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This book is a tribute to Otmar Trentz, MD, a European pioneer of the "Acute Care Surgery" model in the 1980s and 1990s. Otmar Trentz represents the prototype of a dedicated academic trauma surgeon and passionate teacher and advocate for the integrated care of the trauma patient with associated musculoskeletal injuries.

Born in Trier, Germany, in 1942, Otmar Trentz graduated from Medical School at the University of Würzburg in 1967. After residency in Würzburg and Hannover, he was board certified in general surgery in 1974. Dr. Trentz then joined Harald Tscherne's renowned "Hannover School" as an attending trauma surgeon. In 1980, at the young age of 38 years, he was elected as the director of trauma surgery at the Hannover Nordstadt Hospital. From 1983 to 1990, Otmar Trentz was the chairman of the Department of Trauma Surgery and professor of surgery at the University Hospital of Homburg/Saar. In 1990, he was elected as chairman and professor of trauma surgery at the University of Zurich, Switzerland. Under Otmar Trentz's leadership, the University Hospital Zurich grew to one of the nationally and internationally renowned "premier" academic level 1 trauma centers in Europe.

Some of the seminal achievements of Professor Trentz's tenure in Zurich from 1990 to 2008 include the implementation of an integrated approach to the care of the multiply injured patient and the introduction of the first ATLS course in Switzerland in 1999. His research focus was centered on the pathophysiology of shock, polytrauma, and the pathogenesis of "host defense failure" in multiply injured patients. Otmar Trentz's scientific oeuvre encompasses more than 300

peer-reviewed publications in the pertinent trauma literature. He is furthermore the editor of multiple textbooks in the field of general surgery and trauma surgery, including the encompassing work entitled "Unfallchirurgie" which represents the "bible" for all trauma surgeons in German-speaking countries. Otmar Trentz is furthermore the Editor Emeritus of the European Journal of Trauma and Emergency Surgery.

Today, at the respectful age of 71 years, Professor Trentz continues to work as a consulting surgeon at the Madras Institute of Orthopaedics and Traumatology (MIOT) in Chennai, India, where he performs about 100–120 surgical procedures per month.

Beyond a doubt, Otmar Trentz's legacy as a charismatic, passionate, modest, and endlessly hardworking pioneer in the field of trauma surgery will endure in the generations of European trauma surgeons whom he mentored during the "golden years" in Hannover and Zurich.

Otmar Trentz

Foreword: The Master's Perspective

This comprehensive textbook provides a timely, integrated view on the impact of musculoskeletal injuries in the multiply injured patient and provides the rationale for coordinated interdisciplinary care. In contrast to isolated orthopedic injuries, which are managed by most general orthopedists with interest in fracture care, the polytraumatized patient requires a distinct, custom-tailored approach related to the optimal timing and modality of fracture fixation. The book is authored by trauma-trained, dedicated surgeons who recognize this critical distinction. Clearly, *polytrauma* represents more than just the sum of all individual injuries. The complex underlying pathophysiology renders multiply injured patients vulnerable to an uncoordinated fragmentation of care by individual specialists, which ultimately results in suboptimal patient outcomes. More than a century ago, the father of modern medicine, Sir William Osler (1849–1919), stated that "Specialism has fragmented the specialties themselves in a way that makes the outlook hazardous."

Management of Musculoskeletal Injuries in the Trauma Patient provides a unique perspective of coordinated trauma care, which takes into account the fundamental concept that specific orthopedic injuries have a dramatic impact on the systemic pathophysiology of major trauma. For example, a fractured femur aggravates the systemic burden to the pulmonary endothelium and the blood-brain barrier through release of inflammatory mediators, including fat embolism syndrome. Patients with chest contusions and/or head injuries are therefore more vulnerable to secondary organ failure (ARDS, brain edema), unless femur fractures are stabilized in a proactive fashion through multidisciplinary *damage control* protocols. Similarly, patients with severe pelvic ring disruptions are at risk for exsanguinating hemorrhage and the acute coagulopathy of trauma unless early bleeding control is achieved by standardized measures, including external pelvic fixation and retroperitoneal packing. Such proactive protocols evolved from the "European model" which historically considered the integrated trauma team as the single *specialist* responsible for the care of critically injured patients. In the United States, this evolving model is reflected by the civilian acute care surgeon and the military combat trauma surgeon.

This book outlines the concept of optimized multidisciplinary care for trauma patients with orthopedic injuries in a logical and comprehensive fashion, with a focus on high-energy limb- and life-threatening injuries, extremes of age, and critical associated injuries to the head, spine, and torso.

Both editors are renowned experts in integrated trauma care systems, with a track record of synergistic team building across the main disciplines involved in the care of the trauma patient (orthopedic surgery, trauma surgery, and neurosurgery). Unquestionably, the primary goal of the initial multidisciplinary management of polytrauma is *patient survival*. As trauma care has matured, the ultimate functional outcome of the patient has focused importance on the need for *state-of-the-art* orthopedic trauma care. This new textbook will hopefully contribute to a meaningful prioritization and optimization of the care of the injured patient with associated musculoskeletal injuries and strengthen the bond between the responsible teams through improved communication and standardized interdisciplinary protocols.

Denver, CO, USA

Ernest E. Moore, MD, FACS
Editor-in-Chief
The Journal of Trauma and Acute Care Surgery

Preface

Management of Musculoskeletal Injuries in the Trauma Patient is a first-edition textbook written in the multidisciplinary spirit of modern trauma care. The genesis of the project originated during a course at the annual *American College of Surgeons* meeting in Chicago entitled *Orthopedic Trauma Emergencies*. This symposium was aimed at a mixed audience of general, trauma, and military surgeons. The lecture hall was packed and the audience was primed with practical and highly sophisticated questions, which revealed the widespread interest in the overall management of musculoskeletal injuries in trauma patients. During the ensuing discussion in and out of the lecture hall, we were impressed by the level of knowledge about musculoskeletal injuries of our non-orthopedic colleagues. We also became aware of how critical it was for on-call general and trauma surgeons to understand the modern advances in orthopedic trauma care in order to build better integrated trauma systems and to improve the daily collaboration with their orthopedic colleagues. Concomitantly, orthopedic surgeons articulated their need to understand the evolving rationale for treatment of chest, abdominal, brain, and vascular injuries in order to ensure a high-quality multidisciplinary approach to the care of the trauma patient. From these shared anecdotal experiences evolved a plan for a new textbook dedicated to the decision making of when and how to treat musculoskeletal injuries in the trauma patient.

The book's first edition is organized into three sections based on the priorities of trauma care, specific injuries, and specialized approaches in the presence of significant associated injuries. While we recognize that a one-size-fits-all approach is not appropriate for the diverse field of trauma care, we also maintain that a "do-it-different, every place, every day" attitude defies logic with regard to the underlying principles of the pathophysiology of shock, organ failure, and death. Therefore, we have focused on the complex interplay of musculoskeletal injuries and their effect on optimized patient-centered care. Given that a prospective randomized study is not possible for every clinical trauma question, we must apply data plus logic plus individual experience to formulate new treatment strategies aimed at reducing error and improving outcomes. Toward that end, we are extremely privileged to have a selection of world-renowned authors with stellar reputation and outstanding experience. All authors are traumatologists in their respective subspecialties who have devoted their lives and careers to the care of the injured patient.

Throughout the world, trauma care standards and systems differ widely. What does not differ is the (patho)physiologic response to injury. Bleeding must be stopped, shock must be corrected, the lungs must be protected, and the brain must receive sufficient oxygen. Once hemostasis is achieved, fractures need to be realigned and stabilized to preserve future function. The precise means and order of accomplishing these tasks will differ from place to place, yet the principles underlying the specific choices of treatment are relatively constant. Trauma surgery and orthopedic trauma surgery have evolved dramatically in recent years and decades, with the introduction of new, innovative concepts and techniques of unquestionable benefit to the injured patient. The downside of such evolution, however, is the divergence in understanding between different specialties and the fragmentation of care by overspecialization of surgical disciplines. Our intent in this text is to help bridge the gaps between the (sub)specialties involved in the care of the trauma patient and to present our colleagues with up-to-date, practical information that can be implemented and applied in real time. Understanding the advances in trauma and musculoskeletal injury care will facilitate better decision making, appropriate and safe treatment protocols, and overall improved coordination between trauma team members. Our hope is that elucidation of these shared principles, combined with informed decision making, will aid in the treatment of the injured patient, no matter where and when.

