2015;90(3):362–394.)

Cahill and Palmer⁶⁷ first reported in 1983 the diagnosis and surgical treatment of 18 patients with this entity. Compression of the posterior humeral circumflex artery within this space occurs with the arm in the “cocked” position (abduction and external rotation). Chronic compression and trauma to this artery in overhead motion athletes, particularly pitchers and volleyball players, can lead to aneurysmal dilation or occlusion (Fig. 184.7). Aneurysms in this location are prone to distal embolization in the hand.^{68,69}

Experience with nine patients at the Mayo Clinic suggests a new classification of quadrilateral space syndrome into vascular and neurogenic etiologies. Vascular quadrilateral space syndrome (vQSS) is caused by repetitive mechanical trauma to the posterior circumflex humeral artery resulting in thrombosis and/or aneurysmal degeneration leading to distal embolization. vQSS is most often seen in overhead athletes. Neurogenic

QSS (nQSS) is caused by fixed structural impaction of the quadrilateral space by fibrous bands, muscular hypertrophy, or other space-occupying lesions. This occurs from repetitive microtrauma to connective tissue in the quadrilateral space and can be seen in recreational athletes, such as swimmers, or in occupations requiring some overhead movement. Presentation can include upper extremity muscle atrophy, paresis, paresthesia, poorly localized shoulder pain, or tenderness with palpation of the quadrilateral space.⁷⁰

For vQSS, thrombolytic therapies may be used initially to dissolve the distal emboli, but surgery to treat the aneurysm should then be addressed. Surgical treatment of aneurysms in this location involves ligation of the posterior humeral circumflex artery via incision in the high axilla (Fig. 184.8).^{7,71–73} The anterior and posterior humeral circumflex arteries provide blood supply to the humeral head, and at least one of the two vessels must be preserved or repaired to prevent avascular necrosis. Many athletes return to practice after operation, and surgical treatment may be supplemented with 3 to 6 months of anticoagulation.⁷⁰

Treatment of nQSS can initially be conservative with oral anti-inflammatory medications, physical therapy, and limitation of activities; however, surgical decompression involving neurolysis and excision of fibrous bands or other space-occupying lesions often allows patients to return to full activity without pain or limitation after several weeks.⁷¹

Humeral Head Compression of the Axillary Artery

Lord and Rosati⁷² first reported compression of the third portion of the axillary artery by the head of the humerus. With overhead throwing or striking motions in which the arm is abducted and externally rotated, there is downward compression of the humeral head against the axillary artery.⁷³ Sonographic studies have demonstrated the impedance of flow through the axillary artery by the humeral head with these overhand motions.^{74,75}

Symptoms typically include arm fatigue and loss of pitch velocity after several innings. Finger numbness, Raynaud syndrome, and cutaneous embolization have occurred as well. Evaluation includes duplex studies and arteriography with the arm at rest and in the provocative position (Fig. 184.9).⁷⁶

Treatment is based on extent of injury. For those with compression of the artery alone, modification of throwing motion has resulted in improvement of symptoms. Saphenous vein patch angioplasty has been used for those whose symptoms do not improve.^{75,76} In patients with structural injury to the vessel, resection with saphenous vein bypass has been performed either anatomically tunneled or extra-anatomically above the pectoralis minor.⁷⁷ In addition to arterial repair, mobilization of the second and third portion of the axillary artery from the surrounding fascia assists with decompression. Long-term results are good, and many of the athletes treated at major centers return to competition after rehabilitation and short duration of anticoagulation.^{74–77}

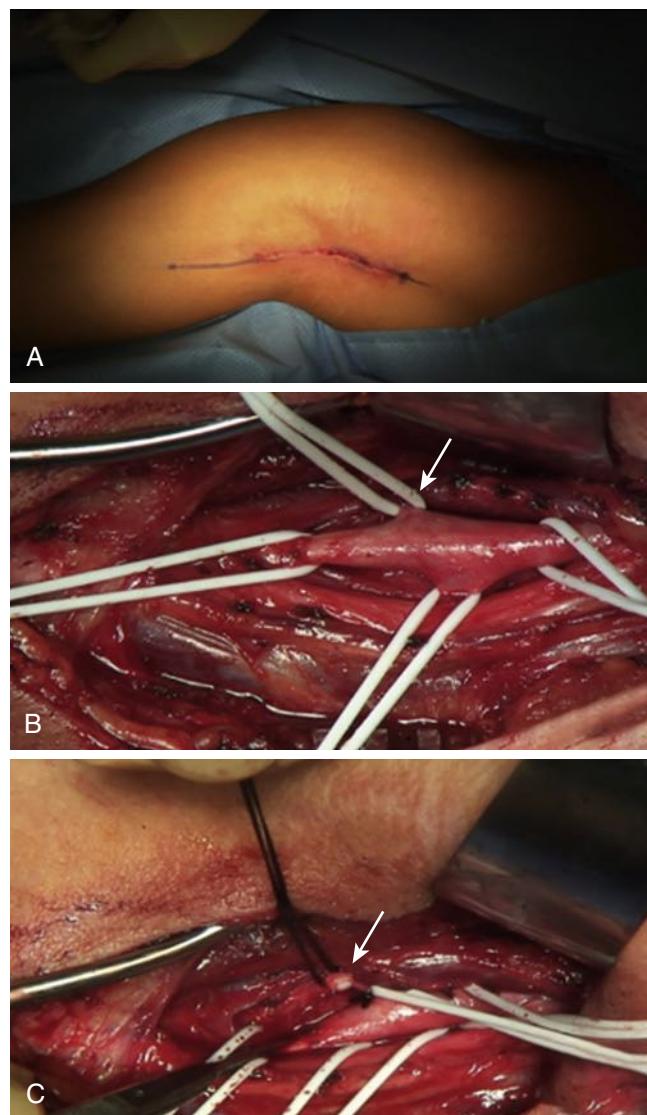
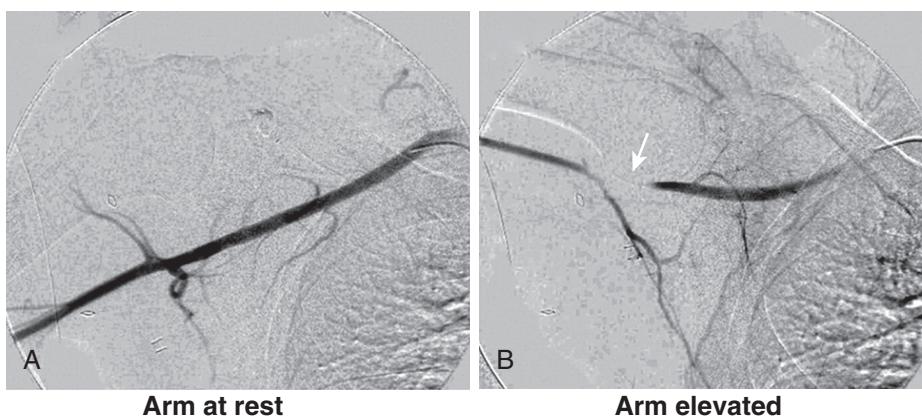


Figure 184.8 Open Ligation of the Posterior Circumflex Humeral Artery. An incision is made in the high axilla (A). Exposure and inferior retraction of the axillary vein and brachial plexus allow control of the axillary artery and the posterior circumflex humeral artery (PCHA) and anterior circumflex humeral artery (ACHA) (B). The PCHA is ligated while the ACHA is preserved to prevent avascular necrosis of the humeral head (C). (From Rollo J, Rigberg D, Gelabert H. Vascular Quadrilateral Space Syndrome in 3 Overhead Throwing Athletes: An Underdiagnosed Cause of Digital Ischemia. *Ann Vasc Surg.* 2017;42:63.e1–63.e6.)

Thoracic Outlet Syndrome

Athletes who engage in overextended shoulder motion, such as baseball pitchers, butterfly swimmers, weightlifters, and oarsmen, are potential candidates for thoracic outlet compression. Although not as common as neurogenic and venous thoracic outlet syndrome, injuries to the subclavian artery have been reported in these athletes. Because a comprehensive review of thoracic outlet syndrome is provided elsewhere (see Chapters 123–126, Thoracic Outlet Syndrome: Pathophysiology and Diagnostic Evaluation; Neurogenic; Arterial; Venous), only a brief discussion as it relates to athletic activities is given here.



Clinical Findings and Risk Factors

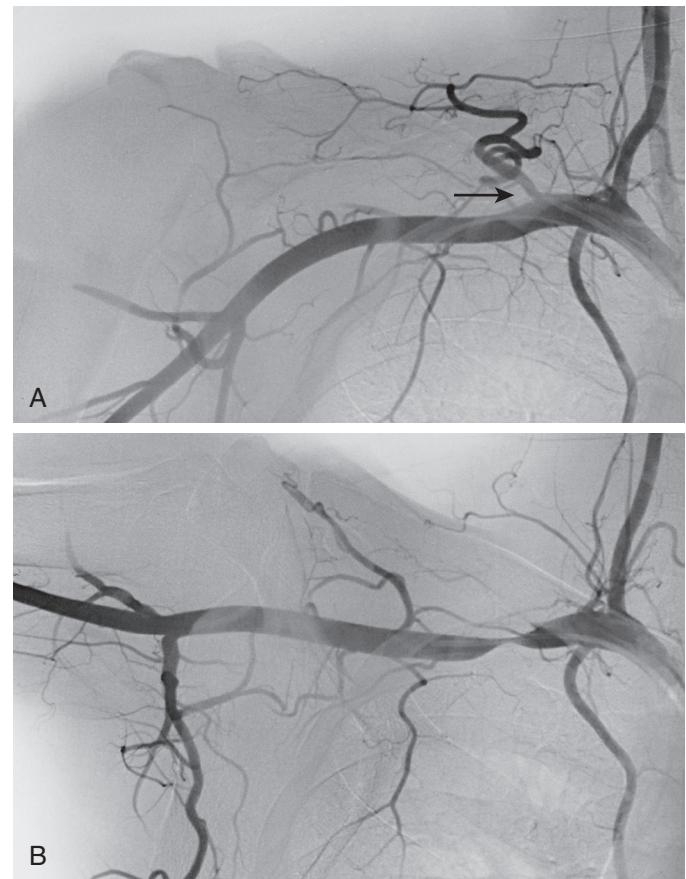
Compression of the subclavian or axillary artery can occur at the scalene muscles, costoclavicular space, or pectoralis minor muscle. A significant portion of cases of arterial thoracic outlet syndrome are due to underlying bone abnormalities, such as a cervical rib or anomalous first rib; but in athletes, hypertrophy of the scalene muscles or pectoralis minor muscles can lead to symptoms as well.⁷⁸ With chronic compression and vessel wall damage, a spectrum of disease can develop, including thromboembolism, occlusive disease, and aneurysm formation.

Although arterial thoracic outlet syndrome has been recognized for more than two centuries, the association with elite athletes has only been recent. Tullos et al.⁷⁹ in 1972 reported an axillary artery thrombus secondary to compression by the pectoralis minor in a major league pitcher. In 1978 Strukel and Garrick⁸⁰ reported on three competitive baseball pitchers with thoracic outlet compression. However, until the 1986 report of Fields et al.⁸¹ on athletic injury in the thoracic outlet, the injury had received little attention. In their fascinating report, they describe a major league pitcher who suffered a catastrophic stroke resulting from subclavian artery thrombosis with proximal clot propagation.

Symptoms are like those previously described in humeral head compression of the axillary artery and include arm fatigue, loss of pitch velocity, finger numbness, Raynaud syndrome, and cutaneous embolization. It is often difficult to make the diagnosis, and evaluation by an orthopedic surgeon to rule out musculoskeletal abnormalities is useful. Duplex scanning and transcutaneous Doppler studies with the athlete in the pitching position help to detect compression of the subclavian or axillary artery. A definitive diagnosis is established by arteriography with positional exposure (Fig. 184.10).

Treatment

Treatment depends on the extent of injury. Nonbone compression alone can be treated by division of the offending muscle or tendon.⁷² If an anomalous first rib or cervical rib is the culprit, a transaxillary approach can be used for decompression. If the subclavian artery requires reconstruction because of severe occlusive disease or aneurysmal disease, combined



supraclavicular and infraclavicular incisions provide better exposure. Saphenous vein is the preferred conduit for bypass, although prosthetic grafts or the femoral vein can also be used.^{80–82} Long-term patency rates are good, approaching 90% to 100%. Return to competition after arterial thoracic outlet syndrome repair is not well documented.

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Brown SN, Doolittle DA, Bohanon CJ, et al. Quadrilateral space syndrome: the mayo clinic experience with a new classification system and case series. *Mayo Clin Proc.* 2015;90(3):382–394.

New classification system for quadrilateral space syndrome explaining the differences between vascular and neurogenic etiologies, and the best treatment options for each process.

Ferris BL, Taylor Jr LM, Oyama K, et al. Hypothenar hammer syndrome: proposed etiology. *J Vasc Surg.* 2000;31:104.

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This contemporary multicenter study demonstrates the sensitivity and specificity of noninvasive systolic blood pressure measurements to make the diagnosis of hand-arm vibration syndrome. Included are the techniques of performing this testing.

A complete reference list can be found online at www.expertconsult.com.

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Special Techniques in Pediatric Vascular Surgery

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INTRODUCTION

Although conditions afflicting the extracardiac vascular system during infancy and childhood have been described for well over two centuries, a comprehensive literature dedicated to the management of pediatric vascular surgery patients remains lacking. Despite the presence of a number of textbooks devoted to the focused topic of pediatric cardiac surgery, a unified resource collating common themes and describing surgical techniques and principles does not exist for vascular surgery of children and the young adult. This can partly be explained by the range of vascular surgery conditions encountered in the modern era of pediatric surgery and by the diversity of providers who render treatment in this therapeutic area. Beyond pediatric surgeons with expertise in advanced vascular surgery reconstruction, adult vascular surgeons, pediatric and adult cardiothoracic surgeons, transplant surgeons, urologists, plastic surgeons, and adult general surgeons may all at one time or another operate on the blood vessels of pediatric patients. It is our hope in this introductory chapter to provide a broad overview of concepts and techniques that are particularly relevant to the pediatric population and that may prove useful to those who, either rarely or frequently, may be called upon to treat the vascular problems of this unique age group.

HISTORICAL BACKGROUND

One might trace the origins of pediatric vascular surgery to the 16th century when Guido Guidi first described a congenital vascular malformation after observing pulsatile varices protruding from a young man's scalp.¹ Giovanni Morgagni, a precocious Italian anatomist and a pioneer in the development of pediatric surgery, was instrumental in further expanding this early experience with vascular anomalies. His original description of coarctation of the thoracic aorta in 1760 was just one of his many original discoveries.² Sixty years later, August Meckel reported the associated collateral circulation and rib erosion that is seen in this condition.³ In 1835 Schlessinger was the first to discover that aortic coarctation is not limited to the aortic isthmus⁴ and, later in the same century, Pierre-Carl Pontain reported on arterial hypertension as a pathophysiologic consequence of coarctation.⁵ Although much less frequently encountered, constriction of the subisthmic aorta was noted by Schlessinger during his autopsy of a 15-year-old girl whose distal thoracic aorta was nearly obliterated.⁴ Quain reported a similar malady of the abdominal aorta in a 50-year-old man in 1847.⁶ The first clinical recognition of renovascular hypertension in a child was in 1938 by Wyland Leadbetter and Carl Burkland.⁷ They further noted that removal of the child's kidney, which

had become ischemic from fibromuscular dysplasia, corrected the hypertension. These are just a few of the myriad discoveries that have provided a framework for our current understanding of many pediatric vascular disorders.

For many years, operative repair of vessels in infants was deemed too hazardous to undertake.⁸ Thomas Starzl overcame this barrier in July 1967 with his pioneering efforts to complete the first successful pediatric liver transplant. This operation, carried out on a 19-month-old child with a large hepatoma necessitating multiple vascular anastomoses, was a major milestone in pediatric vascular surgery.⁹ Further technical advances were developed by Dean, Stanley, and Fry in their landmark work on pediatric patients with renovascular hypertension.^{10,11} Over the past 3 to 4 decades, pediatric, cardiac, and vascular surgeons have continued to accrue significant clinical and technical experience that has translated into more consistent results and allowed more sophisticated vascular reconstructions to be undertaken in infants and children.

BASIC PRINCIPLES

William Ladd and Robert Gross are often cited as the founders of pediatric surgery, and much of the current-day training of pediatric surgeons can be traced to these two men and their many pioneering contributions. Publication of their textbook *Abdominal Surgery of Infancy and Childhood* in 1941 was instrumental in establishing pediatric surgery as a unique and independent surgical subspecialty.¹² This text emphasized the paramount importance of gentle tissue handling, one of William Halsted's fundamental surgical principles, when operating on children. Although there are many similarities between children and adults regarding surgical management and operative technique, there are also a number of important differences that warrant highlighting. These range from fluid management and anesthetic concerns to issues related to the future growth of organs and their surrounding critical elements. From a vascular surgical standpoint, one must take into consideration that as an infant or child grows, the demands on blood vessels will also increase.

Timing of Surgery

If clinically permissible, vascular reconstruction in small infants should be delayed to allow for further development as reconstructing vessels less than 2 mm in diameter is technically challenging and has been associated with comparatively inferior results. For example, deferring renal revascularization in children with renovascular hypertension until the child is at least 3 years old has been shown to increase the likelihood of a successful outcome.¹³ Fortunately, the incidence of neonatal hypertension is quite low, ranging from 0.2% to 2%,¹⁴ and prolonging the use of antihypertensive medications in an effort to delay surgery has not been associated with significant long-term adverse effects.¹⁵ However, occasionally, it can prove challenging to control high blood pressure in an infant, given the variable onset and duration of oral antihypertensive medications as well as unpredictable responses to these agents.

Persistent, uncontrolled hypertension in these young patients can lead to congestive heart failure and cardiogenic shock, which may compel surgical reconstruction of the renal arteries regardless of the child's age. Although the preference is to defer surgical repair until the patient is of appropriate age and size, renal artery reconstruction has been safely performed in the very young; the University of Michigan has described their successful treatment of a 3-month-old, 4700-g baby with bilateral renal artery occlusions.¹³

In contrast to the recommendation to delay the surgical correction of renovascular hypertension, early repair of thoracic coarctation is typically advised, given low reported perioperative mortality rates and a relatively low risk of reintervention or recurrent coarctation following reconstruction.¹⁶ More importantly, early repair minimizes the incidence of vascular dysfunction and persistent hypertension.¹⁷

Anticoagulant and Antiplatelet Agents

The management of antithrombotic therapy in children has both similarities and important differences from that in adults. The major distinctions in the pediatric population relate to their dynamic and evolving coagulation system, age-dependent distribution and clearance of antithrombotic drugs, limited vascular access and the impact of this on monitoring efforts, the limited availability of medications in liquid formulations, dietary differences complicating oral vitamin K antagonists (VKAs), and general compliance issues.¹⁸

Unfractionated heparin has been used safely in children for decades. As in adults, heparin is typically systemically administered intraoperatively when vessels are temporarily clamped. A bolus dose of 75 to 100 units/kg results in a therapeutic activated partial thromboplastin time (aPTT) in 90% of children.¹⁸ However, this percentage may in fact be an overestimation because aPTT therapeutic ranges are calculated using adult plasma and aPTTs are slightly elevated at baseline in young children. As such, the therapeutic range in a neonate correlates to a reduced relative increment in aPTT compared with adults; as a result, aPTT values correctly predict therapeutic heparin concentrations only 70% of the time.¹⁹ Consequently, maintenance doses are age dependent, with infants requiring an increased hourly dose (28 units/kg per hour) compared with children older than 1 year of age (20 units/kg per hour).

Protamine sulfate can be safely given to reverse heparin intraoperative anticoagulation in the pediatric population. It is typically administered at a rate of 1.0 mg/100 units of heparin received. Given that neonates and young children have a more rapid clearance of heparin compared with adults, protamine is typically not administered in these age groups.^{13,18} Avoiding protamine administration and allowing heparin to wear off over the early postoperative period confers an additional theoretical benefit of minimizing thrombotic events in particularly small-caliber blood vessels.

Similar to adults, postoperative anticoagulation regimens in children have evolved over the past decade. This is due to both difficulties inherent to the use of VKAs and the advent of

newer agents such as low-molecular-weight heparin (LMWH) and novel oral anticoagulants (NOACs). VKAs can be particularly problematic in newborns, given their decreased levels of vitamin K-dependent coagulation factors. The fact that VKAs are available only in tablet form, and that vitamin K is nearly absent from breast milk but typically present in infant formula, further complicates their use.²⁰ LMWH has now become the preoperative and postoperative anticoagulant agent of choice in the pediatric population. This class of drugs is appealing, given a decreased need for monitoring compared with VKAs, minimal drug–drug interactions, and a reduced risk of heparin-induced thrombocytopenia.²¹ However, LMWH has considerable dose variability in children, which can result in fluctuations in antithrombin levels. In addition, the general unpopularity of frequent cutaneous injections obligated by LMWH therapy has led some centers to begin trialing NOACs. Although quite promising, the data on the use of NOACs in children remains limited at present and their appropriate role in this population remains to be defined.²²

Antiplatelet drugs have been used for decades in children. If reconstructing very small vessels, aspirin is typically administered intraoperatively through a nasogastric tube to minimize the chance of thrombus formation at anastomotic sites.¹³ Doses ranging from 1 to 5 mg/kg have been deemed safe.¹⁸ Reye syndrome is rare in this setting because its association with aspirin is at much higher dosage levels (>40 mg/kg).²³ Clopidogrel has been used more frequently in recent years, particularly in children with heart disease. One randomized trial indicated that clopidogrel at a dose of 0.2 mg/kg per day was efficacious in young children.²⁴

Pediatric Anesthesia and Perioperative Care

The subspecialty of pediatric anesthesia owes much of its foundation and progress to M. Digby Leigh, who published the first dedicated text on the discipline in 1947.²⁵ Although the full breadth of content pioneered by Leigh and further developed by subsequent generations of pediatric anesthesiologists is well beyond the scope of this chapter, there are several basic principles worth mentioning.

First, given that a neonate's ability to maintain normothermia is inadequate, the operating room environment should be optimized to minimize a baby's heat loss. As part of this effort, the ambient room temperature should be maintained between 23°C and 25°C (80°F and 85°F), inhaled gases should be warmed and humidified, fluids should be delivered via a warming device, and a forced warm air delivery device or warming blanket should be used. Second, given the obvious size differences between young children and adults, a full complement of appropriately sized anesthesia-related equipment and devices (pulse oximeter probes, facemasks, tracheal tubes, suction catheters, laryngoscope blades, etc.) must be readily available.

A critical component of anesthesia administration to infants and young children is expertise in airway management. This skill set requires a detailed knowledge of the developmental anatomy and physiology of the pediatric skull, nose,

and upper airway. Equally as important to safely performing facemask ventilation, laryngoscopy, and subsequent tracheal intubation is the ability to effectively address postoperative airway complications, some unique to the pediatric population. Laryngospasm, hypoxemia, croup, and aspiration are among the respiratory events that are responsible for nearly 50% of pediatric anesthesia complications.²⁶ In contrast to adults, the usual method of inducing anesthesia in children is with inhalational agents, typically nitrous oxide followed by sevoflurane. Upon emergence from anesthesia, the child is typically placed in the “recovery position” (lateral decubitus) because it promotes airway patency and is helpful should the child develop emesis. This latter complication is not trivial because postoperative nausea and vomiting are significantly more common in children than adults and, among other negative consequences, can delay discharge from the recovery room. In addition to proper positioning in the lateral recovery position to minimize the aspiration risk, children are often prophylactically administered an antiemetic along with dexamethasone, which can minimize both tracheal edema and postoperative vomiting.²⁷

BASIC VASCULAR TECHNIQUES

As in the adult world, there are multiple elements that comprise any successful pediatric vascular surgery operation. Proper patient positioning, adequate exposure and lighting, optimal incision location and size, safe and effective clamp selection and placement, appropriate suture material and conduit choice, and technically sound anastomoses are all important for a positive outcome. Of course, the aforementioned factors are all of secondary significance behind selecting the correct procedure from the onset.

Vascular Instruments and Retractors

In general, vascular instruments used during pediatric reconstructions are similar to those typically used in the adult realm. However, small versions of forceps, clamps, Castroviejo needle holders, scissors, and other specialty instruments are available and can be helpful in manipulating smaller-sized pediatric blood vessels. For example, “baby” Jacobson or Potts bulldog clamps are useful atraumatic vascular clamps, and Yasargil or Heifitz clips can be particularly beneficial for small or fragile vessels at risk for tearing or prone to spasm. Delicate handling of tissue with Debakey and fine Gerald forceps is advised. Metzenbaum, Stevens tenotomy, and Potts or Dietrich scissors are useful for dissecting vessels as well as extending arteriotomies.

With regard to retractor systems, both self-retaining and handheld instruments are used much like in adult surgery. For abdominal procedures, typically a Thompson Pediatric Abdominal System (Thompson Surgical Instruments, Traverse City, MI) is used because its selection of lower-profile blades with variable depths and widths affords considerable versatility in young patients (Fig. 185.1). For the extremities and neck, Weitlaner retractors, which come in various sizes, are most commonly used.

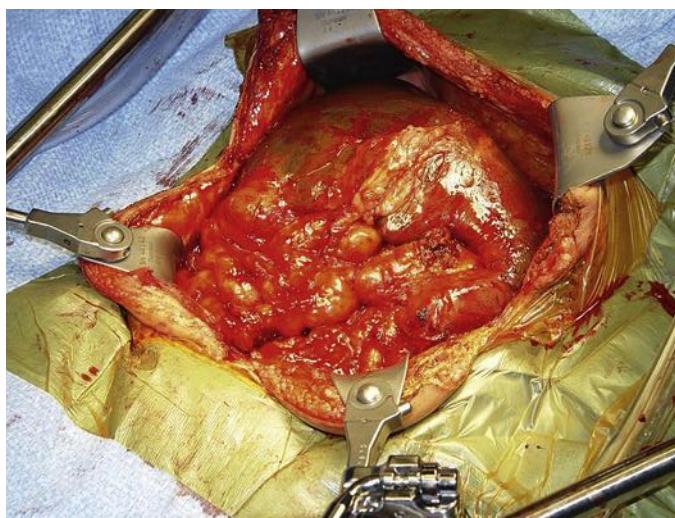


Figure 185.1 A Thompson Pediatric Abdominal Retractor System (Thompson Surgical Instruments, Traverse City, MI), due to its lower profile blades with variable depths and widths, provides excellent exposure to the abdominal contents in young patients.

Vascular Exposure and Clamping

Information relating to specific vascular exposures is beyond the scope of this chapter and is described elsewhere in this textbook. In general, exposure of the relevant blood vessels should be done in the most direct route possible and with the least amount of tissue manipulation. Broadly prepping the patient is recommended in the event that more complex exposure or vascular control than originally anticipated becomes necessary. Anatomic landmarks and key structures should guide the surgeon's incision and subsequent dissection. In the absence of a palpable pulse to further direct the course of the dissection, a Doppler probe should be used in the effort to avoid unnecessary tissue disruption and the creation of tissue flaps that may negatively impact wound healing. Given that the abdomen of a young child is barrel shaped, a transverse incision often provides better exposure to the abdominal viscera and the abdominal aorta and its major branches than a midline incision (Fig. 185.2). There is also evidence that a transverse incision is less likely to develop fascial dehiscence compared with a vertical incision in children less than 1 year of age.²⁸

Gentle handling of tissues is paramount in the effort to avoid vessel disruption, hematoma formation, and other bleeding complications. Particular caution should be undertaken in patients with known connective tissue disorder or in the setting of infected or dissected vessels. The clinical and anatomic scenario at hand should guide selection of appropriate clamp type and size. Applying clamps in a side-biting fashion should generally be avoided given the small caliber of pediatric vessels and the increased risk of "back-wall" suture placement with subsequent vessel thrombosis.

Arteriotomy Closure

Primary closure of an arteriotomy or venotomy in a pediatric patient is the simplest and most expedient means of arterial



Figure 185.2 Proposed location of a transverse abdominal incision in a young child, which can provide better exposure of the abdominal aorta and viscera given the barrel-shaped nature of the abdomen.

or venous reconstruction. However, given the small caliber of pediatric vessels and their need for future growth, primary closure can result in luminal narrowing with a hemodynamically significant stenosis. As such, similar to the situation with adult vascular reconstructions, judgment must be used in determining the safety and appropriateness of closing a given vessel primarily versus using a patch closure that may be necessary to ensure an adequate luminal size. Although a patch repair in adults is typically undertaken by beginning a running suture at each apex, some have advocated for an interrupted suture technique in children (see Figs. 61.39 and 61.40); such a strategy will theoretically better accommodate native vessel growth and luminal expansion over time. Similarly, the patch size should be made sufficiently large so as to not cause vessel constriction over time as the child grows. In one report of pediatric trauma patients less than 10 years of age and with a vascular injury, patch repair was the most common operative management strategy, being used three times more frequently than bypass grafting (28% vs. 9.6%).²⁹

Arterial Replacement and Bypass Procedures

Several different techniques can be used to address young patients afflicted with aneurysmal degeneration or arterial occlusive disease. Although relatively infrequently encountered in children, connective tissue disorders, tuberous sclerosis, vessel infection, inflammatory middle aortic syndrome, and trauma are all examples of clinical conditions that may necessitate a major vascular reconstruction. Replacement of the diseased vessel with a bypass procedure is the most commonly performed revascularization procedure in these settings. Depending on the extent of vessel involvement, segmental resection



Figure 185.3 Ostial stenosis of the left renal artery in a 4-year-old girl with renovascular hypertension. (From Stanley JC, Criado E, Upchurch GR Jr, et al. Pediatric renovascular hypertension: 132 primary and 30 secondary operations in 97 children. *J Vasc Surg*. 2006;44(6):1219–1228.)

and reconstruction with vessel reimplantation or primary end-to-end reanastomosis are additional options. Reimplantation has been used in the surgical management of peripheral aneurysms, as well as for children with renovascular hypertension. This latter clinical entity typically develops from renal artery stenosis as a result of progressive medial dysplasia and intimal fibroplasia. The anatomic pattern where disease is limited to the renal ostia is particularly conducive to repair with renal artery implantation (Fig. 185.3). In a series of 97 children treated surgically for renovascular hypertension at the University of Michigan, reimplantation of the renal artery was the most common means of repair, being performed in 45% of procedures.¹³ With this technique, the anterior and posterior aspects of the transected renal artery should be spatulated to ensure creation of a generous anastomosis. Typically, the renal artery is reimplanted onto the aorta; although infrequent, the iliac artery can be used if performing a renal autotransplantation. An aortic punch device can be helpful in fashioning a desired oval aortotomy that is at least twice the diameter of the renal artery. A tendency toward oversizing the aortotomy in this manner will decrease the chance of developing an anastomotic stricture over time (Fig. 185.4). As noted previously, an interrupted suture technique should be considered for the anastomosis unless the patient is a mature adolescent with normal-sized vessels. Medial mobilization of the kidney may be necessary to ensure the anastomosis is free of tension.

Peripheral and visceral aneurysms can often be treated by resection and primary end-to-end reanastomosis. This technique has been used for aneurysms of the renal, splanchnic, brachial, femoral, and popliteal arteries.³⁰ When using this technique, both vessel ends should be spatulated to increase the anastomotic circumference (see Fig. 61.4B).

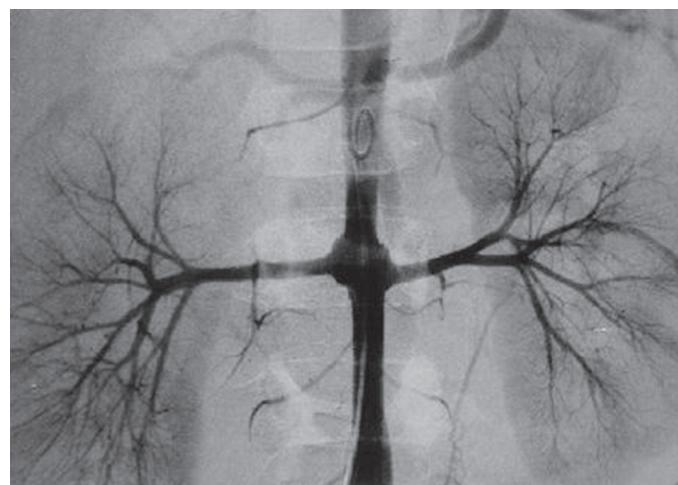


Figure 185.4 Bilateral renal artery–aortic implantations in a 7-year-old boy. Note the capacious aortic anastomoses from spatulation of the renal arteries. (From Stanley JC, Criado E, Upchurch GR Jr, et al. Pediatric renovascular hypertension: 132 primary and 30 secondary operations in 97 children. *J Vasc Surg*. 2006;44(6):1219–1228.)

Reconstruction with a bypass or interposition graft adds complexity to the procedure, given the need for an additional anastomosis and important issues related to conduit selection. The most common indications for performing an arterial bypass or interposition graft in the extremity of a child are peripheral aneurysms and trauma. Traumatic causes include both blunt injury and iatrogenic injury, such as arterial disruption or thrombosis associated with percutaneous access or catheter placement. Abdominal aortic coarctation, also referred to as midaortic syndrome, and renovascular hypertension due to renal artery stenosis, on the other hand, are the most frequent indications for bypass grafting originating from the descending thoracic or abdominal aorta.

Children with abdominal aortic coarctation typically develop aortic narrowing in a supraceliac location and present during the first or second decade of life with refractory hypertension (Fig. 185.5A). Up to 80% of these children have coexisting splanchnic or renal artery occlusive disease. In rare cases the degree of obstruction can result in symptomatic mesenteric ischemia.³¹ In the largest series to date of 53 children surgically treated for midaortic syndrome, Stanley and colleagues corrected the developmental narrowing with either a thoracoabdominal bypass graft ($n = 26$) or an interposition aorto-aortic graft ($n = 3$).³² Notably, more than half of the bypass procedures necessitated a concomitant renal or splanchnic revascularization procedure (see Fig. 185.5B). The conduit of choice for thoracoabdominal bypass grafting in this series was expanded PTFE because it was believed to be less likely to undergo post-implantation dilatation compared with Dacron. It is important that the graft be oversized in relation to the aortic diameter at the time of implantation to avoid the development of critical stenoses with future aortic growth. As such, 8- to 12-mm grafts, 12- to 16-mm grafts, and 14- to 20-mm grafts are recommended for use in young children, early adolescents, and late adolescents/adults, respectively. In contrast to

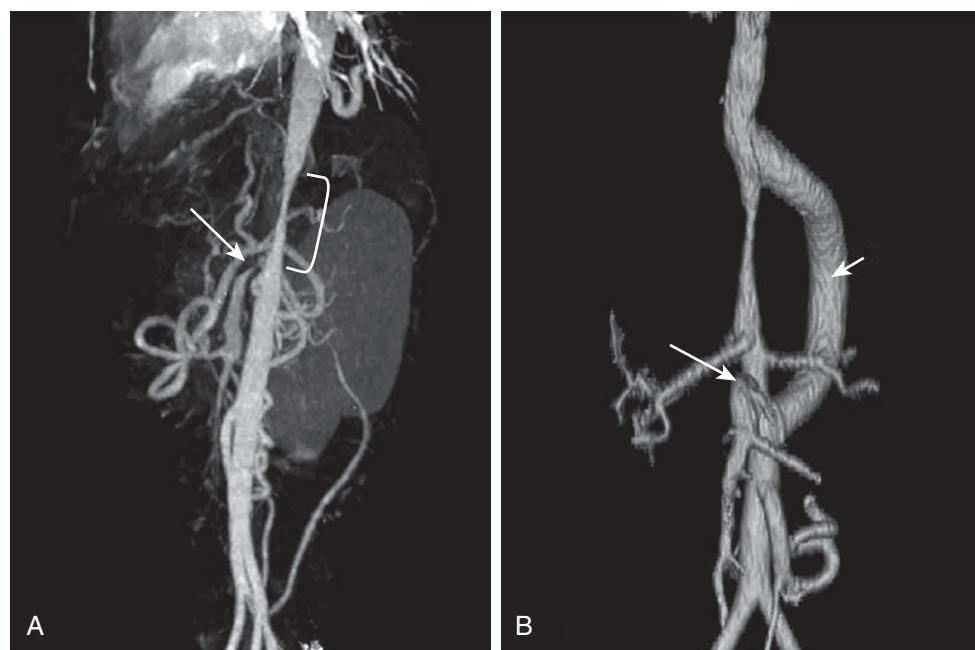


Figure 185.5 (A) Midaortic syndrome (bracket) with superior mesenteric artery stenosis (arrow). (B) Thoracoabdominal bypass (large arrow) with aortic implantation of superior mesenteric artery (small arrow). (From Stanley JC, Criado E, Eliason JL, Upchurch GR Jr, Berguer R, Rectenwald JE. Abdominal aortic coarctation: surgical treatment of 53 patients with a thoracoabdominal bypass, patch aortoplasty, or interposition aorto-aortic graft. *J Vasc Surg*. 2008;48(5):1073–1082.)

graft diameter, the length of the graft is not as important as there is minimal longitudinal aortic growth after the age of 10 years. Although the long-term overall graft patency was excellent in Stanley's series (97% at 5 years), lifelong surveillance is necessary to ensure that reoperation for graft outgrowth, anastomotic stenosis, or failed visceral artery reconstruction is not required.³²

The use of a prosthetic conduit in children is traditionally limited to major aortic reconstructions. Autogenous conduit, namely the great saphenous vein (GSV), is preferred for visceral and peripheral reconstructions. It is important to be aware that GSV grafts can undergo late expansion and even aneurysmal degeneration, especially if the vein is unsupported when used in young children. A review of 100 aortorenal grafts created with GSV revealed that 16% developed late aneurysm dilatation or stenosis.³³ In a more recent series of 16 lower extremity bypass procedures undertaken with GSV in children, the vein grafts proved more durable, with only 11% demonstrating expansion over a 10-year follow-up period.³⁴ Given the risks of late weakening and aneurysmal degeneration, some advocate for supporting GSV grafts originating from the abdominal or pelvic vessels in a Dacron mesh wrap. Alternatively, some prefer to use the internal iliac artery as a conduit when originating a bypass from the aorta or iliac vessels because it offers greater long-term durability compared with GSV.¹³ A novel and quite creative technique using the principles of tissue expansion has recently been developed for children with midaortic syndrome that can obviate the need for any of the aforementioned conduits.³⁵ By periodically inflating a tissue expander implanted posterior to the distal aorta over time, longitudinal growth of the distal aorta and iliac vessels is promoted, ultimately allowing for resection of the diseased segment of aorta and reconstruction with a primary end-to-end anastomosis (Fig. 185.6).

Endovascular

Paralleling the tremendous improvements in endovascular technology seen in adult cardiovascular care, recent technical advances have now made it possible to perform complex percutaneous arterial and venous interventions in infants and children. There are several fundamental differences in technique between adults and children with regard to contrast angiography. In general, contrast volume should not exceed 4 to 5 mL/kg in neonates or 6 to 8 mL/kg in infants. A hand injection technique, rather than power injection, is generally preferred when the patient is less than 15 kg to maximize control of the arterial bed opacification and minimize contrast reflux or excessive injection rates. Power injectors can safely be used in children weighing more than 15 kg, although the injection parameters are typically reduced to half the adult rates.³⁶ Systemic heparinization is usually administered following arterial access in children less than 15 kg to prevent femoral artery thrombosis. Access is usually gained with ultrasound guidance using a 21-gauge access needle and an 0.018-inch micropuncture wire.³⁷ A 3- to 5-F vascular sheath can then be placed for diagnostic and/or therapeutic interventions. An array of dedicated pediatric-length catheters has been developed to facilitate cannulation of the artery or vein of interest but notably there are currently no dedicated arteriotomy closure devices for pediatric-sized vessels.

Complications related to percutaneous arterial access are generally uncommon, but their frequency can approach 10% in children younger than 1 year of age.³⁶ The most frequently reported complications include hematoma, dissection, thrombosis, pseudoaneurysms, and arteriovenous fistulae. Vessel thrombosis as a result of vasospasm is of particular concern in small patients (<15 kg); incidence rates as high as 16% have been reported.³⁸ Prophylactic intraarterial nitroglycerin (1 to 3 µg/kg) can be administered to help prevent this complication in small children.³⁶

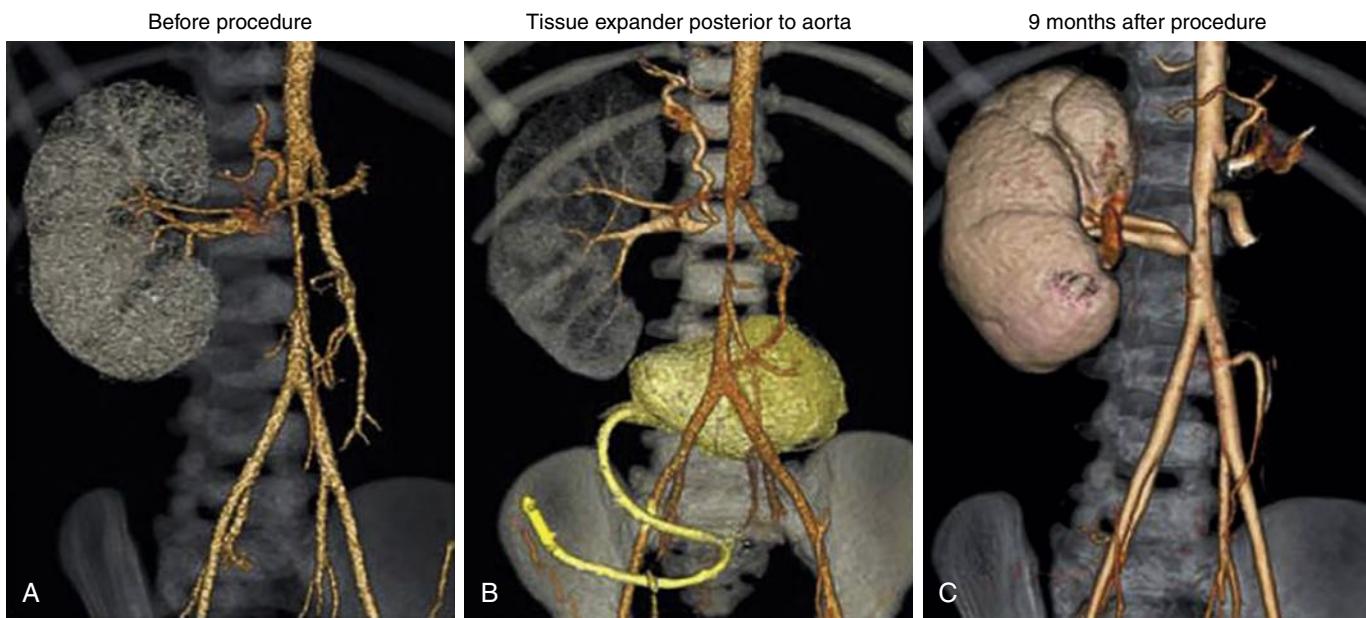


Figure 185.6 (A) Frontal angiographic view of 3-year-old girl with midaortic syndrome. (B) Placement of retro-aortic tissue expander prior to aortic surgery. (C) Removal of tissue expander followed by primary aortic reconstruction. (From Kim HB, Vakili K, Modi BP, et al. A novel treatment for the midaortic syndrome. *N Engl J Med*. 2012;367(24):2361–2362.)

In general, percutaneous balloon angioplasty is preferred to the insertion of balloon-expandable stents, for several reasons. First, the sheath size required for angioplasty catheters (4–5 F) is typically smaller than that required for covered or bare-metal stents (6–7 F). Sheath size is certainly an important consideration in young children with small access vessels. Additionally, balloon-expandable stents do not accommodate vessel growth with time. Consequently, the use of balloon-expandable stents often requires serial reinterventions over time with incremental angioplasty to keep up with somatic vessel growth. Given the inherent limitation that most balloon-expandable stents can only expand to 12 mm, it is not uncommon for a stent placed in a child's aorta to need surgical explantation by the time the child reaches adult age and size.

Balloon angioplasty has been used as a treatment for children with renovascular hypertension for more than 20 years, with varying degrees of success. In one series of 33 patients, Shroff and colleagues achieved blood pressure control in only 54%, and 37% of the patients developed restenosis.³⁹ It is recommended to avoid placing stents in pediatric renal arteries unless there is significant elastic recoil after angioplasty or a postangioplasty dissection is identified. Hepatic artery stenosis can develop in up to 20% of pediatric liver transplant recipients,⁴⁰ and these lesions can also be successfully treated with percutaneous angioplasty techniques. Typically, balloons between 2 and 6 mm in diameter are used, and the associated complications include acute vessel dissection, vasospasm, and thrombosis.⁴¹ The literature on this technique is limited to several case series, and currently no long-term data are available. Although balloon angioplasty of aortic coarctation in pediatric patients has been a safe and effective treatment option since the 1980s, its durability remains in question. In a series of 67 neonates and

children with coarctation treated by endovascular means, 25% developed recoarctation requiring reintervention and 5% developed an aneurysm at the angioplasty site at long-term follow-up (median, 5 years).⁴²

Much like in adults, endovascular procedural success in children will likely continue to improve as the associated technology becomes further advanced over time. However, the experience and outcomes from the adult population, which is based mostly on patients with atherosclerotic occlusive disease, cannot be easily extrapolated to children, who most frequently have congenital vascular anomalies. Nonetheless, endovascular therapy will likely confer the benefit of decreased perioperative morbidity at the potential expense of lower long-term durability, a trade-off that parallels the adult experience with endovascular treatment options.

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A complete reference list can be found online at www.expertconsult.com

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Aortic and Arterial Aneurysms in the Pediatric Population

DAWN M. COLEMAN, JONATHAN L. ELIASON, and JAMES C. STANLEY

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SUMMARY 2469

Pediatric arterial aneurysms include many rare and heterogeneous diseases. Etiologies vary widely among the different types of aneurysms, with important differences in the aneurysm's character and clinical relevance often dependent on the specific vessel affected (Table 186.1). Aneurysms in children, with few exceptions, should be considered dangerous. Although optimal management strategies and established criteria for operative treatment remain ill defined, rupture and thrombosis are reported frequently enough to justify early intervention in most cases. Unfortunately, children having an active systemic process responsible for their aneurysm carry the highest chance of rupture, but they are also at greatest operative risk. Many surgical procedures in the treatment of these aneurysms are complex, and management strategies based upon the specific arterial bed involved deserve individual review.

AORTIC ANEURYSMS

Aortic aneurysms are classically defined as enlargement (dilation) of the aorta's diameter greater than 1.5 times normal. Diagnosis of a pediatric aortic aneurysm requires a careful consideration of location and aortic size, for which standards are not well established. Nevertheless, useful data on aortic dimensions exist in two contemporary studies of normal pediatric aorta sizes by tomographic examinations based on a number of factors including age, gender, the site of aortic involvement, and body surface area.^{1,2} These measurements have particular value in assessing suspected genetic etiologies of aortic aneurysms wherein the entire aorta may be dilated and a segment of

normal aorta may be nonexistent to compare to the most aneurysmal site. Pediatric aortic aneurysms are generally categorized as genetic, developmental (idiopathic), infectious, inflammatory, or traumatic.

Genetically-induced aortic aneurysms are well recognized. Mutations in fibrillin 1, transforming growth factor- β (TGF β) receptor type 2, collagen 3A1, and *TSC1* and *TSC2* cause Marfan syndrome, Loeys–Dietz syndrome, vascular Ehlers–Danlos syndrome, and tuberous sclerosis, respectively. In addition to aortic aneurysms, all of these genetic diseases are usually responsible for other tissue abnormalities affecting nonvascular organs, most frequently involving the skin, musculoskeletal system, neural tissues, as well as the gastrointestinal tract and kidneys. Other genetic syndromes are exceedingly rare causes of pediatric aortic aneurysms,^{3,4} but the more common of these syndromic diseases deserve individual discussions.

Marfan syndrome is due to an autosomal dominant disease resulting from a defect in the *FBXO35* gene which leads to abnormal collagen cross-linking. Dilation of the aortic root and thoracic aorta in both children and adults is a hallmark vascular feature of this syndrome.⁵ Although infantile disease is rare and children are less commonly affected than adults, it is noteworthy that 50% to 83% of children with Marfan syndrome exhibit aortic dilation, including 20% having aortic aneurysms. The administration of an angiotensin receptor blocker (ARB) or beta-blocker is recommended to slow the aortic root enlargement in children.⁶ Neonatal Marfan syndrome, resulting primarily from *de novo* mutations, is a particularly severe and rapidly progressive phenotype of this genetic disease.^{7–9}

TABLE 186.1 Pediatric Aneurysms

Category	Arteries Commonly Affected	Aneurysm Characteristics	Clinical Manifestations
I Genetic Marfan syndrome Loeys–Dietz syndrome Ehlers–Danlos syndrome Tuberous sclerosis Arterial tortuosity Cutaneous laxa syndrome	Aorta, muscular arteries	Medial elastic tissue disorganization, cystic mucinous deposits, both solitary fusiform and multiple saccular aneurysms	Aortic rupture or dissection relatively common; arteriography
II Developmental – Congenital	Aorta, splanchnic, renal, iliofemoral arteries	Medial thinning and fibroplasia; solitary and multiple saccular aneurysms at arterial bifurcations	Usually asymptomatic, rupture uncommon, extremity aneurysms often present as painless pulsatile mass
III Infectious	Aorta, iliac arteries	Acute inflammatory infiltrates, chronic fibrosis with saccular aneurysms	Contaminated umbilical artery catheterizations, bacterial endocarditis, rupture common
IV Inflammatory Takayasu disease Kawasaki disease Behçet disease Systemic lupus Periarteritis nodosa	Aorta, brachiocephalic, renal, splanchnic, iliofemoral arteries, coronary (Kawasaki Disease)	Acute necrotizing inflammation, chronic panmural inflammation with multiple saccular aneurysms	Constitutional symptoms common during active disease, aneurysms in quiescent phase usually asymptomatic
V Traumatic	Aorta, extremity arteries	Disruption of all three layers of artery (pseudoaneurysm), thrombosis of small aneurysms common	Protean manifestations, aortic aneurysms often rupture, peripheral aneurysms often asymptomatic

Loeys–Dietz syndrome is an autosomal dominant connective tissue disorder resulting from a defect in the TGF- β pathway that causes abnormal pathway signaling, tissue friability, craniofacial abnormalities, and a characteristic panvasculopathy.^{10,11} The vascular phenotype is characterized mainly by descending thoracic aortic and peripheral aneurysms, arterial tortuosity, and dissections. Aortic dissections occur in aortas having smaller diameter than observed in Marfan syndrome. Progressive aneurysmal expansion and rupture are commonplace with this syndrome and operative therapy is pursued earlier rather than later in most cases. Beta-blocker and angiotensin II type I receptor blocker (ARB) therapy should be considered for both Marfan and Loeys–Dietz patients, to potentially reduce progressive aortic dilation.^{12,13}

Ehlers–Danlos syndrome, vascular (type IV), is an autosomal dominant disease resulting from a defect in the *COL3A1* gene responsible for type III collagen. The syndrome is associated with spontaneous rupture of hollow viscera, as well as large arteries such as the aorta. Aortic root and thoracic aortic aneurysms or dissections are usually seen in older children and are not rare findings in this syndrome.¹⁴ An earlier report noted that of 132 vascular complications encountered in 24 patients, 17 had thoracic or abdominal aortic aneurysms, dissections, or ruptures.¹⁵ Vascular repair in these patients may be hazardous because the arteries are extremely fragile, and reconstructions should be undertaken with extreme caution.

Tuberous sclerosis is an autosomal dominant disorder characterized by complex multi-organ involvement affecting the brain, retina, kidneys, heart, and skin, with vascular manifestations occurring less often. Hamartomas in multiple organ

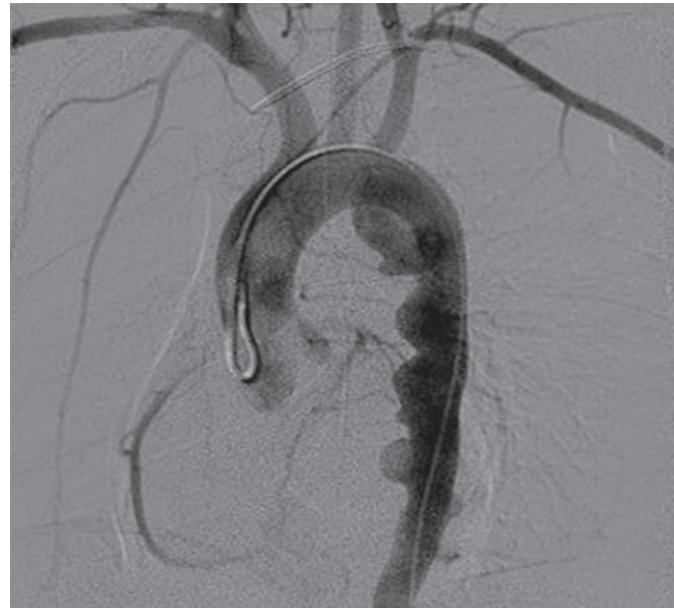


Figure 186.1 Multiple saccular aortic aneurysms along the anteromedial aspect of the descending thoracic aorta in a 5-year-old with tuberous sclerosis.

systems are a common feature, and central nervous system involvement occurs often with associated seizures and developmental delay. The vascular complications of this entity tend to occur in younger children. The abdominal and thoracic aorta are more commonly affected than peripheral arteries (Fig. 186.1). Nearly two dozen pediatric aortic aneurysms attributed to tuberous sclerosis have been reported.^{16–18} Instances

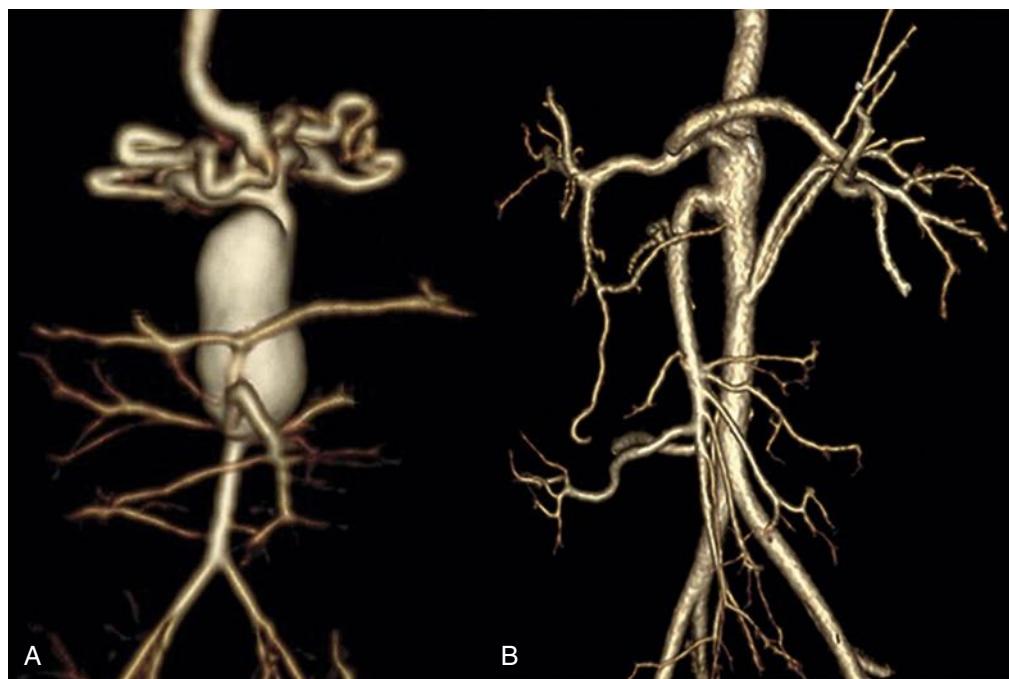


Figure 186.2 (A) Saccular supraceliac abdominal aortic aneurysm with involvement of the celiac, superior mesenteric, and renal arteries associated with focal and nearly complete occlusion (coarctation) of the diaphragmatic aorta in a 6-month-old (computed tomography angiography). (B) Appearance following resection of the coarcted aortic segment with primary aorto-aortic reanastomosis, open aneurysmorrhaphy, and aortic reimplantation of one of the affected arteries with no evidence of progressive aortic dilation 7 years postoperatively. (From Eliason JL, Coleman DM, Criado E, Stanley JC. Surgical treatment of abdominal aortic aneurysms in infancy and early childhood. *J Vasc Surg*. 2016;64:1252–1261.)

of rupture and death have been reported in both infancy and childhood.¹⁸ Although the pathogenesis of the vasculopathy remains unclear, there is evidence for dysplastic and degenerative changes within the arterial wall and a loss of heterozygosity involving genes *TSC1* and *TSC2*.^{18–20} Abdominal aortic aneurysmectomy and aortic reconstruction in children with tuberous sclerosis may be accomplished without some of the hazards that may accompany other genetic arteriopathies.^{21,22}

Developmental (idiopathic) aortic aneurysms are likely due to some misadventure during fetal development, without a currently recognized heritable or spontaneous genetic etiology.^{21,23–25} In cases of thoracic aortic aneurysms that do not meet criteria for the more common syndromic aneurysms, there are data to suggest that acceptance of a global phenotype is useful to stratify risk and guide clinical interventions, including pharmacologic means to lessen the rate of aneurysmal progression.²⁶ It is noteworthy that children having other systemic features involving the craniofacial, ocular, or cutaneous tissues have aneurysms more apt to undergo rapid enlargement.

Certain developmental aortic aneurysms may be attributed to post-stenotic turbulent flow causing aortic dilation. Such aneurysms are most often observed in children with thoracic isthmic coarctations. Treatment of the latter has been by conventional open or endovascular means.²⁷ Abdominal aortic narrowings caused by abnormal fusion of the embryonic dorsal aortas during the 4th week of fetal development are less common. In these children, it is likely that an unknown genetic defect, yet to be identified, results in a major fault in the vessel wall formation. Because of the frequent renal and splanchnic arterial involvement, open surgical interventions are often complex (Fig. 186.2).

Infectious aortic aneurysms in the past accounted for nearly one-third of pediatric aortic aneurysms, but are less commonly encountered in contemporary times, most often associated

with umbilical artery catheterization.²¹ Infectious aneurysms are typically saccular and may involve the thoracic aorta, abdominal aorta, and iliac vessels (Fig. 186.3). *Staphylococcus aureus* and *albus* are predominant organisms responsible for these infections. These aneurysms present most commonly during the first 2 years of life. In such cases, it is critical for infection to be quiescent if a prosthetic conduit is to be used for the aortic repair. Unfortunately, such an option does not exist in the face of many acutely expanding or ruptured infected aortic aneurysms. Noniatrogenic periaortic infections are also recognized as a rare cause of childhood aortic aneurysms.

Inflammatory aortic aneurysms are uncommon, being most often attributed to Takayasu aortoarteritis and in rare instances to Kawasaki disease. Even rarer causes of pediatric aortic aneurysms are Behcet disease and systemic lupus erythematosus.²⁸

Takayasu aortoarteritis is an idiopathic, chronic, large vessel arteritis involving the aorta and its primary branches.²⁹ It begins with panarteritis in the adventitia which progresses to the intima, eventually causing vascular narrowing, occlusion, and aneurysms in the later phases of the disease. Aneurysms usually affect older children and adolescents. Initial systemic manifestations of Takayasu disease include headaches, fever, and dyspnea.³⁰ Hypertension with reduced carotid or femoral arterial pulses affect the majority of children.²⁹ Conventional open or endovascular surgical procedures for both occlusive and aneurysmal disease occurring as a sequela of Takayasu disease may be successfully undertaken.^{30,31} However, with the exception of emergencies, elective surgery should occur only in children with quiescent inflammatory disease following contemporary drug treatments.³² Operative interventions in the face of active inflammation markedly increase the likelihood of later anastomotic stenoses or aneurysms.

Kawasaki disease (mucocutaneous lymph node syndrome) is a systemic arteritis occurring in younger children of unknown



Figure 186.3 Infectious infrarenal abdominal aortic aneurysm (right arrow) and a right iliac artery aneurysm (left arrow) in a 2-week-old associated with infected umbilical catheterization and sepsis (computed tomography angiography). (From Eliason JL, Coleman DM, Criado E, Stanley JC. Surgical treatment of abdominal aortic aneurysms in infancy and early childhood. *J Vasc Surg*. 2016;64:1252–1261.)

etiology although it is thought to be triggered by an infectious agent. The latter initiate an immune response in genetically susceptible children. It is characterized by a febrile state typically with erythematous extremities, fingertip desquamation, conjunctivitis, and lymphadenopathy. The disease more commonly affects the abdominal segment of the aorta, usually during the first year of life. A critical component of Kawasaki disease is coronary artery aneurysms, affecting approximately a fifth of children with an attending risk of acute thrombosis.^{33,34} Aneurysm regression with immunosuppression is well described in this disease, especially following the early administration of intravenous immunoglobulin.³⁴ Aortic or peripheral arterial reconstructions should be avoided in the face of active disease at the risk of anastomotic disruption or stenosis.³⁵

Traumatic aortic aneurysms in childhood are rare, with most penetrating aortic injuries resulting in hemorrhage and death, although deceleration tears of the thoracic aorta are often contained and serve as the site of pseudoaneurysm formation. Such aneurysms may be treated with open aortic reconstructions or in select patients by endovascular means.

Open surgical interventions for aortic aneurysms in children include aneurysmectomy with interposition graft reconstructions, non-anatomic aorto-aortic bypasses, and closed or open aneurysmorrhaphy.²¹ These reconstructions are often complex, given the frequency of splanchnic or renal artery branches originating from the aneurysms. Aneurysmectomy with interposition graft aortic reconstruction is the most direct means of treating pediatric abdominal aortic aneurysms limited to the infrarenal aorta (Fig. 186.4). The largest diameter graft that can safely be implanted should be used, but

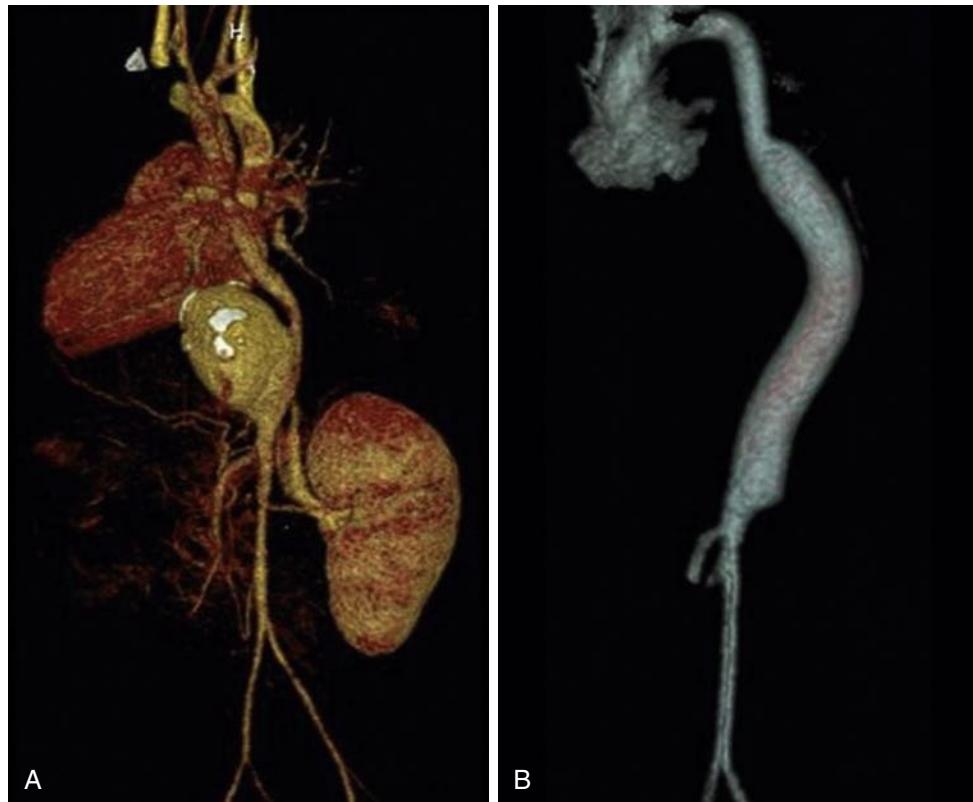


Figure 186.4 (A) Saccular mycotic aneurysm of the supraceliac abdominal aorta in a 20-month-old associated with infected umbilical artery catheterization (computed tomography angiography). (B) Magnetic resonance angiography following aneurysmectomy and repair with interposition 12-mm expanded polytetrafluoroethylene graft. (From Eliason JL, Coleman DM, Criado E, Stanley JC. Surgical treatment of abdominal aortic aneurysms in infancy and early childhood. *J Vasc Surg*. 2016;64:1252–1261.)

not one so large that slow flow along the graft's inner surface results in the accumulation of thrombus with the potential for thromboembolism.

Aortic grafts placed in older children and adolescents must allow for sufficient blood flow as adulthood is reached. In infants and younger children, larger conduits permit further growth prior to the predicted need for reoperation as the child outgrows the initial reconstruction. In general, this translates to 6- to 10-mm grafts in newborns and small infants, 10- to 12-mm grafts in larger infants, 12- to 14-mm grafts in young children, and 14- to 20-mm grafts in older children and adolescents. Teflon (expanded polytetrafluoroethylene [ePTFE]) prostheses are favored over woven or knitted Dacron (polyethylene terephthalate) due to the inherent postimplantation dilation of the latter conduits. Cryopreserved homografts may be an appropriate conduit in select children, although their long-term durability has not been established.^{18,25}

Aneurysmectomy with nonanatomic aortic reconstructions in the form of thoracoabdominal or abdominal aorto-aortic and aorto-iliac bypasses are undertaken most often when splanchnic or renal arteries have their origins from the aneurysm itself, mandating their revascularization (Fig. 186.5). Usually, the aortic reconstructions are undertaken first, before reconstructions of the branch arteries. This provides continuous flow to the pelvic and lower extremity tissues during respective splanchnic and renal artery reconstructions, and eventual aneurysmectomy.

Closed or open aneurysmorrhaphy in treating pediatric abdominal aortic aneurysms is ill defined.²¹ Nevertheless, if the underlying basis of the aortic dilation can be eliminated, such

as with post-stenotic aneurysms, the inexorable progression of aortic expansion following aneurysmorrhaphy may not occur. This assumes the residual aortic wall will remain stable. Although the latter may be reasonable, no long-term data exist to confirm such. Closed aneurysmorrhaphy is performed most often in treating small fusiform aortic aneurysms. Two specific circumstances negate the safe performance of closed aneurysmorrhaphy: (1) the first being the presence of thrombus or loose fibrinous debris lining the aneurysm sac, and (2) the second being an exceedingly large aneurysm, in which the plicated wall would be difficult to collapse. In the latter setting, an open aneurysmorrhaphy with excision of a portion of the wall would be more appropriate (see Fig. 186.2).

Endovascular aneurysm repair (EVAR) has a limited role in the definitive management of most pediatric abdominal aortic aneurysms. An exception to this tenet is the treatment of traumatic pseudoaneurysms in older adolescents in whom further aortic growth is unlikely, or in those emergent circumstances of an aortic disruption. Thoracic endovascular aneurysm repair (TEVAR) for thoracic aneurysm in select patients of appropriate age and size may be also appropriate, although caution exists in children whose aneurysm disease is a result of a progressive arteriopathy.

Surgical treatment of abdominal aortic aneurysms is successful in most infants and young children.^{21,25,36} This involves complex operative techniques that must take into account the child's growth potential, the aneurysm location and whether the splanchnic and renal arteries are involved. Regardless of the intervention undertaken, long-term follow-up is mandatory for all surgical interventions.

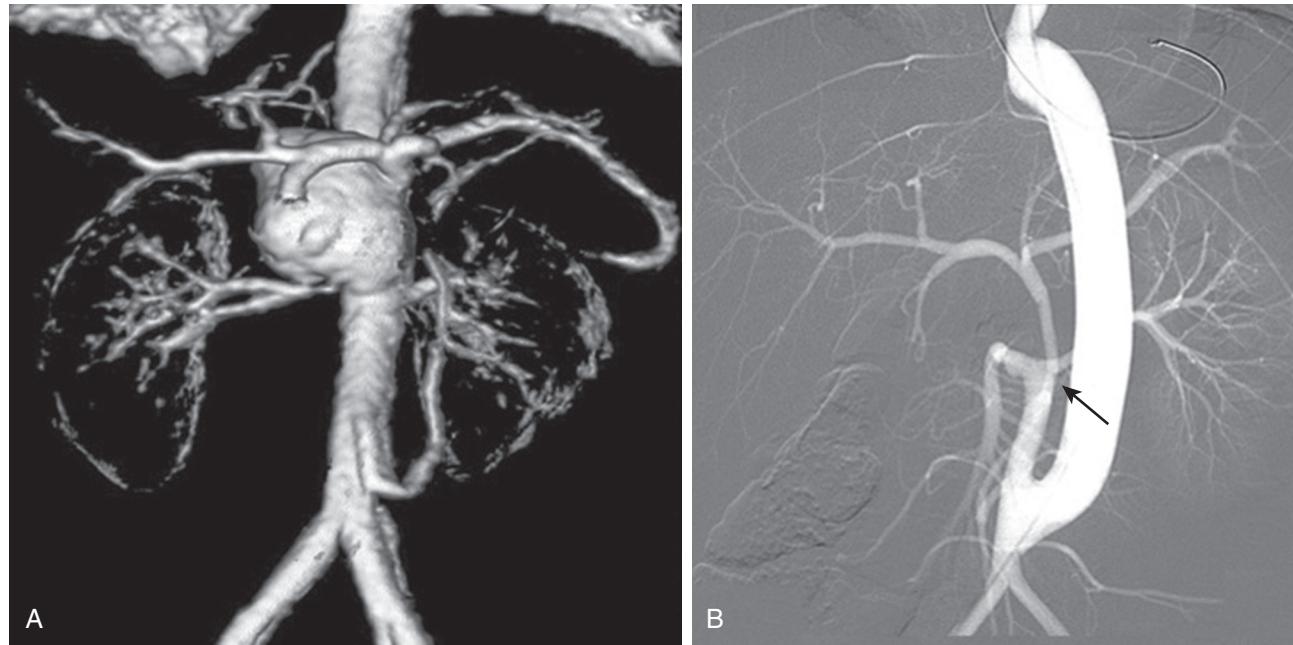


Figure 186.5 (A) Central abdominal aortic aneurysm with involvement of the celiac, superior mesenteric, and renal arteries associated with tuberous sclerosis in a 5-year-old (computed tomography angiography). (B) Treatment included aneurysmectomy, 14-mm expanded polytetrafluoroethylene thoracoabdominal bypass, aortic reimplantation of a left renal artery and superior mesenteric artery (SMA), and aortoceliac bypass with a hypogastric artery graft, all from the distal abdominal aorta (arrow), as well as a right nephrectomy (digital subtraction arteriography). (From Eliason JL, Coleman DM, Criado E, Stanley JC. Surgical treatment of abdominal aortic aneurysms in infancy and early childhood. *J Vasc Surg*. 2016;64:1252–1261.)

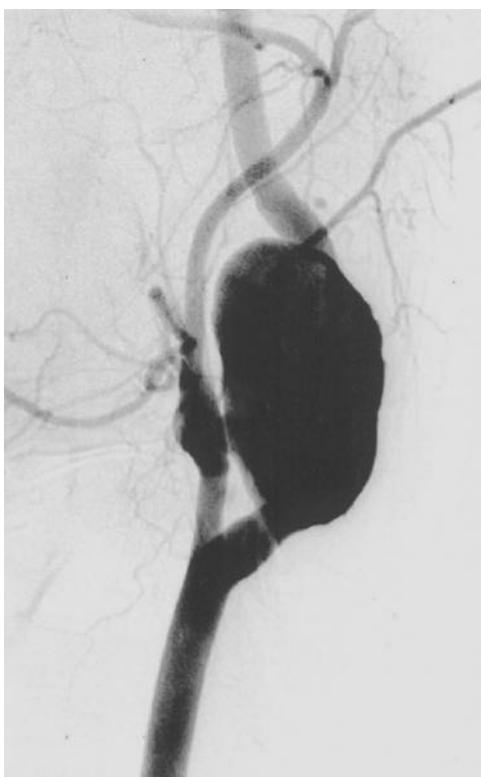


Figure 186.6 Carotid Artery Aneurysm. Preoperative angiogram demonstrating a large traumatic saccular internal carotid artery aneurysm in a 14-year-old. (From Davis FM, Eliason JL, Ganesh SK, Blatt NB, Stanley JC, Coleman DM. Pediatric nonaortic arterial aneurysms. *J Vasc Surg*. 2016;63:466–476.)

NONAORTIC ARTERIAL ANEURYSMS

The natural history of nonaortic pediatric arterial aneurysms remains ill defined, as is the categorization of these aneurysms.^{37,38} The risk of expansion, rupture, thrombosis, and distal thromboembolism of these nonaortic aneurysms warrant intervention in most instances. These peripheral arterial aneurysms are extremely uncommon but often share etiologic risk factors similar to aortic aneurysms, including infection, inflammation, inherited vasculopathies, and trauma. Polyarteritis nodosa is relatively common among the inflammatory arteriopathies, and in contrast to aortic aneurysms, neurofibromatosis 1 is often associated with nonaortic arterial aneurysms in childhood. It is appropriate to discuss peripheral arterial aneurysms by the vascular bed affected.

Extracranial cerebrovascular artery aneurysms have been previously defined to exist: in the region of the carotid bulb having a diameter greater than 150% of the normal common carotid artery or 200% of the normal internal carotid artery, and in the internal carotid artery with dilations greater than 120% of a normal cross-section of the ipsilateral artery.³⁹

True aneurysms of the extracranial carotid artery are likely a manifestation of a genetic disorder, inflammatory disease, or traumatic pseudoaneurysms resulting typically from blunt injury, or iatrogenic accompanying head and neck operations or during vascular access (Fig. 186.6). The most commonly reported symptom is a pulsatile neck mass.

Horner syndrome and cranial nerve dysfunction rates have been reported to be as high as 4.6% to 11%.⁴⁰ Given that the risk of central neurologic complication and rupture rates are reported to be as high as 42%, early surgical treatment is usually pursued.⁴¹

Aneurysmectomy with an interposition bypass is the most commonly described procedure in treating carotid artery aneurysms, using both autogenous and prosthetic conduits.^{42–45} The authors favor the use of hypogastric artery given the risk of late aneurysmal deterioration of vein grafts when placed in low resistance arterial beds with high diastolic flow.⁴⁶ Procedural morbidity has been associated with cranial nerve injury (4% to 22% incidence) and central neurologic complications, such as stroke (4% to 11% incidence).⁴⁰ Ligation may be considered in select patients but only with confirmation of an intact circle of Willis.⁴⁷ Endoluminal therapy which has become frequently used in treating intracranial aneurysms in children,⁴⁸ may have a place in extracranial cerebrovascular disease with the use of covered stents in certain cases.^{49,50}

Splanchnic artery aneurysms most commonly have a mycotic, inflammatory, traumatic, or connective tissue disease origin.^{51–53} Abdominal pain is common, and luminal gastrointestinal bleeding has also been described.^{54–56} Blunt and deceleration injuries to the liver and spleen can result in small pseudoaneurysms that may thrombose without clinical sequelae or be treated with endovascular embolization if symptomatic or associated with hematobilia.

Although surgical intervention to exclude an aneurysm and reestablish arterial continuity is ideal (Fig. 186.7), in cases of infection or most genetic diseases, vessel friability may preclude reconstruction, mandating ligation (Fig. 186.8) with reliance on collateral flow to lessen intestinal ischemic complications. In the case of superior mesenteric artery aneurysms, the latter is likely to be successful if the aneurysm is proximal to the middle colic and inferior pancreaticoduodenal arteries' origin, in close proximity to the aorta.

Renal artery aneurysms are generally attributed to a connective tissue disorder or a congenital vasculopathy, such as arterial dysplasia related to neurofibromatosis. A contemporary radiologic review of renal angiography performed for pediatric fibromuscular dysplasia and neurofibromatosis characterized the phenotype to include 12 cases (28%) of renal aneurysms with a background incidence of occlusive disease affecting 91% of patients (39/43).⁵⁷ The clinical relevance of these aneurysms remains ill defined, including the natural history and rupture risk. Aneurysms are often saccular in appearance and occur most commonly at bifurcations. Overt rupture is unlikely and covert rupture into an adjacent renal vein even less common.²³ Surgical options for treating these aneurysms are complex and include resection with primary angioplasty closure, bypass, or reimplantation of the affected artery following excision of the aneurysm (Fig. 186.9). The authors favor hypogastric artery for conduit in cases of aortorenal bypass for reasons aforementioned. Ethanol ablation and coil embolization may also be considered for distal, subsegmental, and cortical aneurysms.^{58,59} Primary partial or complete nephrectomy may be required for irreparable

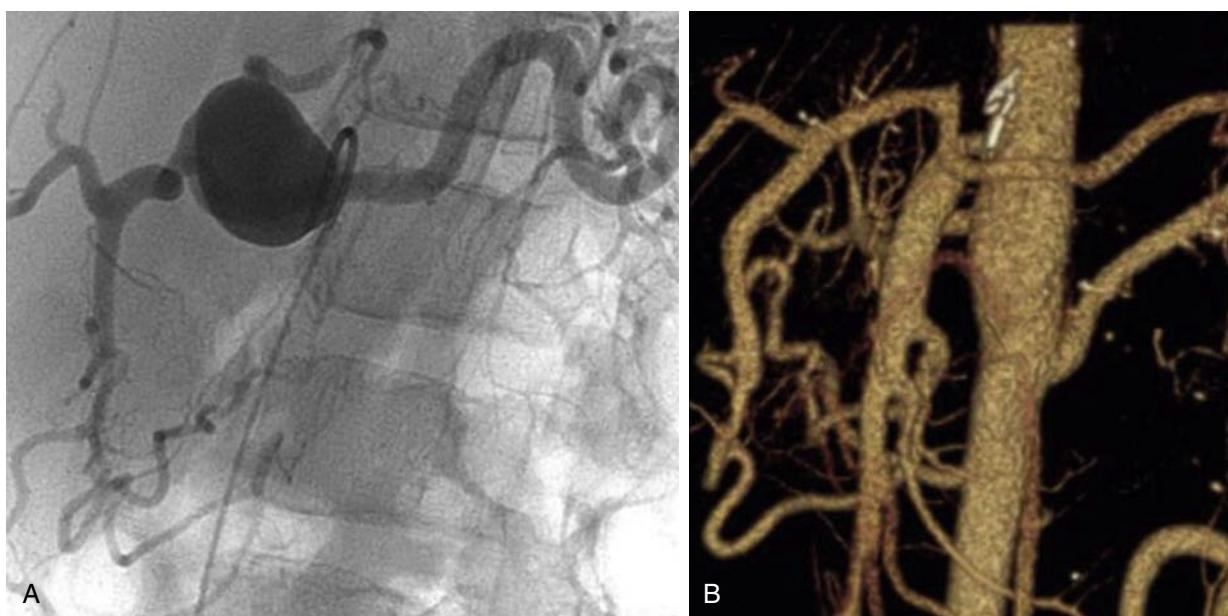


Figure 186.7 Celiac Artery Aneurysm. (A) Preoperative angiogram of a post-stenotic celiac artery aneurysm in a 13-year-old. (B) Postoperative computed tomographic arteriography image following resection of the aneurysm and primary end-to-end anastomosis of the proximal hepatic to proximal splenic artery. (From Davis FM, Eliason JL, Ganesh SK, Blatt NB, Stanley JC, Coleman DM. Pediatric nonaortic arterial aneurysms. *J Vasc Surg*. 2016;63:466–476.)

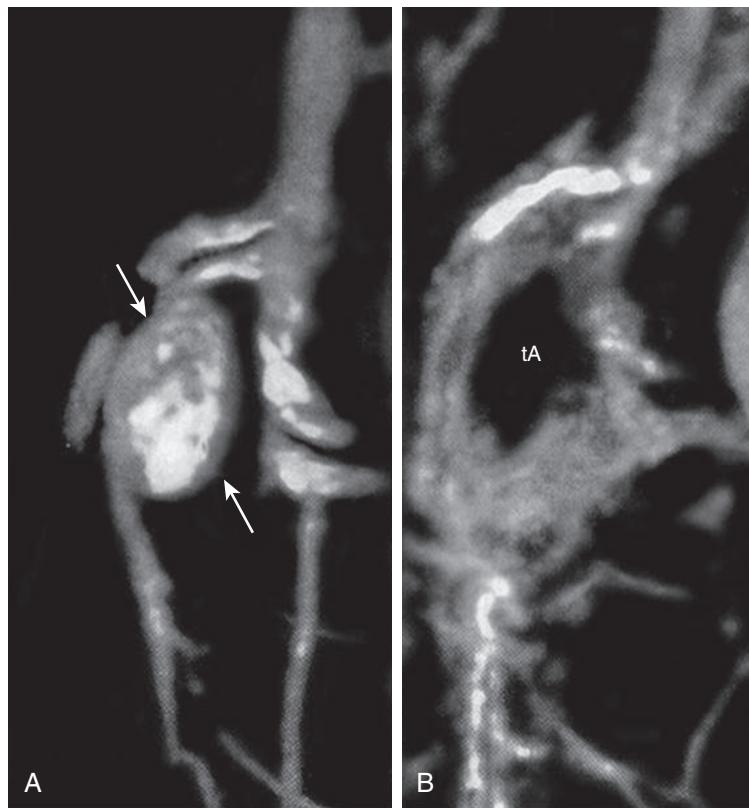


Figure 186.8 Superior Mesenteric Artery (SMA) Aneurysm. (A) SMA aneurysm in a 10-year-old child with Ehlers–Danlos syndrome (arrows). (B) Postoperative appearance of thrombosed aneurysm (tA) following proximal and distal ligation alone. (From Upchurch GR Jr, Zelenock GB, Stanley JC. Splanchnic artery aneurysms. In: Rutherford RB, ed. *Vascular Surgery*, 6th ed. Philadelphia, PA: Elsevier Saunders; 1973:2015.)

disease, including multiple aneurysms not amenable to any form of reconstruction.³⁷ Closed aneurysmorrhaphy of small (2 to 3 mm diameter) renal artery aneurysms with fine monofilament suture may be appropriate for those aneurysms encountered during treatment of other larger, more clinically

relevant aneurysms, or during renal artery revascularization for occlusive disease.