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Part I

Priorities of Trauma Care

Musculoskeletal Injury

1

James F. Kellam

Introduction

Musculoskeletal injury, has significant effects on the local soft tissues around the injury as well as an effect on the entire physiology of the patient [1]. Musculoskeletal injuries involve an extremity and/or the pelvic girdle. A limb segment can be defined as that portion of the axial skeleton that exists between two joints including the respective articular components of that segment. This segment is made up of skin, subcutaneous tissue, muscle, nerves, vessels (arteries and veins), bone, and articular cartilage. An understanding of the physiology of each of these components is mandatory to guide the assessment and treatment of the pathology caused by the musculoskeletal injury. The combination of these injured components may become significant in their effect on the overall condition and complications that the patient may incur. There is also a significant interaction between the severity of the local musculoskeletal injury and the patient inflammatory response leading to pulmonary and multiple organ dysfunction and potential death [2]. In order to understand and treat musculoskeletal injury, this chapter will look at these various components with regard to their assessment and management.

An injury to a limb segment will result in a spectrum of damage to these components. The range of this spectrum is determined most importantly by the energy applied to cause the injury. The greater the energy force, the greater the damage and hence the more components involved, and the more the injury to this axial limb segment, the more likely the compromise to the patient's overall condition and care. This concept is best exemplified by the multiple investigations looking at the interaction of fracture care and patient outcomes and in particular that of damage control versus early total care [3].

General Conditions Related to Musculoskeletal Injury

Any individual managing musculoskeletal injury must be aware of two conditions in particular that will affect the general physiology of the patient. The first condition is the development of adult respiratory distress syndrome (ARDS) and in particular the systemic inflammatory response. The second condition is fat embolism.

Systemic Inflammatory Response

The systemic response to musculoskeletal injury results in the stimulation of the inflammatory response. This systemic response has

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two components: the well-known inflammatory response and a now-recognized counter regulatory anti-inflammatory response (CAR). These two processes work in balance with each other to control the inflammatory response so that the patient's final outcome will be normalcy. Should this balance be lost, significant complications will occur. These responses are based upon the release of a variety of inflammatory mediators which will drive either the systemic inflammatory response or the counter response. As long as these remain balanced, homeostasis will be present and the patient will recover in a relatively predictable manner. If this balance is disturbed, then significant complications can develop such as the systemic inflammatory response syndrome (SIRS) or/and multiple organ dysfunction [4]. Giannoudis has described four potential mechanisms for the development of these post-traumatic complications. The microenvironment theory, the gut hypothesis, the two-hit theory, and the microenvironment theory [5]. The microenvironment theory is presently felt to provide the logical cause for these complications. This is essentially the adherence of activated neutrophils to the endothelial lining creating a protective environment for toxic metabolites secreted by the neutrophils. As these toxic products cannot be neutralized by their appropriate antioxidants and antiproteinases, the endothelial lining of the vessels is destroyed, permitting extravasation of fluid and the migration of cells along with toxic mediators into the parenchymal tissue of the lung and other organs. The consequence of these activities is cell death and organ failure, leading to the demise of the patient. Another common theory is the two- or double-hit model. This is based upon the fact that the injury represents a first *hit* or insult, turning on the appropriate inflammatory response. Early orthopedic operative treatment, among other factors, can cause a second hit. The physiology of the second hit is that neutrophils previously primed by the initial injury undergo activation by the secondary insult, causing a release of mediators which target endothelial tissue. Increased endothelial permeability leads to "leaky capillaries" in the lungs and begins the cycle of adult respiratory distress syndrome (ARDS) and multiple organ failure (MOF). Additionally, depending upon the magnitude of this surgical assault, the inflammatory response may either be increased to a pathological level or the counter inflammatory response may be stimulated, thus shutting down the needed inflammatory response necessary to manage the injury This has led to the concept of damage control orthopedic surgery where one is particularly cognizant of the patient's physiological status after injury and titrates the amount of surgical intervention necessary to create a stable patient throughout this phase [6]. Another more traditional approach is early total care where it felt that the patient's best opportunity for success is by managing all fractures at the same time as long as the patient remains stable [7]. Controversy arose between these two concepts when Pape reported a high mortality rate in patients with femoral shaft fractures, treated with early total care who were severely injured. He suggested that some patients may not tolerate early total care and that damage control would be a safer approach. Today both of these treatment methods are compatible with each other. It is now a much better thought to consider early appropriate care as suggested by Vallier [8]. This is care which is based upon the physiological status of the patient following the injury and then determining what is best done in order to provide effective care of musculoskeletal injury while at the same time not compromising the overall patient. This requires teamwork with trauma surgeons, anesthesiologists, and neurosurgeons and an understanding of the physiology of injury.

Fat Emboli Syndrome

This is a very specific syndrome that is associated with long bone fractures particularly of the femur and tibia [9]. It usually occurs in those patients who have had one or more fractures of the lower extremity including the pelvis in whom there has been no history of hypotensive shock, chest injury, or ARDS. This is felt to be secondary to the embolization of fat or fat type material from the marrow of the injured bone or soft tissue. The end result of the fat emboli syndrome is the same

as the systemic anti-inflammatory response in that respiratory distress develops in the lungs due to increased alveolar capillary permeability [9]. Neurological sequelae also occur as the fat can cross the blood-brain barrier. Coagulation disorders and an overall picture of early MOF may also occur. One concern for the orthopedic surgeon is that intramedullary fixation may worsen or cause a fat embolism syndrome by driving the fat from the medullary canal into the vascular system and then to the lungs. Fat embolism is recognized by the gradual drop of oxygen concentration or hypoxia in the first 24–48 h following injury and/or treatment. Development of upper chest petechiae and embolic fat seen in the retina may also be presenting symptoms [10]. The usual treatment of this syndrome is increasing supplemental oxygen as necessary and potentially providing full respiratory support, including intubation. The use of certain drugs, such as corticosteroids, has not been effective and is usually contraindicated. In most cases, with appropriate diagnosis and treatment, symptoms slowly resolve.

Thrombosis

The third potential systemic complication from musculoskeletal injury is the development of venous thrombotic disease [11]. This area has remained controversial within the trauma literature. Most of the information that is available has come from the total joint literature concerning the recognition and management of patients. The problem with the injured patient is that all three of Virchow's triad—stasis, venous injury, and hypercoagulability—usually exist to some degree or other in any injured limb segment. Stasis is secondary to the injury, immobilization, and low flow states associated with hypovolemic shock. Venous injury occurs as a direct result of the injury to the extremity as well as a byproduct of anesthesia, infection, and placement of intravenous access. Hypercoagulability can occur from the variety of different physiological consequences induced by the trauma [12]. There has always been difficulty in correlating the occurrence of fatal pulmonary emboli (PE) to the presence of deep vein thrombosis (DVT). However, the fatal PE rate for trauma patients without prophylaxis is significant and specific types of trauma patients should be placed on prophylactic anticoagulation as soon as the patient is safe from acute bleeding. Long-term anticoagulation remains controversial. Most trauma patients receive prophylaxis until ambulatory. Longer-term anticoagulation is usually reserved for those with symptomatic PE or DVT. For those patients who are not ambulatory or unable to be mobile with ambulatory aids or only mobile from bed to chair, the use of prophylactic anticoagulation, either with low-molecular-weight heparin, Coumadin, or an inferior vena cava filters, should be considered [13]. However, there is little to prove that those patients who are mobile or have injuries below the knee require ongoing prophylaxis after the discharge from the hospital [14].

The Musculoskeletal Injury

The Patient

Musculoskeletal injury is usually the result of some form of injurious force of a mechanical nature. Whether from a simple fall or high-energy car collision, the patient is always at risk for other non-appendicular injuries to the chest, head, and abdomen. Healthcare providers assessing an extremity injury should always perform a complete examination of the patient to assure that non-appendicular injuries are not missed. Based on the mechanism and nature of the presenting injury, this examination can be in-depth or limited. However, a logical, complete head-to-toe evaluation is critical, consistent with the principles of the Advanced Trauma Life Support® system as promulgated by the American College of Surgeons.