Upper extremity aneurysms most commonly result from trauma, including iatrogenic catheterization-related injury and the inflammatory arteritides, such as Kawasaki disease

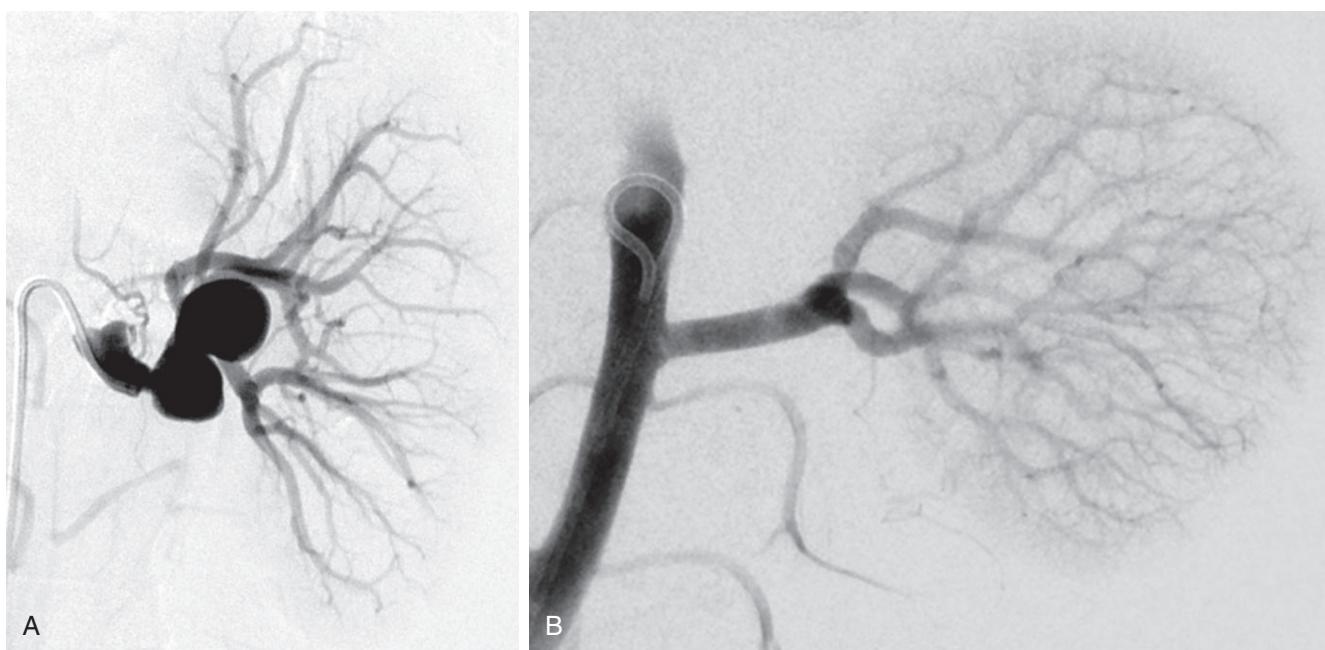


Figure 186.9 Renal Artery Aneurysm. (A) Preoperative angiogram of a 14-year-old with neurofibromatosis (NF-1) demonstrating left renal artery ostial and distal stenosis associated with two bilobed distal left renal artery aneurysms. (B) Postoperative angiogram following aneurysm resection with *ex vivo* reconstruction requiring syndactilization of three segmental renal artery branches anastomosed to an aortorenal bypass using hypogastric artery graft.

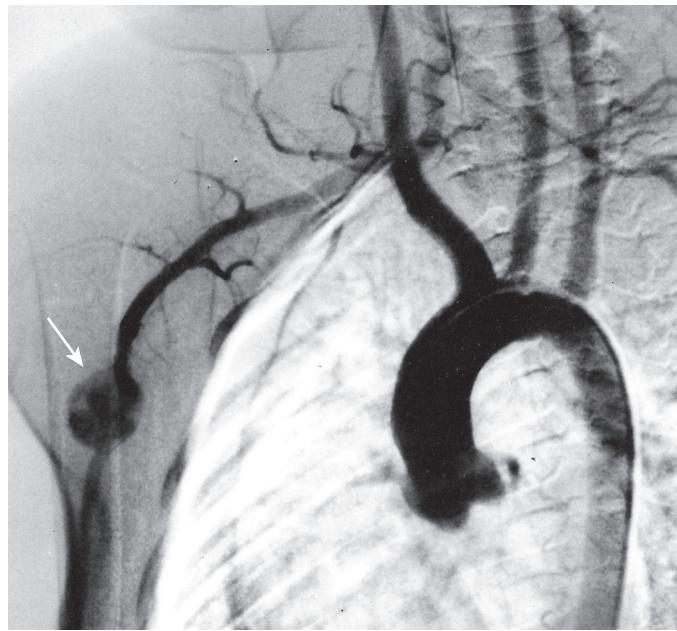


Figure 186.10 Brachial Extremity Aneurysm. Preoperative arteriogram of a 4-year-old with a right brachial artery aneurysm (arrow) resulting from Kawasaki disease. (From Stanley JC, Eliason JL. Pediatric arterial diseases. In: Coran AG, Adzick NS, Krummel TM, et al., eds. *Pediatric Surgery*, 7th ed. Philadelphia, PA: Elsevier; 2012:1643.)

(Fig. 186.10). These aneurysms often present as a pulsatile mass, although pain and paresthesia have been reported.^{37,60,61} Although rupture of noninfectious aneurysms is rare, the risk of distal hand and digit thromboembolism merits early surgical intervention.^{60–63} Aneurysm resection

distal to the axillary artery with successful revascularization by direct re-anastomosis, bypass with reversed saphenous vein, or ligation pending the presence of adequate collateral vessels have all been well described with negligible morbidity and mortality.^{61,64,65} Aneurysms involving arteries within the hand may be treated with microvascular interventions.^{66,67}

Lower extremity aneurysms most frequently involve the iliac arteries (Fig. 186.11), sharing similar etiologic risk factors with aortic aneurysm.^{68–73} Although mainly asymptomatic, these aneurysms have been reported to cause abdominal pain and lower quadrant abdominal mass.⁷² Although internal iliac artery aneurysms may be safely ligated in the setting of robust pelvic collateralization, common and external iliac arteries require treatment with resection and re-anastomosis, interposition grafting, aorto-iliac bypass, or plication. A prosthetic conduit is appropriate in this setting, recommended to be sized as large as possible to facilitate future growth without risking luminal thrombus and distal thromboembolism.^{37,68} Cadaveric homografts may have a role in the management of infected iliac artery aneurysms.⁷³

Femoral artery pseudoaneurysms and true aneurysms (Fig. 186.12) are subject to acute thromboses and warrant early operative intervention with reconstructions using autologous vein following the aneurysmectomy. Similarly, pediatric popliteal artery aneurysms are resected or isolated from the arterial circulation because of potential thromboembolism, followed by primary arterial reconstruction or bypass.^{37,74} Involvement of the more distal tibial artery with aneurysm disease is exceedingly rare.⁷⁵

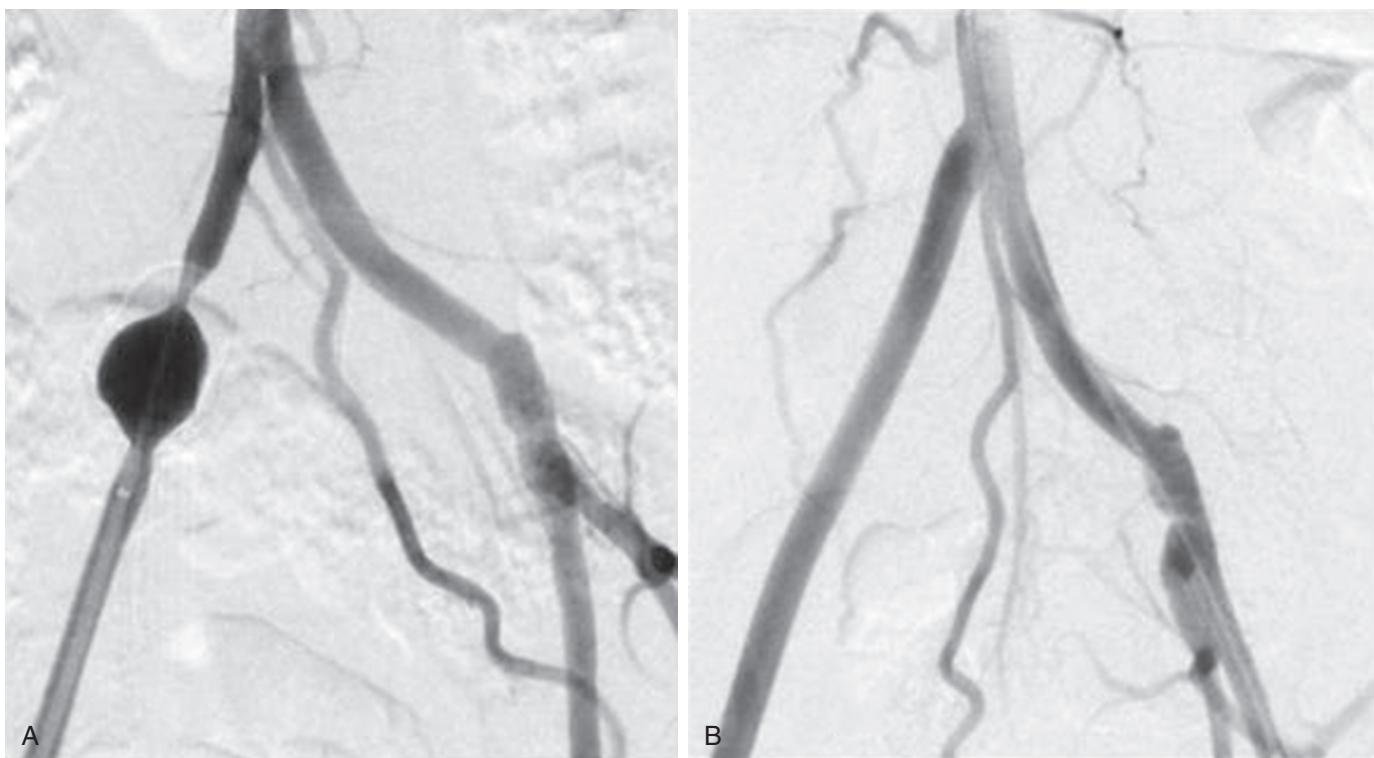


Figure 186.11 Iliac Artery Aneurysm. (A) Preoperative arteriogram of a 10-year-old child with a right common iliac artery aneurysm. (B) Postoperative arteriogram following aneurysm resection with aorto-iliac bypass using 7 mm polytetrafluoroethylene prosthesis. (From Davis FM, Eliason JL, Ganesh SK, Blatt NB, Stanley JC, Coleman DM. Pediatric nonaortic arterial aneurysms. *J Vasc Surg*. 2016;63:466–476.)



Figure 186.12 Superficial Femoral Artery Aneurysm. Superficial femoral artery aneurysm of unknown etiology in a 2-year-old treated by aneurysmectomy and interposition vein graft reconstruction.

SUMMARY

Pediatric aortic and arterial aneurysms represent a variety of complex diseases affecting multiple vascular territories. Individualized surgical treatment that considers patient age and anatomic factors may be undertaken with negligible perioperative morbidity and mortality. The University of Michigan experience supports the need for long-term follow-up of children treated for aortic and nonaortic arterial aneurysms. Among children undergoing successful aortic aneurysm procedures, 10% required secondary operations.²¹ Although no reinterventions were required following the treatment of extremity aneurysms or extracranial cerebrovascular aneurysms, 15% of children treated for visceral aneurysms underwent later reoperations.³⁷ A multidisciplinary team of vascular surgeons, pediatricians, radiologists, and anesthesiologists provide the best setting for the most optimal care of children with these aneurysms.

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Davis FM, Eliason JL, Ganesh SK, et al. Pediatric nonaortic arterial aneurysms. *J Vasc Surg*. 2016;63:466–476.

The largest contemporary single institution experience reporting on the clinical presentation and management of 41 children with 61 nonaortic aneurysms across diverse vascular beds. This study from the University of Michigan suggests that individualized surgical treatment, ranging from simple ligations to major arterial reconstructions, are durable and can be undertaken with minimal risk.

Eliason JL, Coleman DM, Criado E, Stanley JC. Surgical treatment of abdominal aortic aneurysms in infancy and early childhood. *J Vasc Surg*. 2016;64:1252–1261.

The largest contemporary single institution experience reporting on the clinical presentation and surgical management of 11 pediatric patients with aortic aneurysms. This study from the University of Michigan suggests that successful surgical management of AAAs in infants and young children requires careful execution of a diverse group of surgical techniques based on the etiology, the child's size and growth potential, and the aneurysm's location and coexisting branch involvement.

Sarkar R, Coran AG, Cilley RE, et al. Arterial aneurysms in children: clinicopathologic classification. *J Vasc Surg*. 1991;13:47–57.

A relevant classification schema proposed for pediatric arterial aneurysms that considers: (1) arterial infection, (2) giant-cell aorto-arteritis, (3) autoimmune connective tissue disease, (4) Kawasaki disease, (5) Ehlers–Danlos syndrome or Marfan syndrome, (6) other forms of noninflammatory medial degeneration, (7) arterial dysplasia, (8) congenital–idiopathic factors, and (9) false aneurysms.

A complete reference list can be found online at www.expertconsult.com

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Pediatric Vascular Tumors

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INTRODUCTION

Vascular tumors are different from malformations in that they have proliferative endothelium, while malformations occur from abnormal tissue growth as the child grows and has quiescent endothelium. Both primary vascular tumors and vascular malformations may look similar, raised or flat lesions, blue, red or purple in hue. Growth and/or expansion of such may risk clinical complications like disfigurement, chronic pain, recurrent infections, systemic manifestations like thrombocytopenia, coagulopathies, and congestive heart failure in the minority of patients with large high-flow lesions, and very rarely death.^{1–3} Most of these tumors are seen in infants and children, except pyogenic granulomas, which can be seen at any age.

Historical treatment options were limited, and often reserved for palliation. Infantile hemangiomas, with a prevalence of 4%–5%, are by far the most common benign tumors of infancy with other vascular tumors exceedingly rare. Low disease frequency, diverse natural history and unusual morphology have challenged tumor classification. The International Society for the Study of Vascular Anomalies (ISSVA) classification expands on the 2013 World Health Organization (WHO) Classification with additional phenotypes and precise terminology² (Table 187.1). This chapter reviews in further detail the more common benign vascular tumors including: infantile hemangioma (IH), congenital hemangioma (CH), spindle cell angioma, epithelioid hemangioma (EH), pyogenic granuloma, and kaposiform hemangioendothelioma (KHE).

Vascular tumor diagnosis rarely requires biopsy and treatment strategies hinge on multidisciplinary care. Application of

topical therapies, the use of antiangiogenic drugs, sclerotherapy, or laser therapy, and the role for surgical excision continue to evolve. Vascular anomaly or malformation centers have been created at many referral centers to centralize and coordinate care.^{4,5} Similarly, resection of secondary tumors may involve pediatric, orthopedic, oncologic surgeons, and neurosurgeons alone or in combination, with vascular surgery involvement relegated to select patients with large or infiltrative tumors near the aorta or vena cava and their major branches, or the major head, neck or extremity vessels. In contrast to adult patients, major vascular reconstruction is uncommonly required in the pediatric population.

BENIGN TUMORS

Infantile Hemangioma

Most infantile hemangiomas (IH) are sporadic, however rare cases are associated with an abnormality of chromosome 5 and 34% of patients had a family history of infantile hemangioma, most commonly in a first-degree relative.³ The pathogenesis of IH is development from progenitor cells which normally would evolve into blood vessels.⁶ The precursor cell seems to be a multipotent hemangioma-derived stem cell, which produces an erythrocyte-type glucose transporter (GLUT1).^{7,8} These stem cells are similar to placental endothelium but genetically derived from the child.⁹ Hypoxia is widely accepted as the primary stimulus for growth of IH.¹⁰ Endothelial proliferation likely occurs from complex interactions between activated T cells, alterations in vascular endothelial growth factor receptor

TABLE 187.1 2018 International Society for the Study of Vascular Anomalies (ISSVA) Classification of Vascular Tumors

Category	Vascular Tumor Type	Causal Gene
Benign	Infantile hemangioma Congenital hemangioma (RICH, NICH, PICH) Tufted angioma ^a Spindle cell angioma Epithelioid hemangioma Pyogenic granuloma Others ^b	GNAQ/GNA11 GNA14 IDH1/IDH2 FOS BRAF/RAS/GNA14
Locally Aggressive or Borderline	Kaposiform hemangioendothelioma ^a Retiform hemangioendothelioma Papillary intralymphatic Angioendothelioma (PILA), Dabska tumor Composite hemangioendothelioma Pseudomyogenic hemangioendothelioma Polymorphous hemangioendothelioma Hemangioendothelioma NOS Kaposi sarcoma	GNA14 FOSB
Malignant	Angiosarcoma Epithelioid hemangioendothelioma	(Post radiation)/MYC CAMTA1/TFE3

Adapted from ISSVA Classification of Vascular Anomalies. ©2018 International Society for the Study of Vascular Anomalies. Available at: issva.org/classification.

^aMany experts believe that tufted angioma and kaposiform hemangioendothelioma are part of a spectrum rather than distinct entities.

^bOther benign tumor types include: hobnail hemangioma, microvenular hemangioma, anastomosing hemangioma, glomeruloid hemangioma, papillary hemangioma, intravascular papillary endothelial hyperplasia, cutaneous epithelioid angiomatic nodule, acquired elastotic hemangioma, littoral cell hemangioma of the spleen.

NOS, not otherwise specified

(VEGFR) expression, and the binding of endothelial growth factor A to VEGFR-2 receptors. Sometime between early infancy and 4 years of age, these tumors involute, the mechanism of which is unclear. Endothelial proliferation slows and apoptosis increases during this time, with potential triggers related to a decrease in proangiogenic maternal estrogens or an increase in angiogenesis inhibitors in the epidermis. The tumor is replaced by fat cells, which also derive from the aforementioned stem cells.^{11–13}

IH is the most common benign vascular neoplasm of infancy with an incidence of 4%–5%.¹ IH affects non-Hispanic white infants more often than those with darker skin. Premature infants with low birth weight, particularly girls, also have a higher risk of developing these tumors. For every 500-g decrease below a 2500-g baby, the risk of IH increases 40%. Additional risk factors include: advanced maternal age, placenta previa, pre-eclampsia and other placental anomalies.¹ Lesions tend to be solitary (80%) and involve the head and neck, trunk, or extremity in decreasing order of frequency. The tumors appear most often at 3–6 weeks of age, but may be evident as small pale, telangiectatic, or ecchymotic spots in nearly one-half of infants at birth.^{14,15}

IH has a proliferative growth phase during the first 9 months of life, with most rapid growth in the first 8 weeks of life. Eighty percent of volumetric growth is achieved by age 5 months.^{16,17} Tumor appearance during this stage varies based on whether it involves the superficial or deep dermis. If the tumor is in the superficial dermis, it appears bright red, but when deeper, the lesion either has a bluish or normal color. Many IH are mixed with both superficial and deep components (Fig. 187.1A–C). Proliferation often slows by 5 to 6 months of age;

however, in those with a primarily deep component, proliferation can be prolonged, sometimes up to 18 months of age. Subsequent growth coincides with growth of the child.¹⁷ The involution phase begins by 1 year of age, at which point the tumor shrinks, the color fades, the center of the lesion pales, and the tumor softens. Involution is complete by 4 years of age in most children. Some patients with a large deep component are left with fibro-fatty tissue and redundant skin. Scarring is most often associated with ulcerative lesions. Telangiectasias and discoloration are other sequelae after lesion regression is complete.^{16,18}

IH may be multiple and involve the skin or liver. The cutaneous lesions are often less than 5 mm in diameter and dome-like (Fig. 187.1D). If more than five hemangiomas are present, the child has a 16% chance of harboring visceral lesions, with the most common extracutaneous location being the liver. The brain, gut, and lung are rarely involved. Most hepatic IH are small, discovered incidentally, and are classified as multifocal or diffuse. Multifocal hepatic hemangiomas are usually asymptomatic, although high-output cardiac failure by arteriovenous or portovenous shunting occurs rarely. Similar to cutaneous IH, involution begins by 1 year of age and often earlier in most children.^{19,20}

Diffuse IH can be seen in the liver and often do not need active treatment and rather simply close observation. At times, large, diffuse IH may replace much of the liver parenchyma and cause massive hepatomegaly. Compression of the inferior vena cava or upward pressure on the diaphragm with reduction of the pleural space causes respiratory symptoms or abdominal pain and pressure in select patients. High-output cardiac failure is rarely seen. Almost all infants with profound liver involvement



Figure 187.1 Various Types of Infantile Hemangioma. (A) Superficial lesion of the face. (B) Deep lesion of the trunk. Notice the difference in outward appearance and color. (C) A mixed lesion with both superficial and deep components. (D) Multifocal superficial lesions on the trunk and buttocks.

develop hypothyroidism because this hemangioma expresses a deiodinase that inactivates thyroid hormone, necessitating thyroid-stimulating hormone monitoring.²¹ As the hemangioma regresses, the need for replacement therapy ebbs. Differential diagnosis includes hepatic arteriovenous malformation (AVM) and malignant neoplasm. Hemangiomas and AVMs exhibit

fast-flow characteristics, with 90% of fast-flow lesions being hemangiomas and the remaining ones AVMs. Hepatoblastoma and metastatic neuroblastoma do not have the shunting characteristics of hemangioma.²⁰

IH may be associated with specific syndromes in which there are anomalies of the cardiovascular, gastrointestinal,



Figure 187.2 Segmental lumbosacral (A) and facial (B) infantile hemangiomas. Such findings should raise suspicion of LUMBAR or PHACE associations.

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and urogenital systems; brain or spinal cord; eye; and/or bone. A child with a large, segmental midline lumbosacral region IH carries a 33% chance of having a spinal anomaly, such as a tethered cord, lipoma, or intraspinal hemangioma (Fig. 187.2A). Magnetic resonance imaging (MRI) between 3 and 6 months of age is important to exclude spinal abnormality in these cases.²² Lower body IH may be associated with urogenital anomalies or ulceration, myelopathy, bone deformities, anorectal malformations, arterial, and/or renal anomalies. This association is termed LUMBAR, and disproportionately affects females. The IH is usually large, segmental, and superficial; has minimal postnatal growth, and often ulcerates. The hemangioma may be seen in the sacral or lumbar regions, the perineum and genitalia, or lower extremity.²³

Segmental plaque-like IH of the face may be associated with one or more anomalies in the brain or cerebrovascular system, heart, eye, sternum, or supraumbilical region, categorized as PHACE syndrome (see Fig. 187.2B). The acronym represents posterior fossa brain malformation, hemangioma, arterial anomalies, coarctation of the aorta and cardiac defects, eye abnormalities, and sternal clefting or supraumbilical raphe. Ninety percent of affected children are female. The most common anomaly is a cerebrovascular malformation in 72% of patients, and approximately 8% of infants sustain a stroke. PHACE syndrome is found in less than 3% of all

patients with IH, and extracutaneous features in this group are seen in less than one-third of the children.²⁴ Recognition of PHACE syndrome is critical because these patients have higher risk of complications and sequelae than those with isolated IH.

Diagnosis of IH is made by history and physical examination in nearly all patients. Ultrasound shows a well-circumscribed hypervascular mass. Low-resistance arterial waveforms are present, and venous drainage is seen. MRI can be used if ultrasound imaging is equivocal, but is rarely necessary. IH is seen as a parenchymal mass with dilated vessels and signal voids during the proliferative phase, unlike an arteriovenous malformation. The lesion is isointense on T1 sequences and hyperintense on T2 images and enhances homogeneously after the administration of contrast. An involuting IH is lobular and has adipose tissue and a reduced number of vessels, with signal void or enhancement.²⁵ MRI or MR angiography is used to image the brain and neck for children with PHACE syndrome. Echocardiography and an eye examination are important adjuncts. Although ultrasound may be useful when LUMBAR associations are suspected, MRI often is needed to image the lumbosacral spine, abdomen, and pelvis. Choice of study is based on age of the infant or child and type of lesion.

Less than 1% of IH require histopathologic evaluation for diagnosis. Biopsy is indicated if malignant disease is suspected by atypical appearance or if the diagnosis remains unclear after

the lesion is imaged. Microscopic evaluation of a proliferating lesion shows tightly packed capillaries with plump endothelial cells and minimal intervascular stroma. In contrast, an involuting lesion shows reduction in the number of capillaries that have enlarged channels, increased stroma, and fibrofatty tissue.^{26–28} A hallmark of IH is that it shows GLUT-1 positivity throughout all stages of proliferation and involution.

Observation is the mainstay of management as most tumors are small and localized and do not involve esthetically or functionally important areas. Parents can be reassured by showing them examples of proliferative, involuting, and regressed lesions that were managed in other infants. Close observation is needed during the proliferative phase to be certain the tumor does not ulcerate or pose risk of destruction of adjacent important structures.²⁹

An early therapeutic intervention at 1–3 months of age has been cited as critical for complicated infantile hemangiomas to prevent medical complications and permanent disfigurement. Photos have been used to triage low-risk versus high-risk infantile hemangiomas, and a scoring system has been used for primary care physicians to encourage early referral to multidisciplinary hemangioma specialists.^{4,5}

Topical beta-blockers are used primarily for the treatment of small, localized, superficial hemangiomas as an alternative to observation. They have also been used in combination with systemic therapy in complicated hemangiomas or to prevent rebound in a hemangioma being tapered off of systemic treatment.^{6,7} The same precautions as noted below for propranolol should be followed for topical beta-blockers as the systemic absorption of timolol is variable. Cautious administration is required for ulcerated and deep hemangiomas because higher plasma concentrations of timolol can be seen. Topical dosing requires the ophthalmic gel-forming 0.5% solution of timolol with one drop applied to the hemangioma twice daily until a treatment response is achieved. Data from a multicenter, retrospective, cohort study of 731 children with predominantly superficial IH treated with topical timolol 0.5% twice daily supported that 92% of patients showed significant improvement in color, and 77% of patients showed improvement in size, extent, and volume.⁷

Approximately 16% of lesions develop skin ulceration during the proliferative phase, at an average of 4 months of age.³⁰ Ulceration is more common on the lips, neck, anogenital, and other flexural regions; larger and segmental IH are also at risk of ulceration (Fig. 187.3). Standard wound care is topical antibiotic and Vaseline, followed by gauze. These lesions may be very painful, so topical lidocaine may be needed. Topical pharmacotherapy may be appropriate treatment for small IH that do not require systemic therapy. Topical corticosteroid, such as clobetasol, may prove effective for small, superficial lesions, although hypopigmentation and skin atrophy can occur. Application of timolol, also has been proven effective for small, superficial lesions.³¹

Systemic therapy is indicated for large hemangiomas that cannot be treated with local injections and is considered when topical therapy has been ineffective. Several drugs have been

used, including beta-blockers, steroids, and anti-metabolic agents.²⁹ Propranolol, a nonselective beta-blocker, is the first-line therapy for infantile hemangiomas. Potential mechanisms of action include vasoconstriction and/or decreased expression of VEGF and bFGF, leading to apoptosis.^{8,9}

In a large industry-sponsored randomized trial, 456 infants aged 5 weeks to 5 months with a proliferating infantile hemangioma of at least 1.5 cm received either a placebo or propranolol (dose = 1 mg/kg per day or 3 mg/kg per day) for 3 or 6 months. After interim analysis of the first 188 patients who completed 24 weeks of trial treatment, the regimen of 3 mg/kg per day for 6 months was selected for the final efficacy analysis.³² Eighty-eight percent of infants treated with propranolol demonstrated improvement by week 5, compared with only 5% of patients who received placebo. In another study of 635 infants with IH, the overall response rate was 91% following propranolol treatment (dose = 2 mg/kg per day), with most patients showing regression and only 2% with side effects, none of which were severe.¹¹ Finally, a recent meta-analysis evaluated 5130 patients from 61 studies and concluded that propranolol was more effective and safer than were other treatments for IH.¹² Consensus treatment guidelines are presented in Table 187.2.^{1,13,14}

Rebound tumor growth occurs if the medication is abruptly discontinued during the proliferative phase. Rebound refers to the growth of IH after propranolol cessation. A multi-institutional, retrospective review of 997 patients with IH identified a rebound rate of 25.3% in 912 patients.¹⁷ Multivariate analysis revealed risk factors for rebound to be deep IH and female sex.

Corticosteroids were the historic first-line of treatment for IH, first used in the late 1950s, but never approved by the U.S. FDA. Corticosteroid therapy has become less popular secondary to the acute and long-term side effects of steroids, but used at times when there is a contraindication to beta-blocker therapy. If systemic beta-blocker or steroid therapy proves ineffective, third-line systemic drugs such as interferon or vincristine are considerations but are rarely, if ever, used. These medications have a high risk of side effects; interferon is not recommended in children younger than 1 year of age because of the risk of neurologic problems such as spastic diplegia.^{5,20}

Embolization is considered for large high-flow IH that cause congestive heart failure, such as multifocal or diffuse hepatic hemangiomas that have macrovascular shunts, particularly when there is little or only partial response to systemic beta-blocker therapy. The goal of embolization is to provide prompt control of heart failure as the therapeutic benefits of systemic agents accrue. This treatment has little or no role in hepatic hemangioma without heart failure. Drug therapy is used post-embolization until the child is approximately 1 year of age, when involution generally begins.

Pulsed dye laser therapy is primarily used to treat residual telangiectasia post-involution but can also be used to treat areas of ulceration in lesions slow to respond to other treatments. Carbon dioxide laser has been used to treat subglottic

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Figure 187.3 Images of ulcerative infantile hemangioma on the neck (A) and upper back (B).

hemangioma during the proliferative phase as drug treatment is initiated.^{33–36}

Excision of proliferating lesions is rarely indicated, given the success of pharmacotherapy.²⁹ The tumor is highly vascular, so there is a risk of blood loss. Anesthetic complications, scarring, and cosmesis in infants must be considered. A well-localized or pedunculated, ulcerated, or bleeding lesion may be excised if the resulting scar is esthetically placed and no worse than would be predicted if excision is done after involution is in process or complete. The ideal time for operative intervention is between 3 and 4 years of age. At this age the tumor is smaller and less vascular and it will no longer involute. In general, the deformity improves with time, which makes long-term memory of the procedure and self-esteem issues less likely.^{18,21}

Circular hemangiomas with residual fibrofatty tissue or excess skin are best managed with circular excision and purse-string closure.³⁷ The resultant scar can be revised 6 to 12 months later and closed linearly. This technique reduces the length of the linear scar relative to the initial diameter of the hemangioma. Initial linear excision of a circular hemangioma generates a scar nearly 3 times the diameter of the tumor.

Congenital Hemangioma

CH are benign vascular tumors that proliferate *in utero*, are fully formed at birth and do not illustrate the same postnatal

growth as previously described for IH. This tumor is most often solitary, although multiple lesions have rarely been described and may range from 1 or 2 cm to more than 20 cm in diameter. CH has a red-violaceous color with coarse telangiectasias, central pallor, and a peripheral pale halo. Unlike IH, CH is more common in the extremities and has an equal sex distribution.^{38–40} There are three forms of CH, further defined in Table 187.3.

Rapidly involuting CH changes quickly after birth, with one-half the lesions completely regressed by 7 months of age and the others by 14 months. Rapidly involuting lesions are noted in the limbs or head and neck in 40% to 50% of cases and the trunk in 6% (Fig. 187.4).^{38,39} Involution may result in atrophic skin and little underlying subcutaneous adipose tissue, in contrast to regression of IH.

CH remains a clinical diagnosis, although to the clinician unfamiliar with these lesions, CH can be difficult to diagnose. Diagnostic criteria include a purpuric lesion fully formed at birth, frequently with a halo around the lesion, with high flow noted on ultrasound imaging. Essential to the diagnosis is observation of stability or decrease in size over time and, importantly, these lesions do not enlarge unless there is hemorrhage into the tumor. Ultrasound and MRI findings are similar to those of IH in that lesions demonstrate fast flow, shunting, and enhancement. Histopathologically, CH is differentiated from IH because CH does not immunostain for GLUT1.^{26,41} Somatic activating

TABLE 187.2 Guidelines for Propranolol Treatment for IH

Initiation of Treatment	Treatment should be undertaken in consultation with a pediatric vascular anomaly specialist with expertise in the diagnosis and treatment of pediatric vascular tumors and in the use of propranolol in children. An expert consensus panel suggested that hospitalization for initiation of oral propranolol be considered in the following circumstances: <ul style="list-style-type: none"> Infant aged 4 weeks or younger (corrected for gestational age). Infant of any age with inadequate social support. Infant of any age with comorbid conditions affecting the cardiovascular or respiratory system, including symptomatic airway IH. Infant of any age with conditions affecting blood glucose maintenance. Pretreatment evaluation should include: <ul style="list-style-type: none"> History, with focus on cardiovascular and respiratory abnormalities and family history of heart block or arrhythmia. Physical examination (include cardiac and pulmonary assessment and measurement of heart rate). No need for echocardiogram or electrocardiogram for standard-risk patients. Electrocardiogram should be considered in children with heart rate lower than normal for age and history of arrhythmia or arrhythmia detected during examination. Family history of congenital heart disease or maternal history of connective tissue disease.
Dosing	<ul style="list-style-type: none"> 1 mg/kg per day to 3 mg/kg per day divided into 2 or 3 doses; starting dose varies depending on risk factors and location of initiation. Outpatients and inpatients are initially started at a dose of 0.5 mg/kg per day to 1 mg/kg per day and increased over time. Initially, dosing of 3 times per day is recommended for infants younger than 5 weeks and for patients with PHACE syndrome.
Monitoring	Oral propranolol peaks at 1 to 3 h after administration and most centers measure heart rate and blood pressure 1 and 2 h after each dose with initiation and then when the dose is increased by at least 0.5 mg/kg per day. Parent and patient education includes: when to hold the medication, signs of hypoglycemia, feeding necessity through the night, and when to call the physician with issues, such as illness, that may interfere with oral intake or lead to dehydration or respiratory problems.
Contraindications	Sinus bradycardia, hypotension, heart block greater than first degree, heart failure, asthma, hypersensitivity, PHACE syndrome. (<i>PHACE syndrome with CNS arterial disease and/or coarctation of the aorta may be a relative contraindication.</i>)
Adverse Effects	Hypoglycemia, hypotension, bradycardia, sleep disturbance, diarrhea/constipation, cold extremities. A retrospective review of 1260 children with IH treated with propranolol revealed a 2.1% rate of adverse effects that required discontinuation of treatment. ⁷¹
Duration of Treatment	While no consensus guidelines exist for the treatment duration of propranolol, a prospective, multi-institutional study that assessed efficacy and safety of propranolol in high-risk patients for treatment identified that treatment administered for a minimum of 6 months to 12 months increased treatment efficacy. ⁷³

mutations of GNAQ and GNA11 have been associated with CH.⁴²

Large CH may exhibit high-flow cardiac output. Additionally, thrombocytopenia has been reported with a large, rapidly involuting CH, but that association is not Kasabach–Merritt phenomenon (KMP; see later).⁴¹ A solitary hepatic hemangioma in an infant represents a rapidly involuting CH. The lesions are discovered incidentally on prenatal or antenatal ultrasonography, and there is no association with cutaneous hemangiomas. They do not stain positive for GLUT1, a marker for IH.²⁶ Noninvolving CH persist long term, as the name implies, and involve the head or neck in 43% of cases, the extremities in 38%, or the torso in 19%.⁴⁰

Most CH are managed initially by observation. A RICH is regressed by 1 year of age. Reconstruction with dermal, fat, or acellular dermis grafts is considered for atrophic scarring, best done before 4 years of age. Rarely, large lesions that cause high-output cardiac failure are treated by embolization or excision.

NICH is a stable lesion and does not respond to pharmacologic treatment. These lesions typically require resection.²⁹

Spindle Cell Hemangioma

Spindle cell hemangiomas (SCH), previously called spindle cell hemangioendotheliomas, often occur as superficial, painful lesions involving the distal extremities in children and adults.^{1,43,44} These tumors appear as red-brown or bluish lesions that can begin as a single nodule and develop into multifocal painful lesions over years. The lesions can be seen in Maffucci syndrome associated with IDH1 or IDH2 mutations (cutaneous spindle cell hemangiomas occurring with cartilaginous tumors, enchondromas) and Klippel–Trénaunay syndrome (capillary/lymphatic/venous malformations), generalized lymphatic anomalies, lymphedema, and organized thrombus.^{45,46} These tumors, a significant percentage of which are completely intravascular, are well circumscribed, occasionally contain

TABLE 187.3 Three Forms of Congenital Hemangioma¹

Rapidly involuting congenital hemangiomas (RICH)	Large high-flow lesions that are completely formed at birth but rapidly involute by 12–15 months. They can ulcerate and bleed, and can cause transient heart failure and mild coagulopathy. After involution, usually some residual changes in the skin are present. ^{74,75} An increased risk of bleeding associated with venous lakes and venous ectasia. Infants with RICH should be evaluated with ultrasonography and monitored closely if these high-risk features are noted. ⁷⁶
Partial involuting congenital hemangiomas (PICH)	These lesions are completely formed at birth and involute only partially.
Noninvoluting congenital hemangiomas (NICH)	These lesions are formed at birth and never involute. Depending on the location of the lesions and whether they cause functional impairment, the lesions may be excised surgically.

**Figure 187.4** Progressive Regression of a Rapidly Involuting Congenital Hemangioma of an Extremity.

phleboliths, and consist of cavernous blood spaces alternating with areas of nodular spindle cell proliferation. Surgical resection is usually curative, with a risk of recurrence.

Epithelioid Hemangioma

Epithelioid hemangiomas (EH) are benign, typically superficial vascular tumors, that can occur in other areas (i.e., bone) with focal and multifocal lesions.^{1,47} EH may be a reactive process, as they can be associated with local trauma and can develop in pregnancy. Patients typically present with local swelling and pain at the involved site. In the bone, they present as well-defined lytic lesions that involve the metaphysis and diaphysis of long bones, and can have a mixed lytic and sclerotic pattern of bone destruction.⁴⁸

**Figure 187.5** Typical Appearance of Pyogenic Granuloma as a Solitary Red Papule, Which Often has a Stalk.

Histopathology reveals small caliber capillaries with eosinophilic, vacuolated cytoplasm and large oval, grooved, and lobulated nuclei. The endothelial cells lack cellular atypia and mitotic activity. EH is associated with FOS gene variants, and this genetic abnormality can be helpful in distinguishing epithelioid hemangiomas from other malignant epithelioid vascular tumors.⁴⁹ Various modalities of treatment have been described to include surgery and curettage, endovascular embolization, sclerotherapy, cryoablation, radiation therapy and medical management (including treatment with sirolimus and interferon).^{48,50}

Pyogenic Granuloma

Pyogenic granuloma, also known as lobular capillary hemangioma, is a solitary red papule that averages 6.5 mm in diameter, rapidly grows, has a stalk, and is complicated by bleeding and ulceration (Fig. 187.5).^{51,52} Sex distribution is 2:1 male to female, with mean age at onset of approximately 7 years. The lesion rarely occurs during the first year of life, and the prevalence is inversely correlated with age. Approximately 40% occur at age 5 years or less, another 30% between 5 and 10 years of age, and less than 25% after age 10. Nearly 90% of lesions are cutaneous, with mucous membrane involvement in almost 12%. Distribution is head or neck in 62% of cases, trunk in 19%, upper extremity in 13%, and lower extremity in 5%. Head and neck lesions involve the cheek, oral cavity, scalp, forehead, eyelid, or lips, in decreasing order of frequency.⁵² Pyogenic granuloma may be induced by trauma, insect bite, viral infection, or dermatologic disorder in 25% of patients.