Limb Segment Assessment

The assessment of the injured leg segment must follow a logical sequence starting with skin,

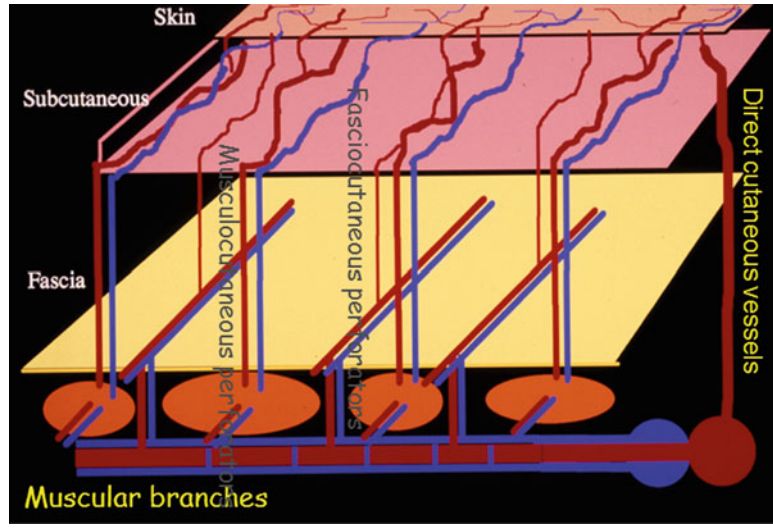

Fig. 1.1 Blood supply to the skin

subcutaneous tissue, muscle, nerve, vessel, and then bone.

Skin

The skin provides the coverage for any limb segment and is important for cosmesis, the prevention of infection, and the prevention of dehydration and loss of fluid. Consequently, an assessment of the limb segment coverage must be undertaken. It is the first aspect that the treating surgeon will see and hence should be dealt with first. Generally speaking, with musculoskeletal injury, it is either an open injury in which the skin has been violated through laceration or puncture or a closed skin injury where the skin will be crushed, bruised, or degloved. A grading system for closed soft tissue injuries was developed by Harald Tscherne [15]. This is related to the amount of damage done to the soft tissues and best evaluated by assessment of the skin. A grade zero injury is none or minimal injury to the soft tissue and skin, which is manifested by little evidence of injury. A grade 1 injury has more damage to the skin as evidenced by skin bruising and swelling. A grade 2 injury has deep contusions to the skin with early serous blistering and muscle contusion and impending compartment syndrome. The grade 3 injury is full thickness contusions, crush, avulsion injury, and swelling with loss of wrinkles and bloody blisters similar to a full thickness burn and maybe associated with a compartment syndrome.

Understanding of the blood flow to the skin becomes important in surgical decision making. The more severe the skin injury, the greater the risk for infection and skin necrosis if a surgical procedure is undertaken too early in the skin recovery phase or the incision is poorly placed. The skin has three mechanisms of blood supply. The most common is through intramuscular vessels that perforate the muscle, the fascia, and come directly to the skin, so skin overlying muscle is well perfused. However, areas in which the skin overlies bone or little muscle, the skin is perfused by arteries that are not protected by the muscle and transverse these potentially damaged areas (Fig. 1.1). Hence, this skin over bony prominences may be easily damaged either with injury and/or with surgical incisions and lead to further complications of skin necrosis and loss. This understanding leads the surgeon to try to place incisions over muscle to maximize the skin blood supply while at the same time avoiding elevating the subcutaneous tissue from the fascia for fear of disrupting the muscular perforators to the skin. When necessary to make an incision in an area which is not over muscle, understanding the local blood flow is imperative so as not to devascularize certain segments of the skin. This is more complex when injury adds further disruption to the skin. It is also important to let

the skin return to normal after an injury. This is best evidenced by the resolution of swelling so that the operative site skin looks like the normal opposite side and that it wrinkles. This may take up to 3 weeks in severe injuries. The treatment of fracture blisters is best done by whatever the surgeon is most comfortable with [16]. There is no preferred method.

Subcutaneous Tissue

The subcutaneous tissue is usually a part of the examination at the time of a skin injury. In most closed injuries, this will never be a concern other than in avulsion injury of the subcutaneous tissue from the fascia layers (Morel-Lavallee injury) [17]. This is usually diagnosed by palpation of the extremity around the injury and feeling a boggy or fluid-filled area under the skin. This is an occult lesion and may not be recognized for days. It is a hidden area for serosanguineous fluid collection or occult blood loss. Its management will depend on the underlying fracture and the need for operative intervention. As these areas can be contaminated by hematogenous spread of organisms, it may be wise to drain these open or percutaneously and culture. When healed, it would be safe to operate the fracture. If found when approaching a fracture, it is best to debride, culture, and then fix the fracture, but the area must be drained.

During debridement of an open fracture or at the time of fixation of a closed fracture, the subcutaneous tissue maybe found to be completely crushed and dead with dead or poorly viable skin overlying the area. These areas will usually go on to break down and required local debridement. If at the acute debridement of an open fracture fixation the area can be safely debrided and closed, it is wise to do so as to avoid later complication. This will hold true in a closed fracture undergoing operative treatment as well. If debridement will compromise coverage of fracture fixation, then flap coverage will be necessary and it may be advisable to wait out the natural evolution of this soft tissue injury. These decisions all require experience in soft tissue assessment and handling. However, the ultimate decision should be documented as further excision, even amputation, may be required, depending upon the patient, the condition, and the methods of treatment.

Muscle

Assessment of the muscle is a critical aspect in any musculoskeletal injury. The reason for this is that muscle provides the most amount of blood for the area for healing and for the prevention of infection. Also, muscle is responsible for function, and with muscle damage and loss, ultimate function deteriorates, and patients' outcomes are less than ideal. Consequently, the assessment of the muscle viability in any open injury should be made at the time of debridement. At this point one must look at its color and its consistency in that it is soft and not hard or firm. This feel for consistency is important as muscle dies from the inside out. A muscle may demonstrate contacting fibers on its surface, but the core is dead. This muscle will generally have a firm or rubbery feel, while that muscle that is alive will feel soft throughout. Should the muscle have this rubbery firm feel, it is important to split the fibers and assess the deeper layers for muscle death. This muscle may not be excised at this time, but it is an indication for the need for a repeat look to assure no further death has occurred. Muscle contractility is best tested by tapping on the muscle with an instrument or the finger and seeing it contract, or by touching it with a cautery and seeing the contraction. Circulation can be checked by observing the muscle for punctate bleeding points indicative that the intricate vasculature that is providing blood supply is intact [18].

The assessment of muscle in a closed injury is at the present time extremely difficult. There are few clinical tools able to assess the viability or vascularity of muscle on a routine clinical basis. Certainly the role of compartment pressure measuring for compartment syndrome is one way of indirectly measuring the vascular supply or viability of muscle but is only used in those conditions in which one is suspecting the compartment syndrome. However, the more common circumstance is the significant high-energy injury or crush injury and particularly those with complex, closed fractures in the metaphyseal region. This leads to significant swelling of the

muscle and ongoing swelling of the soft tissues. This has led to the concept of damage control limb surgery in which the use of external fixation is applied to maintain the leg to length as well as the bone and provides stability to the soft tissues to allow them to heal, revascularize, and be prepared for surgical intervention, minimizing the complications [19].

Nerve

Nerve injuries can range from annoying in the case of cutaneous nerves to devastating loss of sensory and motor function. Without ongoing functional nerves, muscles will not function and patient outcomes will be poor. However, nerves injured in continuity will tend to return to function with time. An assessment of the neurological status of the upper and lower extremity is imperative with every injury. It is important to document injury but more important to assess and document ongoing neurological loss which may stimulate further acute emergency treatment to prevent nerve loss. The majority of nerve injuries in closed fractures are usually some form of contusion and rarely a laceration. The standard management has been to decrease compression by reduction of the fracture. There has been no proof that there is a need to explore a nerve injury in closed fracture as an acute treatment. Certainly at the time of operative fixation if the nerve is injured and is readily accessible, then exploration is warranted. However, in open fractures with nerve injuries, part of the debridement process is to explore the nerves in the area if injured and determine what has transpired and treat accordingly [20].