Pyogenic granuloma is suggested by its appearance, location, and association with trauma or other factors. Microscopic analysis shows an exophytic mass attached to a narrow stalk on low-power examination and a normal number of mast cells, in contrast to a proliferating IH. The superficial part of the tumor shows immature capillaries with interspersed fibroblastic tissue, similar to granulation tissue, and edema in the matrix. The deeper segment has proliferating capillaries arranged in a lobular pattern extending into the deep dermis, and a dense, fibrous stroma.²⁶

Pyogenic granuloma has been treated by curettage, electrocautery, shave excision, laser therapy, or full-thickness excision. Of these, the most common treatment is shave excision followed by cautery. Incomplete treatment is fraught with re-growth of the lesion.^{52,53} Topical beta-blockers have been used to treat facial lesions in children.⁵³

INTERMEDIATE TUMORS (LOCALLY AGGRESSIVE)

Kaposiform Hemangioendothelioma and Tufted Angioma

This rare vascular neoplasm has prevalence in infancy of 1 per 100,000, is reddish purple with an ill-defined border, and



Figure 187.6 Kaposiform Hemangioendothelioma of the Extremity.

may be mistaken for ecchymosis on first glance (Fig. 187.6). Sixty percent of KHEs manifest in the neonatal period, and more than 90% are evident by infancy.⁵⁴ The tumor is usually greater than 5 cm in diameter, larger than the typical IH, and there may be hair noted in the involved skin, which tends to be a transient phenomenon. KHE has an equal sex distribution, is solitary, and affects the head or neck in 40% of children and the trunk or extremity in 30% each.⁵⁵ The lesion is locally invasive but does not metastasize. Multifocal cases are rare but should prompt biopsy when suspected. The natural history of this tumor is expansion in infancy and early childhood and then partial regression. Fibrosis occurs and may cause chronic pain and contractures. Many affected children exhibit KMP.⁵⁶ The syndrome is more common in children with large tumors and in those with visceral and muscle involvement.⁵⁷ KHE is pathologically similar to tufted angioma, the latter considered by some authors to be a milder form of this tumor.⁵⁸ Profound thrombocytopenia (<25,000/mm³) and consumptive coagulopathy in this condition may prove life-threatening in 12% to 24% of children.^{56,57} An adult variant occurs and is male predominant, smaller in size but with similar distribution as children, and without risk of KMP.⁵⁹

KHE can be diagnosed by history and physical examination, but biopsy is often used to confirm diagnosis. MRI is used to confirm diagnosis and determine extent of disease. Lesions are infiltrative and hyperintense on T2 images and enhance with contrast. Subcutaneous stranding and ill-defined borders are common, and prominent vessels with hemosiderin cause signal voids.²⁵ Histopathology shows an infiltrating lesion with lobules of spindled lymphatic endothelial cells, some of which may be dilated or have a glomeruloid appearance. Vessels contain thrombi and hemosiderin, and mitoses are uncommon. Tumors will immunostain positive for lymphatic markers D240 and PROX1.²⁶ High serum levels of angiopoietin-2 (Ang-2) have been found in high-risk patients with KHE and kaposiform lymphangiomatosis.⁶⁰ Ang-2

levels have also been noted to decrease in response to therapy with sirolimus.

Treatment should be individualized considering tumor size, location, presence of symptoms, and severity of coagulopathy. Observation is reasonable for small, asymptomatic and low-risk tumors as spontaneous regression and stability has been described. Uncomplicated and localized KHE and tufted angioma can be treated with surgical excision, pulsed dye laser, or topical agents (i.e., steroids, sirolimus, or tacrolimus).^{61–63} Surgical excision may be possible for lesions that have failed medical management or are life-threatening, while embolization may serve as a temporizing measure.

Patients with KMP and/or functional compromise with symptoms require more aggressive therapy, with guidelines issues by an American and Canadian multidisciplinary expert panel.⁶⁴ The most common treatment option has traditionally been steroid therapy with or without vincristine or other agents; however, many institutions are now using the mTOR inhibitor sirolimus, with or without steroid therapy, as primary treatment for high-risk patients.^{65–70} Steroid therapy has not shown to be effective as a single agent for complicated KHE, even at high doses, as patients treated with steroid therapy have a response rate of only 10% to 20% with a significant number of side effects. Steroid therapy is currently being used in combination therapy with vincristine or sirolimus.^{1,71} Even with therapy, these lesions do not fully regress and can recur; worsened symptomatology (pain, inflammation) can occur

with age, especially around the time of puberty, and long-term effects may include chronic pain, lymphedema, heart failure, and orthopedic issues.⁷²

SECONDARY TUMORS

Full review of the types, diagnosis, imaging studies, and management strategies for infants or children with secondary tumors that may have vascular involvement is beyond the scope of this chapter. The general precepts of management, including multidisciplinary assessment, are similar to those in [Chapter 194](#) (Vascular Tumors and their Management). In short, tumor type, location, and extent influence therapy and surgical approach. Even though segmental resection and replacement of major arteries and veins is uncommon, proximal and distal vascular control is important, should vessel injury and bleeding occur in the course of dissection. Autogenous patches or conduits are preferred, except in some cases of aortic or caval replacement. Interrupted sutures are necessary when an interposition or bypass graft is placed, to accommodate growth of the child. If visceral, renal, or supra-aortic trunk grafts are necessary, hypogastric artery is generally preferred over saphenous vein because of long-term aneurysmal degeneration in the latter. This caveat is similar to how pediatric aortic branch vessel reconstructions are done for occlusive or aneurysmal disease. [Figures 187.7 and 187.8](#) highlight illustrative cases in which vascular surgeon involvement was needed.

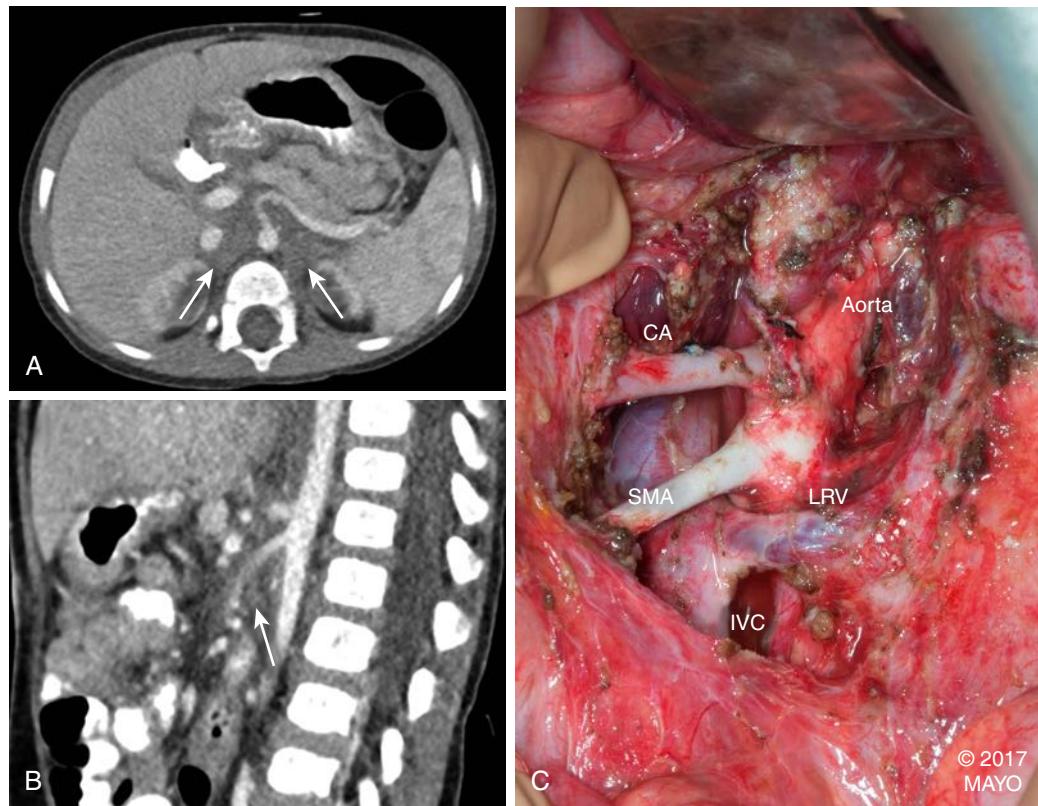


Figure 187.7 Infant with a neuroblastoma surrounding the paravisceral aorta, celiac, superior mesenteric, and left renal arteries and left renal vein, as shown by the arrows on axial and coronal CT images (A, B). The tumor could be separated from the aorta, branch arteries, and renal vein (C). CA, celiac artery; CT, computed tomography; IVC, inferior vena cava; LRV, left renal vein; SMA, superior mesenteric artery.

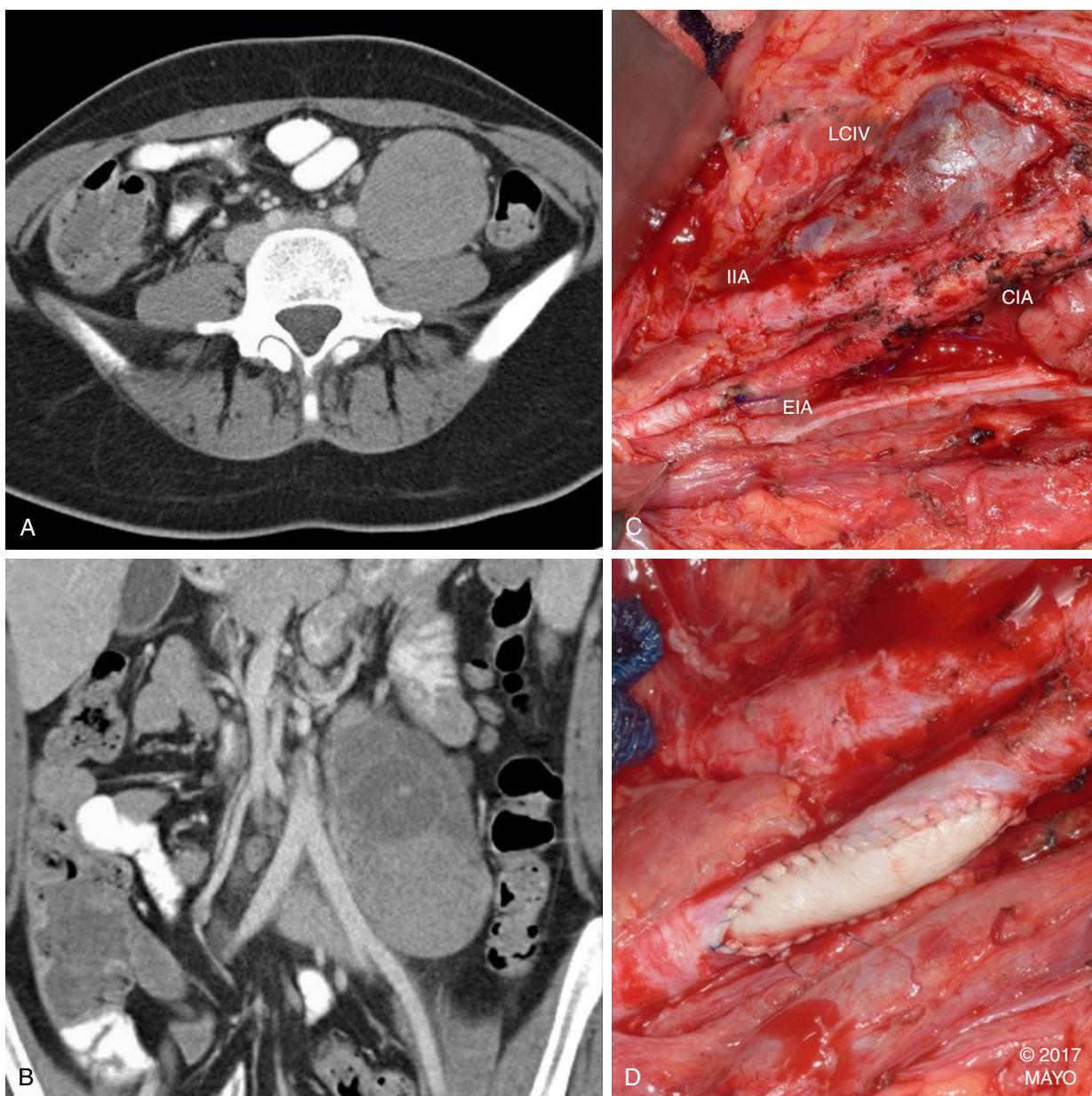


Figure 187.8 Large, lobulated left pelvic neuroblastoma abutting iliac vessels as shown on axial (A) and coronal (B) CT images. The tumor was resected but was adherent to the external iliac artery, which required a bovine patch for reconstruction (C, D). CIA, common iliac artery; CT, computed tomography; EIA, external iliac artery; IIA, internal iliac artery; LCIV, left common iliac vein.

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A complete reference list can be found online at www.expertconsult.com

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Vascular Trauma in the Pediatric Population

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INTRODUCTION

Pediatric trauma is the leading cause of death in children older than one year in the United States.¹ Although vascular injuries are infrequent, occurring in 0.6% to 1% of trauma patients,^{2,3} they constitute an important cause of mortality after trauma. Efforts to curb death and morbidity after automobile accidents, such as wearing seat belts and using car seats, may have mitigated the mortality due to pediatric vascular injuries⁴ but other studies suggest that the rate of vascular injuries has not been significantly affected.^{3,5} Likely, the most common cause of pediatric vascular injury is iatrogenic injury after peripheral interventions. Outside of the United States and Europe, warfare which does not discriminate civilians from combatants has also resulted in significant vascular injuries among noncombatants, many of whom are, unfortunately, young children.^{6,7}

The spectrum of treatment choices for vascular injuries includes expectant therapy, vessel ligation, direct vessel repair, or interposition graft repair. In adult patients, both endovascular and open vascular options are available. It is difficult to standardize the management of pediatric patients with vascular injuries due to a diverse set of factors. First, these vascular injuries are rare, occurring in less than 0.6% of pediatric patients who present with trauma.² Additionally, many different specialties participate in repair of these injuries ranging from pediatric

surgeons, adult trauma surgeons, vascular surgeons, orthopedic surgeons, neurosurgeons and plastic surgeons.³ Each specialty brings their unique perspective and background in how to deal with these injuries, which makes arriving at a consensus difficult. Furthermore, pediatric vascular injuries are far more technically challenging to treat, and this adds significant complexity to the choice of treatment for these injuries. The unique issues with these patients include size (diameter) of the injured vessel, spasticity of young vessels, and the choice of treatment must accommodate ongoing axial growth in this pediatric population. It is possible to apply the same principles and techniques of adult trauma to the older child, but the younger child may require different approaches. For example, in pediatric patients with blunt vascular injuries, historically, definitive arterial reconstruction has not always been the preferred management approach. Ligation or expectant therapy (systemic heparin without repair) was the most common choice of treatment. Adverse outcomes, such as loss of axial growth leading to debilitating gait disturbances, limb overgrowth due to traumatic arteriovenous fistula (AVF), and amputation have prompted calls for a more aggressive approach in management of pediatric extremity vascular injuries.²

This chapter is structured to describe the multiple components associated with pediatric vascular injuries including epidemiology, diagnosis, treatment, and outcomes of these injuries.

EPIDEMIOLOGY

The reported incidence of any vascular injury among patients younger than 16 years is 0.6% in the US National Trauma Data Bank.² This, however, does not account for iatrogenic injuries that are sustained during instrumentation for vascular or cardiac procedures (Fig. 188.1). Although the exact prevalence of iatrogenic injuries is unknown, since these occur during hospitalization for admissions other than trauma, few case series have evaluated their experience with these types of injuries.^{8–11} In tertiary pediatric hospitals, iatrogenic vascular injuries range from 33%⁹ to 100%⁸ of an institution's experience with pediatric vascular trauma. These penetrating vascular injuries can occur from misadventures during intravenous (IV) insertion, arterial puncture for blood gas analysis, transfemoral diagnostic or intervention procedures, umbilical artery catheterization or during surgery (e.g., during cardiopulmonary bypass operations or during orthopedic procedures). It is *estimated* that half of pediatric vascular injuries across all ages are iatrogenic penetrating injuries. The prevalence of iatrogenic injuries decreases with advancing age.¹² Neonates have the highest frequency of iatrogenic vascular injuries which then declines in the 2- to 6-year-old range (50% iatrogenic), followed by those children over 6 (33% iatrogenic).¹³ Lin and colleagues identified four factors in their series of patients with iatrogenic injuries that were associated with increased risk of iatrogenic femoral complications.¹² These included: age younger than 3 years, type of therapeutic intervention, ≥3 earlier catheterizations and the use of a 6-F or larger guide catheter.¹² Even with heparinization and use of appropriately sized catheters, the thrombosis rate

ranges from 1% to 25%.^{12,14} Both artery and vein accessed can be thrombosed after intervention. In a report of neonatal injuries after instrumentation, for example, two of seven patients presented with phlegmasia cerulea dolens who were interestingly successfully treated by leech therapy.¹⁵

Other causes of vascular trauma become more prevalent as a child's age increases and, as noted above, more than two-thirds of vascular injuries in children over the age of 6 are due to noniatrogenic causes.¹⁶ Mechanism of pediatric vascular injuries differs in and out of war zones. In a review of the National Trauma Data Bank, the most common cause of trauma in children was motor vehicle accidents.^{2,4} Other causes of trauma in decreasing order of frequency included firearm injuries, stab wounds, and falls.^{2,4} Blunt injuries constituted the majority of injuries in this study of the US National Trauma Registry^{2,4} which was similar to Swedish registry data.⁵ In most large contemporary single institutional studies, penetrating causes were the most frequent causes of noniatrogenic vascular injuries in children over 6 years of age.^{4,10,17,18} It is observed that pediatric patients with blunt injury had an overall higher injury severity score than those presenting with penetrating injuries.¹⁰ Needless to say in areas of the world in which children are exposed to warfare, these unfortunate children are more prone to sustain penetrating vascular injuries due to high-energy gunshot wounds and IEDs with complex wounds and combined bony and tissues injuries.⁶

Anatomic location of injuries is somewhat dependent on the mechanism of injury. Overall, however, the most common site of noniatrogenic vascular injuries in pediatric population is upper extremity vessels.^{2–6,10,13,17,18} Penetrating upper

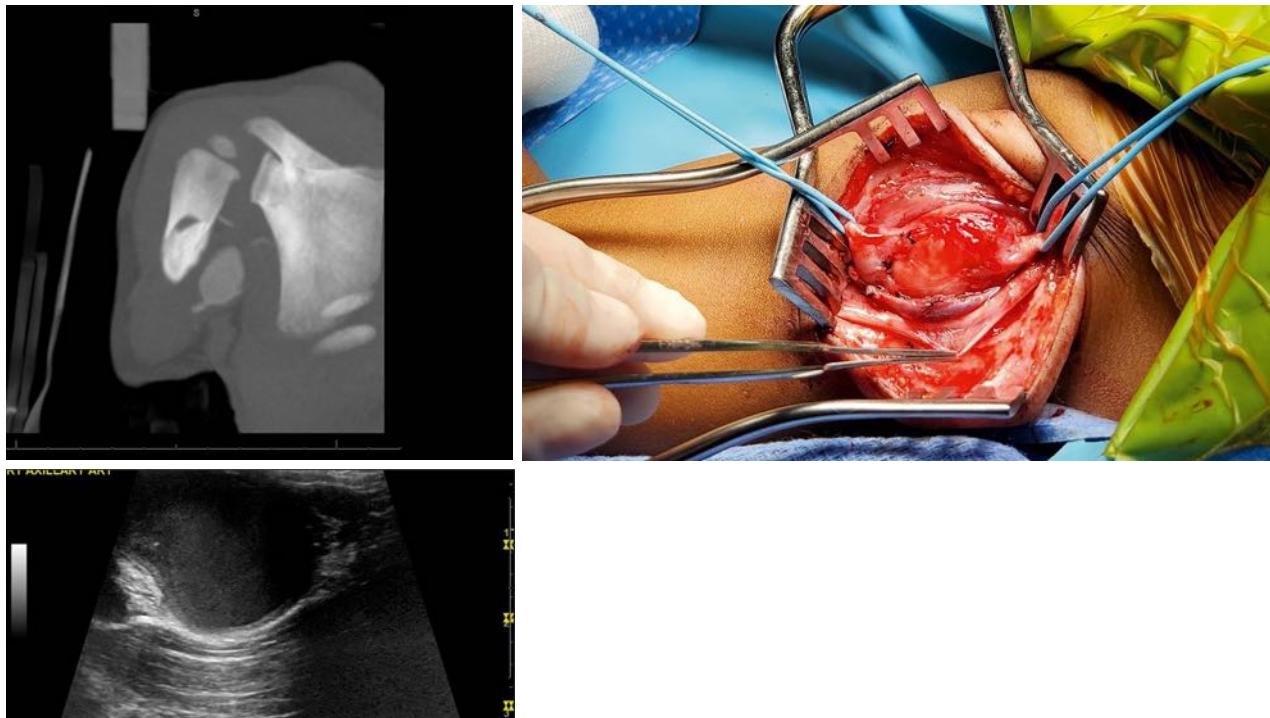


Figure 188.1 Iatrogenic injury in a 7-month-old child who had multiple lines and developed a large brachial pseudoaneurysm that was resected with end-to-end anastomosis of the brachial artery. (Courtesy Dr. Lindsey Haga, UPMC Vascular Surgery.)

extremity injuries are significantly more common than the truncal vessels, which include abdominal, thoracic, and cervical vessels. Among patients with blunt mechanism of injury, upper extremity vessel injuries constitutes a third of vascular injuries often combined with an orthopedic trauma.^{2,4} In patients with noniatrogenic injuries, penetrating injuries are significantly more common in the vessels of the upper extremity than other vessels.^{2-6,10,13,17,18} Neck vascular and truncal artery injuries, however, carry the highest mortality rates, in excess of 50%.^{4,19,20} Most of these patients sustain concomitant significant organ injuries^{4,19} and these patients have significantly higher injury severity scores (ISS) than patients with other injuries (e.g., 45.4 ± 19.8 vs. all injured patients 12.3 ± 11.7 in one study¹⁰). In one study, truncal injuries significantly increase the odds of mortality independent of all other confounders.¹⁰ In a contemporary NTDB study, site of injury was not an independent statistically significant factor of mortality in multivariate analysis although the odds of mortality increased ($P = \text{NS}$) with neck and truncal vascular injuries.⁴ However in this study, ISS score, Glasgow coma scale of less than 9 and shock (defined as SBP <90) were all independent predictors of mortality.⁴

It should be noted that venous injuries are less common than arterial injuries and more difficult to diagnose in pediatric population. For that reason, there are few studies on the natural history of these injuries and consequently not too many scientifically proven guidelines on how to manage this type of injury. It is noted that combined arterial and venous injuries significantly increase the odds of mortality. In most contemporary studies of pediatric vascular injuries, the most common site of observed venous injury was the IVC^{2,10,21} followed by other truncal vessels, such as cervical veins,¹⁹ portal veins,²² and iliac veins.²⁰ The most commonly injured extremity vein is the femoral vein.²¹ In one study of injuries to the truncal vessels, Hamner et al. noted a mortality rate of 67% among patients with IVC injury²² making these injuries significantly morbid.

ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS

Anatomical landmarks used to access arteries and veins in adults for insertion of large-bore IV catheters or arterial access are not as helpful in children, and this is one of the major contributing factors to iatrogenic injuries in children. In children, for example, there are reports of 12%²³ to 33%²⁴ minor overlap of femoral vein and artery which can lead to inadvertent arterial injury during venous access obtained without ultrasound guidance. Use of ultrasound during peripheral intervention has shown to significantly decrease complications after interventional procedures in adults,²⁵ but the effects of routine use of ultrasound on preventing these injuries among the pediatric population merit further exploration.

Another significant contributor to the iatrogenic injuries is the size of vessels used for pediatric interventions. Using ultrasound, Warkentine et al. showed a significant correlation between size of the femoral artery and child's weight and body

surface area.²³ In a large study of pediatric patients undergoing cardiac catheterization, the authors developed a risk scoring system for the prediction of serious adverse events (SAE) among these pediatric patients.²⁶ In this model, age <30 days and weight <2.5 kg, and procedures that required endovascular intervention (i.e., required larger sheaths) were highly predictive of SAEs that included vascular injuries.²⁶ These findings correlate well with the observation by Lin et al. noting a high correlation of vascular injuries with the use of larger (>6 F) sheath.¹² The effects of sheath size and complications afterward in pediatric population may be due to significant spasm encountered in these arteries after catheterization. In an older study, Mortensen has demonstrated complete arterial occlusion due to arterial spasm around a catheter in children.²⁷ It was observed by Franken and colleagues that size of catheter in relationship to the size of artery can significantly affect arterial spasm.²⁸ Franken and colleagues observed that when either catheter diameter was greater than 50% of arterial diameter or when catheter diameter was less than 1.9 mm smaller than arterial diameter significant spasm was observed.²⁸ The smaller size of pediatric vessels and highly spastic nature of these vessels can lead to spontaneous thrombosis and severe persistent vasospasm that may last for hours.²⁸ Other physiologic factors that contribute to arterial thrombosis in this patient population are related to the low intravascular volume and other medical conditions that exist in these children. Children requiring cardiac catheterization often suffer from low cardiac output that can also exacerbate thrombosis by relatively low flow to the distal tissue beds.

Iatrogenic and traumatic vascular injuries may lead to disruption, obstruction, or spasm of injured vessels. Obstruction of the vessel will lead to occlusion of the vessel and downstream ischemic symptoms that require urgent attention and restoration of flow. When vasospasm is suspected, conservative therapy after removal of the offending agents (e.g., a catheter) may be adequate to alleviate vasospasm and restore arterial flow. The most common pediatric vascular injury is blunt brachial artery injury after supracondylar fracture. Arterial spasm after orthopedic injury may lead to ischemic arm after this injury. These injuries are often self-limiting and improve after orthopedic repair, and unless the arm is significantly ischemic, observation and conservative therapy may be equally effective as surgery (see later).^{11,29} Adjuncts, such as use of intra-arterial vasodilators or papaverine, may reverse spasm and restore blood flow if spasm does not improve. Warming the patients with injured lower extremity arteries may also reverse arterial spasm.

Unique to vascular injuries is the creation of traumatic AVFs due to iatrogenic or other traumatic causes. Since these fistulae are often asymptomatic initially, they may go undetected for years. These injuries can then lead to high-flow cardiac failure by gradual enlargement of the fistula communication and subsequent increase in demand for cardiac output. Flow disturbances due to these fistulae can lead to both high-pressure venous³⁰ and ischemic arterial ulcers³¹ in the affected limb. Additionally, in certain cases, high flow to the affected extremity may lead to limb overgrowth. Once diagnosed, the majority of these AVFs can be ligated or coiled.

DIAGNOSTIC EVALUATION

Diagnosis of penetrating vascular injuries is easier since these patients present with hard signs of vascular injuries. Hard signs, including external bleeding and/or expanding hematoma, are reliable indicators of vascular injuries. When these signs are absent, the diagnosis of pediatric vascular injuries requires a high index of clinical suspicion and careful vascular examination. As noted above the majority of noniatrogenic traumatic pediatric vascular injuries in countries at peace are due to blunt mechanisms.^{2,4,5} Without evident hard signs of vascular injuries in many patients with blunt arterial injuries, the diagnosis of pediatric vascular injuries can be difficult and requires careful vascular exam and radiographic studies. As in adults, examination of the affected and contralateral extremity includes skin color, capillary refill, and pulse examination even in the absence of actual vascular injuries, because, as noted previously, pediatric vessels are highly reactive and can go in spasm due to trauma leading to reduced or absent pulses. In one series of noniatrogenic traumatic injuries, Myers and colleagues noted 26% of intraoperative spasm as the cause of low pulse exam in pediatric patients.¹⁷ Additionally, combined orthopedic and arterial injuries, a very common occurrence in pediatric population, can lead to significant arterial spasm which can often be corrected after such injuries have stabilized.¹⁸ It is therefore important in the multiply injured child with trauma, lacking hard signs of vascular injury, to address life-threatening injuries first, resuscitate and warm the child, and then re-evaluate pulses to avoid unnecessary diagnostic tests and possible operative intervention.

In the absence of hard signs of vascular injury, serial exams using continuous wave hand-held Doppler can be an effective tool. Doppler can be used to listen to the pulse if it is absent but more importantly it can be used to measure ankle-brachial index (ABI) in the injured extremity. Doppler can also be used for measurement of the injured extremity index (IEI), which like ABI, allows for the Doppler occlusion of an injured limb compared to a non-injured extremity. Katz and colleagues showed that ABI in children younger than 2 is not as reliable as in older children.³² These authors observed that ABI is less than 1.0 until the child is above 25 months of age or has attained a body surface area of greater than 0.5 m².³² For this reason, Cannon et al. recommend using IEI in children instead of ABI and recommend strong consideration of poor perfusion as due to vascular injuries or systemic causes when IEI is less than 0.9 in children older than 2 and less than 0.88 in children aged 2 and under.¹³ No direct comparisons of these two measurements are made in any study, but given the limitations of ABI in children younger than 2, IEI seems to be a more accurate objective test of ischemia due to injury. Pulse oximetry has also been shown to be effective in following patients with arterial injury, for example, in those who have blunt brachial injury due to supracondylar fracture.³³ Regardless of the technique used, if resuscitation, warming, and correction of bony injuries does not correct the malperfusion of an injured limb, diagnostic tests should be used to identify the location and severity of the arterial injury.

Duplex ultrasound can identify the location and severity of these injuries in children suspected of vascular injuries (Fig. 188.1). High specificity and sensitivity of arterial duplex suggests that Duplex should be the first investigation study in evaluating arteries and able to detect arterial injuries.³⁴ Limitation of duplex is related to the location of injury and the operator's ability to perform it. For example, duplex can be highly useful in detection of cervical carotid injuries, but it cannot assess other more proximal truncal or cephalad arterial injuries. Computed tomography (CT) angiogram (CTA) has been shown to be a very reliable study for detection of blunt truncal injuries, such as when a child has signs of direct trauma to neck, chest, or abdomen such as the seat belt sign.³⁵ CTA is often employed in patients with multiple injuries and can often detect occult truncal injuries that were missed during physical exam or not appreciated due to the location of the injury (Fig. 188.2). CTA has also become very useful in the detection of extremity arterial injuries in children regardless of the child's size (Figs. 188.1 and 188.2). When CTA is not helpful and in selected cases, angiography may be indicated. Angiography should be performed only as the last resort given the significant risks of angiography in young children. Angiography is very useful in identifying the location of arterial injury and may assist in distinguishing arterial injury from vasospasm. If diagnostic tests are not conclusive, in the setting of limb hypoperfusion, exploration of the suspected artery is indicated regardless of patient's age and size.

MANAGEMENT OF PEDIATRIC VASCULAR INJURIES

Management of the injured vessel should be coordinated with the other providers and should not jeopardize the injured child's survival. As noted previously, many tenets of adult vascular trauma are applicable to pediatric patients. Like adult vascular trauma, the principles of care related to vascular injury include exposure of injured vessel, control of hemorrhage, and restoration of blood flow to the end organ. In that regard, several adjuncts in adult vascular trauma can be translated to the pediatric patients with vascular injuries. These include the following:

1. *Use of temporary vascular shunts.* This adjunct has been quite useful in adult trauma by reducing the ischemia time and possible consequences of ischemia (e.g., nerve injury, muscle loss, compartment syndrome) while other injuries are treated. As with adult patients, a shunt should be inserted, after distal embolectomy and infusion of heparinized saline in the distal artery. Argyle shunts used for carotid surgery or a pediatric feeding tube can be used as a shunt in these smaller vessels (Fig. 188.3).
2. *Role of heparin.* Heparin is used extensively in adult vascular trauma provided there is no other contraindication to its use, such as excessive bleeding from polytrauma or documented allergy to heparin. Heparin mediates platelet aggregation and decreases the propagation of formed thrombosis and may reduce formation of thrombosis in a

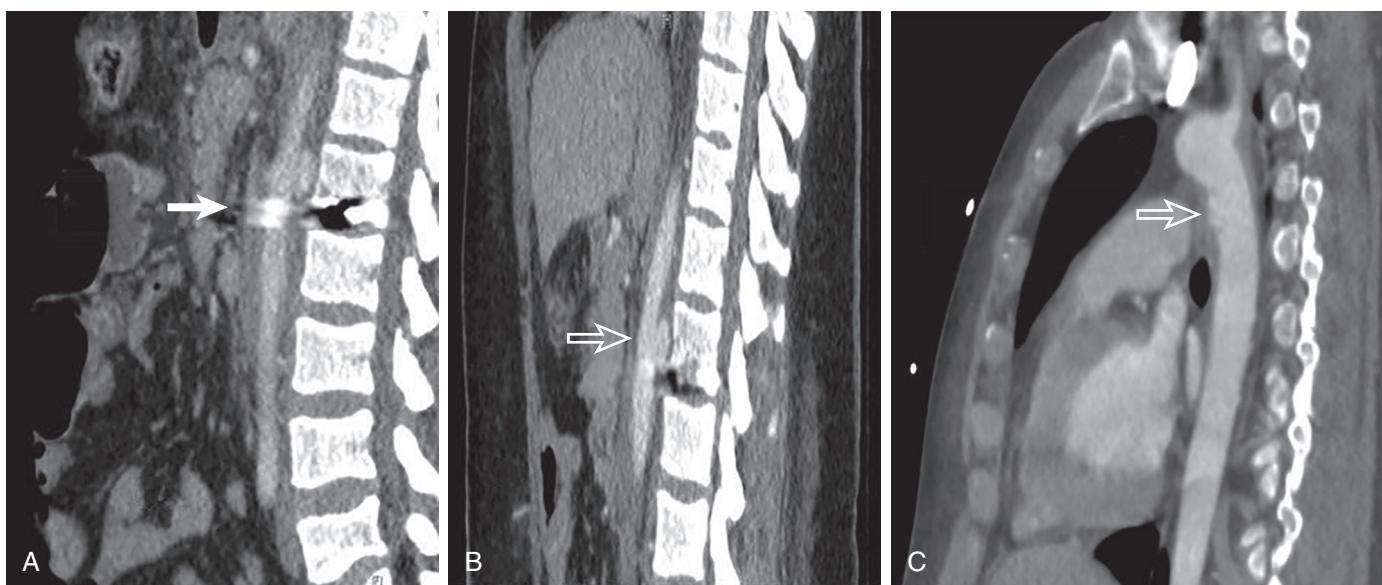


Figure 188.2 Utilization of CTA in Detecting Arterial Truncal Injuries. (A,B) CTA of a 17-year-old male shot through the abdomen. The solid arrow points to bullet trajectory whereas the open arrow points to an aortic pseudoaneurysm due to heat/blast injury. (This was not appreciated during abdominal exploration.) (C) Thoracic aorta injury (arrow) distal to the left subclavian artery in a 10-year-old male involved in a motor vehicle accident.

narrowed spastic artery. The same principle applies to pediatric patients who should receive systemic heparin if there is no contraindication to its use. Temporary use of systemic heparin may also be used in cases of pediatric vascular injury where vasospasm has been a complicating factor, reducing distal outflow.

3. *Nonoperative management of arterial injuries.* In adults, minimal arterial disruption observed in patients with arterial injury is often managed nonoperatively. This may be the case of both blunt and minor penetrating vascular injuries identified during the work-up for vascular injury with CTA, angiogram, or duplex. Similar to adults, if the distal circulation is intact, and the injury is minor (i.e., minimal vessel wall and intimal disruption or no active bleeding), the injured child can be observed. The basis of this practice is the observation by Stain and colleagues that these intimal injuries healed without any intervention during the follow-up in adult patients.³⁶ Extrapolating from adult literature, it is reasonable to observe minor arterial injuries provided that there is intact distal circulation and the child can be observed carefully and monitored by follow-up imaging studies.
4. *Lower extremity arterial injuries in infants and young children.* Historically, early exploration of pediatric vascular injuries was avoided unless there was an evidence of ongoing exsanguinating bleeding. However, poor long-term outcomes from this conservative therapy strategy have led to more aggressive approaches with some recommending exploration even in the setting of vasospasm.¹² Although this approach may not be appropriate in all cases,²⁹ the consequences of delayed exploration and revascularization compel many surgeons to intervene and explore early to avoid these consequences. Operative management of the injured

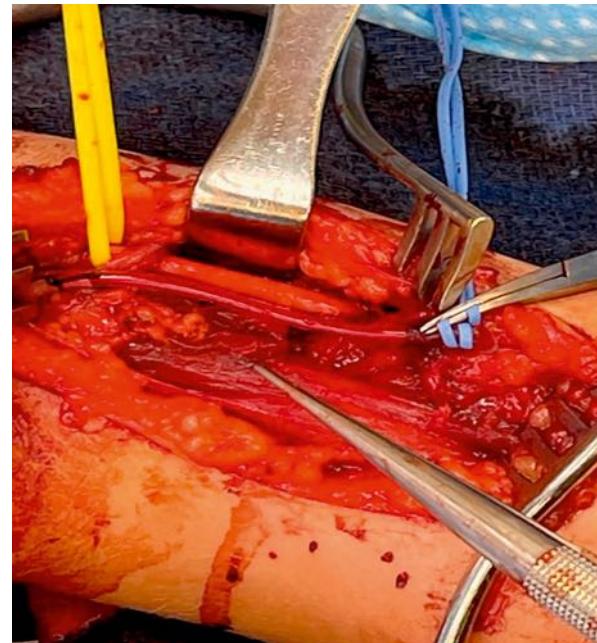


Figure 188.3 Use of pediatric feeding tube in a 5-year-old patient with severe brachial artery disruption.

vessel in children is challenging as the vessels are spastic and small, and any repair must take into consideration the need for future axial and radial growth of blood vessels as well as the long-term patency of vascular repairs.³⁷ More contemporary series similarly suggest that truncal ischemia can be tolerated in infants and children and managed with anticoagulation alone with acceptable results.^{11,38} In a case series from Michigan, the child's weight was a significant risk factor for surgical intervention in children with acute limb ischemia after injury, suggesting a delay until the child

is bigger.¹¹ In another series from Indiana University, the authors noted that in 10 out of 14 children the artery was recanalized in follow-up.³⁸ Both series indicate that there is an unknown risk of limb discrepancy or symptoms when the artery is not revascularized^{11,38} but the overall results of anticoagulation alone were acceptable. These children must be carefully observed with repeat duplex evaluations to assess distal perfusion. Duration of anticoagulation is anecdotal and depends on the quality of distal perfusion. If initial anticoagulation fails, as evidenced by progression of ischemia and/or tissue loss, surgical exploration in these children should be attempted regardless of the child's size.

5. Other adult trauma adjuncts in children. Other tenets of management of adult vascular trauma should also be applicable as needed in pediatric patients. These include liberal use of fasciotomies and use of endovascular techniques to temporize bleeding (i.e., proximal balloon occlusion) or reestablish flow to the injured extremity if injured vessel size can accommodate endovascular options.

Management of Iatrogenic Vascular Injuries

Iatrogenic injuries may lead to complete or partial occlusion of an injured vessel. When this injury leads to complete occlusion, limb hypoperfusion can set in with devastating circumstances. In adults, conservative management of arterial injuries may lead to devastating long-term consequences perhaps due to the short "ischemic threshold" time beyond which limb quality deteriorates rapidly, as shown in animal models.³⁹ In cases of arterial occlusion, arterial exploration, embolectomy, and patch angioplasty will lead to acceptable outcomes even in children younger than 2 years of age.¹²

If iatrogenic injury has not caused complete arterial occlusion, definitive repair can be delayed as long as the limb is well perfused. Small flaps and dissection often can be followed by serial exam and systemic heparinization to avoid arterial thrombosis. These patients must be monitored carefully and operated upon if the limb perfusion deteriorates. Interval repair of such injuries may also lead to acceptable outcomes,^{11,12,14,38} but immediate restoration of perfusion should be the gold standard.

Extracorporeal membrane oxygenation (ECMO) cannulation is a common cause of iatrogenic pediatric vascular injuries in tertiary pediatric centers.^{11,40,41} These injuries that occur in about 20% to 52% of ECMO patients^{41,42} can affect either femoral or carotid cannulation sites.^{11,40,42} It is interesting to note that, not surprisingly, ECMO is a significant risk factor of adverse outcome after operative intervention for acute limb ischemia.¹¹ Carotid injuries have shown to lead to cognitive disabilities, which can be avoided by carotid repair after ECMO, and this has led centers to perform routine carotid reconstruction after ECMO, with good patency and neurologic outcomes.⁴⁰

Management of Noniatrogenic Injuries

Management of noniatrogenic injuries is dependent on the location and mechanism of the injury. As noted previously most

vascular trauma after the age of 6 is related to noniatrogenic causes.¹⁶ The epidemiology of these injuries was previously discussed.

Penetrating trauma to vascular injury requires immediate attention to temper blood loss and repair injured vessels in order to save life and limb. High-energy penetrating blast injuries in urban warfare or war zone areas may require extensive soft tissue debridement, use of vascular shunts, and liberal use of fasciotomy while other injuries are addressed using a multi-specialty approach to these injuries, as reported by Peck and colleagues.⁴³ The successful experience by these surgeons operating in Iraq and Afghanistan and reporting of 12 unfortunate children who sustained combat-related injuries⁴³ lends significant support to the philosophy of early diagnosis and operative management of vascular injuries in children.

The tenets of arterial repair, applicable to adult arterial injury, apply to children with the special consideration that children's vessels are smaller, spastic and any repair or interposition graft must accommodate the axial growth of the injured vessel and limb. Similar to adult injuries, if the injury is confined to a short segment of the arterial wall, primary repair of the injured artery with interrupted nonabsorbable polypropylene sutures is the procedure of choice. Patch angioplasty may be required in cases where simple repair is not possible due to lateral wall injuries. This type of repair avoids luminal growth issues at the anastomosis site and has been used with good success in low-velocity penetrating injuries and blunt injuries.^{3,44} Patch angioplasty can be safely performed with a segment of non-injured greater saphenous vein (GSV).