Vascular Injury

Vascular injury is associated with a fracture. It is an acute situation which requires the treating musculoskeletal surgeon to have in the back of his mind a plan that can be implemented quickly. In fact, it is imperative that in any institution that will manage these injuries that there should be protocols developed between the musculoskeletal surgeon and vascular surgeon to assure that prompt, adequate assessment and treatment is carried out [21]. The first form of assessment is the monitoring of the distal pulses from the injury. Obviously if these are equal to the opposite side, there is probably little evidence of any injury. If they are not present or decreased, one must then look at the perfusion distal to the fracture and assess if this is compromised. This assessment is done through functional evaluation of both neurological injury and muscle function as well as capillary refill. If both nerve and muscle function are intact, then there is enough perfusion to that extremity that investigations may be carried out to assure that one has made diagnosis of the vascular injury and knows what is happening. However, if there is no perfusion to the distal segment and it is cold and pulseless, with no neurological function and no muscle function, this is an acute emergency which requires prompt action. The management of this acute injury requires from the orthopedic surgeon a reduction of any deforming forces across the artery. This usually means a closed reduction and splintage of the fracture. Following this, a reassessment of the pulses is performed. If pulses do not return in the majority of isolated injury circumstances, one can make the decision that the vascular injury is at the site of the fracture. There is little need to waste time looking or determining where it is and obtaining arteriography to do this. However, if there are multiple injuries to the leg or if one is unsure of oneself, then the use of arteriography or contrast enhanced computer tomography (CT) is extremely helpful in quickly determining the lesion location. In the acute situation with acute vascular injury, an arteriogram may be done on the operating table by injecting the dye through femoral artery for the lower extremity or the axillary artery for an upper extremity injury. This is a procedure done by vascular surgeons which is helpful to determine the level of injury. In the operating room, the vascular surgeon and orthopedic surgeon need to work as a team to assure the appropriate debridement and exposure that allows both to work and that also will manage the injury of the bone correctly and at the same time allow prompt revascularization. This may mean that the orthopedic surgeon goes first or the vascular surgeon, but this is a team approach and needs to be discussed with the

qualified individuals at the time of treatment. There are many ways of handling this such as temporary external fixation followed by vascular repair then the appropriate definitive fixation of the fracture, external fixation with vascular repair and delayed definitive fixation, vascular repair, followed by definitive fixation or external fixation. All these choices need to thought out and planned by both surgeons. One must also remember that after 6 h of cold ischemia time, serious consideration must be given to the use of fasciotomies distal to the arterial repair. Within 6 h of complete ischemia, reperfusion of the muscle will lead to significant swelling and potential for a compartment syndrome. As these patients are critically ill and tend to be intubated and monitoring of their compartments is difficult if not impossible, the use of prophylaxis fasciotomies is highly recommended in this circumstance to prevent limb loss or muscle death and poor function [22, 23].

Compartment Syndrome

A compartment syndrome is the death or compromise of viability of muscle secondary to closure of the capillary beds of muscle in any contained area in an extremity [24]. The causes of a compartment syndrome are many. Consequently, what must be remembered is that for any injury to an extremity, the treating surgeon must rule out a compartment syndrome. This first is done on a clinical and physical examination basis. Because the muscle has a decreasing blood supply, the muscle itself responds by becoming increasing irritable which means that it is painful. In fact, as the swelling increases, it becomes excessively painful and feels like the leg will be bursting. One might describe this as a "heart attack of skeletal muscle." Hence, the most common symptom of compartment syndrome is pain out of proportion to what one would expect with the injury and also excessive use of analgesic medication. Physical exam is helpful in diagnosing a compartment syndrome, but one must be wary of the findings. The common method of testing compressibility of the injured limb is extremely subjective. Although a very tense hard compartment is easily recognizable, something between that level and soft compartments may be difficult to determine and hence mislead the treating physician. Pain with passive stretch is difficult to interpret secondary to the fracture site pain and irritation due to damaged muscle. If truly present, it is a definitive sign of this condition. Loss of pulse, paresthesia, and neurological injury are all indicative of a compartment syndrome but occur as the end result of the damage secondary to loss of vascularity. Decreased sensation and nerve function usually indicate that permanent damage has already occurred and hence treatment is too late. In alert and oriented patients, the diagnosis is made on clinical grounds and a high degree of suspicion for this condition. Should doubt exist or the patient is noncooperative due to injury, drugs, intoxication, or anesthesia, compartment pressure measurement is a technique that is useful to confirm the diagnosis. There are many monitors and one should choose one that is easily available, and the surgeon knows how to use. The accepted pressure level for decompression is usually the difference (Delta P) between the diastolic blood pressure and the compartment pressure. This must be less than 30 mmHg. This method is important because it takes into account the patient's physiological circumstance such as hypotension or hypertension [25].

The treatment of a compartment syndrome is fasciotomy. There is no other treatment and there is no reason to wait. If the diagnosis is contemplated and written on the chart, a fasciotomy must be carried out [26].

Bone

Bone is the supporting structure for all the soft tissues. Without a solid pillar, the different soft injuries will not heal correctly, so assessment and stabilization of the bony injury is imperative to assure a functional recovery of the musculoskeletal injury. The assessment of the bony injury is the final component of the musculoskeletal injury. The general accepted method of determining this is radiographic evaluation. Two orthogonal views are necessary for all fractures. These orthogonal views should be reviewed very

carefully to determine an understanding of the injury. Should the treating surgeon not be able to their satisfaction to determine what the injury is, then the use of further methods of radiographic assessment is helpful. This is usually computer-assisted tomography (CAT) scan. This is particularly helpful in articular injuries to determine the intra-articular involvement and position of fragments. It is not of much help in diaphyseal fractures as most plain x-rays are usually sufficient. Although there are numerous articles that have shown the benefit of CAT scan for the evaluation of articular fractures, it is not a mandatory examination and should only be ordered if indicated. Increasing health costs and the recently published dangers of too many CAT scans make the routine use of CAT scans for every injury unacceptable. CAT scanning should be more based upon the lack of understanding of the plain radiographs or a predetermined protocol-based approach.

The bony injury must be assessed for location, fracture pattern, displacement, bone quality, and prior injury. Each of these factors will be critical in developing a treatment plan. The diagnostic process of determining the bony injury will lead to a description of the injury that will finally result in classification of the injury. However, until the fracture is visualized at surgery or finally treated nonoperatively, the surgeon is always working with limited information and cannot truly classify the fracture but can make treatment decisions. During this process of diagnosis and planning, a description of the injury as to location, pattern, and displacement which represents the forces applied is the best working description. Although many classifications exist, it is important to use one that is universal, with a consistent language and if possible validated as to being accurate. At present, the best available long bone classification to meet these needs is AO Foundation/Orthopaedic Trauma Association (AO/OTA) classification based on Müller's principles of his comprehensive long bone classification. This system provides a standard terminology for communication between surgeons and at the same time an alpha numeric code for documentation for registry and research purposes [27, 28].

There are two bone injuries with different outcomes. The diaphyseal fracture is a fracture that does not demand an anatomical reduction with regard to the ultimate treatment. This fracture needs length, rotation, and angulation to be correct without considering an anatomical reduction of the fracture. In general, diaphyseal fractures are treated by indirect reduction and relative stability (controlled fracture site motion within the strain tolerances of the healing tissue) [29]. Exceptions to this plan are usually noted in simple fracture patterns of the upper extremity diaphysis where anatomical reduction via a direct approach is performed followed by plate fixation, creating absolute stability (no motion at the fracture site) [30]. This concept may also apply to simple metaphyseal fractures. However, articular fractures are more critical as malalignment of the articular surface leads to post-traumatic arthritis and potential disability. It has been shown that the best method of treatment for an articular fracture is to reduce the fracture anatomically and provide compression to cancellous bone [31].