Special considerations should be given to cases where interposition graft is required in children. The issues to consider are details pertinent to techniques of anastomosis creation, choice of suture material, and the type of conduit to accommodate the axial growth in children and avoid luminal narrowing at the anastomosis site. Since there is a need for these arteries to grow both axially and radially, the running-type suture technique commonly used in adults may not be ideal in the pediatric population (Fig. 188.4). This type of suturing technique, in theory, may cause purse-string narrowing of the anastomosis

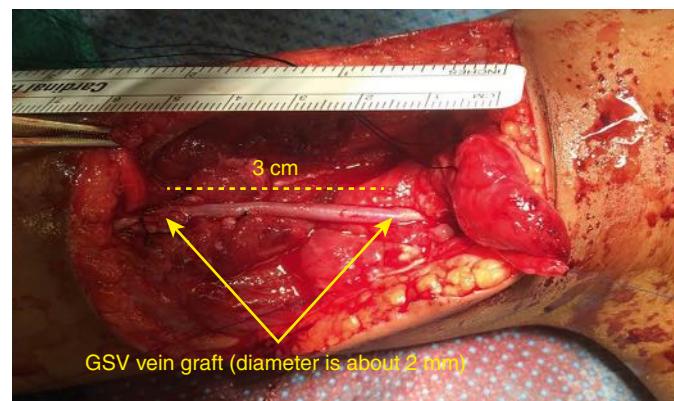


Figure 188.4 Penetrating injury to proximal brachial artery in a 7-year-old male. Repaired with an interposition graft using greater saphenous and interrupted 8-0 Prolene. GSV, greater saphenous vein. (Courtesy Dr. John Eidt.)

and may hinder the radial expansion of the artery at the site of anastomosis. This has prompted some authors to recommend interrupted sutures instead of running sutures in pediatric patients.⁴⁵ Although there are no comparative studies in pediatric patients comparing the types of anastomosis among pediatric patients, in animal studies, the use of running-type anastomoses has been shown to cause arterial narrowing in growing pigs at the site of anastomosis.⁴⁶ These authors also noted that nonabsorbable sutures were less thrombogenic than absorbable sutures.⁴⁶ These observations suggest that interrupted nonabsorbable sutures on a beveled anastomosis further enlarge the anastomotic connection between conduit and artery, and may be the best method of arterial reconstruction in pediatric patients. The GSV has shown to be a durable conduit among children who underwent vascular reconstruction.⁴⁷ Therefore like adult trauma patients, the contralateral GSV should be the conduit of choice if an interposition graft is needed. This conduit should be preferentially used when there is high blast injury to the artery, which requires debridement of artery and the surrounding tissues to provide a clean and tension-free repair. In children, these types of injuries are often encountered in combat zones and invariably are associated with other injuries. In the setting of other injuries, a shunt can be used while complex neurologic and bony fractures are addressed.⁴⁸ It should be noted that dog bites in the pediatric population could also present with both blunt and penetrating vascular injuries.⁴⁹ These devastating injuries, similar to high blast injuries, require significant tissue debridement and arterial injuries often with interposition graft.⁴⁹

Early and liberal use of lower extremity four-compartment fasciotomy in patients with lower extremity ischemia has shown to decrease limb-related adverse outcomes.⁵⁰ This is universally practiced in wartime extremity injuries and is well described

in the civilian pediatric literature.³ This should be practiced in pediatric patients with lower extremity ischemia to improve the quality of limb salvage.

Special Case of Noniatrogenic Arterial Injuries – Blunt Brachial Artery Injury

Upper extremity arterial trauma is the most common site of arterial injury in children in a recent analysis of the US National Trauma Data Bank registry.⁴ In the setting of combined arterial and orthopedic injury, treatment of arterial injury requires a team approach. The majority of combined arterial and orthopedic injuries are improved once the orthopedic fracture is reduced. Blunt brachial artery injury is a common site of pediatric arterial injury where an orthopedic injury compromises the brachial artery. It occurs in the setting of a fall onto an outstretched hand and can be associated with neurovascular injury. Blunt brachial artery is the most common type of injury in a recent publication from Sweden⁵ and is reported in 10% of children with supracondylar fracture⁵¹ with potentially devastating consequences of upper extremity amputation and Volkmann ischemic contracture. Stretch injuries to the artery, which can disrupt the vessel intima or impingement of the vessel by surrounding tissue, are the usual causes of this (Fig. 188.5). The presentation can range from normal to pulseless pink hand (PPH) to severe ischemia. After the orthopedic injury is corrected, pulses may return, obviating any necessity of intervention on the brachial artery. In patients with no pulse and evidence of ischemia after orthopedic reduction, the brachial artery must be explored and repaired *in situ* after thromboembolectomy or interposition graft with proximal GSV. Controversy exists about the best course of therapy among patients with PPH after reduction. Traditionally, PPH was managed expectantly with anticoagulation and serial exam of the



Figure 188.5 (A,B) A common orthopedic injury is supracondylar fracture. Both of these patients presented with a pulseless pink hand. Both improved after orthopedic repair. But the patient in (B) required vascular exploration, embolectomy, and patch repair. (Courtesy Dr. Timothy Ward, Dept. of Orthopedics, UPMC, Pittsburgh, PA.)

hand that appeared well perfused (pink) and arterial exploration considered *only* if the patient developed neurologic signs of ischemia.⁵² Other authors recommend arterial exploration for *every* case of PPH.⁵³ Although no prospective randomized trial exists, we suggest that there may be equipoise between observation and exploration for patients who present with isolated blunt brachial artery injury using a large administrative database.²⁹ This study was somewhat limited, however, being a retrospective analysis of an administrative database, and much clinical information beyond the index admission was not available.²⁹ These findings were confirmed in contemporary series as well. For example, in a review of 404 patients with suprcondylar fractures, 68 children were found to have vascular injuries.⁵⁴ 42 of 63 patients had immediate return of radial pulse and 18 had return of pulses a few hours to eleven days after fracture reduction.⁵⁴ It is, therefore, acceptable to manage these patients with PPH conservatively with anticoagulation after closed reduction. This approach warrants close observation for any signs of perfusion compromise and if perfusion is compromised, emergent arterial revascularization should be performed. Lack of pulses in a cold white extremity after orthopedic reduction should prompt exploration of the brachial artery and restitution of pulses with arterial repair or interposition graft.

Truncal Vascular Injuries

Similar to the adult vascular injuries, injuries to the truncal vessels in children are life-threatening and require immediate attention. The overall mortality rates of vascular injuries in US children is about 8%–13% using NTDB.^{2,4} These rates are significantly higher among patients with truncal arterial injuries.^{2,4} Barmparas et al. observed a 16% to 41% mortality rate for truncal vessels: neck 16%, chest 41%, abdomen 25%.² In a more recent study, among children who died from vascular trauma, we observed that more than 90% had isolated neck and truncal vascular injuries.⁴ In a large trauma center, Corneille et al. observed that *all* cases of mortality out of 116 children with significant vascular trauma were due to truncal arterial injuries.¹⁰ The most important predictor of outcome in pediatric patients with truncal injuries is hemodynamics at the time of presentation with other factors affecting survival, including caval injuries, anatomic location of the injury, and the mechanism of injury.^{4,19} Unlike adult patients, blunt injury to the aorta is uncommon in the pediatric population and, as this is typically accompanied with a variety of other injuries, is universally incompatible with survival.¹⁹ The use of seat belts has decreased the rate of these injuries in children.⁴ In injured pediatric patients, due to the small diameter of injured arteries, endovascular options, commonly employed in adults with great success, are very limited although the rate of endovascular treatment utilization is increasing.⁵⁵

Truncal injuries can often be diagnosed with high resolution CTA without any need for angiography (Fig. 188.2).¹⁹ Most cases of pediatric thoracic aortic injuries and great vessel trauma are managed with open surgery. Thoracic aortic injuries are treated with clamp-and-sew technique without any significant

adverse neurologic outcomes.^{19,20,55,56} Although among patients with severe truncal artery injuries who present in shock outcomes are extremely poor, using this clamp and sew technique Allison et al. reported survival rates of 81% to 92% among patients who were hemodynamically stable at presentation with *no* paraplegia.¹⁹ Similar to adults with blunt thoracic aorta, delayed repair with early initiation of beta-blocker therapy has shown a survival benefit to patients.⁵⁵ Smaller diameter thoracic stents can be used in some adolescent children with blunt thoracic artery injuries,^{57,58} but long-term results are lacking and should be used only cautiously (Fig. 188.6).

Management of abdominal vascular injuries is dictated by hemodynamic stability and the severity of associated injuries and thus ranges from suture repair of injured vessels to interposition replacement with a synthetic graft for aortic and caval injuries and use of GSV or hypogastric arteries for other intra-abdominal vascular injuries.

Mechanism of injury and anatomical location dictates the appropriate repair of cervical injuries. For lesions due to a penetrating mechanism and located in the anatomically accessible lesions (zone II), simple repair or interposition graft of injured vessel is possible. More proximal injuries (zone I) with penetrating injuries may require median sternotomy. More distal injuries (zone III) may be treated by ligation of the internal carotid artery or endovascular techniques (coiling or use of covered stent) if anatomically feasible to save the child's life. Blunt injuries to the carotid artery are more difficult to diagnose without a high-definition CTA, but they rarely benefit from surgical intervention. These patients do worse than patients with penetrating injuries,^{3,4} and treatment is often careful observation, antiplatelet therapy and serial imaging.⁵⁹ Injury to the innominate artery requires median sternotomy. This approach also allows exposure of the proximal right subclavian artery. The proximal left subclavian artery can be approached via left anterior lateral thoracotomy. Endovascular treatment of the subclavian or innominate artery can be attempted in children with appropriate size arteries (Fig. 188.7). Endovascular technique can easily accommodate arterial control with a balloon, which is gradually replacing the need for proximal control by these open techniques.

ROLE OF ENDOVASCULAR TECHNIQUES IN PEDIATRIC PATIENTS

As noted in the section on iatrogenic injuries, endovascular interventions in children can be fraught with complications. The smaller vessels in children are also not suitable for large stent graft coverage. So, despite significant use of endovascular interventions for all forms of traumatic vascular injuries in adults, endovascular intervention plays a limited albeit increasing role in pediatric population and then, mainly in adolescents. The adolescent thoracic aorta, for example, can accommodate smaller diameter thoracic stent grafts,^{58,59} but most traumatic aortic injuries in pre-teen children require open repair. Endovascular techniques can be used to control bleeding by placing a proximal balloon and therefore simplifying exposure; they



Figure 188.6 Endovascular Repair of GSW to Distal. (A) Thoracic aorta in a 14-year-old male (arrow). (B) This was successfully repaired. (C) A few months later, follow-up CT shows that the stent has migrated and covered celiac access (arrow points to the initial location of stent). (Courtesy Dr. John Eidt.)

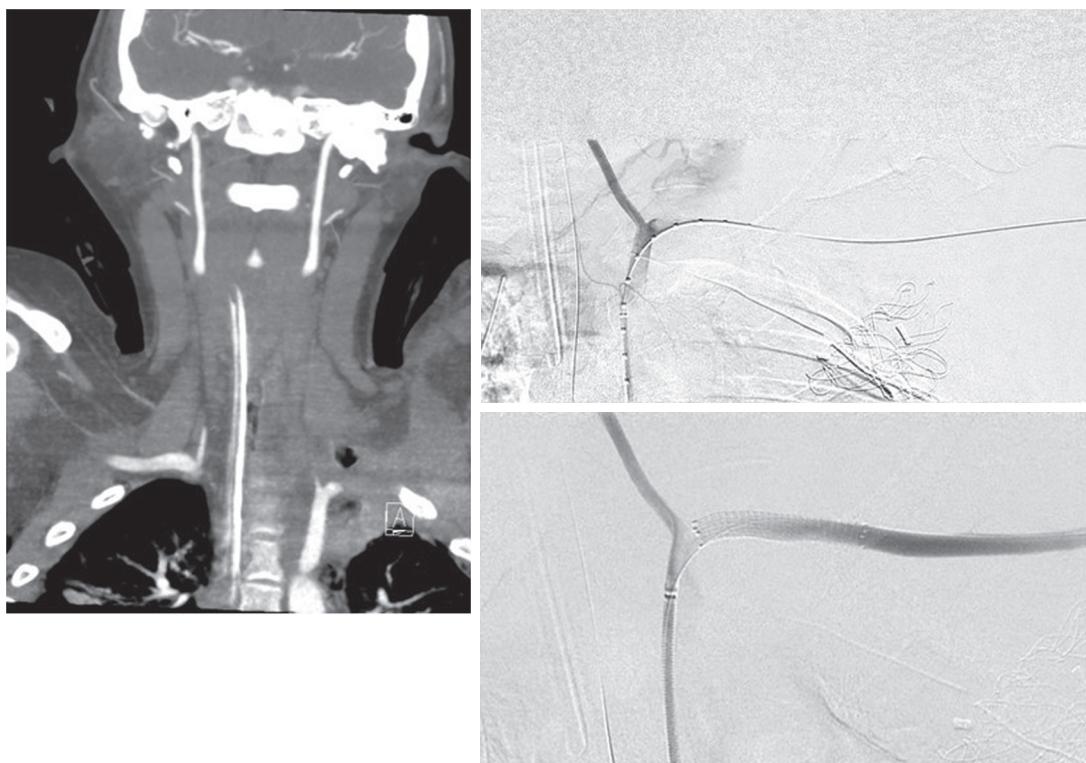


Figure 188.7 Endovascular repair of a blunt injury to the left subclavian artery in a 10-year-old female with severe multi-trauma and ischemic left arm.

can also be used for embolization of inaccessible bleeding vessels (e.g., see above injuries to zone III cervical injuries); and in some cases there are reported cases of use of covered stents to manage traumatic cerebrovascular injuries in pediatric patients (Fig. 188.7).⁶⁰ Indeed there has been an increase in the utilization of endovascular treatments in the pediatric population.⁵⁵ These authors noted that these interventions have increased by threefold from 2007 to 2015 and the majority of increase in the utilization of endovascular treatments was related to higher utilization of endovascular therapy for injuries in thoracic, abdominal/pelvis and upper extremity regions.⁵⁵ Undoubtedly with advances in endovascular technology, endovascular treatment will play a significant role in the management of children with vascular trauma.

POSTOPERATIVE MANAGEMENT AND OUTCOMES

Routine postoperative management of injured children requires collaboration with pediatric specialists who can better manage the details of care. With specific reference to traumatic vascular injuries, these patients require routine hourly vascular examination with any changes prompting immediate attention. Similar to adult trauma patients, and barring any medical contraindication, antiplatelet therapy is advisable. Although there are no clear scientific reasons for this, antiplatelet therapy may be beneficial to the injured vessel or the new anastomosis, which is often devoid of endothelium and thus prone to platelet adhesion to the injured vessels. The duration of this therapy is not scientifically proven, but it appears that a short course (30 days) is adequate. Routine use of anticoagulation is not indicated and may cause bleeding, particularly in patients with polytrauma.

Other than routine pulse exam and clinical assessment, postoperative surveillance should be tailored to the vascular repair. The issues of appropriate postoperative surveillance beyond the index hospitalization and compliance of these trauma patients is multifaceted. The compliance of trauma patients for follow-up is low,⁶¹ and since trauma care is provided with multiple services, the burden of compliance with follow-up is passed between services.⁶² Additionally no clear surveillance follow-up protocol is agreed upon. Patients with thoracic aortic repair with thoracic stent should undergo annual CTA to evaluate the stented aorta until adulthood. Other than this type of injury, routine CT or CTA is not indicated. For lower extremity revascularization, simple pulse exam or IEI can detect a failing graft in the immediate postoperative period.³ Duplex surveillance of the graft at one month postoperatively and then at routine intervals is advisable to expose early technical issues or stenosis or aneurysmal changes of the graft at later time points.

Difficulties with compliance and low number of patients with pediatric trauma makes a discussion about long-term postoperative outcomes difficult. The discussion about long-term outcomes after pediatric vascular trauma therefore centers on retrospective case series or observational database studies. In-hospital mortality rates of these patients are reported to

be 8%–13.2%,^{2,4} with 2.4% of patients dying at the time of presentation to the emergency department based on the US National Trauma Data Bank data.² Case series studies reported crude mortality rates which are similar (9.7%¹⁰ to 12%³ in two large case series). The highest mortality among the pediatric patients was among infants (18.2%) and of these, the highest mortality was among patients with chest vascular injuries.^{2,4} The overall rate of in-hospital amputations was low, but the highest amputation rates were observed among patients with brachial and popliteal artery injuries for upper and lower extremity injuries, respectively.^{2,4} Lin et al. reported the long-term results of a series of 34 patients with iatrogenic injuries.¹² In this series, patients were followed for a mean follow-up period of 38 months. No limb loss with surgical repair was reported and gait disturbance was observed among the three patients who presented with chronic limb ischemia and gait disturbance.¹²

CONCLUSIONS AND FUTURE DIRECTIONS

Pediatric vascular trauma is currently managed by different groups of specialists with different training and backgrounds. Klinkner and colleagues noted that vascular surgeons treated only 17% of pediatric vascular injuries.³ Since these events are rare, there are no clear guidelines and little algorithmic care of these patients. Most data reported here is based on evidence from animal studies, case series, and expert opinion. It is therefore imperative that in every institution, algorithmic care of these patients be developed by all the involved specialists and based on the best available data so all the involved specialties follow the same guidelines in taking care of these fragile patients. It is important that data for these cases be recorded in a registry dedicated to pediatric patients. The National Trauma Data Bank now collects demographic information on these patients and Barmparas and others have used this registry for a comprehensive review.^{2,4,55} Ideally, collaborative efforts by vascular and trauma registries to collect data on vascular injuries, outcomes, and long-term follow-up may provide the best source of information for best quality care. Additionally, the U.S. Military Medical Corps has been involved in many cases of pediatric vascular trauma since 2003. The information about their experience should be added to NTDB and any future vascular trauma registries to help translate their successful management techniques to the civilian pediatric population. Such a registry must also record the information about in-hospital neonatal vascular injuries and their outcomes. Given the significant advantages of the use of ultrasound among adult patients in reducing vascular injuries,²⁵ guidelines about use of this technology to reduce injuries in these pediatric patients should be investigated.

Despite the limitations of the data noted above, modern data emphasize aggressive operative management of vascular injuries in pediatric patients in clear contradistinction to the historical, more conservative management approach. This approach, similar to adult vascular trauma, comprises a

comprehensive team approach that includes bleeding control, vascular shunting to circumvent ischemia while other injuries are addressed, liberal use of fasciotomy for lower extremity injuries, and use of endovascular approaches as the definitive or contributive mode of treatment. Future research in the field of pediatric trauma should focus on creating endovascular approaches to both possibly mitigate the injury or ultimately treat the injury definitively. Such treatment options must accommodate the small size and expected growth of the arteries of these patients.

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Placement of Arteriovenous Dialysis Access and Central Catheters in the Pediatric Patient

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BACKGROUND

Pediatric end-stage renal disease (ESRD) is a challenge for caregivers, nephrologists, and surgeons. The number of children and adolescents beginning ESRD care is steadily decreasing from a high of 17.5 per million in 2004 to 13.8 per million in 2016, representing a decrease of 21.1%.¹ Despite the decreasing number of pediatric patients with ESRD, there is still a large burden of healthcare costs, accounting for \$194,059,769 Medicare dollars in 2016.¹ Although 25.1% of pediatric patients initiating treatment for ESRD do so with index transplantation and 18.3% initiate with peritoneal dialysis, a large majority of patients (51.2%) utilize hemodialysis (HD) as their first modality of renal replacement therapy. Currently, the National Kidney Foundation's (NKF) Kidney Disease Outcomes Quality Initiative (K-DOQI) guidelines recommend placing permanent HD access in all patients aged 0 to 19 who are greater than 20 kg and are not expected to receive a kidney transplant within 1 year.² Thus, patients expected to be on dialysis more than 1 year that meet the age and size criteria should receive a permanent arteriovenous fistula (AVF). Over 50% of pediatric patients who initiate ESRD treatment with HD meet these criteria; however, the large majority of these suitable patients commence HD treatment via central venous catheters (CVCs).

The Fistula First Breakthrough Initiative (FFBI) was established in 2003 to improve the rates of arteriovenous fistula placement for HD. Combined with the FFBI, the K-DOQI practice guidelines set a goal of 40% AVF placement for first time access patients, and this goal was exceeded by 2011 with 60% fistula use. However, the FFBI does not specifically address the low rates of fistulas in the pediatric population. To combat this problem, the International Pediatric Fistula First Initiative (IPFFI) was established in 2005 with the aim of addressing the lack of AVF use in the pediatric population. The IPFFI was a collaborative effort with the Midwest Pediatric Nephrology Consortium, whose aim was to increase awareness among pediatric providers (nephrologists, surgeons, and dialysis staff) that AVF is the preferred access method for long-term HD in pediatric patients. The initiative has influenced practice in the United States and evidence supporting fistula first placement in the pediatric population is emerging with evidence of reduced blood stream infections secondary to increased fistula placements.^{3,4}

INCIDENCE

The incidence of ESRD in the pediatric population appears to have peaked in 2003 and has since then been steadily decreasing, although compared with the early 1980s and 1990s, the

incidence curve has largely plateaued. In 2016, the incidence of ESRD in patients aged 0 to 21 years old was 1388, slightly decreased from 1619 in the year 2010.¹ In comparison, the incidence in adults 22 and older was 123,287 patients the same year. The cumulative prevalence of ESRD in pediatric patients as of December 31, 2016 was 9721 children from 10,136 in 2010. Since its initiation of data collection in 1992, the North American Pediatric Renal Trials and Collaborative Studies (NAPRTCS) reports no significant change in the pattern of incidence of ESRD in the pediatric population. When looking specifically at certain age groups, race, or gender, these rates are anticipated to remain stable.⁵

The United States Renal Data System (USRDS) reports hemodialysis as the most common index treatment for patients initiating care for ESRD; in 2016, 51.2% of pediatric patients initiated care with HD. Twenty-two percent had preemptive transplantation, and 26% started with peritoneal dialysis (PD).¹ Compared to adults, where index transplantations are rare, there is a wider distribution of index treatment modalities in the pediatric population. The index treatment for adults in 2013 was HD in 88.2%, peritoneal dialysis in 9%, and index transplantation in only 2.6% of patients. Additionally, the pediatric population has shown a trend towards higher rates of transplantation after initiating any HD treatment modality. As many as 49% of children with ESRD underwent kidney transplantation within the first 2 years of starting ESRD care from 2014 to 2016; this is a higher figure than the 2009–2013 cumulative data showing 37%. Cumulatively, there were 9619 children and adolescents under the age of 22 with ESRD as of December 31, 2016. Despite HD being the most common form of initial treatment for ESRD, the most common prevalent ESRD modality is kidney transplant (6927 or 72%), followed by HD (1651, 17.2%), and PD (1019, 10.6%) (Fig. 189.1).

ETIOLOGY

In contrast to the adult population where the predominant etiology of ESRD is diabetes and hypertension, ESRD in the pediatric and adolescent population is caused by congenital and acquired disorders. Primary glomerular disease was the leading cause of ESRD in children during 2012–2016 (22.3%), followed by congenital anomalies of the kidney/urinary tract (CAKUT) 21.9%, cystic/hereditary/congenital disorders (11.7%), and secondary glomerular disease/vasculitis (10.7%)¹ (Fig. 189.2).

The most common causes of ESRD include focal glomerular sclerosis (11.5%), renal hypoplasia/dysplasia (10.4%), congenital obstructive uropathies (9.3%), lupus nephritis (5.6%), and unspecified renal failure (7.0%). In African American patients, there is a significantly higher percentage of children with certain nephropathies related to systemic diseases. For example, African American children make up 95% of all children affected with sickle cell nephropathy. In addition, AIDS nephropathy patients in the current reporting period are 97% African American, which is an increase from 91% in previous reporting years. Among those affected by lupus nephropathy, 53% are African American.

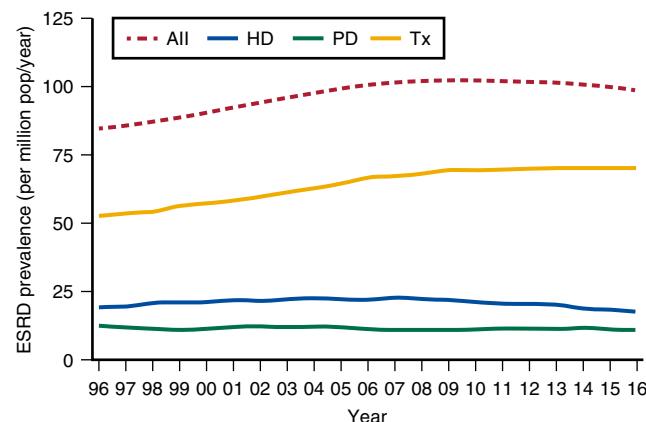


Figure 189.1 Point prevalence of ESRD among pediatric patients (0-21) per million population per year. HD, hemodialysis; PD, peritoneal dialysis; Tx, transplant. (Source: United States Renal Data System: 2018 USRDS Annual Data Report: ESRD among children. Bethesda, MD; 2018.)

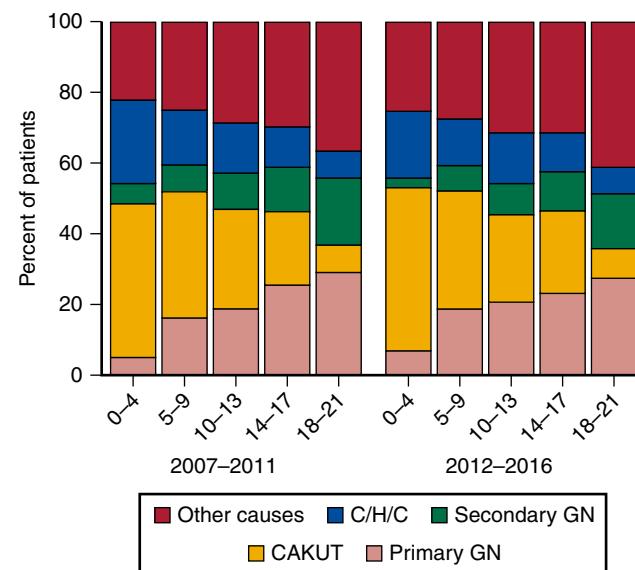


Figure 189.2 Primary reported etiology of ESRD in pediatric patients aged 0 to 21 years old, 2012–2016. CAKUT, congenital anomalies of the kidney/urinary tract; C/H/C, cystic/hereditary/congenital disorders; GN, glomerulonephritis. (Source: United States Renal Data System: 2018 USRDS Annual Data Report: ESRD among children. Bethesda, MD; 2018.)

MORBIDITY AND MORTALITY

ESRD in children, as it does in the adults, confers an increased morbidity and mortality on those affected compared to the general population. However, decreasing overall mortality rates in the pediatric population are being seen. The 2011 to 2015 one-year adjusted all-cause mortality rate was 39 per 1000 patient-years, a 20.4% decrease from 2006–2010 data of 49 per 1000 patient-years (Fig. 189.3A). Additionally, there was a 39.9% improvement in the one-year mortality in the age <2 years group. Adjusted one-year all-cause mortality rates by modality showed decreases of 16.9% among HD patients, 35.5% among PD patients, and 30.8% among transplant patients when comparing the 2006–2010 data to the 2011–2015 period (Fig. 189.3B).

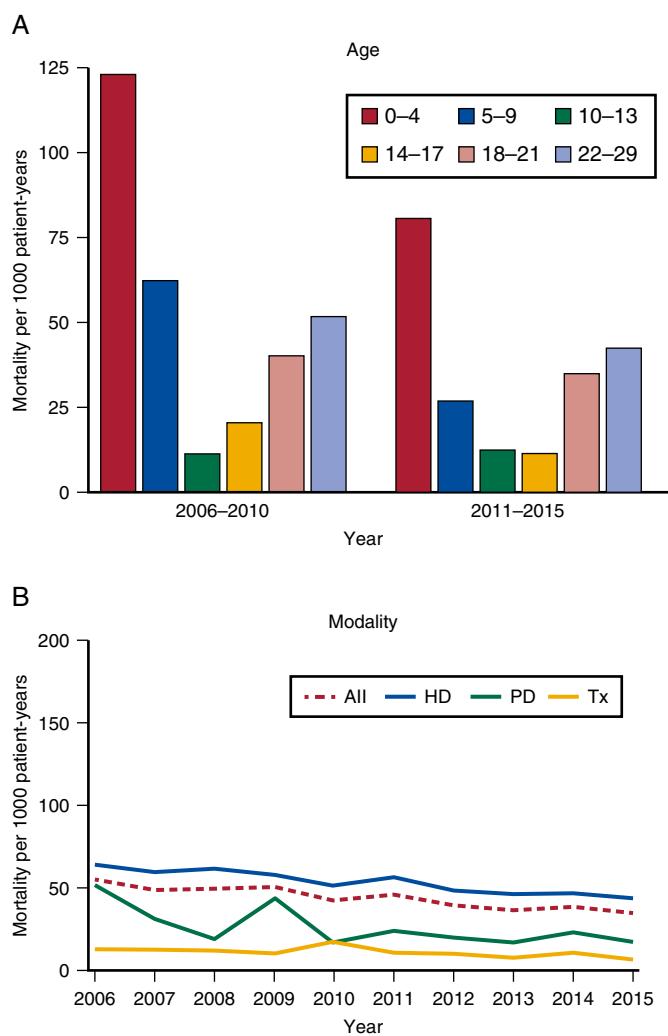


Figure 189.3 One-year adjusted all-cause mortality in incident pediatric patients with ESRD by (A) age with comparison to young adults (aged 0–29 years), 2006–2010 and 2011–2015 and (B) modality, 2006–2015 (aged 0–21 years only). HD, hemodialysis; PD, peritoneal dialysis; Tx, transplant.

Although there was overall improvement in one-year all-cause mortality from 2011–2015, there was still a difference in mortality by ESRD modality. HD- and PD-associated mortality rates were 5.4 and 2.2 times higher than for transplant patients (Table 189.1), respectively, highlighting the importance of transplantation in the pediatric population. Overall, the expected remaining lifetime in years of patients by initial ESRD modality shows that patients receiving kidney transplantation as their primary treatment modality have the longest life expectancy.

The combined 5-year survival for pediatric ESRD patients evaluated from 2008 to 2012 was 89%, with the youngest age group having the lowest survival. The 5-year survival percentages were 84%, 93%, 95%, 93%, and 87% for children aged 0 to 4 years, 5 to 9 years, 10 to 13 years, 14 to 17 years, and 18 to 21 years, respectively (Fig. 189.4A). As expected, the 5-year survival is best for those who have undergone transplantation (95%), followed by peritoneal dialysis and HD, both with a 5-year survival of 82% (Fig. 189.4B). The causes

TABLE 189.1

Expected Remaining Lifetime in Years of Prevalent Patients by Initial ESRD Modality, 2015.

Age group	Dialysis patients	Transplant patients	General population
0–4	22.0	57.7	77.0
5–9	22.8	56.2	72.1
10–13	23.3	52.1	67.6
14–17	20.6	48.9	63.7
18–21	17.6	45.6	59.8
22–29	15.7	42.3	54.1

ESRD, end-stage renal disease.

Data source: Special analyses, USRDS ESRD Database, USA SSA (Social Security Administration) Period Life Table 2015. Includes period prevalent ESRD dialysis and transplant patients in 2015.

of mortality in pediatric patients are primarily cardiopulmonary issues and infections. Cardiopulmonary complications are responsible for 21% of all pediatric ESRD deaths, followed by infections of which bacterial infections cause 11.1% of deaths.

Rates of hospitalization are used as a surrogate for morbidity and a direct indicator of complications. Pediatric ESRD patients average 2005 admissions per 1000 patient-years; the most common causes again are cardiopulmonary issues, resulting in 374 admissions per 1000 patient-years, and infectious complications resulting in 606 admissions per 1000 patient-years.¹ Broken down into hospital admission by modality of treatment, patients who have undergone transplantation have the lowest admission rates at 276 per 1000 patient-years, followed by those on HD at 556 per 1000 patient-years and by those on peritoneal dialysis at 779 hospitalizations per 1000 patient-years (see Fig. 189.4).

PATIENT SELECTION

The inherent technical challenges unique to pediatric patients and shorter expected waiting time for transplantation access selection presents a dilemma. Incident access selection depends largely on information provided by caregivers, but it also relies on preconceived notions about HD, parent and caregiver biases, and the age and maturity of the patient.⁶ Studies are limited, but issues such as small patient size, having a maturing AVF or graft, and communications issues among stakeholders, have been identified as limiting factors to successful autogenous fistula placement and use in pediatric patients.^{7,8} Chand and colleagues identify several major barriers to successful AVF use including communication problems between providers, a lack of standardized referral practices for chronic kidney disease (CKD) patients, a lack of standardized accepting surgeon, as well as a lack of early communication between those placing and repairing fistulas (surgeons and interventional radiologists) and

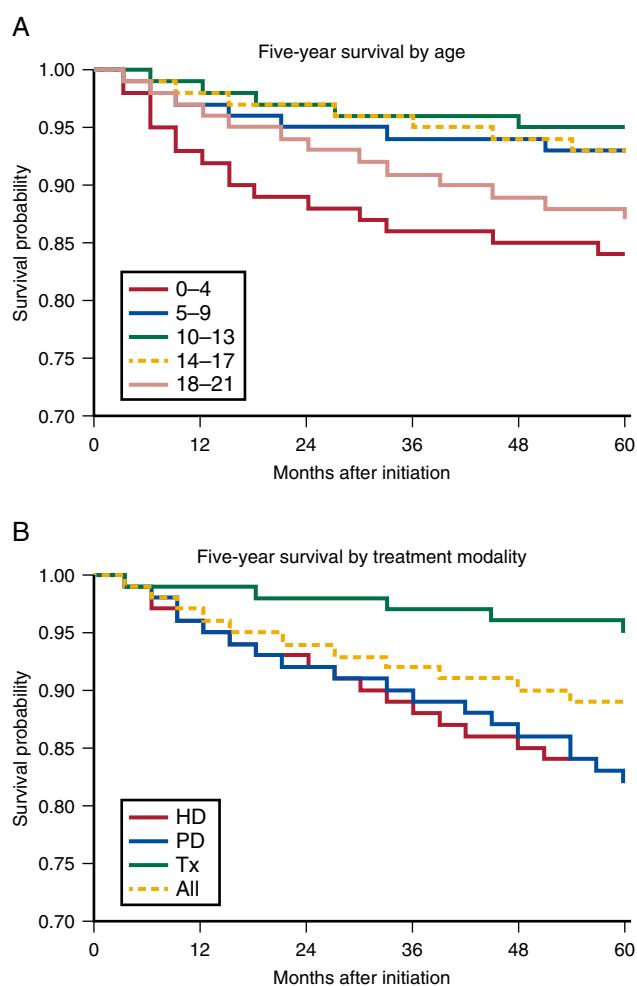


Figure 189.4 Adjusted 5-year survival in incident pediatric patients from 2004 to 2008 shown by age (A), and by treatment modality (B). HD, hemodialysis; PD, peritoneal dialysis; Tx, transplant. (Source: United States Renal Data System: 2015 USRDS Annual Data Report: epidemiology of kidney disease in the United States, Bethesda, MD, 2015, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, Ch. 7; USRDS ESRD Database. Incident dialysis and transplant patients defined at the onset of dialysis or the day of transplant without the 60-day rule; followed to December 31, 2013. Adjusted for age, sex, race, Hispanic ethnicity, and primary cause of ESRD. Ref: incident ESRD patients aged 0 to 21, 2010 to 2011.)

dialysis staff (nephrologists, nurse practitioners, dialysis nurses) regarding problematic fistulas.⁸ Similarly, few studies have looked at psychosocial issues affecting access selection in this population.

Medical decisions can be complex and burdensome; however, when children are allowed to make a decision, long-term implications may not be well understood, although as patients become adolescents, studies have demonstrated decisions are made in conjunction with parents regarding care.⁶ In our own practice, patients report fear of needles, inability to wear jewelry or participate in sports, and the physical appearance of fistulas as barriers to fistula placement. Parents have their own concerns with fistula placement including the uncertainty of knowing what is best for their child despite receiving all the data supporting fistulas over catheters. Brittinger and colleagues evaluated patient discomfort with

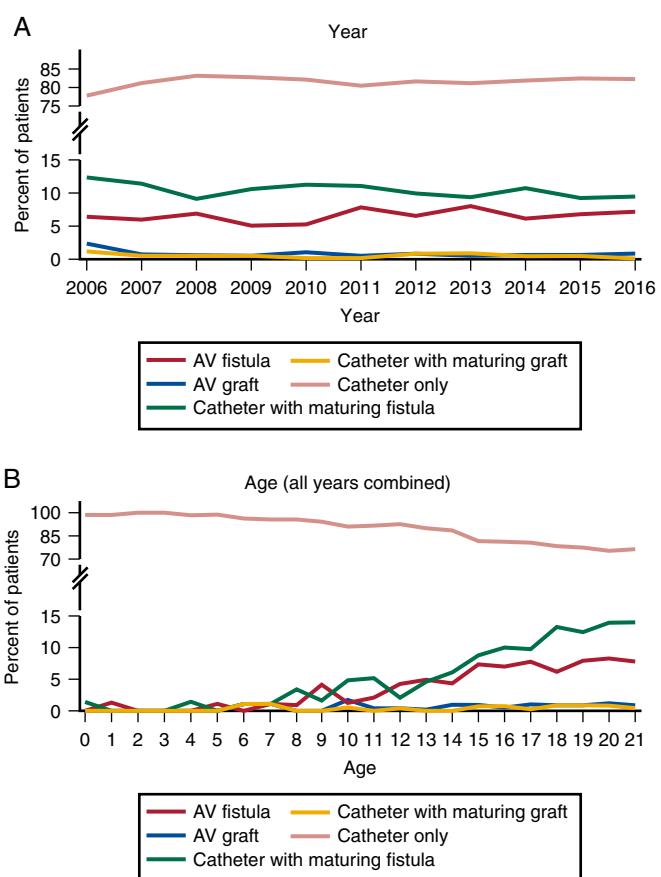


Figure 189.5 Vascular access type at initiation of incident pediatric hemodialysis patients (aged 0–2 years) by (A) year and (B) age, 2006–2016.

HD cannulation and 39% of pediatric patients reported no discomfort, 39% had a tolerable amount of discomfort, and only 22% reported significant discomfort; however, 95% of the participants also reported they would prefer not to revert to CVC for access.⁹

Pediatric patients are initiating HD treatment exclusively with a CVC over 80% of the time, a statistic largely unchanged in the last decade. This percentage is higher when considering those with maturing arteriovenous fistulas or grafts, elevating the percentage to 99.2% in patients aged 0 to 4 years, 98.5% in patients 5 to 9 years, 97.3% in patients 10 to 13 years, 92.2% in patients 14 to 17 years, and lowest at 90.4% among patients 18 to 21 years (Fig. 189.5A). The overall trend of catheter presence at index HD has changed very little since 2006 (Fig. 189.5B). Similar to adults, pediatric fistula placement several months before renal replacement therapy initiation is ideal; thus, nephrologists and surgeons should communicate early about CKD patients such that surgery can be completed in a timely fashion and that fistulas are able to reach functional maturation. Currently, KDOQI guidelines recommend all patients with CKD stage 4 (GFR <30 mL/min per 1.73 m²) be referred for education about modality of ESRD treatment and that AVF be placed 6 months before the anticipated start of HD. Although the progression of CKD to ESRD can vary widely, providers should make early referrals whenever possible.

TREATMENT WITH HEMODIALYSIS

Establishing access for HD is one of the main considerations in initiating treatment for patients of all ages. Issues such as central venous stenosis and catheter-associated infections were among the main concerns leading to the FFBI as well as the International Pediatric Fistula First Initiative (IPFFI). According to the NAPRTCS 2011 dialysis access data report, 78% of patients receiving HD had external percutaneous catheters, 0.3% had external AV shunts, 11.8% had internal fistulas, and 6.7% had internal arteriovenous grafts. Of the external percutaneous catheters, the majority (51.1%) were placed in the subclavian vein exacerbating associated central vein stenosis, 43.7% were placed in the jugular vein, and 4.2% in the femoral vein.¹⁰ Up to 80% of central venous catheters in use are “permanent” catheters instead of “temporary,” decreasing the urgency for autogenous AVF placement.¹⁰ Despite the recognized efforts to increase AVF placement, use of external percutaneous catheters for HD at initiation of therapy has increased from 73% in 1992 to more than 90% of all HD access in 2010.¹

A cross-sectional analysis of prevalent ESRD patients from the ages of 0–21 in May 2017 showed that 51.3% of patients had either an AV fistula or an AV graft as their primary HD access (Fig. 189.6). The type of vascular access was strongly influenced by age, showing a progressive increase in AV fistula/grafts with increasing patient age. Additionally, the USRDS examined the race and etiology of ESRD in age-adjusted analysis and found that whites/Caucasians had a higher use of catheters (52%) compared to other races (African Americans/blacks 44%, other 44.2%). African Americans had a higher proportion of AV graft use (8.7%) when compared to whites (4.5%). Patients with primary glomerular disease as etiology of ESRD had the highest proportion of surgical access in place (AV fistula 48.1% or graft 7.1%).⁴ Catheter use was highest amongst those grouped in “other” etiologies after age-adjusted analysis.