The Open Fracture

The open fracture represents a musculoskeletal injury that brings the assessment and treatment of all the components of the musculoskeletal injury together. In order for a successful result, a thorough assessment of the soft tissue injury to each component of the limb segment is necessary. The final result of this injury will be determined by the ability to salvage as much viable tissue especially muscle while avoiding infection and achieving bone union in appropriate alignment and length. The initial assessment of the soft tissues is performed at the time of debridement which must occur as soon as the patient and surgical team are satisfactorily prepared. Coverage with appropriate board spectrum antibiotics should be commenced as soon as a physician diagnoses the injury as open. It has been shown that the earlier the antibiotics are given, the lower the infection rate. Debridement must achieve a clean surgical wound and be as

Table 1.1 Orthopaedic Trauma Association classification of open fractures

Skin
1. Can be approximated
2. Cannot be approximated
3. Extensive degloving
Muscle
1. No muscle in area, no appreciable muscle necrosis, some muscle injury with intact muscle function
2. Loss of muscle but the muscle remains functional, some localized necrosis in the zone of injury that requires excision, intact muscle-tendon unit
3. Dead muscle, loss of muscle function, partial or complete compartment excision, complete disruption of a muscle-tendon unit, muscle defect does not approximate
Arterial
1. No injury
2. Artery injury without ischemia
3. Artery injury with distal ischemia
Contamination
1. None or minimal contamination
2. Surface contamination (easily removed not embedded in bone or deep soft tissues)
3a. Imbedded in bone or deep soft tissues
3b. High risk environmental conditions (barnyard, fecal, dirty water, etc.)
Bone Loss
1. None
2. Bone missing or devascularized but still some contact between proximal and distal fragments
3. Segmental bone loss

extensive as necessary to assure that there is viable tissue at either end of the open wound. This may take several debridements depending on the amount of contamination and force applied to cause the injury. Stabilization of the bone is imperative to establish a solid pillar upon which the soft tissue can start to heal. This stabilization will be determined at the time of initial debridement based on contamination, needs for further surgical debridement, and ability to close wound. A viable and clean surgical wound will allow the bone to be definitively stabilized with internal fixation. Any concern about further muscle death and failure to completely debride the fracture will lead to the use of external fixation to allow the injured area to be adequately re-explored at the subsequent debridements. Reconstructive soft tissue procedures must be done as soon as possible with a clean and viable injury bed. The bone reconstructive procedures are delayed till the soft tissue envelope has healed and can often be performed up to 3 months.

Although the Gustilo open fracture classification has been used for years to document and guide care, it has been shown to be unreliable in its application [32]. The OTA has developed a more predictive and reliable universal classification system [33]. With this validated system, it is hoped that a better understanding of the open injury and its treatment modalities and results will be forthcoming (Table 1.1).

The aim of modern injury care for that musculoskeletal injury is to not only achieve bony union but more importantly to achieve the maximum function that any injured extremity can obtain. In order for these goals to be achieved, an understanding of the injury to all the components of a limb segment must be understood completely. With this understanding, appropriate methods of treatment of the soft tissue injury, bony injury, and articular injury can maximize the outcome of any given musculoskeletal injury.

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Perioperative Management

2

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General Aspects of Perioperative Trauma Management

The Patient

Although anyone can be a trauma patient, traumatic injury predominately affects the younger population. However, the elderly are also at risk due to increasing mobility and activity later in life. The etiologies of injury are as diverse as their comorbidities. Therefore, a patient admitted to the trauma bay at any time of day may be of any age and may have any combination of injuries and resulting problems, as well as preexisting disease, which may complicate the course of the hospital stay.

The Team

The best management of trauma patients is achieved through a coordinated and multidisciplinary approach. To achieve this goal, the combined workload of the initial assessment, primary resuscitation, diagnostic procedures, and immediate response to life-threatening situations must to be distributed. Because time is crucial, especially in the first phase after admission, joint action on diagnostic procedures and urgent treatment are necessary. Therefore, trauma surgeons, anesthesiologists, and radiologists work simultaneously and in close cooperation. Specialized assistants such as nurses or technicians provide support. The specific division of duties reduces the field of activity for each single provider and enables a better focus on individual aspects of care. Thus, the potential risks of personal overload and of resulting mistakes may be decreased. The duties and responsibilities of each team member must be defined by consensus in advance and must be communicated to the entire team. Ideally, all team members know each other, including their strengths and weaknesses. However, in large trauma centers, this may not be the case. Whenever possible, the team members should briefly introduce themselves and wear color-coded vests to help organize and identify the team members.

The Team Leader

All teams need a leader, especially if different specialties and multiple levels of hierarchy are involved. A predesignated team leader is responsible for oversight and coordination of the multidisciplinary treatment of the trauma patient. All relevant information should be communicated to the team leader. Requests posted from any member of the team (e.g., an urgent need for transfer to the OR) will influence the decision making on sequential management and priority setting. The

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clinical condition of a trauma patient is dynamic, and changes may occur quickly (e.g., a pneumothorax turns into a tension pneumothorax with hemodynamic changes). The response to any alteration of patient's condition must be immediate and targeted. Therefore, priorities should be set dynamically and flexibly.

Use of Algorithms

Various methods to optimize medical and logistic processes in trauma care have been developed worldwide. Algorithm-based guidelines help to maintain an organized workflow and allow for dynamic, situation-based, and comprehensive teamwork. The most popular international course system is Advanced Trauma Life Support (ATLS) of the American College of Surgeons [1]. Trauma physicians from various disciplines are educated and trained together as a team. Basic knowledge of such concepts is necessary for any physician involved in trauma care. However, local conditions and possible structural limitations of each trauma center may hamper the implementation of such concepts. Local standard operational procedures (SOPs) must be formulated to guarantee best practices and care.

Taking Over the Patient

The emergency medical technician (EMT) and/or emergency physician who transports the patient to the emergency department has the earliest contact with the patient. During the initial treatment at the scene, EMTs spend more time with the patient than any member of the trauma team. They perform a systematic physical examination and obtain comprehensive information on the medical history of the patient. By contact with bystanders who observed the patient's accident, they may have gathered and organized valuable information. These data may provide clues to facilitate the identification of specific injuries by providing details on the injury mechanism and kinematics. Therefore, this information must not be lost at the interface between prehospital and clinical management. The EMTs verbally describe the patient to the entire trauma team at a defined time point, in a clear and orderly manner. The team should focus on this brief overview that includes the details listed in Table 2.1. Active listening avoids unnecessary queries or misunderstandings. The team leader calls on the EMT to provide the briefing either just before or after the repositioning of the patient. Furthermore, all relevant details on prehospital findings and treatment must be documented in an adequate manner for later access.

Table 2.1 Important information from the pre-hospital phase

Scene and situation	
Kinematics and mechanisms	Height of fall, death of other passengers
Use of safety devices	Airbag, helmet or other protective gear
Time of injury	
Initial patient status	
Vitals including	ABC
Mental status (GCS or AVPU)	D: Prior to anesthesia or sedation
Treatment and progress	
Measures performed	Compression bandage applied
Medication given	Analgesics, sedatives, fluid
Were these measures successful?	Vitals and mental status, external bleeding control
Concomitant medical information	AMPLE
Difficulties at scene	Entrapment in car, difficult airway
Personal data (patient and affiliated)	Who can be contacted for more information?

A airway, *B* breathing, *C* circulation, *D* disability, *GCS* Glasgow Coma Scale, see also Table 2.9; *AVPU* alert, verbal, pain, unresponsive, see also Table 2.10; *AMPLE* allergies, medications, past medical history, last oral intake, events preceding

Initial Assessment and Treatment

The focus of the initial assessment and treatment is on identifying life-threatening conditions as soon as possible and initiating the right treatment at the right time. It is advisable to follow a standardized procedure such as the ABCDE

Table 2.2 Common reasons for obstructed airway

Tongue	Severe cognitive impairment following brain injury, cerebral hypoperfusion, intoxication or sedation
Gastric content	Aspiration
Blood	Hemorrhage from mouth or nose
Distorted anatomy	Direct trauma to the head
Foreign body	E.g., dental prosthesis, broken teeth, misapplied oral airway devices

approach and thereby to focus to the most critical disorder first. Otherwise, important information needed to deliver lifesaving therapy may be missed. "Treat first what kills first!" is an advisable principle [2].