CENTRAL VENOUS CATHETERS

General Considerations

The 2018 USRDS reported CVC as vascular access at the time of initiation of HD has remained constant since 2006 (78%–83%).¹ Reasons for high prevalence of CVCs include technical challenges of AV fistulas in small children/vessel size, anticipation of short/temporary dialysis course prior to renal transplantation, and avoidance of needling access secondary to pain in children or those with behavior problems. Fadrowski and colleagues reported that 59% of patients followed by their center had central venous catheters.⁷ The reasons given for catheter use included “small body size” in 18.8%, having “AVF/AVG maturing” in 7%, and having a “transplant scheduled” in 10.9% of the patients. Of those patients who listed having a pending transplant, 69% had in fact received a transplant within a year, yet 31% of them were still waiting for an organ while receiving HD via CVC. Another retrospective investigation by the same group looked at risks attributable to central venous catheters compared to fistulas in 12- to

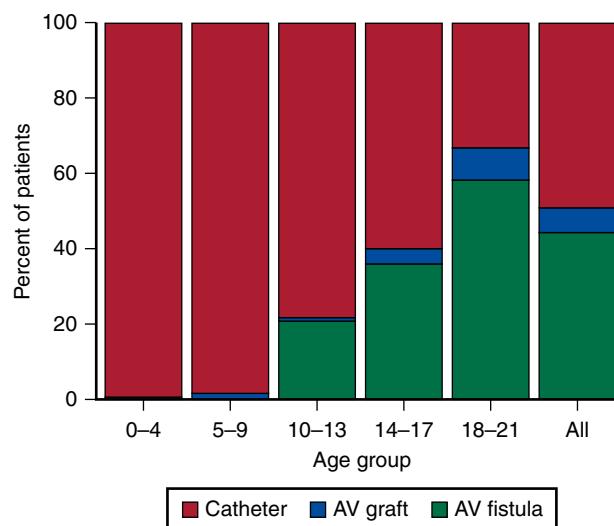


Figure 189.6 Distribution of vascular access type in prevalent pediatric HD patients (aged 0–21 years as of May 31, 2017).

18-year-old ESRD patients receiving HD for the year 2000.¹¹ The authors included 418 patients, 41% with AVF/AVG and 58% with CVC. The group discovered a significant higher relative risk among CVC patients with regard to all-cause hospitalization (RR 1.84; CI 1.38 to 2.44), hospitalizations due to infections (RR 4.74; CI 2.02 to 11.14), and complications of vascular access (RR 2.72; CI 2.00 to 3.69). The durability of central venous catheters remains inferior to fistulas, averaging from 4 to 12 months and in some cases as low as under a month.¹² Central venous catheter durability ranges from 0% to 62% at 1 year, and failure is attributed to infection (17% to 40%), thrombosis (33%), accidental extrusion (5% to 10%), and kinking, which is more common in smaller non-precurved catheters.^{12,13}

A major complication of central venous catheters with long-lasting implications is central venous stenosis. In an attempt to mitigate long-term damage, the K-DOQI guidelines have delineated the sequence of vein selection that is preferred in the event that a CVC is needed.² The recommendation of vein selection lists right internal jugular vein, right external jugular vein, left internal and external jugular veins, subclavian veins, femoral veins, and lastly and rarely used would be translumbar and transhepatic access to the IVC. Publications elucidating the prevalence of central venous stenosis link its occurrence to history of subclavian central venous catheter placement as commonly as 25% to 50%.¹⁴ Finally, although the presence of a catheter seems to impair the long-term patency of AVF in adults,^{10,15–17} this is not the case in pediatric patients as demonstrated in our investigations.¹⁸ Thus, ipsilateral catheter is not a contraindication to fistula placement and efforts to replace catheters with fistulas would reduce the incidence of central venous stenosis.

Technical Considerations

Central venous catheters by design are either temporary (non-tunneled) or permanent (tunneled), and these are often placed



Figure 189.7 A 12-year-old pediatric end-stage renal disease patient who had a 10-French hemodialysis central venous catheter placed in the internal jugular vein. A 10-French catheter was selected due to the patient being smaller than peers her age, and a counter-incision was necessary for proper placement.

percutaneously under moderate sedation and local anesthetic. Tunneled catheters in pediatric population, however, often require general anesthesia to ensure patient compliance, which comes with its own set of potential complications and side effects. There is no evidence-based rule for catheter size selection, making establishing central venous access for HD a major challenge; compounding the issue is that commercially available pre-curved catheters are only supplied in larger sizes. Larger catheters offer higher volumes (and shorter times) during dialysis; however, the size of the child and his or her vessels limit the size of the catheter that can be used. Smaller catheters necessitate longer dialysis sessions due to slower flow rates; thus, the size must be large enough to be useful for dialysis but also small enough to be tolerated by the vessel. The authors created a catheter sizing formula that proved to be safe and is utilized in our practice: [Size (F) = Age ± 2], taking the age of the child and using it as the catheter's diameter in French measurement and then reducing the size if the patient is small for their age or increasing the size if the child is larger than peers the same age. Pre-curved catheters available for the adult populations make percutaneous and subcutaneous tunneling possible; however, these catheters are largely unavailable in the smaller sizes for the youngest of pediatric ESRD patients. In these patients, placing a tunneled CVC usually means artificially creating the curve during placement and making one or more counter incisions (Fig. 189.7) over the access vessel for accurate and precise placement.

ARTERIOVENOUS ACCESS

General Considerations

Autogenous arteriovenous fistulas are the preferred form of access for pediatric patients undergoing HD. Technical success has been demonstrated in a variety of autogenous AV accesses

in both upper and lower extremities.^{19–23} Early experience with autogenous fistula placement in children demonstrates lower patency rates as shown by Bagolan and colleagues with the placement of Cimino fistulas in children from 1985 to 1994; their reported 4-year patency was 63.5%, and the complication rate was 35%, with thrombosis the most common complication.¹⁹ More recent publications have reported better outcomes. The authors have described factors affecting long-term patency of autogenous fistulas of 101 fistulas placed in 93 children with a mean age of 14 years (range 3 to 19), and weights ranging 12 to 131 kg.¹⁸ At the time of surgery, 82% of the patients had a CVC catheter in place and were on HD for an average time of 18 months; furthermore, 24% of them had a history of multiple catheters. The group performed 43 radiocephalic, 29 brachiocephalic, 20 basilic vein transpositions, 9 femoral vein transpositions, and had an average follow up of 2.5 years. The reported primary and secondary patency at 2 years was 83% and 92%, respectively, and 65% and 83% at 4 years, respectively. Chand and colleagues showed that the implementation of IPFFI was feasible at their institution.²⁴ While not uniformly utilized, the use of the operating microscope has also been successful in children, with fewer primary fistula failures.^{25,26}

Technical Considerations

As with central line placement, the K-DOQI publishes guidelines for a structured approach for the placement HD access.² They report a preference for radiocephalic AVF followed by brachiocephalic AVF and lastly by basilic vein transposition. The techniques for standard radiocephalic, brachiocephalic, and basilic vein transposition are widely described and can be applied to pediatric populations with some additional considerations for optimal results.

The initial consultation, similar to adults, starts with a complete history including previous central venous access use, a thorough physical exam with detailed vascular examination, and vein mapping on all patients. The operating surgeon should evaluate the vein mapping personally to determine vein suitability. Due to significant vasospastic response in pediatric patients, vein imaging should also be performed intraoperatively once the patient is placed under anesthesia. Size cutoffs of 2.0 mm for forearm veins, and 2.5 mm for upper-arm veins have had successful rates of maturation.¹⁸ One of the attributes unique to pediatric vessels is their intense vasospastic response with handling; for example, tourniquet occlusion can be used for arterial control during fistula construction in lieu of arterial clamping. Loupe magnification ($\times 2.5$ or greater) should be standard in all pediatric fistulas. Sanabria and colleagues demonstrated 10% early failure rate, and 70% 5-year patency with the use of either operating microscope or loupe magnification.²⁶ Subsequently, Bourquelot and colleagues also published a lower primary fistula failure with use of microsurgery.²⁵ Standard end-to-side anastomosis using a continuous running monofilament suture is recommended, since interrupted suture placement is not imperative. Finally, vein transpositions (brachial or femoral in the

event that superficial arm vasculature is not suitable) can be performed in either a single or two-stage procedure. Kim and colleagues have described the use of two-stage basilic vein transposition with increased percentage of maturation (87%), lower failure rates (7%), and 91% 1-year patency rates.²⁷ Femoral AV fistulas have also been successfully placed in pediatric populations, in the setting of unavailable upper extremities or patient preference as described by Gradman and colleagues with a technique similar to that described in adults.²⁰ Their reported primary patency was 100% and 96% at 1 and 2 years, respectively; secondary patency was reported to be 100%.

CONCLUSION

A total of 81.1% of patients with childhood onset of ESRD who initiated ESRD care before the age of 19 from 1978 to 2016 were still alive as of December 31, 2016.¹ However, establishing pediatric HD access is complex and has yet to become standardized. National data indicate that a large majority of patients initiate HD with CVCs despite results demonstrating that AVF are superior. Central venous catheters are associated with higher complications, more hospitalizations, and shorter lifespans when compared to AVF. Since the initiation of the IPFFI, there has been little change in fistula use for incident HD in the pediatric population. A handful of barriers to fistula placement have been identified, but these have yet to be addressed. Given that the majority of pediatric ESRD patients will outlive at least one transplant and return to HD, providers should steer away from using catheters as the primary treatment modality and avoid the life-long implications of catheter-related complications. Aside from emergent need for dialysis and those that already have a CVC with an impending transplant, CVC use should be largely curtailed. Finally, educational material, such as that used in the IPFFI, should be widely distributed to both providers and patients. Standardizing timing of referrals, improving communication among providers, and delineating pitfalls can greatly increase the prevalence of autogenous fistulas in this population.

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A complete reference list can be found online at www.expertconsult.com.

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Acute Pediatric Arterial Occlusion

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INTRODUCTION

While management of acute arterial occlusion is a core skillset of the vascular surgeon, treating a child with an acutely ischemic extremity can be especially challenging. This is an uncommon problem so few surgeons have significant experience, particularly with infants and neonates with acute limb ischemia, and local expertise will vary. Etiology and epidemiology of acute arterial occlusion in children is distinct from adults where atherosclerosis predominates.^{1–5} Etiology also varies substantially within the pediatric population by age.^{1–5} Traumatic injuries account for the majority of arterial occlusions in older children while iatrogenic iliofemoral artery occlusion is the most common presentation in younger children, and the main focus of this chapter.

Children are not simply smaller versions of adults. Vessel size correlates with age^{6,7} but underlying wall structure is incompletely developed in younger children. Arteries are more fragile and thus more prone to significant injury during invasive procedures or trauma and more challenging to repair with standard instrumentation. Children may also exhibit intense vasoconstriction in response to vessel manipulation, which further increases the challenge of open or endovascular surgery on small vessels that become even smaller from intense spasm.^{5,8} Children also differ in general physiology and pharmacology. Coagulation, intrinsic thrombolysis, drug metabolism and

drug activity can vary significantly for younger children and neonates.^{5,9}

Few prospective comparative studies exist to guide management of children with acute arterial occlusion so treatment recommendations are generally based on expert opinion. In this setting a collaborative approach can be invaluable with teams leveraging local expertise from various combinations of specialists in pediatrics (e.g., critical care, hematology, CT surgery, or interventional cardiology), as well as traditional adult specialists (e.g., vascular surgery, interventional radiology, and plastic or hand microvascular surgery).⁵

EPIDEMIOLOGY OF ACUTE ARTERIAL OCCLUSION IN CHILDREN

Acute arterial occlusion in children is rare. Totapally and colleagues recently analyzed data from over 4 million hospital discharges of pediatric patients using the Kids' Inpatient Database (KID, 2012).² KID includes data for children ages newborn through 20 years from approximately 4000 hospitals in the US. From these data the authors identified 961 children with extremity arterial thrombosis and estimated the rate of acute occlusion to be 2.35/10,000 hospital discharges. This study and others have shown that the etiology of arterial occlusion in children varies significantly by age.^{2–5,10} Iatrogenic arterial

injuries predominate in neonates and infants <6 months of age, while blunt and penetrating trauma are the most common cause of acute arterial occlusion in teens. While these two mechanisms predominate, sporadic case series or reports describe embolization, dissection, and spontaneous arterial occlusion associated with systemic inflammatory conditions, autoimmune disorders, or thrombophilias.^{5,11,12}

Iatrogenic Arterial Injury

Children generally suffer acute occlusion of normal arteries where etiological factors include artery wall injury and stasis of blood flow. Arterial cannulation injures the vessel wall and denudes the endothelium exposing prothrombotic subendothelial matrix to flowing blood resulting in platelet adhesion, aggregation and activation of the clotting cascade. The more delicate structure of incompletely developed arteries in younger children likely predisposes them to more severe wall disruption from cannulation.^{5,6,8} The relatively small caliber of the femoral and iliac arteries creates a size mismatch with arterial sheaths, catheters and cannulas, often made worse by intense vasospasm.^{5,6,8} The result is lumen obstruction with stagnant flow contributing to thrombosis. Other factors suggested to increase risk of catheter-associated thrombosis in young children include inadequate prophylactic anticoagulation, elevated hematocrit common in neonates, and hemoconcentration from sepsis or dehydration (Fig. 190.1).⁵

Femoral artery catheterization carries a 2% risk of an injury requiring operative repair and is the leading cause of acute limb ischemia in children. Femoral cannulation is primarily for invasive blood pressure monitoring in critically ill children, cardiac catheterization, percutaneous treatment of congenital heart and aortic abnormalities, cardiopulmonary bypass, and extracorporeal membrane oxygenation (ECMO).^{2,5,13,14} Thrombosis was reported in 1.3% of infants less than six months of age undergoing cardiac catheterization in the KID database¹⁵ but this is an underestimation, as much higher rates of occlusion have been reported in prospective clinical trials (8%–9%).⁵

Patient factors correlating with acute femoral occlusion from catheterization include younger age and severe vasospasm. Vasospasm is seen in up to 62% of children during catheterization and is more frequent and severe in neonates.^{5,7,8} The combination of catheter size relative to vessel size was found to correlate strongly with degree of vasospasm. Age is a surrogate for smaller vessels with less wall development. In a recent study of children with mean age of 22 ± 8 months of age, the average diameter of the common femoral artery was 3.1 mm.⁷ Tadphale et al. recently constructed nomograms for femoral artery diameter vs. age to aid in decisions regarding accessing vessels for diagnostic or interventional procedures (Fig. 190.2).⁶ They reported average femoral artery diameter at birth of ~2 mm, then ~2.5 mm at 1 year of age, increasing to ~4 mm by 4 years of age.

In a recent study employing routine use of ultrasound to examine access sites after cardiac catheterization the overall rate of femoral thrombosis was 7.9%, but in the subset of children younger than 6 months of age thrombosis increased



Figure 190.1 Angiography in a Child with Catheter-Associated Femoral Artery Thrombosis. (With permission from Cardneau JD, Henke PK, Upchurch GR Jr, et al. Efficacy and durability of autogenous saphenous vein conduits for lower extremity arterial reconstructions in preadolescent children. *J Vasc Surg*. 2001;34:34–40.)

to 23.4%.¹⁶ Thrombosis correlated significantly with lower weight, larger sheath size, and longer procedure duration. Others have reported similar associations with increased numbers of catheterizations.⁵ These factors reflect the complexity of the intervention, extent of vessel injury, and duration of lumen obstruction/stagnant flow.

Umbilical artery catheters carry a 1%–3% risk of symptomatic arterial thrombosis in neonates.⁵ Aortic thrombus can propagate into iliofemoral segments or embolize distally. Risk of thrombosis increases if the patient does not receive adequate heparin prophylaxis while the catheter is in place and varies with catheter positioning. If the catheter tip is positioned above the visceral aortic branches (e.g., above the diaphragm), risk of associated aortic thrombus is significantly reduced compared to tip placement below the diaphragm.⁵

Traumatic Arterial Injury

Vascular trauma is covered extensively in separate chapters that provide comprehensive discussions on the pathogenesis and treatment of blunt and penetrating arterial injuries in adults and children (see Chs. 183, Vascular Trauma: Extremity and 188, Vascular Trauma in the Pediatric Population). A recent report from Wang and colleagues studied 1399 pediatric trauma patients and found 23 children who suffered concomitant vascular injuries requiring surgical intervention.¹⁷ As in previous reports, pediatric vascular trauma was more common in the upper extremities than lower extremities (61% vs. 30%) and penetrating injuries were less common than in adults. In a review of the National Trauma Databank, Barmparas and colleagues studied the epidemiology of pediatric vascular injuries

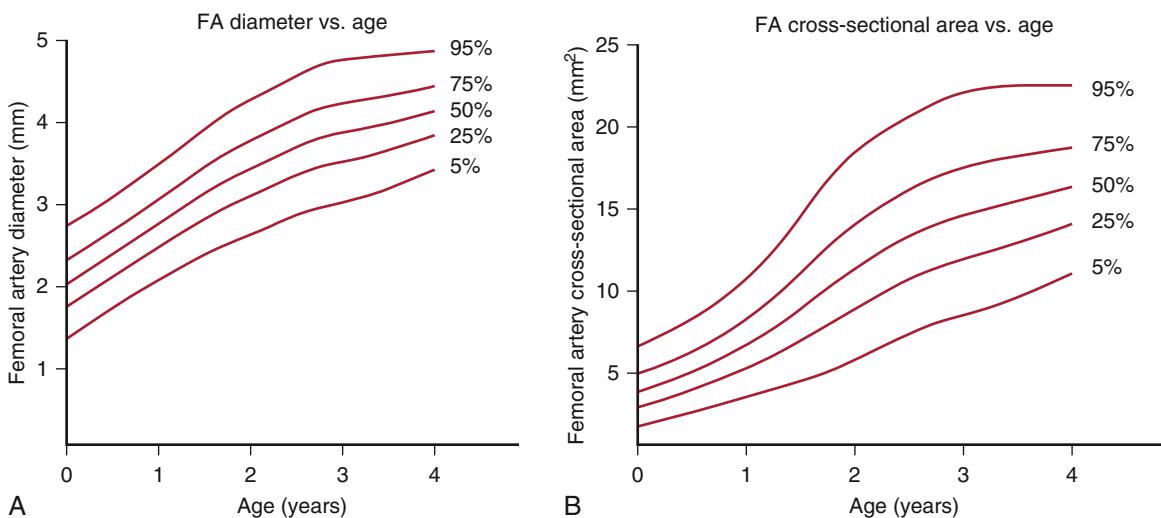


Figure 190.2 (A) Nomogram comparing femoral artery (FA) diameter to age. (B) Nomogram comparing FA cross-sectional area to age. (From Tadphale SD, Zurakowski D, Bird LE, et al. Construction of Femoral Vessel Nomograms for planning cardiac interventional procedures in children ages 0–4 years old. *Pediatr Cardiol*. 2020;41:1135–1144. Springer.)

in 251,787 traumatized children under age 16.¹⁸ The incidence of vascular injury was 0.6%, nearly threefold lower than for adults. The majority of injuries occurred in males (74%) and most often in the upper extremity (35.7%), abdomen (24.2%) and lower extremity (18.6%). Children with vascular trauma had a much lower mortality than adults (13.2% vs. 23.2%), but mortality among children was highest in infants (18.2%).

Spontaneous Occlusion

Sporadic small case series and case reports have described unprovoked arterial occlusion in association with a variety of inherited and acquired conditions in children (Table 190.1). Spontaneous occlusions are rare and typically associated with conditions causing a hypercoagulable state.⁵ These include inherited (e.g., antithrombin and protein C or S deficiency)¹⁹ or acquired thrombophilia (e.g., DIC, nephrotic syndrome, antiphospholipid syndrome),^{5,20} acute inflammation (e.g., sepsis), chronic inflammatory diseases (e.g., Behçet disease), autoimmune disorders (antiphospholipid syndrome), collagen vascular diseases, and heparin-induced thrombocytopenia (HIT).^{5,20–22} The pattern of unprovoked arterial thrombosis often differs from iatrogenic or traumatic occlusions as more distal arteries are often affected,^{21,23} as in the example of purpura fulminans in the newborn (e.g., from protein C or S deficiency), or in patients with DIC or HIT.⁵ Children with a thrombophilia are also more likely to present with concomitant venous thromboses.

CLINICAL PRESENTATION

History and Physical Examination

As noted above, children with acute iliofemoral occlusion most commonly present after arterial instrumentation or acute extremity trauma.^{2,5} In patients undergoing arterial line

placement for monitoring or cardiac catheterization, a high index of suspicion is required when evaluating limbs with signs or symptoms of impaired blood flow. Similarly, patients on ECMO or cardiopulmonary bypass require close monitoring for impaired limb perfusion during cannulation and also immediately following decannulation. More subtle forms of vascular puncture such as femoral or brachial puncture for arterial blood gas samples can also lead to arterial occlusions in susceptible patients. Femoral or upper extremity venipuncture for IV access or central line placement can result in accidental cannulation of an adjacent artery. In patients with spontaneous arterial occlusions, a history of inherited thrombophilia may be obtained from first-degree relatives. Patients may also have had prior thrombotic events such as catheter-associated or spontaneous venous thromboembolism.

Physical examination findings in older children are typical of those seen in adults and with an appropriate index of suspicion the diagnosis can be straightforward. Cool, mottled extremities with pallor or cyanosis, and decreased pulses compared to the opposite extremity suggest impaired flow. Differential Doppler opening pressures in the distal extremity can confirm reduced perfusion from stenosis, occlusion or spasm. Pain can be variable and loss of motor and sensory function is generally a late finding. Reduced perfusion can be more subtle in younger children and neonates. Hand-held continuous wave Doppler of extremity arteries can be an invaluable component of the examination, contrasting signal characteristics in involved and unininvolved extremities. Assessing changes in motor or sensory function in neonates and infants can be challenging but decreased limb movement can be a sign of severe ischemia. Rizzi and colleagues in a systematic review on catheter-associated arterial occlusions found that 78% had signs of acute ischemia at presentation.²⁴

If collateral circulation around the common femoral artery is impaired by occlusion of the hypogastric artery and/or common femoral branches, appearance of the extremity is often

TABLE 190.1Spontaneous Arterial Occlusion in Children:
Associated Factors

Inherited	
Reduced levels/activity	Increased levels/activity
Antithrombin III, protein C, protein S	Factor V Leiden and prothrombin G20210 mutations Increases in factors VIII, IX, XI Hyperhomocysteinemia
Acquired	
Antiphospholipid antibody syndrome	+/- Systemic lupus Erythematosus Lupus inhibitor Anticardiolipin antibodies B2-glycoprotein 1 antibodies
Sepsis	Dehydration DIC Vasopressors
Nephrotic syndrome	
Vasculitis	Behcet syndrome Kawasaki disease Takayasu arteritis RA
IBD	
HUS/TPP	
Cancer	ALL Solid tumor +/- metastasis Chemotherapy/biologics
Blood dyscrasia	Thrombocytosis Polycythemia Homozygous sickle cell anemia
Drugs	Heparin-induced thrombocytopenia (HIT) Contraceptives

that of profound ischemia (Fig. 190.3). Appearance of the limb can also be influenced by underlying congenital cardiopulmonary defects with poor cardiac output, intracardiac shunting, hypoxemia, hypotension and vasopressor use. In a recent study of infants with iliofemoral involvement, 89% had complete arterial occlusion at the time of diagnosis and 46% were without Doppler-detectable blood flow below the ankle.²⁵

Laboratory Findings

Laboratory testing is often nonspecific to acute arterial occlusion but essential for patient management. CBC with platelets and differential may show evidence of hemoconcentration. Leukocytosis with an abnormal differential may support sepsis or a hematological malignancy. There may be evidence of thrombocytosis. Patients on anticoagulation for prophylaxis of indwelling arterial lines and catheters should be monitored regularly to ensure a therapeutic range as low values predispose to catheter-associated artery thrombosis.⁵ Abnormally low platelet count or a sudden drop can indicate DIC, or heparin-induced thrombocytopenia (HIT) in children with prior heparin exposure. HIT can be confirmed with functional platelet activation assays (e.g., heparin–IgG–PF4 complex) or serologic assays (e.g., serotonin release assay).²⁶



Figure 190.3 Catheter-Associated Ilioemoral Thrombosis in a Neonate.

In children with significant ischemic changes the creatine phosphokinase level may be elevated and, if so, it can be trended to monitor impact of revascularization or to monitor for progression. Urine myoglobin can also inform the degree of ischemic muscle injury.

In patients with sepsis or with cutaneous signs of DIC, appropriate coagulation panels should be obtained. In spontaneous thrombosis, appropriate assays for autoimmune or systemic inflammatory disorders may be guided by the clinical picture and family history. With recurrent or otherwise unexplained spontaneous thrombosis, genetic and functional assays should be considered to assess for inherited or acquired thrombophilia (Table 190.1).^{5,11,20}

Diagnostic Imaging

Duplex ultrasound can be invaluable in the diagnosis of acute arterial occlusion in all age groups. It is generally available for bedside exam and in experienced hands can be definitive in diagnosing acute extremity artery thrombosis. It is rapid, inexpensive and avoids having to move tenuous patients outside of the ICU. It can be repeated frequently without concerns for exposure to ionizing radiation or contrast.

Axial imaging with either CT angiography (CTA) or contrast-enhanced MR angiography (ceMRA) may be helpful in defining location and extent of arterial injury and occlusion. Axial imaging in young children requires general anesthesia and secure IV access. CTA is often used in children presenting with serious multiple injury trauma. Its advantages include wide availability, speed, and the ability to define associated injuries, and precise location and extent of vascular involvement. CTA has high spatial resolution and is often helpful in planning for open vascular surgery and endovascular procedures. However, radiation exposure is significant with CTA, an important consideration in children, and contrast carries risk of nephropathy in the critically ill population. ceMRA has a more limited role in managing acute arterial occlusions in children as it is generally more time-consuming and cumbersome for unstable patients, particularly neonates and infants. ceMRA studies have lower spatial resolution than CTA and are less well tolerated in older children. MRA may be an alternative to CTA in patients with moderate renal impairment but, as in adults, gadolinium is contraindicated in severe renal insufficiency.

Digital subtraction angiography (DSA) remains the gold standard for defining lumen irregularities, degree of stenosis, extent of obstruction, and collateral flow around and run-off beyond a site of occlusion. DSA also provides the option for endovascular revascularization when appropriate. Being invasive, DSA carries the risk of injuring an additional artery, particularly in smaller children, and exposure to ionizing radiation is significant. Contrast exposure with DSA can often be less than for extensive CTA exams.

TREATMENT

Treatment of acute arterial occlusion in school age children can generally be guided by principles and strategies outlined in previous chapters pertaining to adults (see Chs. 103, Acute Limb Ischemia: Evaluation, Decision Making, and Medical Treatment; 104, Acute Limb Ischemia: Surgical and Endovascular Treatment; and 183, Vascular Trauma: Extremity). Neonates, infants and toddlers, however, present unique challenges due to small delicate vessels prone to spasm. Adult instrumentation, particularly endovascular equipment, is large relative to arteries in young children but newer access devices developed for adult pedal and radial artery access may be helpful when working with children. Open repair in the very young may be particularly suited to microvascular techniques when available.²⁷

The natural history following acute femoral arterial occlusion in younger children appears to differ from that for adults. Children exhibit more tolerance to prolonged mild to moderate ischemia, greater potential for recanalization of thrombus in otherwise structurally normal arteries, and greater potential for collateralization.^{5,28–30} These and other factors, as well as relatively poor patency after open procedures in the very young, have shifted the risk–benefit calculation toward nonoperative algorithms for the majority of iatrogenic iliofemoral occlusions in children. Pediatric vascular trauma, however, is still for the most part approached surgically.¹⁷

Anticoagulation

Contemporary management of iatrogenic femoral artery thrombosis in children most commonly includes a period of anticoagulation.^{5,28,29,31} This strategy is used less frequently in pediatric vascular trauma, but more so than for adults.¹⁷ In children with strong contraindications to full anticoagulation, open surgical thrombectomy and direct artery repair is appropriate for severe limb ischemia. But in children with mild or moderate limb ischemia and a contraindication to anticoagulation, a period of close observation may be considered.

Anticoagulation in this setting is most commonly achieved with continuous infusion of unfractionated heparin (UFH), as it can be stopped and reversed quickly for serious bleeding complications or to prepare for invasive procedures. Low-molecular-weight heparin (LMWH) has also been used with success.⁵ Metabolism of heparin and other antithrombotic agents is different in children than in adults and it is prudent to have a pediatric hematologist or pharmacist assist with drug dosing and monitoring.⁹ The most recent ACCP/CHEST consensus

statement on the use of antithrombotic agents in children recommends pediatric patients with acute iliofemoral thrombosis from iatrogenic injury be started on UFH, and if a catheter is still in place it should be removed promptly.⁵ The authors provide standard dosing nomograms for anticoagulation in children and guidelines for monitoring and adjusting to therapeutic targets (Table 190.2).

The therapeutic range for activated partial thromboplastin time (aPTT) in adults receiving UFH by infusion may not be valid in children due to altered drug activity and metabolism. For this reason, many experts suggest monitoring anti-Xa activity assays instead.⁵ Levels of 0.35 to 0.7 units/mL are typically considered therapeutic during continuous infusion of UFH (Table 190.2).^{5,9} Infants receiving twice daily subcutaneous LMWH should have dosing adjusted to achieve anti-Xa levels of 0.50 to 1.0 units/mL, drawn 4 to 6 h after an injection (Table 190.2).⁵

Careful serial examination of the involved extremity is critical, with regular assessment of blood flow, sensory motor status and for ischemic skin or muscle injury. If there is clear progression of thrombosis on serial imaging (e.g., Duplex ultrasound) or clinical progression toward irreversible tissue/organ injury, despite therapeutic heparinization, then thrombolytic therapy or surgical thrombectomy should be considered.

Recommendations for duration of anticoagulation following iatrogenic iliofemoral occlusion have varied widely. ACCP/Chest guidelines suggest 5 to 7 days of anticoagulation⁵ while other groups recommend longer duration of treatment. The group from Michigan studied their results with nonoperative management and suggest a 6-week period of anticoagulation followed by duplex ultrasound imaging to determine if additional anticoagulation is required.²⁹ A report from the group at UCSF documented resolution of femoral artery occlusion using ultrasound in one-third of children after 2 weeks of anticoagulation, and then roughly another third in each of the following 2-week intervals.²⁵ Based on their data they recommend continuing anticoagulation and repeating ultrasound surveillance every 2 weeks until resolution of thrombus.

Acute outcomes with anticoagulation for iatrogenic femoral occlusion include restoration of flow in ~70% of children at 30 days without further intervention. Minor bleeding is the most frequently reported complication.⁵ Kayssi and colleagues reported a 94% success rate in treating acute limb ischemia with anticoagulation alone in children up to 18 years old.³² In the series reported by Ramirez and colleagues, 50 children were identified with iatrogenic femoral occlusion (mean age 10 weeks) of which 83% were heparinized and followed without intervention.²⁵ Approximately 70% of children receiving anticoagulation showed thrombus resolution at 30 days, in contrast to the remaining 17% of children with contraindications to anticoagulation, in which only 1 of 9 showed thrombus resolution. Mortality was 10% but none of the deaths were attributed to iliofemoral occlusion. Bleeding complications occurred in 8% and there were no amputations or tissue loss. Cohen and colleagues reported on 99 infants under 1 year of age (mean age 7 weeks) with catheter-associated iliofemoral thrombosis treated with anticoagulation for up to 28 days (mean 3 weeks).

TABLE 190.2 Systemic Heparin Dosing and Adjustment for Children*

Unfractionated Heparin				
aPTT, seconds	Bolus, units/kg	Hold, min	% Rate Change	Repeat aPTT
<50	50	0	+10	4 h
50–59	0	0	+10	4 h
60–85	0	0	0	Next day
86–95	0	0	-10	4 h
96–120	0	30	-10	4 h
>120	0	60	-15	4 h

Obtain aPTT 4 h after administration of the heparin loading dose and 4 h after every change in the infusion rate				
Low-Molecular-Weight Heparin				
Drug	Weight	Age	Initial treatment dose	Initial prophylactic dose
Age-dependent dose of enoxaparin	na	<2 months	1.5 mg/kg/dose q12h	0.75 mg/kg/dose q12h
	na	>2 months	1.0 mg/kg/dose q12h	0.5 mg/kg/dose q12h
Pediatric (all ages) dose of dalteparin	na	all	129 ± 43 U/kg/dose q24h	92 ± 52 U/kg/dose q24h

na, not applicable.

Enoxaparin has 110 anti-factor Xa units/mg.

Dalteparin has 100 anti-factor Xa units/mg.

*Adapted from Monagle et al. Antithrombotic therapy in neonates and children: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest*. 2012;141(2 Suppl):e73S–e80S.

They noted four bleeding complications of which one was considered major. Four patients received open thrombectomy (4%) and one thrombolysis. Outcomes were 60% complete thrombus resolution, 33% partial resolution, 6% no resolution, and 8 with thrombus progression. There were four deaths unrelated to anticoagulation and no mention of amputation.³³

Thrombolysis

For children who fail to improve or in whom ischemia progresses despite adequate anticoagulation, thrombolysis with tissue plasminogen activator (tPA) may be considered. Contraindications to thrombolysis in children are similar to adults but also include extreme prematurity (Table 190.3).^{5,34} There is no standardized dosing algorithm for thrombolysis in children with acute arterial occlusion. There are no laboratory assays with therapeutic ranges available to guide the intensity of thrombolytic activity. Rather, dosing and duration of treatment are targeted to clinical efficacy balanced against the risk of serious bleeding.^{5,34–36}

A number of case series have been published describing use of thrombolysis in children and their treatment algorithms and results have been cataloged in recent reviews on this subject.^{5,34,35} Systemic lysis has been the predominant route of delivery but a few reports describe catheter-directed thrombolysis to treat arterial occlusion in children. Dosing protocols for systemic intravenous tPA have varied widely from 0.01 to 0.6 mg/kg/h with treatment duration ranging from 0.5 h to more than

24 h³⁵ and a number of reports mixed data for venous and arterial indications when reporting success and complications.³⁵

The 2012 ACCP/Chest guidelines reviewed the literature on systemic lysis in children and found the most common protocol was 0.5 mg/kg/h for 6 hours.⁵ A subsequent report from Bratincsák and colleagues on use of tPA for cardiac catheterization access site complications in 12 children showed thrombus resolution in all cases with no major bleeding complications using a much lower dose range of 0.01 to 0.04 mg/kg/h for 0.5 to 4 h.³⁶ A more recent review of the literature on thrombolysis in children presents data on three approaches including low and high dose protocols for systemic dosing and a protocol for endovascular catheter-directed tPA (Table 190.4).³⁴

The risk of bleeding and treatment of bleeding during thrombolysis should be informed by appropriate laboratory monitoring. Before thrombolytic therapy is initiated, hemostatic problems should be corrected including thrombocytopenia and vitamin K deficiency. Endogenous antithrombin levels are reduced in infants so plasma infusion may be necessary before initiating tPA to allow for adequate plasminogen activity.^{5,34} Children should also be typed and screened before initiating thrombolysis and monitored in the ICU.

Most authors recommended concurrent low-dose UFH infusion during systemic thrombolysis with infusions of 5–10 U/kg/h being common. Monitoring UFH during lysis should be via anti-Xa levels as aPTT values can be unreliable in the presence of fibrin split products. Careful laboratory monitoring should also include Hg/HCT, platelet count, fibrinogen,

TABLE 190.3**Potential Contraindications to Thrombolysis Children****Absolute Contraindications**

Active bleeding	
Gastrointestinal bleeding within 10 days	
Stroke or intracranial hemorrhage within 3 months	Some suggest 6 months
Intracranial or spinal surgery within 3 months	
Severe head trauma within 3 months	
Platelets <100	
Fibrinogen <100 mg/dL	

Relative Contraindications

Extreme prematurity	
Uncontrolled severe hypertension	
Major surgery within 10 days	
Major trauma within 10 days	
CPR or asphyxia within 7 days	
Puncture of a noncompressible vessel within 10 days	
Organ biopsy within 10 days	
Intracranial vascular malformation or tumor	
Infective endocarditis	
Hepatic failure	
Pregnancy or puerperium	
Gastrointestinal bleed within 21 days	
Hepatic failure	
Contraindication to unfractionated heparin	
Contraindication to contrast media	
Other Considerations	
Direct thrombin inhibitors or direct factor Xa inhibitors should be held 48 h before tPA	Or normal coagulation parameters

fibrinogen degradation products, D-dimer, and PT/PTT every 6 hours. Fibrinogen levels below 100 mg/dL should prompt consideration of cryoprecipitate or plasma replacement. Measurement of fibrin degradation products or D-dimers are helpful in documenting presence of systemic fibrinolytic activity. Maintaining a platelet count >100 K/ μ L during thrombolysis has also been recommended.^{5,34}

Mild bleeding (e.g., oozing from a wound or puncture site) can often be treated with local pressure and supportive care. More extensive bleeding may require lowering the dose or interrupting thrombolysis and/or UFH. Major bleeding requires stopping thrombolysis and UFH then administering cryoprecipitate (1 unit/5 kg or 5–10 mL/kg), an antifibrinolytic, or both; and administering other blood products as indicated. Heparin can be reversed with protamine.^{5,34} Emergent surgery may be required to correct and evacuate active bleeding.

Kayssi and colleagues reported 75% success with systemic thrombolysis when used as a bailout in children with inadequate responses to therapeutic anticoagulation.³² The most frequent problem was bleeding at sites of invasive procedures. A large single-institution study reported bleeding in 68% of their patients, and transfusion in 39%. Prolonged duration of thrombolytic infusion was associated with increased bleeding. Zenz et al. reported bleeding requiring transfusion in 3 of 17 (18%) patients treated for 4–11 h and minor bleeding in another nine (54%).³⁷ Another recent prospective study reported bleeding requiring transfusion in three of 26 children (11.5%) and minor bleeding episodes in 11 (42%). Prolonged duration of thrombolytic infusion was associated with increased bleeding.^{5,37}

Catheter-directed thrombolysis may be considered, when appropriate, delivering tPA directly into the occlusion. Targeted delivery has the advantage of high local drug levels, less systemic exposure, and less risk of bleeding.^{34,38}

Open and Endovascular Revascularization

Strategies outlined in previous chapters on adult limb revascularization may be applied to children with an acutely ischemic limb. Choice of treatment will depend on a combination of factors including patient age, severity of symptoms, and local availability of experienced specialists.

As noted above, iatrogenic acute arterial occlusion predominates in neonates and infants and most often children respond favorably to nonoperative management. Surgical intervention in this age group is generally reserved for situations of clear progression to limb- and life-threatening ischemia after a trial of antithrombotic therapy. Small delicate vessels and vasospasm often limit what is technically feasible, even with loupe magnification. Direct femoral embolectomy can be attempted using 2-F Fogarty catheters followed by primary artery repair. Direct vessel closure or repair can be facilitated with microvascular techniques, when available. We have found our hand surgeons valuable in completing primary repair or interposition vein grafts and bypasses of injured arteries in infants and toddlers, taking advantage of their skills with an operating microscope.

Older school-aged children have larger vessels^{6,7} generally amenable to standard open and endovascular strategies for arterial revascularization, as in adults.^{17,18,39,40} Direct exploration, thrombectomy and vessel repair are first-line therapy to reestablish limb perfusion. With blunt or penetrating trauma, saphenous vein caliber is commonly adequate for direct artery reconstruction or bypass in the extremities. Prosthetic conduits can be employed as well, as for initial damage control procedures in children with multiple injuries. Endovascular techniques complement open reconstruction. Angiography can help to guide management and enable catheter-directed mechanical and/or chemical thrombolysis, over-the-wire balloon thrombectomy, angioplasty or stenting in treating children.

Minimizing artery spasm can aid in both open and endovascular procedures. Strategies include warming the OR and the patient and minimizing manipulation of the vessel or cautery. A number of solutions applied topically to vessels during open

TABLE 190.4 Thrombolysis Dosing in Children

Mode of Thrombolysis	Alteplase Dosing Infusion	Duration of Thrombolysis	Concomitant UFH Therapy	Laboratory Monitoring	Bolus
Systemic thrombolysis	Low-dose: 0.01–0.06 mg/kg/h (max 2 mg/h)	6–72 h	Prophylactic UFH with goal UFH anti-Xa level of 0.1–0.3 or UFH at 10 U/kg/h	Every 6–12 h: fibrinogen, CBC, FDPs, PT, aPTT, UFH anti-Xa	None
	High-dose: 0.1–0.5 mg/kg/h (max 2 mg/h)	2–6 h, may repeat if indicated			None
Site-directed thrombolysis	0.01–0.03 mg/kg/h or max 1–2 mg/h	Up to 72–96 h	Therapeutic UFH with goal UFH anti-Xa level of 0.3–0.7 or UFH at 10 U/kg/h	Every 6–12 h: fibrinogen, CBC, FDPs, PT, aPTT, UFH anti-Xa, renal profile, urinalysis	0.1–0.3 mg/kg (max dose 10 mg)

Note: There are no standardized dosing protocols for thrombolysis in children. These are examples gleaned from the literature. Each patient should be assessed individually for risks, benefits and goals of treatment and whether thrombolysis is indicated. A dosing strategy can then be tailored accordingly, generally in concert with a pediatric hematologist.

(Adapted from Tarango C, Manco-Johnson MJ. Pediatric thrombolysis: a practical approach. *Front Pediatr.* 2017;5:260.)

procedures can help to reduce the degree of spasm, including: warm saline irrigation, nitroglycerine, papaverine, milrinone, lidocaine and others. Catheter-directed intraluminal nitroglycerine administration can also help promote dilation and distal flow during endovascular procedures.

Patency following open revascularization in infants and neonates is poor in contrast with older children or adults. Andraska et al. and others reported durable restoration of pulses in a minority of cases.²⁹ Lin and colleagues reported their outcomes with open revascularization in 34 children following iatrogenic thrombosis.¹⁴ Among the 36 procedures, 14 were for iliofemoral thrombosis (6 thrombectomy with primary repairs and 6 with vein patch, and 2 resections with primary repair). Outcomes beyond 30 days included claudication in 7 of 14 children; gait disturbance or limb length discrepancies in 4 but 84% eventually regained normal circulation.¹⁴

Acute outcomes for treatment of traumatic arterial injuries in children are reviewed in detail in Chapter 188 (Vascular Trauma in the Pediatric Population). Klinkner and colleagues reported on 103 children with vascular trauma with overall mortality of 9.7%.⁴¹ Twenty-three children underwent arteriography, 1 endovascular reconstruction, 59 open repairs ranging from ligation to bypass with prosthetic or vein, and 11 patients were managed nonoperatively. There were 15 amputations in 11 patients, 3 in upper extremities. Two arms were deemed beyond salvage on arrival and another was amputated after attempted reimplantation.⁴¹ In the review of the National Trauma Data Bank by Barmparas and colleagues mentioned above (over 1500 pediatric vascular injuries), mortality was 13.2% (18.2% in infants). The incidence of amputations, when adjusted for site of vessel injury, was similar between children and adults (7.5% in the lower extremity and 0.7% in the upper extremity).¹⁸ Prieto and colleagues studied a database of children discharged from California hospitals and found 775 children with vascular trauma in an extremity. The majority were upper extremities (60%) and amputations were reported in 7%, all in the lower extremities.⁴⁰ Other single-center series

have reported amputations in 0% to 18% lower extremity arterial injuries.¹⁸

In a comparison between open surgery and medical management for traumatic vascular injuries, Lazarides and colleagues reported open repairs in 11 of 12 older children (10 vein interposition grafts), while 6 of 11 children under 6 years of age were managed nonoperatively. They had no deaths, 2 amputations (both in the surgery group), and one late limb length discrepancy.³⁹

LATE SEQUELA

The majority of children treated for acute arterial thrombosis will do well from a vascular perspective. Goals for acute management are life and limb salvage and restoration of arterial flow. In contrast, long-term goals are primarily normal limb growth and development and normal limb function. These goals may be realized in some cases without artery patency if collateralization is robust. However, problems with limb development and function have been described and if not caught and corrected in a timely manner may result in permanent limb impairment.^{28–30,39}

Hemodynamic forces play a major role in tissue development in the fetus and younger children. As tissues and organs grow, their increased demand for volume blood flow, in turn, drives supporting vessels to grow in response to hemodynamic cues from blood flow velocity and pressure (i.e., rising lumen shear stress and vessel wall strain). In the extremities a mismatch between perfusion and tissue demand can blunt musculoskeletal development, even in the absence of symptoms of ischemia such as claudication. A threshold of perfusion above which limb development will proceed normally has not been defined so decisions regarding late revascularization in children are largely based on clinical judgment.^{28,30}

Arterial occlusion after catheterization can go unrecognized, particularly if children are managed based on physical exam findings rather than routine post-catheterization testing (e.g., duplex

ultrasound).¹⁶ Celermajer and colleagues found that ~30% of previously catheterized children had access site occlusions or stenosis identified with later attempts at catheterizations.⁴² Distal limb pressures are not typically documented in children, and limbs that appear well-perfused can have significant asymmetry in limb perfusion that is unrecognized. Claudication, gait abnormalities and limb length discrepancies or atrophy may first become evident after periods of significant growth. The incidence of claudication varies from 0% to 6.3% and remains an important indication for late intervention.^{5,28–30} For this reason, it is important to educate parents, and to follow children with the vascular laboratory, in order to identify and correct perfusion deficits early enough to allow for compensatory growth before long bones fuse their growth plates.

Few studies have addressed late outcomes in limb function in a prospective or systematic way. Late complications are uncommon but symptoms and signs include gait disturbances from claudication or discrepancies in limb girth and/or length. Reports on late complications are typically small retrospective series or case reports and children are often first seen when referred for problems later in life rather than identified prospectively.⁴³ The incidence is poorly defined. A report by Taylor and colleagues identified 58 children that had femoral catheterization when less than 5 years of age.³⁰ When examined 5 to 14 years later with duplex ultrasound, they found over 40% had femoral occlusions and the mean ABI in catheterized limbs was only 0.79.³⁰ Among the 24 limbs with iliofemoral occlusion, only 1 had claudication, but another 4 demonstrated a limb length discrepancy. Similarly, Rizzi and colleagues reported on 95 children treated for catheter-associated thrombosis and documented late stenosis or occlusion in 33%. Mean follow-up in this report was only 2 years which may explain the low rate of claudication (one child) or limb length discrepancy (one limb).⁴⁴ More recently, Eliason and colleagues reported on treatment of chronic ischemia in 33 children following prior iatrogenic or traumatic arterial injuries.²⁸ The majority of these children were preadolescent (age range 3–17) and 25 children had claudication-like symptoms; 21 had limb length discrepancies; and 5 presented with compensatory scoliosis. Decreased muscle mass and smaller foot size were late findings in 7 of the children. Wang and colleagues reported late outcomes in 25 children after revascularization and found 1 late amputation, 2 chronic wounds, and 1 limb length discrepancy. No patients were identified with claudication during a mean follow-up of 53.5 months.¹⁷

In summary, late outcomes in children following iatrogenic or traumatic arterial occlusions are not well studied. The

incidence of late complications varies widely in retrospective series. Claudication and impaired limb growth can occur so parents should be educated about potential problems and vascular status should be documented at discharge with ultrasound or other imaging or physiological testing to define the baseline early after initial treatment. Given relatively high rates of re-occlusion reported in late follow-up, it would be prudent to establish regular vascular lab surveillance for a period of time after revascularization and refer children promptly for consideration for further revascularization procedures should abnormalities be identified.

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Erectile Dysfunction

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Based on a previous edition chapter by Boback M. Berookhim and John P. Mulhall

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Erectile dysfunction (ED) is defined as the recurrent or consistent inability to develop and/or maintain a penile erection sufficient for satisfactory sexual performance.¹ It is a common condition among men that can have significant negative downstream impact on quality of life and interpersonal relationships, associated with poor general health, and can have role limitations because of physical and emotional problems.²

EPIDEMIOLOGY

Recent studies estimate that ED affects roughly 20 million men in the US with a projected growing incidence rate of 26 new cases per 1000 annually.^{3,4} Globally, the prevalence of ED is expected to grow to an estimated 322 million by the year 2025, with the largest projected increases in the developing

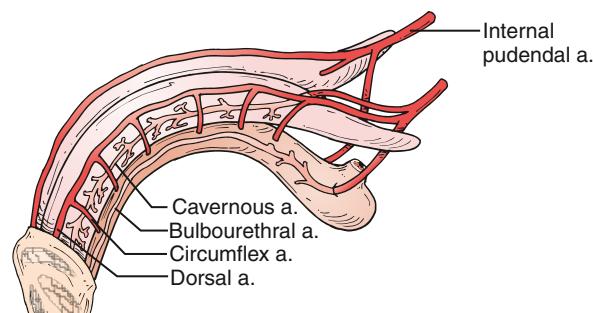
world, i.e., Africa, Asia, South America, etc.⁵ The prevalence of ED, however, appears to be consistent among different ethnic groups, with population-based studies demonstrating a 22% rate of ED in white men, 24% in black men, and 20% rate among Hispanic men all over 40 years of age.⁶

PHYSIOLOGY OF PENILE ERECTION

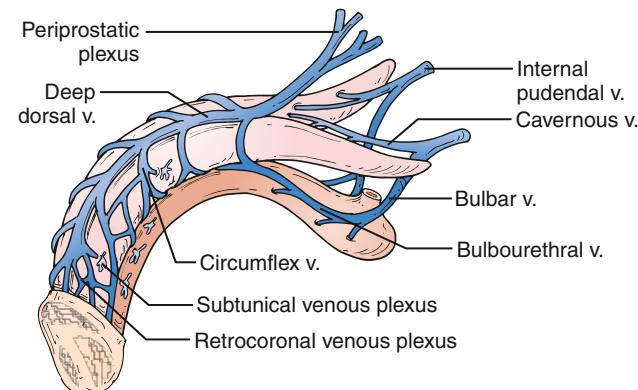
Structurally, the penis is composed of three cylinders of erectile tissue, which includes the paired dorsal corpora cavernosa and the ventral corpus spongiosum, encompassing the urethra. The corpora cavernosa are encased by the dense tunica albuginea, which provides flexibility, rigidity, and strength to the penis. Blood flow to the penis typically originates from the internal pudendal artery, a branch of the internal iliac artery. Distally, it becomes the common penile artery, further subdividing into the dorsal, cavernosal, and bulbourethral arteries. Accessory pudendal arteries arising from the external iliac, obturator, vesical, or femoral arteries can all contribute significantly to the arterial supply to the penis.⁷ The cavernosal arteries end at the helicine arteries that form part of the vascular reservoir in the corpora cavernosa. In the same area, venous drainage begins within the lacunar spaces, which drain into subtunical venules and emerge as emissary veins. The deep venous system continues with these emissary veins draining into the cavernosal veins and deep dorsal or spongiosal veins until they join either the prostatic venous plexus or the internal pudendal veins. Superficial veins coalesce to form the superficial dorsal vein, which drains into the saphenous veins (Fig. 191.1).

Erections are the result of a highly coordinated process mediated by the dynamic interplay of psychological, neuronal, hormonal, vascular, and cavernous smooth muscle systems. Innervation to the penis includes somatic nerves, responsible for sensation and contraction of the bulbocavernosus and ischiocavernosus muscles, and the parasympathetic and sympathetic autonomic nerves located at levels S2–S4 and T12–L2, respectively, which work antagonistically for tumescence or detumescence. These segments form the hypogastric and pelvic plexi. The fibers that innervate the penis compose the cavernous nerve, which travels along the posterolateral aspect of the prostate and then accompanies the membranous urethra through the urogenital diaphragm.⁸ The cavernous nerves then innervate the helicine arteries and trabecular smooth muscle.⁹

The cavernous smooth muscle plays a pivotal role in erectile function and remains contracted in the flaccid state of the penis under α -adrenergic control. The erectile process begins with sexual stimulation, which increases parasympathetic activity and stimulates the release of acetylcholine from cavernous nerve terminals and consequently nitric oxide (NO) from the endothelium of the cavernosal arteries.^{10,11} The nitric oxide (NO)/cyclic guanosine monophosphate (cGMP) pathway ultimately results in smooth muscle relaxation and increased blood flow through the penile arteries (Fig. 191.2).^{12,13} The increased blood leads to engorgement of the sinusoids, compressing subtunical venules and peripheral sinusoids thereby reducing venous outflow (veno-occlusive mechanism). The culmination of these events is an increase in intracavernosal pressure, resulting



A



B

Figure 191.1 Penile Vascular Anatomy. (A) Penile arterial supply. (B) Penile venous drainage. *a.*, artery; *v.*, vein. (Source: Wein AJ. *Campbell-Walsh Urology*, 10th ed. Philadelphia, PA: WB Saunders/Elsevier; 2011:691–692.)

in penile erection. Penile detumescence is regulated by sympathetic release of norepinephrine, stimulating alpha-receptors in the penile vasculature. This promotes contraction of both the arterioles and smooth muscles, decreasing blood inflow and opening venous outflow.

PATHOPHYSIOLOGY OF ERECTILE DYSFUNCTION

Given the complex coordination necessary to induce erections, it is easy to see how any derangements along the pathway can lead to ED. ED is classified based on likely etiology: psychogenic, neurogenic, endocrinologic, vasculogenic, and drug-induced. Though one cause may be predominant, ED is often multifactorial.

Psychogenic Erectile Dysfunction

Psychogenic (nonorganic) ED was once believed to account for most forms of ED. However, the current belief is that organic ED is more common and likely coexists in a large portion of psychogenic ED patients.¹⁴ Psychogenic ED can be further classified as either situational, caused by either partner-related, performance-related, or associated with psychological distress, or generalized psychogenic ED, which is secondary to a lack of sexual arousability.¹⁵ Psychogenic ED is often a diagnosis of exclusion. The clinical features include sudden-onset ED,

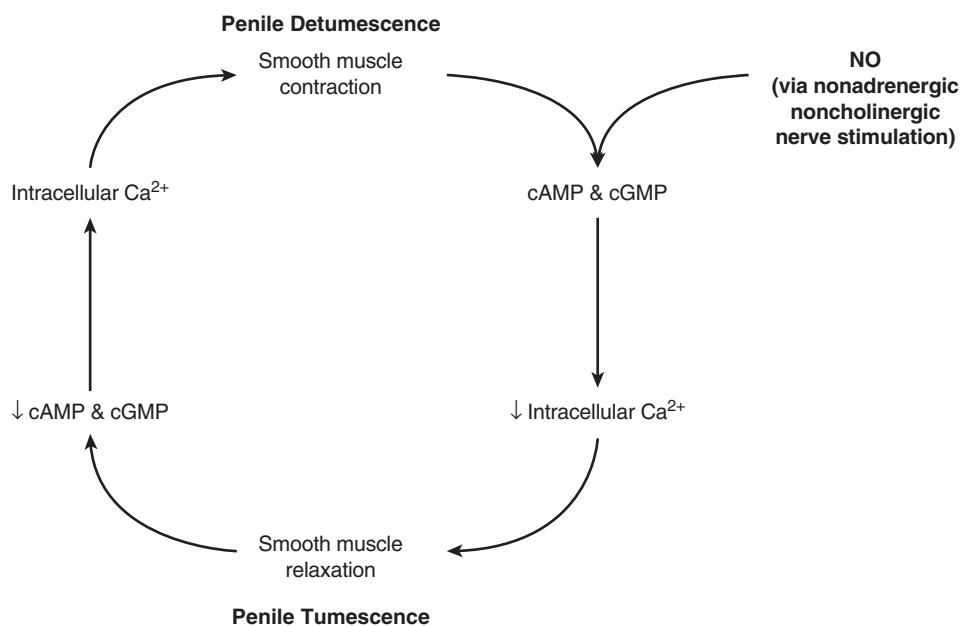


Figure 191.2 Erectile Physiology. Ca^{2+} , calcium; cAMP, cyclic adenosine monophosphate; cGMP, cyclic guanosine monophosphate; NO, nitric oxide.

with intermittency of function or a situational nature to the erectile problems, as well as reports of good nocturnal erections.¹⁶ Likewise, given the mental aspect of the disease; it is often associated with other sexual dysfunctions such as premature ejaculation or occurs during periods of increased mental stress such as depression or trauma.¹⁷

Neurogenic Erectile Dysfunction

Neurogenic ED occurs secondary to deficits along the neuroaxis from brain down to cavernosal nerves. Therefore, it can be further subclassified by the level of interference (supraspinal, spinal, and peripheral neurogenic ED).¹⁸ Supraspinal ED is generally caused by damage to the brain including tumors, stroke, Parkinson disease, or temporal lobe epilepsy.¹⁸ Diseases at the spinal level include multiple sclerosis, spinal cord injury, transverse myelitis, and lumbar disc disease or surgery.^{18–20} Peripheral causes include diseases associated with lower motor neuron lesions, trauma, pelvic pathology, and radical pelvic surgery.¹⁸ Pelvic surgery, particularly radical prostatectomy, has a high risk of causing cavernous nerve injury and has been demonstrated to have a significant impact on erectile function.²¹

Endocrinologic Erectile Dysfunction

Testosterone is a critical component in men's health and sexual behavior, though its exact relationship with ED is not well-defined and far from linear. Studies do show that testosterone replacement therapy significantly improves erectile function in hypogonadal men and therefore should be considered part of initial therapy in these cases.²² However, increasing testosterone in eugonadal men does not result in any improved function.²³ Likewise, other dysfunctions along the hypothalamic pituitary gonadal (HPG) axis including hyperprolactinemia, which causes inhibition of luteinizing hormone (LH),

hypothyroidism (low LH levels), and even profound hyperthyroidism (high estradiol levels) can all be associated with sexual dysfunction.

Vasculogenic Erectile Dysfunction

ED secondary to vascular disease is one of the most prevalent causes of organic dysfunction. It commonly occurs secondary to chronic atherosclerotic disease leading to arterial insufficiency. Suppressed perfusion of the hypogastric–cavernous–helicine leads to lower intracavernosal pressures, longer fill times, and decreased penile rigidity. In a subset of patients, this arterial insufficiency can also be caused by arterial injury (e.g., trauma). ED can also occur from inadequate venous occlusion.²⁴ Corporal veno-occlusive dysfunction (CVOD) includes development of large venous shunts draining the cavernous tissue (e.g., priapism venous shunts) or degenerative or functional changes in the tunica albuginea, as seen in Peyronie disease.^{25,26}

Studies show ED and cardiovascular disease (CVD) share common risk factors including hypertension, diabetes mellitus, hypercholesterolemia, obesity, and smoking.²⁷ A Spanish study of over 2400 patients has demonstrated each of these to be independently associated with ED and age-adjusted odds ratios of having ED of 4.0 in diabetics, 1.58 with hypertension, 1.63 with high cholesterol, 2.63 with peripheral vascular disease, and 2.5 among smokers.²⁸ Therefore, the Princeton III consensus guidelines recognize ED as a strong predictor of CVD and specifically coronary artery disease (CAD).²⁹ Additionally, ED has been described as an independent marker of cardiovascular events and all-cause mortality after adjusting for age, weight, hypertension, diabetes, hyperlipidemia, and cigarette smoking.³⁰ ED is also an independent predictor of peripheral artery disease (OR 1.97), with a significant stepwise increase in prevalence of peripheral artery disease with increasing severity of ED (28% of men with mild ED, 33% with moderate ED, and 40% with severe ED).³¹ Therefore, the American Urology

Association (AUA) guidelines recommend counseling that ED is a risk marker for underlying CVD and may warrant further evaluation and treatment.³²

Multiple theories have been proposed to explain the association between ED and CVD. The artery size hypothesis proposes that since atherosclerosis is a systemic disease, affecting all vascular beds, symptoms present at different time points according to the diameter of the arterial blood supply.³³ Therefore, a 50% stenosis of the coronary arteries would amount to a near complete occlusion of the penile arteries. Likewise, arterial insufficiency is related to lower oxygen tension in the intracavernous blood.³⁴ This has been associated with decreased prostaglandin E₁ (PGE₁) and E₂ formation, leading to transforming growth factor (TGF) β₁-induced collagenization of cavernous smooth muscle.^{18,35} Increased connective tissue limits the dynamic flexibility required for good erections and may lead to CVOD.³⁶

CVD can also lead to endothelial dysfunction furthering ED. In vascular disease, damage to the endothelium not only diminishes vasodilation and promotes inflammation, but also damages the NO pathway. Endothelial NO is critical for increased blood flow and vasodilation of the penile arteries, and necessary for erectile function. Therefore, the downstream impact of NO downregulation can substantially decrease vasodilation as well as modulation of smooth muscle cells and inhibition of cellular adhesion.^{37,38}

ED has also been demonstrated to be a strong predictor of subsequent cardiovascular events. Data from the Prostate Cancer Prevention Trial (PCPT) demonstrated that incident ED was associated with a 25% increased likelihood of subsequent cardiovascular events during the 5-year study follow-up and that men with incident ED during the study period were at a 45% increased risk of cardiovascular events.³⁹ This association was similar to the risk associated with current smoking or a family history of myocardial infarction. Another study addressing men referred with ED and vasculogenic ED documented on penile ultrasonography demonstrated a 20% rate of abnormal stress echocardiography on cardiac evaluation.⁴⁰ Therefore, vasculogenic ED is closely related to CVD and related morbidity.

Drug-Induced Erectile Dysfunction

Drug-induced ED may be a contributory factor in almost a quarter of affected cases.⁴¹ The medications most commonly associated with ED are antihypertensives, psychotropics, and antiandrogens. Among the antihypertensive, multiple studies have demonstrated significant increases in ED among patients on thiazides as compared with placebo.^{42,43} Studies of β-adrenergic antagonists demonstrate mixed results, with nonselective drugs such as propranolol showing clear associations with ED and agents with higher selectivity for the β₁ adrenoreceptor, such as acebutolol, showing a reduction in ED compared with placebo.⁴⁴ Angiotensin receptor blockers have also been consistently shown to have proerectile effects.⁴⁵ Antidepressants, specifically selective serotonin reuptake inhibitors (SSRIs), can negatively affect all steps of the sexual response

cycle, most notably ejaculatory latency. Differences are noted in the incidence of ED among different medications, with higher rates noted in patients on paroxetine.⁴⁶ Antiandrogens cause partial or complete blockade of circulating androgens either by inhibiting their production or antagonism at the level of the androgen receptor; they are frequently used in the treatment of prostate cancer. Use of these medications is generally associated with decreased sexual desire, although medical castration with luteinizing hormone-releasing hormone agonists/antagonists and nonsteroidal anti-androgens such as flutamide or bicalutamide can also contribute to veno-occlusive dysfunction.⁴⁷

ASSESSMENT OF ERECTILE DYSFUNCTION

History and Physical Examination

The cornerstone of ED evaluation involves a detailed history taken from the patient and when possible the partner. This evaluation should include a thorough medical, psychosocial, and sexual history, including detailed review of current and recent medications.^{48,49} Given the sensitive subject matter, discussions should be done privately and the clinician should remain nonjudgmental while displaying trust and concern. Likewise, validated questionnaires such as the International Index of Erectile Function (IIEF), the abridged five-item version of the IIEF, and the Sexual Health Inventory in Men (SHIM), are useful to initiate discussion, document the degree of disease, and guide/track future management.^{32,50,51} Since ED is more often multifactorial it is important to explore for possibly related underlying medical conditions or prior medical history (trauma or pediatric surgeries) while also assessing the intra- and interpersonal context of the patient's sexual function.

The physical examination is equally important. This includes basic anthropometrics, body habitus, and evaluation of blood pressure, heart rate, and pulses. More focused evaluation includes male secondary sex characteristics and a genital examination.^{52,53} This would include examination of the penis for size and position of the urethral meatus (assessing for micropenis or congenital chordee), presence of tunical plaques (Peyronie disease), evaluation of testicular size and consistency (hypogonadism), and a digital rectal examination in the appropriately aged man.⁵³ In a patient with suspected vascular disease particular attention should be paid to carotid bruits, fundal arterial changes, abdominal pulsations, and femoral and pedal pulses.²⁹

Laboratory Evaluation and Adjunctive Testing

Additional laboratory testing may be also indicated in select patients either to work up or to confirm the presence of underlying diseases. This includes fasting blood glucose, a lipid profile, and morning serum testosterone levels.^{32,53} Given the high correlation of CVD and ED the Princeton III consensus statement recommends that all men over 30 years old with organic ED be considered at increased CVD risk until otherwise proven. They should receive further testing including resting

electrocardiogram and serum creatinine level.²⁹ For patients in whom CVD is present or suspected, consultation with a cardiologist or primary care physician is advised and additional work-up may then be warranted.

Noted largely for historic purposes, nocturnal penile tumescence (NPT) monitoring, was used to study the quality of nocturnal erectile function, either in a sleep laboratory setting or with a portable, home use device such as the Rigiscan (Timm Medical Technologies, Inc., Minneapolis, MN).⁵⁴

Vascular Evaluation

Vascular evaluations assess the corporeal structure's ability for arterial blood inflow, engorgement, and retention. Therefore, diagnostically these studies aim to detect elements of arterial insufficiency and CVOD.

Office Injection Testing

Intracavernosal injection (ICI) testing is a very basic method of erectile assessment. It is performed by injection of a vasodilatory drug (or combination of drugs), combined with genital or audiovisual stimulation, in a clinical setting. The erectile response is then evaluated by a clinician to assess both the rigidity and duration of response. After the test, patients are monitored for detumescence and those who do not spontaneously detumesce within the hour are given intracavernous injections of a diluted phenylephrine solution. Vasodilatory medications used may include PGE₁ alone or in a combination: papaverine, and phentolamine. Although ICI testing was at one point used to differentiate organic from psychogenic ED, currently its value is to define a functional veno-occlusive mechanism in men who develop rigid, sustained erections and serves as a method to elicit erections for more advanced imaging and procedures.⁵⁵ Failure to obtain a rigid erection may indicate vascular disease but may also be the result of an excessive sympathetic response associated with patient anxiety, needle phobia, or inadequate dosing during ICI testing.⁵⁶

Duplex Ultrasonography of the Penis

Duplex Doppler ultrasound (DDUS) of the penis is the most reliable noninvasive diagnostic method for evaluating ED. Beyond ICI it adds an imaging component and quantifies blood flow thereby providing key functional information. Data from DDUS can guide therapy for poor oral ED therapy responders, differentiate psychogenic versus organic ED, aid in the evaluation of a young man with primary ED or a history of pelvic trauma, and provide data prompting cardiovascular evaluation in the man with vasculogenic ED without overt CVD risk factors.⁵⁷

Given the private nature of eliciting erectile function, DDUS should be performed in a quiet, comfortable room, isolated from intrusion and distraction. An erection is then pharmacologically induced using ICI. If an adequate erection is not achieved, patients should be redosed to ensure maximal smooth muscle relaxation for accurate results. DDUS is performed using a high-resolution (7.5–12 MHz) linear array ultrasound transducer with color pulse Doppler. In the supine

position, the penis is scanned to find the left and right cavernosal arteries. Peak systolic velocity (PSV), end-diastolic velocity (EDV) as well as resistive indices (RI) are measured. The penis can also be evaluated using B-mode images to observe for tunical plaques (Peyronie disease), fibrosis, and calcifications in either the vasculature or the plaque itself.

Different cutoffs have been explored for normal PSV values, but generally a PSV below 25 cm/s suggests cavernous arterial insufficiency.⁵⁸ This level has a sensitivity of 100% and specificity of 86% among patients with abnormalities on pudendal angiography.⁵⁹ A PSV cutoff of 35 cm/s decreases the sensitivity to 76%, but increases specificity to 92%.⁶⁰ Significant asymmetry of PSV, greater than 10 cm/s difference between sides, suggests a significant atherosclerotic lesion or iatrogenic/surgical cause for decrease in arterial flow.⁶⁰ Veno-occlusive dysfunction is evaluated using EDV in the presence of normal arterial inflow. Generally EDV of 6 cm/s or more indicates CVOD (Fig. 191.3).^{57,60} Given concerns for the specificity of EDV alone in the diagnosis of CVOD for patients with arterial insufficiency, RI is also used with a cutoff of less than 0.75 as abnormal.^{57,61} Conversely, due to the way RI is calculated and dependent on PSV, some patients with good arterial inflow and significantly elevated EDV may still have values within the normal range. Therefore, RI alone should also not be used for diagnosis.

Selective Internal Pudendal Angiography/ Penile Angiography

Penile arteriography is usually reserved for patients being considered for penile revascularization surgery. These men usually have: (1) pure arterial insufficiency without CVOD; (2) a focal occlusion of one or both common penile or cavernosal arteries; (3) perforating branches traveling from the dorsal to the cavernosal artery; (4) at least one patent inferior epigastric artery (IEA) of sufficient length to serve as a donor artery; and (5) at least one patent dorsal artery to act as a recipient artery. Another indication for angiography is for men with high-flow priapism, where angio-embolization of the cavernosal artery–corpus cavernosal fistula is potentially curative.

Both technically challenging and invasive, pudendal/penile angiography requires an endovascular specialist to cannulate both the internal pudendal arteries and the inferior epigastric arteries, which are used for revascularization.⁵⁷ Arterial inflow to the penis should be maximized with the use of ICI vasoactive agent, administered prior to contrast injection. The timing of injection should allow for maximal arterial inflow to the penis but not at such a time point as to cause reduced arterial flow at full rigidity. Selective catheterization of the internal pudendal artery is performed to increase detail (Fig. 191.4).⁶² Despite the anatomic detail provided by arteriography, it represents only a road map and is not a functional test. Thus, the results must be interpreted in light of the functional results obtained using DDUS.

Historical Studies of Penile Blood Flow

There are several other methods developed to investigate penile blood flow, but these have either fallen out of favor or been

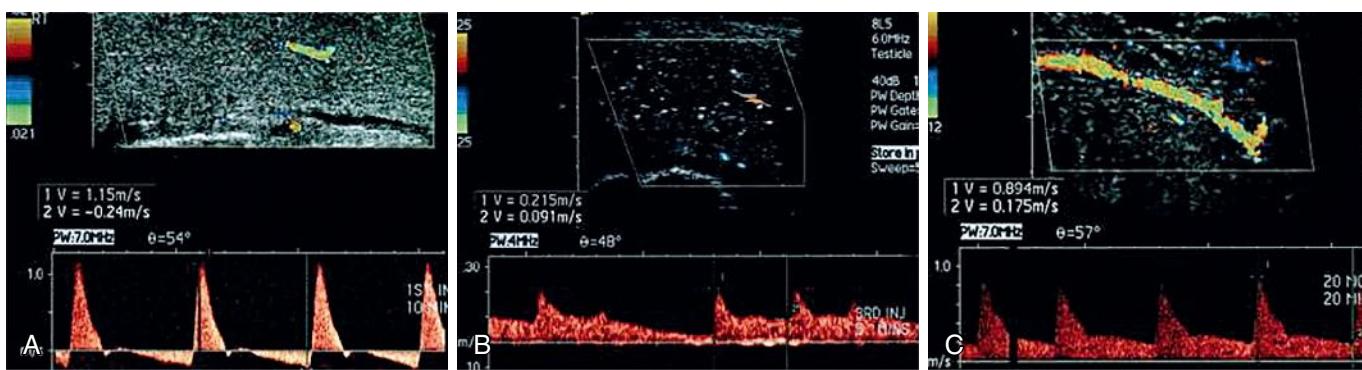


Figure 191.3 Color Doppler Duplex Ultrasound of the Penis. (A) Normal erectile hemodynamics: Doppler waveform analysis demonstrating cavernosal artery peak systolic velocity of 115 cm/s and end-diastolic velocity of -24 cm/s, representing normal erectile hemodynamics. (B) Mixed vasculogenic erectile dysfunction (ED): Doppler waveform analysis demonstrating cavernosal artery peak systolic velocity of 21.5 cm/s and end-diastolic velocity of 9.1 cm/s, indicating the presence of arterial insufficiency and corporal veno-occlusive dysfunction (CVOD). (C) Corporal veno-occlusive dysfunction: Doppler waveform analysis demonstrating cavernosal artery peak systolic velocity of 89.4 cm/s, and end-diastolic velocity of 17.5 cm/s. End-diastolic velocity greater than 6 cm/s indicates corporal veno-occlusive dysfunction. (Source: Wilkins CJ, Sripasad S, Sidhu PS. Colour Doppler ultrasound of the penis. *Clin Radiol.* 2003;58(7):514–523.)

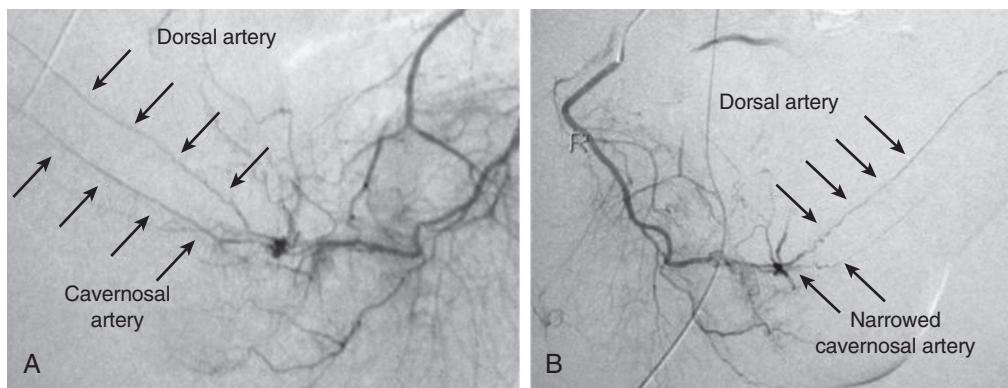


Figure 191.4 Selective Internal Pudendal Angiography. (A) Normal internal pudendal angiogram demonstrating patent dorsal and cavernosal arteries of normal caliber. (B) Internal pudendal angiogram demonstrating patent dorsal artery with occlusion of the cavernosal artery. (Images courtesy Dr. Irwin Goldstein, MD, San Diego Sexual Medicine, Alvarado Hospital, San Diego, CA.)

found to be invalid. Studies that assess pressure differences in a flaccid penis, such as penile brachial pressure index (PBI), are no longer used since they do not assess the hemodynamic properties of an erection.⁶³ Likewise, the clinical relevance of penile plethysmography is also questionable, though some evidence suggests it may be helpful in assessing penile vascular endothelial function.⁶⁴ Other imaging modalities like penile magnetic resonance imaging, radio isotopic penography, and penile near infrared spectrophotometry have been explored, but have largely not been implemented into meaningful clinical practice.^{65–67}

Dynamic infusion cavernosometry and cavernosography (DICC) is worth mentioning since it is the most accurate assessment of erectile hemodynamics. However, given the invasive nature and need for specialized equipment, its clinical use is limited and has largely been replaced by DDUS.^{68,69}

TREATMENT OF ERECTILE DYSFUNCTION

Current treatment recommendations are based on a shared decision-making approach so that the patient and partner are presented with all potential treatment options up front. Then a balanced discussion is led by the clinician to review potential advantages and disadvantages of each treatment modality, tying in how they correspond to the patient's clinical situation and expectations. This approach is unlike prior structured treatment algorithms, which were based largely on risk, and reflects the goal-directed nature of ED management.⁴⁸ Studies between randomized clinical trials and routine care have found that through shared decision making, patients are more knowledgeable about their therapy and have higher congruence between the chosen therapy and their own values.⁷⁰ This

change in approach is also reflected in the most recent AUA guidelines for ED.³²

Lifestyle Modifications

After ruling out adverse medication effects, patients with organic ED should first optimize lifestyle modifications. Since a number of comorbidities like diabetes, hypertension, and CVD are heavily associated with ED, management of these diseases may prevent worsening or ameliorate the extent of ED. Next, patients should target the factors commonly associated with ED and frequently CVD, including smoking cessation, weight loss, and increased aerobic exercise. Numerous trials have demonstrated improvements in erectile function with smoking cessation.^{71,72} Likewise, weight loss can be especially helpful. In a randomized controlled trial of obese Italian men, participants were randomized to receive: (1) intervention based on detailed advice for a 10% weight loss (including diet and exercise), or (2) control, where no advice was given. At 2-year follow-up, the patients in the intervention group had significant weight loss and reported significant improvements in erectile function defined by the IIEF.⁷³

For patients with predominantly psychogenic ED, psychosexual therapy intervening on anxiety, sensate focus, interpersonal therapy, and couples communication among other techniques may be useful.⁷⁴ Likewise, integration of these methods with medical management has also proved successful.^{74,75}

Phosphodiesterase Type 5 Inhibitors

In 1998, the PDE5 inhibitor sildenafil citrate (Viagra, Pfizer Inc., New York) became the first oral therapy for ED approved by the U.S. Food and Drug Administration (FDA). This medication dramatically changed management of ED compared with historic therapies. Utilizing similar mechanisms of action, four PDE5 inhibitors have since been approved by the FDA: sildenafil, vardenafil hydrochloride (Levitra or Staxyn, Bayer Pharmaceuticals Corp, West Haven, CT, and GlaxoSmithKline, Philadelphia, PA); tadalafil (Cialis, Lilly LLC, Indianapolis, IN); and avanafil (Stendra, Vivus Inc., Mountain View, CA) (Table 191.1).

Mechanism of Action and Means of Use

As their names suggest, PDE5 inhibitors work by inhibiting the PDE5 enzyme, which degrades cGMP, the downstream effector of NO. Prolonged activity of cGMP decreases intracellular calcium concentration and maintains smooth muscle relaxation. Therefore, this process augments but does not induce erectile response. The choice of the optimal PDE5 inhibitor for the individual patient is dependent upon the patient's sexual dynamics, particularly the frequency and predictability of sexual activity as well as the presence of comorbid conditions. For instance, a daily dose of a tadalafil is approved for concomitant treatment of benign prostatic hypertrophy (BPH).⁷⁶

Outcomes

Overall, PDE5 inhibitors have demonstrated strong efficacy and tolerability among patients. Collectively, these medications

TABLE 191.1

Pharmacokinetics of FDA-Approved Phosphodiesterase Type 5 Inhibitors

Generic Name	Trade Name	T _{max} (min)	T _½ (h)	Bioavailability
Sildenafil	Viagra	60	4	40%
Vardenafil	Levitra, Staxyn	42	4.5	15%
Tadalafil	Cialis	120	17.5	Not tested
Avanafil	Stendra	45	5.1	30%

FDA, U.S. Food and Drug Administration; T_{max}, time required to attain maximal plasma concentration; T_½, half-life, time required for elimination of half of the medication from plasma.

report a roughly 70% successful sexual intercourse rate.⁷⁷ In pooled estimates, use of sildenafil, vardenafil, and tadalafil had an approximate 2.5-fold increased likelihood of improvement in erectile quality compared to placebo in general ED patients.⁷⁷ The ability of these agents to permit sexual intercourse depends upon the underlying etiology of ED, with men with psychogenic ED responding better than men with veno-occlusive dysfunction.⁷⁸ Optimization of all the medications includes coordinating dosing with food intake to improve absorption, titrating drug dosing as needed and repeat trials of medication, and appropriate sexual stimulation to first induce the erectile process.

Adverse Events and Contraindications

Side-effect profiles are similar among PDE5 inhibitors. Most commonly reported include headache (7%–16%), flushing (4%–10%), nasal congestion (3%–4%), and heartburn (4%–10%).^{77,79} Distinct from tadalafil and avanafil, sildenafil and vardenafil have some cross activity at the PDE6 receptor, located in the retina, explaining the greater complaint of visual disturbances in those medications.⁷⁷ Ischemic nonarteritic optic neuropathy (NAION) and priapism have been rarely reported among users of PDE5 inhibitors. However, systematic reviews show no clear link, though patients should be counseled to seek care if similar symptoms occur.^{77,79}

PDE5 inhibitors are contraindicated in patients who use nitrate medications owing to a risk of severe hypotension.⁸⁰ Although not specifically indicated, patients with serious CVD should use PDE5 inhibitors with caution, primarily due to risks associated specifically with physical exertion (sexual activity) as opposed to risks posed by the PDE5 inhibitors themselves. Vardenafil use is contraindicated among patients taking type 1A or type 3 antiarrhythmics and among those with prolonged QT syndrome.⁸¹

Intracavernosal Injection Therapy

Intracavernosal injection (ICI) therapy was first described for the treatment of ED in 1982, and was the catalyst for the exploration of medical therapies for the treatment of ED.⁸² ICI is a safe and recognized treatment for ED effective in up to 90% of ED patients and in more than 50% of those who fail PDE5 inhibitors.

Mechanism of Action and Means of Use

There are three main medications used in ICI: PGE₁, phentolamine, and papaverine. These medications are frequently used in combination (Trimix – PGE₁, phentolamine, papaverine; Bimix – phentolamine, papaverine) for increased efficacy and potency. PGE₁ (the FDA-approved version is known as alprostadil and marketed as Caverject [Pfizer, New York, NY] or Viradel/Edex [Schwartz Pharma, Milwaukee, WI]) activates prostaglandin receptors, resulting in an increase in the intracellular concentration of cyclic adenosine monophosphate (cAMP) in the cavernosal smooth muscle, which causes tissue relaxation.⁸³ Phentolamine is a nonselective α -adrenoceptor antagonist, which induces relaxation of cavernosal smooth muscle.⁸⁴ Papaverine acts as a nonspecific PDE inhibitor and initiates an increase in both intracellular cAMP and cGMP, resulting in penile erection.⁸² Use of ICI therapy involves direct injection of these vasoactive medications into the corpora cavernosa by the patient. The patient is trained to self-administer the medication at home. Responses are generally seen within 5 to 10 minutes, with dose-dependent changes in duration and rigidity of the erection.