Airway

First, it is essential to ensure an open and patent airway. A lack of oxygen is the most urgent threat to life. If the upper airway is obstructed, all further efforts to transport oxygen to the lungs will inevitably fail. Insufficient oxygenation of the blood will result in a critical undersupply to organs. The brain is most susceptible to permanent damage due to hypoxemia, and trauma patients are at risk for airway obstruction (Table 2.2). A patient who gives vocal responses will most probably have a patent airway up to that time point. If not, a simple chin lift or jaw thrust may quickly reopen the airway. The gentle insertion of nasopharyngeal or oropharyngeal airways into semiconscious patients may further secure the airway as bridging therapy. Patients with intact gag reflexes may not tolerate these devices. If they are tolerated, they may assist oxygen insufflation with non-rebreather masks in spontaneously breathing patients. Furthermore, they often facilitate bag-valve mask ventilation that may be indicated. If the patient is unconscious or has otherwise lost protective airway reflexes, a secure airway must be established immediately to prevent aspiration and ensure proper oxygenation. Whenever feasible, the patient's airway should be assessed in advance for potential difficulties with airway maneuvers. The mnemonic LEMON (look, evaluate, mallampati, obstruction, neck) [3] may be helpful. Factors predictive of difficulties are listed in Table 2.3.

Table 2.3 Some findings that may suggest the presence of a difficult airway

Long upper incisors
Short interincisor distance (<3 cm)
Extreme relation of maxillary to mandibular incisors (e.g., prominent "overbite")
Short thyromental distance (<6.5 cm or < 3 ordinary finger breadths)
Restricted visibility of uvula (e.g., Mallampati class > II)
Macroglossia
Short and/or thick neck
Limited neck mobility (chin to chest or extension)
Highly arched or very narrow palate
Obesity
History of difficult airway
Acute injury to the face, mandible or neck

The table is not intended as an exhaustive list

Endotracheal intubation is considered the gold standard but is also known to be potentially difficult, particularly in trauma patients. Furthermore, all trauma patients are non-fasting, and there is a high risk of aspiration. Orotracheal intubation should be performed using a standardized rapid sequence induction (RSI) technique. This includes preoxygenation [4–7] with 100 % oxygen to wash out nitrogen and maximize the oxygen pool and to delay arterial desaturation during successive apnea. All equipment required in the subsequent process must be at hand and tested for functionality. Next, medication to induce anesthesia is administered through a patent IV access. Bag-valve mask ventilation before intubation should be avoided unless the patient's ventilation is inadequate. The vocal cords are visualized by direct laryngoscopy, and the endotracheal tube (ET) is gently passed through them. If the first attempt to correctly place the ET fails, a re-saturation of the patient by manual ventilation (100 % oxygen with a tight-fitting mask including reservoir) is required. If fluids such as blood or saliva occlude the visual field, cautious suctioning of the mouth and pharynx may improve conditions for the next attempt.

Table 2.4 Common indications for endotracheal intubation

Cardiac or respiratory arrest
Airway obstruction
Respiratory insufficiency
Severe hypoxemia (despite supplemental oxygen)
Severe cognitive impairment (GCS < 8) requiring airway protection
Need for deep sedation or analgesia (also preoperative management)
Severe hemorrhagic shock
Increased intracranial pressure (transient hyperventilation)
Delivery of 100 % oxygen to patients with carbon monoxide intoxication
Facilitation of management (e.g., diagnostics) in combative or intoxicated patients

Immediately after successful intubation, the correct endotracheal position of the tube must be verified by the use of a carbon dioxide detector, ideally by capnography. If capnography is not available, a colorimetric CO_2 monitoring device may indicate proper intubation of the airway or false esophageal intubation. Because neither device is capable of detecting main-stem bronchus intubation, a physical examination including a thorough auscultation in search of bilateral ventilation and thoracic excursion is required. Radiographic imaging can also identify an excessively deep intubation. Securely fixing the ET helps to prevent tube dislocation during later patient movement (e.g., transfer to computer tomography). During the entire airway maneuver, monitoring of oxygen saturation, cardiac rhythm, and blood pressure is mandatory. Common indications for endotracheal intubation are listed in Table 2.4.

Additionally, trauma patients with suspected cervical spine injury require gapless immobilization of the cervical spine until a radiographic diagnosis can securely rule out an injury. Therefore, an additional assistant is required to properly provide manual inline immobilization (MILS) of the cervical spine during the entire airway maneuver [8]. The anterior portion of the cervical collar may be opened while strictly maintaining MILS, but conventional laryngoscopy may still be difficult.

Difficult Airway

There is no standard definition of a difficult airway in the literature. A difficult airway is complex and challenges all physicians involved in airway management. Multiple factors derived from the patient, the clinical setting, and the expertise of the practitioner contribute to difficulty. The "Practice Guidelines for Management of the Difficult Airway" [9] developed by the American Society of Anesthesiologists Task Force on Difficult Airway Management [10] suggests the use of the following descriptions:

1. Difficult face-mask ventilation
2. Difficult laryngoscopy
3. Difficult tracheal intubation
4. Failed intubation (after multiple intubation attempts)

Repeated intubation attempts (>2) are independently associated with increased adverse events such as hypoxemia, dysrhythmia, cardiac arrest, regurgitation or aspiration, airway or dental trauma, and main-stem bronchus or unrecognized esophageal intubation [11, 12]. Hence, the use of alternative airway devices should be considered if multiple attempts to secure the patient's airway by conventional direct laryngoscopy techniques fail. A difficult airway cannot always be anticipated and can easily surprise an unprepared emergency team. Everyone who is or who may become responsible for securing the airway must have a predesigned strategy to cope with an expected or unexpected difficult airway. Standardized protocols tailored to the specific setting of each trauma center facilitate decision making in situations with time constraints and enable the trauma team to be ahead of the emergency. In case of a suspected difficult airway, the difficult airway protocol in effect should be followed immediately. The accepted standard of care for an anticipated difficult intubation is conscious intubation using a flexible bronchoscope.

In addition, numerous supraglottic/extraglottic airway devices are available. Most are part of routine daily anesthesia care, but they are also considered rescue devices in difficult airway scenarios. These devices can be blindly inserted to an extraglottic position, and they allow for indirect ventilation through a glottic opening. Some

devices allow for the placement of a stylet, exchange catheter or flexible fiberscope through the airway device into the trachea. An endotracheal tube can then be railroaded over the provided guide wire to secure the airway.

A variety of fiber-optic or video-assisted rigid laryngoscopes allow for direct visualization of the intraglottic airway in real time to securely place an endotracheal tube. These commercially available devices differ in size and quality with respect to attached or remote video screens, and they have the option of tube guidance. One instrument's particular feature may be advantageous in certain circumstances but disadvantageous in others (e.g., the size of video screen aids viewing but limits portability). Whereas video laryngoscopy generally offers better glottic visualization than conventional laryngoscopy [13–15], the success rates of endotracheal intubation are not necessarily higher due to the difficulties of tube insertion with some devices [15]. Furthermore, blood, emesis, or airway injury may occlude the optical portion of the device and limit the videolaryngoscopic view in trauma patients.

Patients with suspected or known cervical spine injury do not necessarily have a difficult airway, but they may need a technique with the least cervical motion to prevent further injury to the spine. There is ongoing debate about best practices in the traumatized patient [16–18]. A conscious fiber-optic intubation is preferable in cooperative, hemodynamically stable patients who are not in immediate respiratory distress [19]. While the use of flexible fiber-optic bronchoscopes has some advantages, it is time-consuming. If time is essential to securing the airway, more rapid alternatives, such as those described above, should be used.

The clinicians will have to choose among the broad variety of tools and techniques depending on the actual situation and/or algorithm. The physicians responsible for airway management should have experience with at least two or three different instruments. To become familiar with these techniques, the tools can be used in routine cases to prepare for the management of difficult airways. To discuss all available and suitable alternative airway devices here would go beyond the scope of this chapter; they are described elsewhere [8, 13–16, 18–26].