Outcomes

Used in patients who generally have ED refractory to PDE5 inhibitor therapy, ICI therapy is highly efficacious, with reported 89% response rate (patients able to achieve a penetration-hardness erection capable of sexual intercourse). However, there is at least a 24% discontinuation rate across ED patients at 36-month follow-up.⁸⁵ Discontinuation rates and reasons vary according to the patient population and can be secondary to recovery of erections following radical prostatectomy, progression of ED such that the patient requires escalating medication doses, and dissatisfaction with ICI due to loss of spontaneity. Patients with a history of radical prostatectomy, pelvic radiation therapy, diabetes, or a long preexisting history (>5 years) of untreated ED are more likely to fail ICI therapy.⁸⁵ True failure of ICI therapy is generally a clinical indicator of the presence of cavernosal smooth muscle damage (veno-occlusive dysfunction).

Adverse Events and Contraindications

The predominant concern about ICI therapy is priapism. Priapism rates with ICI vary in the literature from 0.25% to 7.3%, with figures in the lower part of the range in centers with careful patient selection and superior patient education and counseling.⁸⁵ Reversal of a prolonged erection is accomplished with the intracavernosal administration of an α -adrenergic agonist agent (phenylephrine). Other minor adverse effects include pain at injection site (11%), ecchymosis/hematoma (1.5%), and prolonged erection (1%–5%).⁸⁶

Given the need for injections, ICI is contraindicated in men with severe coagulopathy or unstable cardiovascular disease, those with limited manual dexterity (though partner can be taught injections), and those using monoamine oxidase inhibitors given the risk of hypertensive crisis if a α -adrenergic agonist were used to reverse priapism.

Intraurethral PGE₁ Suppository

Intraurethral PGE₁ in the form of alprostadil (known as Medicated Urethral System for Erection, or MUSE [Meda Pharmaceuticals Inc., Somerset, NJ]) was an alternative method for minimally invasive drug delivery. A small plastic device is inserted into the urethral meatus by the patient, and the pellet is deposited into the urethra. The medication is then absorbed through the urethral mucosa into the corpora cavernosa, where the PGE₁ promotes tissue relaxation. The efficacy of MUSE is variable, with small trials suggesting superior efficacy for ICI versus MUSE.^{87,88} Penile pain is reported in 33% of patients, with approximately 5% reporting urethral bleeding.⁸⁸

Vacuum Constriction Devices

Vacuum erection devices (VEDs) have been approved by the FDA since 1982 and serve as a treatment for those who decline both oral and ICI therapy.⁸⁹ VEDs consist of a suction cylinder and pump, which creates negative pressure around the corporal bodies, thereby engorging them with venous blood. A prescribed compression band is placed around the base of the penis to restrict venous drainage and maintain rigidity. Compression rings cannot be left in place for longer than 30 minutes in order to avoid ischemic injury to cavernosal tissue. Efficacy rates for VED have been reported as high as 90%, however, given the cumbersome nature of the device and the non-physiologic nature of the erection, satisfaction rates are far lower ranging from 30% to 70%.⁹⁰

Penile Implant Surgery

Penile implant or penile prosthesis surgery was first described 50 years ago and after several improvements in product design and surgical technique is the gold standard for invasive surgical treatment of erectile dysfunction.^{90,91} It is an excellent option for patients who have attempted and failed to achieve satisfactory results with other modalities, have grown weary of second-line therapy use (ICI, intraurethral alprostadil, VED), or are not candidates for invasive vascular procedures. Implants act solely to allow a patient to achieve a rigid erection on demand and have no effect on the patient's sensation, or their ability to ejaculate or achieve orgasm.

Technical Considerations

There are two types of implants: malleable (or semirigid) and hydraulic (or inflatable). Semirigid prostheses consist of two malleable rods that are placed into the corpora cavernosa; they can be manually shaped by the patient at the onset of sexual activity and reshaped after sexual relations. Inflatable implants are further subdivided based on the configuration of the prosthesis: a two-piece inflatable, which consists of two cylinders attached to a scrotal pump; and a three-piece inflatable, which consists of two cylinders attached to a scrotal pump and a separate reservoir, which is placed extraperitoneally in the space of Retzius. Choice of implant is both surgeon- and patient-dependent. In the modern era most patients prefer three-piece



Figure 191.5 Penile Revascularization. (A) Isolated and mobilized dorsal artery of the penis just distal to the funiform ligament. (B) Transected inferior epigastric artery transferred to the dorsum of the penis. (C) Microsurgical anastomosis between the inferior epigastric artery and the dorsal penile artery. (Images courtesy Dr. Irwin Goldstein, MD, San Diego Sexual Medicine, Alvarado Hospital, San Diego, CA.)

implants, given their superior rigidity and girth, better concealability and zero rigidity in the deflated state. Two-piece implants are generally placed in patients who have an obliterated extraperitoneal space (no location for reservoir), have small bowel loops abutting the deep inguinal ring (patients who have undergone radical cystoprostatectomy), have a renal transplant, or have poor grip strength and may have difficulty deflating a three-piece implant. Malleable prostheses are not commonly used, though may serve to hold the corporal space open prior to the placement of a three-piece implant in the case of revision surgery.^{90,92}

Outcomes and Adverse Events

Both patient and partner satisfaction are generally high following penile implant surgery, with satisfaction rates varying between 75% and 97% depending on the patient population and implant used.⁹⁰ This is mostly because of the rigidity and spontaneity these devices provide. The most significant complication of penile implant surgery is infection, with data from the most recent generation of penile prostheses varying from 0.7% to 3%.^{91,93} Management requires complete device removal, complicating subsequent reimplantation due to scarring and decreased penile length and girth. Protocols for salvage implantation (infected device removal with simultaneous reimplantation) demonstrate an 84% success rate.⁹⁴ Other complications include component erosion (most often occurring in the infected implant), reservoir herniation, and cylinder migration. Like all mechanical devices, penile prosthesis eventually break down though five-year mechanical failure rates are low, ranging from 0% to 9%.⁹⁰

Penile Revascularization Surgery

Penile revascularization (penile artery bypass surgery) serves as one of two modalities with the potential to cure the ED without the future need of vasoactive medications or implant surgery (the other is crural ligation surgery). However, penile revascularization, unlike revascularization in patients with coronary disease or peripheral arterial disease, is not successful in patients with CVD, as these patients have developed underlying endothelial dysfunction and corporal smooth muscle

dysfunction as part of the disease progression.⁹⁰ In addition, patients with ED secondary to diffuse atherosclerosis will already have significant disease in the small penile arteries, thereby limiting adequate recipient vessels.⁹⁵ Thus patient selection for revascularization surgery is critically important and surgery is most often successful only in highly selected young men with an isolated arterial stenosis following pelvic trauma. Inclusion criteria for consideration for revascularization generally include age (most authorities would not revascularize men older than 40 years old), absence of vascular risk factors, absence of evidence for other etiologies of ED, absence of evidence of CVOD with evidence of pure arterial insufficiency (as described earlier), and the presence of focal occlusive disease in the common penile artery or cavernosal artery that is amenable to distal bypass by the previously outlined arteriographic criteria.^{90,96}

Technical Considerations

Although many bypass procedures have been identified (historically over 20 procedures in total), the most commonly utilized approach is to perform an inferior epigastric artery (IEA) to dorsal artery bypass.⁹⁵ Given the paucity of data pertaining to the use of venous target vessels, the arterial revascularization adapted from Goldstein et al. is reported here (Fig. 191.5).^{96,97}

A variety of scrotal incisions can be made to expose the dorsal artery at the base of the penile shaft. An abdominal incision is made to expose the rectus muscle, which is reflected medially, and the IEA is identified. It is then mobilized from its origin near the external iliac artery to a point near the umbilicus, where it is transected. It is then transferred through the posterior wall of the inguinal canal onto the dorsum of the penis. The abdominal incision is then closed. The adventitial layer of the distal aspect of the IEA and proximal aspect of the dorsal artery are stripped with micro-scissors, and a microsurgical simple interrupted anastomosis is performed with 10-0 nylon sutures. Following completion of the anastomosis and after achieving complete hemostasis, the penis is replaced in its anatomic position and the wound is closed.

Endovascular management of focal arterial occlusion has also been previously studied, with the majority of the reports published in the 1980s using balloon angioplasty without stenting.⁹⁵ Results are variable and poorly defined.

TABLE 191.2

Most Recent Published Reports on Outcomes of Penile Revascularization

Report	Number of Patients	Follow-Up (Months)	Success Rate (%)
Vardi et al. ⁹⁹	35	60	49
Kawanishi et al. ¹⁰⁰	51	60	66
Munarriz et al. ⁹⁸	71	35	55
Kayigil et al. ¹⁰¹	110	60	64
Zuckerman et al. ¹⁰³	17	37	82

Outcomes and Complications

Data on outcomes following penile revascularization are sparse, since most studies are limited by inclusion and exclusion criteria, lack of validated markers to evaluate success, and generally short-term follow-up.⁹⁵ Additionally, published studies have discrepant patient populations, use different anastomotic and bypass techniques, and frequently do not assess long-term patency. Among these studies, subjective success rates vary depending on type or revascularization procedure, definition of successful outcome, and patient population; they range from 50% to 82% (Table 191.2).^{98–103} Nonetheless, practice guidelines from the AUA do consider penile vascularization for young men with ED and focal pelvic/penile arterial occlusion and without generalized vascular disease or veno-occlusive dysfunction.³² Looking specifically at arterial revascularization surgery, a recent review of 71 patients with 34-month mean follow-up demonstrated that 55% of men had IIEF scores qualifying them as having normal erectile function without the use of a PDE5 inhibitor.⁹⁸

The most frequently reported complications include failure to improve erectile function, inguinal hernias, penile shortening, loss of penile sensation, and – in cases where arterialization of the dorsal vein was used – glans hyperemia.^{91,98}

Surgery for Veno-Occlusive Dysfunction

Current guidelines from the AUA, given an absence of clear data demonstrating benefit, do not recommend surgery to limit the venous outflow from the penis.^{32,104} CVOD is currently considered the result of damage to the cavernosal smooth muscle; thus the venous leak is a symptom of an underlying pathology that is not generally corrected by venous ligation.^{88,92} Despite this, venous ligation surgery has been attempted for decades, with poor long-term results. Nonetheless, there are limited studies that show crural ligation surgery among highly selected young patients with isolated crural venous leak (generally secondary to blunt perineal trauma) has some medium-term efficacy.¹⁰⁵ One study of 15 patients with a mean age of 29 years and isolated crural venous leak demonstrated on DICC resulted in unassisted sexual intercourse in 71% of men after crural ligation surgery and 93% of men reported improved erectile function as quantified by the IIEF.¹⁰⁵ Ultimately, the available data on penile venous surgery is limited and unlikely

to result in long-term successful management of ED, delaying more definitive treatments.

FUTURE THERAPEUTIC STRATEGIES

Despite the great strides in ED treatment developed in the past two decades, there remains a group of patients who either cannot tolerate these therapies or have contraindications. These include patients with late stage peripheral vascular disease and post-prostatectomy ED with severe ED.³² Likewise, the shortcoming of current therapies is the focus on addressing only symptoms of ED. Therefore, a growing area of interest is restorative therapies, aimed at repairing or replacing diseased tissue through regenerative tissue engineering or related technologies. Likewise, for the vascular patient in particular, there have been several recent trials looking at the potential of microvascular stenting and balloon angioplasty.¹⁰⁶

Restorative Therapies

Restorative therapies provide an exciting new paradigm of ED treatment. The goal of all these interventions is to upregulate pathways for reducing inflammation, improving wound healing, driving angiogenesis, and recruiting growth factors and chemokines. Ultimately, these pathways restore the structure and function of the diseased tissue, with the promise of “curing” ED.^{107,108} Current technologies include penile injections of platelet rich plasma (PRP), stem cell therapy (SCT), and stromal vascular fraction (SVF). Despite theoretically sound mechanisms of action and promising preclinical data, current restorative medicine remains largely investigational with only a handful of small clinical trials providing evidence of feasibility, tolerability, and safety.^{108,109} Nonetheless, this is a rapidly growing area of study which may completely change the way we treat ED in the future.

Low-Intensity Shockwave Therapy

Similar to restorative therapies, low-intensity shockwave therapy (LiSWT) is a related technology that applies shockwaves to diseased tissue, which leads to neo-angiogenesis, recruitment of progenitor cells, vasodilation and increase in NO, and even nerve regeneration.¹¹⁰ LiSWT is already being used in tissue wound healing and bone fractures and has wide applications throughout medicine. There is considerable interest in the treatment of ED. To date there have been several clinical trials demonstrating not only tolerability and safety, but suggesting efficacy as well.^{107,108} In a comprehensive meta-analysis of many of these studies, Lu et al. looked at 14 trials, of which seven were randomized control, including 833 patients, and found significant improvement of the IIEF (mean difference 2.00) and EHS (risk difference 0.16). They found that patients with mild–moderate ED had a better response, with efficacy of at least 3 months and a strong correlation between energy flux, number of shocks, and duration of treatment with clinical outcomes.¹¹¹ Though these results are exciting and several products for LiSWT have been brought to market, further research

regarding the type of shockwaves, duration of treatment, and even in-depth basic science work on mechanisms of action are still needed before widespread clinical acceptance.^{108,109}

Endovascular Stenting

Cardiovascular stenting provides an interesting solution to arteriogenic ED given the success seen with similar procedures in coronary artery disease.^{106,112} The ZEN trial (Zotarolimus-Eluting Peripheral Stent System for the Treatment of ED in Males with Suboptimal Response to PDE5 Inhibitors) trial was published in late 2012, looking at the safety and feasibility of a balloon-expandable drug-eluting stent for patients with focal atherosclerotic lesions of internal pudendal artery who had suboptimal response to PDE5 inhibitors. Forty-five lesions were treated in 30 subjects and PSV increased from baseline by 14.4 ± 10.7 cm/s at 30 days and 22.5 ± 23.7 cm at 6 months.¹¹³ Another study, PERFECT-2 (Pelvic Revascularization for Arteriogenic Erectile Dysfunction), published in 2016 looked at isolated penile artery stenosis treated by balloon angioplasty. In 22 patients there were 34 isolated penile stenoses. Though 31 of the 34 lesions were successfully dilated, by 8 months, 14 of the 34 lesions in 13 of 22 patients had CTA documented binary restenosis. Ultimately, at 1 year, there was sustained clinical success in 11 of the 22 patients.¹¹⁴ Though these studies suggest that drug-eluting stents and balloon angioplasty may be safely performed and might be efficacious, there is no long-term data fully evaluating the outcomes. Therefore, before clinical acceptance more studies are needed, though patient accrual will be difficult given the limitations in patient selection criteria.

ERECTILE DYSFUNCTION IN MEN UNDERGOING VASCULAR SURGERY

Prevalence of Erectile Dysfunction in the Vascular Surgery Population

Given the clear link between ED and vascular disease, it is not surprising that patients who present with vascular disease may have elements of ED. In an analysis of four studies involving 1476 men with heart disease, myocardial infarction, or vascular surgery, the incidence of ED was found to range from 39% to 64%.¹¹⁵ Another study looking at the prevalence of ED among a population of 137 vascular surgery patients, identified a 90% rate of ED (defined as IIEF erectile function domain score less than 25), with moderate or severe ED in 70% of cases.¹¹⁶ Among patients with an untreated AAA, the rate of moderate or severe ED was 82%. Another study looking at self-reports of preoperative sexual dysfunction in 56 AAA patients using SHIM scores described a 27% rate of ED in patients undergoing elective open AAA repair, 63% for those undergoing endovascular repair of AAA (EVAR), and 45% for those undergoing open repair for AAA rupture.¹¹⁷ It should be noted that these figures are considerably higher than historically reported preoperative ED rates in AAA and aortoiliac occlusive disease patients (ranging from 22% to 39%), but

previous studies had variable definitions of ED without the use of validated questionnaires or defined ED as only the most severe ED cases (IIEF erectile function domain score <11).^{118,119}

Erectile Dysfunction Following Definitive Therapy

Several studies have been done looking at erectile outcomes following vascular therapies. In the landmark Aneurysm Detection and Management (ADAM) study, 1136 patients with 4.0- to 5.4-cm AAAs were randomized to surveillance versus immediate open repair.¹²⁰ Patients were evaluated by a nurse to rate their sexual function as normal versus impotent without the use of questionnaires. "Impotence" rates were significantly higher across patients randomized to immediate repair from 18 months to 4-year follow-up. Across groups, a steady and significant increase in ED was also noted, suggesting disease progression over time. Another rigorously performed study randomized 881 AAA patients to EVAR and open AAA repair and evaluated sexual function outcomes by use of the IIEF erectile function domain score.¹²¹ Of note, patients had significant preoperative ED at baseline, with a mean IIEF score of 11.4 for the EVAR and 10.3 for the open group. At 1 year, there was a mean decline in IIEF of 2.5 and 2.3 for the EVAR and open groups, respectively, and a decline of 3 and 2.9 points, respectively, at 2 years, demonstrating no significant difference between the EVAR and open AAA repair groups. In a vasculopathic population such a decrease is likely age-related and not related to the surgical intervention.

ED following vascular repair has been associated with bilateral internal iliac interruption during EVAR, as would be expected.¹²² Internal iliac embolization prior to EVAR has also demonstrated significant increases in reported ED rates across a number of studies.¹²³ Therefore preservation of the internal iliac artery has been recommended when possible. In addition, adjunct internal iliac revascularization prior to EVAR may improve erectile outcomes, with reports of no change in sexual function and even improvement in erectile ability in a single patient following this procedure.¹²⁴

In light of these findings, it would be prudent for the vascular surgeon to note erectile function preoperatively and counsel regarding erectile function changes following vascular surgical procedures. Patients without prior knowledge of ED may not understand the important role vascular flow has on erectile function and therefore, are unaware of asking about ED with regards to the procedure. A previous report has shown that 91% of patients undergoing open AAA repair or EVAR do not recall receiving preoperative information regarding the risk of a possible negative impact on sexual function.¹²⁵

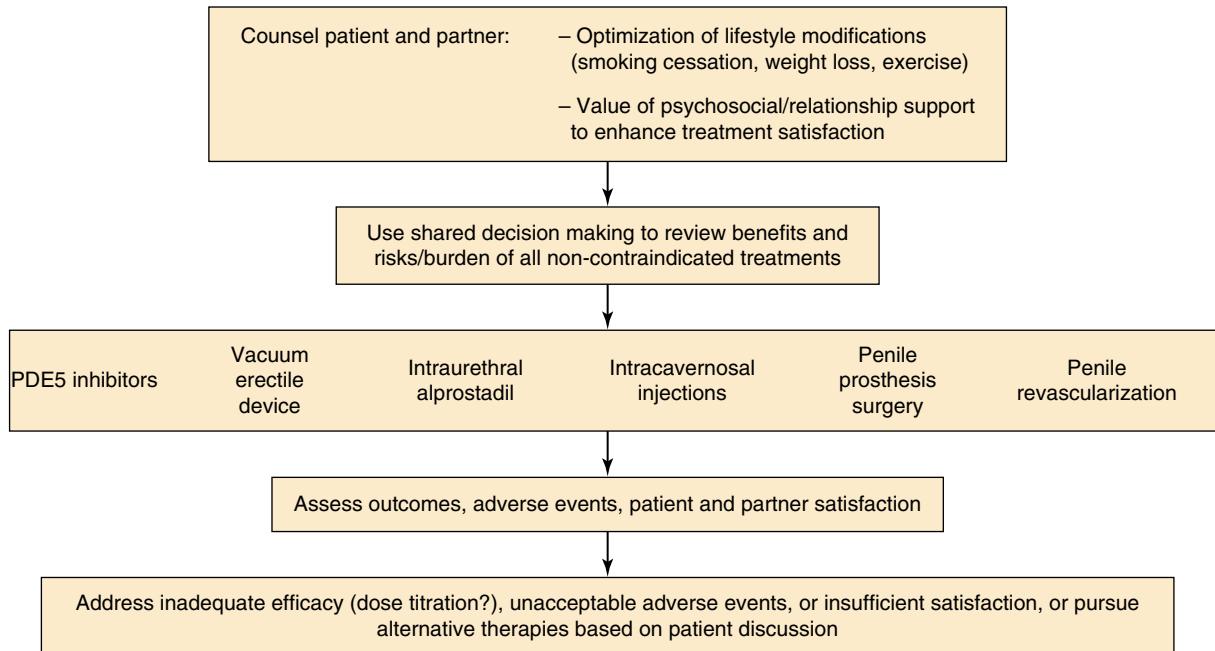
CONCLUSIONS

ED is a highly prevalent condition and a potential marker for significant vascular disease. There are several possible etiologies for dysfunction, though most often it is multifactorial with certain predominant features. History and physical examination are often enough to begin treatment discussion, but

diagnostic evaluation in a subset of patients may be helpful in guiding therapy and identifying patients for vascular interventions. Current treatment algorithm is driven by shared decision making ranging from oral medications to penile prosthetic surgery. Given the close association with vascular disease, vascular

surgeons should be aware of ED when counseling patients regarding surgical interventions. Further studies using validated instruments to quantify erectile function are needed to fully appreciate the effect of vascular reconstructive surgery on ED rates.

CHAPTER ALGORITHM



Algorithm for Shared Decision Making and Treatment Planning for ED. (Adapted from Burnett and Ramasamy. *Campbell-Walsh-Wein Urology*, 12th ed. Philadelphia, PA: WB Saunders/Elsevier; 2020:1516.)

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This seminal article was the first to describe the role of nitric oxide in achieving a penile erection and forms the basis for the targeting of current medical therapy for ED.

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Complex Regional Pain Syndrome

SIKANDAR Z. KHAN and HASAN H. DOSLUOGLU

Based on a previous edition chapter by Ali F. AbuRahma

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INTRODUCTION

Complex regional pain syndrome (CRPS) is a chronic pain condition, usually affecting an extremity and associated with inflammatory and autonomous changes.^{1–10} Pain can be spontaneous or induced by a specific stimulus, usually out of proportion to the inciting event, and associated with skin and soft tissue changes. CRPS is also commonly associated with motor and sensory changes leading to significant disability. Historically, the syndrome has been known by various names including *causalgia*, *reflex neurovascular dystrophy*, *neuroalgodystrophy* and *reflex sympathetic dystrophy* (RSD).^{1,2} These different terms

not only reflect the complex and variable etiology of the symptoms but also limited understanding of its mechanisms. The term *complex regional pain syndrome* was coined by an International Association for the Study of Pain (IASP) task force in 1994,¹ and while extensive research has been done in the last three decades, diagnosis and treatment of CRPS remains challenging in contemporary practice.

DIAGNOSTIC CRITERIA

The diagnosis of CRPS is based on clinical signs and symptoms elicited over several clinical visits and is a diagnosis of

BOX 192.1**The Veldman Criteria**

1. Four out of five of the following symptoms
 - Diffuse pain
 - Difference in skin color
 - Edema
 - Difference in skin temperature
 - Limited range of motion (ROM)
2. Increase in above signs and symptoms with use of extremity
3. Presence of above signs and symptoms in an area larger than the area of primary injury including the area distal to the primary injury

BOX 192.2**IASP Diagnostic Criteria for CRPS**

1. Pain, allodynia or hyperalgesia which is out of proportion for the inciting event
2. Edema, skin changes or evidence of sudomotor dysfunction
3. Absence of any other condition which could explain the presence of pain and associated symptom

CRPS Type I: Presence of an initiating noxious event

CRPS Type II: Associated with nerve injury

exclusion. There are no specific diagnostic tests, however, they may be used to rule out other possible etiologies. However, with a wide array of symptoms and clinical presentations, diagnosis and characterization is difficult. Multiple terms and different diagnostic criteria have been used in the past. Common to all these criteria is a constellation of signs and symptoms due to various neurological, autonomous, and inflammatory abnormalities seen in CRPS. The primary feature of CRPS is pain that can be described as spontaneous burning pain or allodynia, disproportionate to the triggering stimulus. Other features differentiating CRPS from other neuropathic pain syndromes is the regional nature of the symptoms involving body parts beyond the expected distribution of an injury. It usually involves the distal part of the extremities but can also involve the torso and face. The Veldman criteria⁹ (Box 192.1) was based on presence of pain, changes in skin color, temperature and mobility. However, it has not been widely used since the signs and symptoms are not specific to CRPS, leading to over-diagnosis. In an effort to reduce ambiguity and facilitate diagnosis, the IASP proposed consensus-based criteria in 1994 (Box 192.2).¹ While the proposed criteria had high sensitivity, it lacked specificity leading to over-diagnosis of CRPS. The criteria were later revised to include physician-assessed clinical signs in an effort to improve the specificity of the diagnostic tool. The resulting “Budapest criteria” has been shown to be more specific, thus reducing false-positive cases.² The Budapest criteria (Box 192.3) were as sensitive as the original 1994 criteria (0.99 vs. 1.00) with improved specificity (0.68 vs. 0.41).¹⁰ Since our understanding of the pathophysiological mechanisms underlying CRPS continues to evolve, diagnostic criteria can be expected to evolve.

Currently, the diagnostic criteria of CRPS includes:

1. History of a provoking event leading to continuous disproportionate pain

BOX 192.3**Budapest Criteria for CRPS**

1. Continued pain disproportionate to any inciting event
2. Patient must report one symptom in three of the following four categories:
 - Sensory: allodynia or hyperalgesia
 - Vasomotor: temperature asymmetry, skin color change/asymmetry
 - Sudomotor: edema, changes in sweating, edema/sweating asymmetry
 - Motor/trophic: decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)
3. Patient must display one sign at the time of evaluation in at least two of the following categories:
 - Sensory: evidence of hyperalgesia (to pinprick) and/or allodynia (to light touch)
 - Vasomotor: evidence of temperature asymmetry and/or skin color changes and/or asymmetry
 - Sudomotor/edema: evidence of edema and/or sweating changes and/or sweating asymmetry
 - Motor/trophic: evidence of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)
4. There is no other diagnosis that explains the patient's signs or symptoms

CRPS 1 – Without evidence of major nerve damage

CRPS 2 – With evidence of major nerve damage

2. Presence of one symptom in three of the following categories:
 - *Sensory* symptoms including hyperalgesia and allodynia
 - *Motor* dysfunction including limited range of motion and trophic changes such as altered nail and hair growth
 - *Vasomotor* symptoms such as skin color and temperature changes
 - *Sudomotor* symptoms such as edema and altered sweating
3. Presence of one clinical sign in at least two of the following categories:
 - *Sensory* disturbances including allodynia and hyperalgesia
 - *Motor* weakness and dysfunction
 - *Vasomotor* changes such as skin color and temperature alterations
 - *Sudomotor* symptoms such as edema.

CRPS can be subdivided into two types, CRPS I and II, depending upon presence or absence of major peripheral nerve injury.^{2,3} CRPS I includes clinical features of reflex sympathetic dystrophy (RSD) and similar syndromes without any evidence of major peripheral nerve injury. CRPS II develops after a nerve injury, similar to causalgia. However, it should be differentiated from temporary pain and symptoms caused by an injury, including myofascial pain syndrome.

EPIDEMIOLOGY

The incidence and prevalence of CRPS is variable across studies, regions, and populations.^{11–23} Part of it can be explained by the different criteria and definitions used in studies. In a study by Beertuizen et al.,¹⁵ the rate of CRPS ranged from 7% to 48%, depending on the criteria used – the IASP, Veldman and

Harden & Bruehl criteria. Consequently, given the difficulty in diagnosis, the true prevalence of CRPS in the general population remains uncertain and given variable sensitivity of the criteria used, there is likely misdiagnosis of patients. Furthermore, most of these studies have focused on CRPS I and data on CRPS II is lacking.

In a population-based study looking at a US county population, the annual incidence of CRPS I and CRPS II were 5.46 per 100,000 person-years and 0.82 per 100,000 person-years, respectively.¹¹ In another study, looking at a population in the Netherlands, the combined incidence of CRPS I and II was 26.2 cases per 100,000 person-years.¹² While both studies were based on the IASP 1994 criteria, the four times greater rate in the Netherlands study could be explained by the differences in the ethnic and socioeconomic backgrounds of the study populations, and also by variable application of the diagnostic criteria. Sandroni et al.¹¹ strictly applied the IASP criteria while de Mos et al.¹² did not require that all patients fulfill the diagnostic criteria, and included patients with a clinical diagnosis of CRPS, as determined by their clinician. However, both studies showed that it is 3–4 times more prevalent in females, more commonly affects upper extremities with peak incidence at 50–70 years of age.^{11,12} Other risk factors associated with CRPS are menopause, osteoporosis, and smoking.^{13,14}

Fractures are also a risk factor for developing CRPS. The incidence of CRPS after radius fracture has been reported between 1% and 37%,^{15–18} 15% after ankle fracture, and 7.9% after wrist fractures.¹⁵ Sarangi et al. reported an incidence of 30% in their cohort of patients with tibial fractures.¹⁹

CRPS can also develop after surgery including procedures for carpal tunnel release, Dupuytren contracture, distal radius, and shoulder procedures with rates ranging from 1% to 40%.^{20–22} Similarly, CRPS has been reported to occur in 1%–30% after procedures such as external fixation, open reduction and internal fixation of long bone fractures as well as ankle and foot surgery.²³

PATOPHYSIOLOGY

The pathophysiology of CRPS involves an interplay between different systems and factors – neurological (peripheral, central and autonomic nervous systems), inflammatory and immunological. Additionally, there is influence of psychological and genetic factors. The contribution of each individual factor to the overall CRPS picture remains unclear, and it is likely that the diverse clinical presentations are a result of variable contributions of these factors (Box 192.4).

Inflammatory and Immune-Related Factors

The clinical features of early CRPS including pain, edema, erythema and increased temperature point to an exaggerated inflammatory response to an injury. There is evidence of increased concentrations of inflammatory mediators such as bradykinin, substance P and calcitonin gene-related peptide and cytokines including interleukin (IL)-1 β , IL-2, IL-6 and tumor necrosis

BOX 192.4

Possible Pathophysiological Mechanisms of CRPS

- Nerve injury
- Peripheral and central sensitization
- Altered sympathetic nervous system
- Circulating catecholamine
- Inflammatory and immune-related factors
- Reperfusion/oxidative injury
- Psychological factors
- Genetic factors

factor- α (TNF- α).^{24–29} Inflammation is also mediated through oxidative stress pathways and animal studies have shown that ischemia-reperfusion injury can lead to CRPS-like features.³⁰

Autoantibodies against autonomic nervous system structures including β 2-adrenergic and muscarinic type-2 receptors have also been reported in some CRPS patients, suggesting an autoimmune process.³¹

Altered Cutaneous Innervation

Neuronal injury can also lead to development of CRPS.³² Density of epidermal neurites in skin biopsy samples from CRPS-affected extremities has been shown to be lower compared to the unaffected extremity, primarily affecting nociceptive fibers.³³ A decrease in the density of C-type and A δ -type cutaneous afferent neuron fibers in CRPS-affected areas, along with an increase in aberrant fibers of unknown origin, has led to the theory that exaggerated pain sensation may be due to altered function of these fibers.³⁴ In a rat model, needle-stick injury led to a decrease in nociceptive neuron density.³⁵ However, these changes are not seen in all patients with CRPS.⁸ Hence, it remains unclear whether these cutaneous innervation changes lead to CRPS or they represent inconsistent findings in a subset of CRPS patients.

Central and Peripheral System Sensitization

Initial tissue and neuronal injury leads to changes in the central and peripheral nervous systems leading to increased responsiveness to pain. Release of local factors such as bradykinin and substance P increases nociceptor activity and decreases their threshold to fire in response to various stimuli.^{36–39} Due to persistent and exaggerated stimulation of peripheral nociceptors, central sensitization occurs due to increased excitability of secondary nociceptive neurons in the spinal cord.^{38,39} This involves activation of spinal N-methyl-D-aspartate (NMDA) receptors by the excitatory amino acid glutamate.³⁹ These are adaptive mechanisms to avoid additional injury from harmful stimuli. This central activation leads to exaggerated response to painful stimuli (hyperalgesia), and stimulation of pain receptors by ordinary non-noxious stimuli like touch or thermal (allodynia). Additional structural changes may occur in the central nervous system (CNS) including the somatosensory cortex, which may lead to CNS-related symptoms such as motor impairment and neglect, which can be seen in chronic CRPS.^{37,38}

Autonomic Nervous System Dysfunction

Other nervous system changes seen in CRPS include impaired function of the sympathetic nervous system and coupling of sympathetic and nociceptive neurons, leading to pain with stimulation of adrenergic receptors.^{40–42} Animal studies have shown expression of adrenergic receptors on nociceptive fibers in response to injury, which can lead to activation of pain receptors in response to sympathetic activation or circulating catecholamines.⁴⁰ Increased expression of adrenergic receptors in CRPS-affected limbs has also been reported.⁴¹

Additionally, other CRPS symptoms can also be attributed to the sympathetic system. There is an increase in circulating epinephrine during the acute phase of CRPS, potentially explaining the vasodilation, edema and increased temperature, which are hallmarks of early CRPS.⁴² Over time, there is increased sympathetic sensitivity which can cause vasoconstriction leading to cold and clammy limbs, characteristics of chronic CRPS.⁴²

PSYCHOLOGICAL FACTORS

Anxiety and depression may be associated with development of CRPS. However, evidence remains inconclusive and prevalence of psychiatric disorders in CRPS is similar to other chronic conditions.^{37,43} The presence of depression and anxiety seem to have greater impact on pain in CRPS as compared to other pain conditions. This could be explained by increased catecholamine release in response to psychological stressors, which increases pain intensity due to sympathetic– nociceptive receptor coupling. Psychological distress at the time of injury can impact development of CRPS and the severity of symptoms. In patients undergoing total knee arthroplasty ($n = 77$), depressive symptoms in the perioperative period were

predictive of greater severity of CRPS symptoms at 6-month and 12-month follow-up.⁴³

GENETIC FACTORS

There seems to be a familial component of CRPS with greater incidence in family members, involvement of multiple limbs and onset at an earlier age.^{44,45} A specific inheritance pattern or CRPS genotypes are unknown but there is growing evidence of association of certain major histocompatibility complex (MHC) genotypes including human leukocyte antigen (HLA)-B62 and HLA-DQ8.⁴⁴ An upregulation of the matrix metalloproteinase 9 (MMP9) gene has also been reported in CRPS patients.⁴⁵ Further investigation is needed to determine if there is a definitive genetic link predisposing patients to develop CRPS.

CLINICAL PRESENTATION AND EVALUATION

Diagnosis of CRPS is based on clinical signs and symptoms (Fig. 192.1). Diagnostic tests are not necessary to make the diagnosis, but they may be used to support a clinical diagnosis of CRPS and rule out other similar conditions.

Radiographic Studies

Patients with CRPS have diffuse osteopenia, subperiosteal bone resorption and patchy demineralization, especially of the periarticular regions.^{46,47} These changes can be seen on plain radiography. However, these findings appear late and are not specific to CRPS. On triple-phase bone scans, there is increased blood flow in the affected extremity, blood pooling,

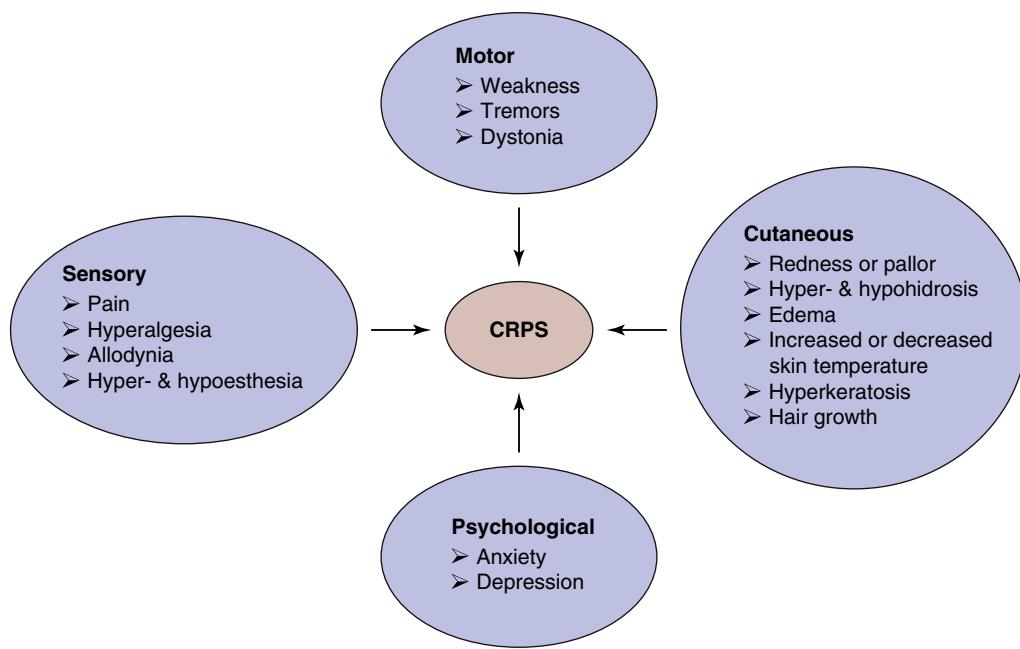


Figure 192.1 Signs and Symptoms in CRPS Patients.



Figure 192.2 Three-phase Bone Scan of Upper Extremities in a CRPS Patient.

and intense periarticular delayed uptake (Fig. 192.2). MRI may show skin thickening, soft tissue edema, muscle atrophy, joint effusions and patchy bone marrow edema.⁴⁷ While these findings are not specific to CRPS, they help in diagnosis in the correct clinical setting, and are also useful to rule out other pathological conditions.

Diagnostic Sympathetic Blockade

Pain relief from sympathetic blockade strongly supports a CRPS diagnosis, and lack of pain relief has a high negative predictive value and essentially rules out CRPS.⁴⁸ Sympathetic blockade can also lead to pain relief in other conditions including ischemia, however, the degree of pain relief in CRPS is substantial unlike in other conditions, in which it is usually mild to moderate.⁴⁹

NATURAL HISTORY

CRPS symptoms develop within the first 3–4 months after the inciting event and the diagnosis of CRPS after this window seems unlikely. The natural course of CRPS is unpredictable with varying rates of resolution. In acute CRPS (<1 year duration), there are higher rates of spontaneous resolution with limited or no specific treatment. In a study looking at 30 patients with CRPS over a course of 13 months, CRPS symptoms resolved in 26/30 patients without any treatment.⁵⁰ In the landmark US study looking at the incidence of CRPS, 74% of cases resolved with limited treatment.¹¹ In contrast, chronic CRPS (>1 year duration) has lower rates of spontaneous resolution. In a study of 102 patients with chronic CRPS, only 30% of patients reported resolution of symptoms, 16% of patients experienced progression with

stable symptoms in 54% of patients over an average follow-up period of 6 years.⁵¹

MANAGEMENT

CRPS if left untreated can lead to chronic pain and significant disability. While many cases of acute CRPS resolve with limited conservative care, a subset of patients go on to develop chronic CRPS. Thus it is important to recognize this subset early and initiate appropriate therapy. Although the number of trials investigating CRPS treatment is increasing, high-quality evidence on CRPS treatment strategies is lacking. Treatment of CRPS is multidisciplinary involving multiple specialties, and includes pharmacotherapy, physical and psychological therapy and interventions to reduce sympathetic activity.

Physical and Psychological Therapy

CRPS patients tend to avoid use of the affected extremity due to significant pain and kinesophobia, resulting in disuse and significant disability. The goal of physical therapy is to improve the functionality and mobility of the affected limb, and reduce pain in combination with pharmacotherapy and psychological therapy.^{52–54} Occupational therapy is aimed at encouraging use of the affected limb in activities of daily living. Different modalities are used in clinical practice and tailored to individual patients including elevation, splinting of the affected extremity, massage, hydrotherapy, range of motion and strengthening exercises, stress loading, relaxation techniques, postural control and edema control strategies, ideally in a multidisciplinary pain clinic or CRPS rehabilitation center.^{8,52–54} Psychological therapy aims at correcting body perception disturbances involving touching, looking and thinking about the affected extremity and techniques at improving awareness of the affected extremity, and reducing fear of using the affected extremity. In a review of 18 trials of physiotherapy-based interventions including 739 patients, two strategies with the best available data showing improvement in pain and function in CRPS patients were mirror therapy (MT) and graded motor imagery (GMI).⁵³

Mirror Therapy

Mirror therapy (MT) is based on the use of a mirror to create a reflective illusion of an affected limb in order to train the brain into thinking that the affected limb has moved without pain, creating a positive visual feedback of a limb movement. This approach exploits the brain's preference to prioritize visual feedback over somatosensory and proprioceptive feedback. It involves placing the affected limb behind a mirror, so the reflection of the opposing normal limb appears in place of the hidden affected limb (Fig. 192.3). MT was first described to treat phantom limb pain⁵⁵ and has been utilized in patients with CRPS with some success. Two trials with MT in CRPS I patients showed improvement in pain and functionality.⁵³

Graded Motor Imaging

Graded motor imagery (GMI) is a three-step program designed to engage the motor cortex without triggering the protective pain response.⁵⁶ The first step involves identifying limb