If all options fail to intubate, ventilate, or oxygenate the patient, cricothyrotomy may be the final lifesaving procedure. These final options include needle cricothyrotomy, percutaneous cricothyrotomy, or surgical cricothyrotomy with emergency tracheostomy.

If a patient is admitted with an airway device in place, its correct positioning must immediately be confirmed by capnography and auscultation. Once the airway is secured and thoroughly confirmed or the conscious patient offers a patent airway, high-flow oxygen should be insufflated before the assessment of breathing and ventilation.

Breathing

The quality as well as the quantity of breathing and ventilation are essential parts of the initial assessment. In spontaneously breathing patients, the evaluation of the respiratory rate, rhythm, and effort to breathe provides a quick overview of the patient's condition. While approaching the patient, the examination should note signs such as nose flaring, agitation, labored respiration, or the inability to speak several words coherently. Any abnormalities may indicate respiratory distress and compromised air exchange. In addition, the respiratory rate should be counted or at least assessed. A slow (<10) or high (>20) respiratory rate urgently needs attention. Changes in the respiratory rate require immediate treatment, as they are reliable markers for insufficient ventilation. The supply of oxygen by a non-rebreather face mask with a reservoir may not be enough to compensate for insufficient ventilation. In these cases, assisted bag-valve mask ventilation facilitates ventilation, optimizes oxygenation, and may avert further deterioration of the patient's respiratory condition. A respiratory rate within the normal range combined with a shallow depth of breathing also indicates that assistance is needed. This type of breathing is considered to be hypoventilation causing respiratory hypercarbia/hypercapnia, and it has the potential to influence

mental status (e.g., somnolent or very fatigued patients). The responsible physician should look for symmetry of chest raises (e.g., flail chest) and thoroughly auscultate for bilateral lung sounds in patients that are spontaneously breathing or on mechanical ventilation (if not performed during airway management). Audible respiratory sounds indicate upper airway obstruction, whereas decreased or absent lung sounds are highly suspicious of a pneumothorax or hemothorax. The suspicion should be ruled out or confirmed and diagnosed more precisely with a chest x-ray [27]. Painful or irregular breaths may be trauma-related and should be monitored to identify any thoracic injuries. Severe impairment of breathing and ventilation is suspected in the presence of a tension pneumothorax, injuries to the spinal cord, or traumatic brain injury with alterations to the regulatory respiration center. Clinical signs of a pneumothorax include rather unspecific findings such as tachypnea and tachycardia but also more specific findings as pulsus paradoxus, decreased breath sounds, and hyperresonance on percussion on the affected side. In addition to these signs and symptoms, some patients present with diminished mental status caused by hypoxia. Audible breath sounds do not rule out a pneumothorax. A hypoxic, cyanotic patient with increased jugular venous distention is highly susceptible to a tension pneumothorax and needs urgent treatment. A tension pneumothorax increases the intrathoracic pressure. Therefore, the need for unusually high airway pressure in patients on mechanical ventilation is another sign of a life-threatening tension pneumothorax. Lifesaving decompression of the chest should be initiated immediately in hemodynamically instable patients (obstructive shock). Rapidly performed needle decompression can bridge therapy until a chest tube can be inserted to restore an air-free pleural space. Whereas radiographic imaging corroborates the diagnosis of a pneumothorax and allows for differentiation of a hemothorax, a tension pneumothorax must be treated without delay, i.e., without waiting for radiologic confirmation. The diagnosis can be made mainly on the basis of clinical examination findings and attentive observance of the patient.

Table 2.5 Causes of inadequate ventilation

Pneumothorax or hemothorax
Direct trauma to chest wall
Injury of the airway (trachea or mainstem bronchi)
Lung contusion
Decreased respiratory drive (resulting from traumatic brain injury, shock, hypothermia, intoxication or excessive sedation)
Aspiration (blood, gastric content)
Cervical spine injury
Toxic lung edema (following gas inhalation)

Inadequate ventilation may also be caused by the other factors listed in Table 2.5. It is important to note that a patient may suffer from various conditions in parallel that interfere with ventilation. Therefore, all possible reasons for inadequate ventilation must be assessed if the patient is in respiratory distress. An intubated patient may also rapidly deteriorate. As a first step, the patient is taken off the ventilator, and manual ventilation is performed. This change adds the sense-based information derived from the use of the bag. At the same time, the use of the mnemonic DOPE [26] to quickly assess the most common causes of an acute decline in patients on mechanical ventilation may be helpful:

D. **Displaced tube**? Bilateral breath sounds still present? Positive capnography?

O. **Obstructed tube**? Does thick mucus obstruct the tube? If so, perform suctioning. Does the patient bite on the tube? Ensure adequate anesthesia depth.

P. **Pneumothorax**? Positive pressure ventilation may exacerbate the valve effect of an existing pneumothorax and progressively build up further pressure in the pleural space. Auscultate and check for elevated airway pressure.

E. **Equipment failure**? Check for oxygen supply. Does the ventilator function correctly? If in doubt, replace any questionable equipment.

Circulation

Once a patent or secured airway and optimized conditions for breathing and ventilation are established, gas exchange and oxygenation of

the blood can take place. Subsequently, sufficient circulation is a mandatory precondition to transport oxygen to the tissues on demand. Although initial monitoring of a trauma patient includes standard monitoring (i.e., ECG, pulse oximetry, and noninvasive blood pressure measurement), a brief, easy-to-perform, and focused clinical examination will provide valuable information on the patient's perfusion and circulatory condition. This includes pulse palpation and an assessment of cardiac rhythm. The absence of a palpable pulse on an uninjured extremity may indicate the decompensated phase of shock. Pale, cold, and damp skin is also associated with shock and severely diminished perfusion and can be evaluated within seconds. Furthermore, a prolonged capillary refill time (>2 s) is suggestive of affected perfusion. However, trauma patients are disproportionately young and without limiting comorbidities [28]. Therefore, they can often compensate severe blood loss for a certain period of time without being hemodynamically compromised. Once the blood loss overcomes the compensatory capacities, a rapid and potentially fatal breakdown of circulation occurs. To assess the severity of insufficient circulation and shock, lactate and/or base excess measurement is recommended [29]. To prevent circulatory collapse and avoid secondary damage to the patient, bleeding control at the earliest possible moment is crucial.

Bleeding Control

Control of ongoing hemorrhages should be initiated on arrival and without delay. Manual pressure or pressure dressings may control continuous external bleeding. If direct pressure fails to arrest life-threatening blood loss from extremities, a tourniquet may be applied early as a lifesaving measure [30–32]. Most evidence for the beneficial use of tourniquets is derived from battlefield injuries [31–36]. However, that type of injury and the injury circumstances differ from the mangled extremities observed in civilian life. Tourniquets can be used temporarily to allow further diagnostics and aid resuscitation until surgery is possible. To avoid potential side effects from tourniquets, such as limb ischemia or nerve paralysis, the duration of tourniquet application should be as short as possible [37, 38]. Furthermore, the use of operative tourniquet systems that are commonly employed in elective surgery is favored over field tourniquets with regard to adverse events [39]. If available, it is advisable to switch to pneumatic devices with an appropriate inflation (above systolic pressure) in case the EMT has applied a field tourniquet to stop a life-threatening hemorrhage.

Severe bleeding may also occur from pelvic fractures. Several circumferential pelvic binders are available for temporary and rapid pelvic closure. In some types of fracture patterns, external pressure applied to the pelvis successfully reduces the pelvic volume. This compression may be sufficient to arrest bleeding from lacerated vessels (mainly veins and smaller arteries) and cancellous bone. The stabilization of pelvic fractures in the emergency department with commercial compression devices or simple bed sheets in hemodynamically instable patients is advised in the Advanced Trauma Life Support guidelines [40]. All of these measures are temporary and are considered adjunct treatment options to minimize blood loss until definitive control of bleeding is achieved.

Whereas surgery is the mainstay of bleeding control, transcatheter angiographic embolization (TAE) is an alternative to arrest hemorrhage in a certain subgroup of patients. TAE is an established, minimally invasive technique to control arterial bleeding from solid organ injury or pelvic fracture [41–45]. In general, TAE is associated with low morbidity [44–47], but complications such as necrosis of the distal colon, ureter, uterine, and bladder as well as perineal wound sepsis [48], ischemic damage of the gluteal muscle[49], and paresis [50] have been reported. These risks should be considered if angiography and embolization are an option for the diagnosis and acute treatment of the bleeding patient and may outweigh the published success rates of over 90 % in arresting pelvic hemorrhage [45, 51–54]. TAE should be performed soon after admission in hemodynamically unstable patients with ongoing or suspected bleeding [55] because mortality rates increase from 14 to 75 % if intervention is delayed (>3 h) [51]. However, there are still ongoing debates regarding how to identify

patients who will benefit from early TAE and how to determine the most beneficial sequence of angiographic embolization to control bleeding relative to surgical interventions [56, 57]. At this time, there are no homogeneous results from clinical studies that precisely aid clinical decision making and refine the optimal timing of TAE versus surgery. Depending on institutional resources, each physician or institution will have to decide which treatment to perform first to control bleeding and avoid further deterioration, such as hemorrhagic shock.

Hemorrhagic Shock

Significant blood loss following trauma may sequentially lead to hemodynamic instability, decreased tissue perfusion, cellular hypoxia, organ damage, and death. According to ATLS [1], the mechanism of injury in combination with the severity of injury, the patient's physiological condition, and the response to volume resuscitation may be used to guide the initiation of surgical bleeding control. For the degree of hemorrhagic shock and subsequent interventions, it is therefore essential to estimate blood loss. Definitions of blood loss are displayed in Table 2.6 [58]. Early signs of shock are: altered level of consciousness as a result of reduced cerebral perfusion, delayed capillary refilling, mottled skin as a consequence of reduced peripheral perfusion, as well as oliguria. An accurate estimation of total fluid loss is further aggravated by urinary loss, insensible perspiration, and tissue edema. Thus, it is important to remember that the average adult blood volume represents approximately 7 % of body weight and that older individuals have a smaller blood volume. In comparison, children have an average of 8–9 % blood volume of body weight, whereas infants have a total blood volume of 9–10 % of their total body weight. An acute blood loss of up to 750 mL is considered as non-shock, whereas a class IV shock is a preterminal state requiring immediate therapy (Table 2.7) [59, 60].

Predictors of Shock and Coagulopathy

Measurements of hematocrit are routinely obtained in bleeding patients. A considerable limitation of the hematocrit is the influence of fluid administration and RBC transfusion [61, 62]. Although frequently measured hematocrit may be an indicator for ongoing blood loss, traumatized patients with significant blood loss may also show a stable hematocrit.

Thus, serial measurements of serum lactate and base excess are more sensitive markers to estimate the extent of bleeding. The level of lactate generated by anaerobic glycolysis and tissue hypoperfusion is an indirect marker for hemor-

Table 2.6 Definitions of massive severe blood loss

Loss of an entire blood volume equivalent within 24 h; or
Loss of 50 % of blood volume within 3 h; or
Continuing blood loss at a rate of 150 mL/min; or
Continuing blood loss at a rate of 1.5 mL/kg/min over 20 min; or
Rapid blood loss leading to decompensation and circulatory failure, despite the support of blood products, volume replacement, and all accepted surgical and interventional treatments to stop bleeding

Modified according to Grottke et al. [58]

Table 2.7 Classification of hemorrhagic shock

	Class			
Parameter	I	II	III	IV
Blood loss (mL)	<750	750–1,500	1,500–2,000	>2,000
Blood loss (%)	<15	15–30	30–40	>40
Heart rate (beats/min)	<100	>100	>120	>140
Blood pressure	Normal	Decreased	Decreased	Decreased
Respiratory rate (breaths/min)	14–20	20–30	30–40	>35
Urine output (mL/h)	>30	20–30	5–15	Negligible
CNS symptoms	Normal	Anxious	Confused	Lethargic

Modified from American College of Surgeons Committee on Trauma [60]
CNS central nervous system

rhagic shock, as shown by Manikis et al. [63]. In this study, initial lactate levels of patients with significant trauma were increased in non-survivors. Aside, it was shown that a prolongation of normalization of elevated lactate levels was also associated with the development of organ failure. Accordingly, base deficit has been shown to be also a potential predictor of mortality in patients with hemorrhagic shock following major trauma [64]. For instance, both in adult and pediatric patients, the base deficit was sensitive for the degree of hemorrhagic shock and mortality [65, 66].

The degree of coagulopathy correlates with the severity of trauma and shock [67]. The high mortality associated with hypothermia, metabolic acidosis, and coagulopathy is also referred to as the "lethal triad" or the "bloody vicious cycle." The metabolic derangements and acidosis affect the coagulation system [68]. A prolongation of clotting time, reduced clot strength, and an increase of the degradation of fibrinogen have been observed after the induction of acidemia induced by hydrochloric acid [69, 70].

The causes of hypothermia are multifactorial and interdependent, including altered central thermoregulation, decreased heat production due to tissue hypoperfusion in hemorrhagic shock, exposure to low ambient temperature, and infusion of inadequately warmed resuscitation fluids and blood components [71]. Apart from an impairment of enzyme activity, lower temperatures correlate with reduced synthesis of coagulation factors and also lead to an alteration of platelet function. Thus, mortality in injured patients with temperatures below 32 °C is increased [72]. Clinically significant effects on coagulation, platelet function, and clinical bleeding are already seen at moderately hypothermic temperatures below 34 °C [73]. TF-FVIIa complex activity is reduced linearly with temperature showing only 50 % of the original activity at 28 °C as compared to normothermia [74]. The effect of hypothermia on platelets is addressed to an impaired signal transduction from initial adhesion to activation of platelets mediated by von Willebrand factor traction on glycoprotein Ib/IX receptors [75].

Fluid Administration and RBC Transfusion

The primary goal of volume resuscitation in traumatized patients is to restore tissue perfusion to maintain end-organ function and to avoid inadequate tissue perfusion manifested by anaerobic metabolism as well as lactic acidosis. Generally, crystalloid or colloid solutions are available. Crystalloids may be categorized as hypotonic, isotonic, or hypertonic. For volume resuscitation, only isotonic and hypertonic fluids are employed because hypotonic solutions do not stay in the intravascular space. Crystalloids are inexpensive and resuscitate both the intravascular and interstitial space. Disadvantages include the formation of edema in patients with capillary leak, and higher volumes are needed to achieve equivalent intravascular volume effects compared to colloids.

Colloids are classified into protein and nonprotein solutions. The colloids with protein are albumin (5 % and 25 %) and gelatin solutions. Available nonprotein colloids are starches with various molecular weights (6 % hetastarch, 10 % pentastarch) and dextrans (e.g., dextran-40). In comparison to crystalloids, colloids remain longer in the intravascular space, exhibit greater volume expansion, and presumably cause less edema. The primary drawbacks include the potential for anaphylaxis (especially with dextrans and gelatin solutions) and a negative impact on coagulation, and they have the potential to cause negative effects on renal function by tubular injury. Despite several studies in the last two decades, it is still unclear what type of volume should be employed as primary resuscitation fluid. Numerous studies have documented an increased risk of death in patients treated with colloids compared with patients treated with crystalloid solutions [76, 77]. This effect was even more pronounced in trauma patients [78, 79]. In contrast, a meta-analysis performed by Roberts et al. showed no difference in mortality between treatment with colloids and crystalloids [80]. The infusion of hypertonic solutions has been shown to lower intracranial pressure and improve survival in patients with penetrating torso injuries requiring fluid resuscitation [81].

Generally, various resuscitation regimes, the heterogeneity of the study populations, and different outcome parameters complicate the accurate analysis of available studies. However, currently, the use of crystalloids in bleeding patients for initial therapy is advised [29]. Hypertonic solutions may also be considered, although the evidence for increased survival is inconclusive. In hemodynamically unstable patients, the infusion of modern hydroxyethyl starch or gelatin can be considered.

Transfusions of red blood cells (RBCs) are a mainstay in trauma management. The concept of specific component therapy was developed during the 1960s. Whole units of blood are separated into plasma, platelets, and RBCs, and these components may be separated further (e.g., by cryoprecipitation). This strategy allows for resource allocation according to the individual needs of the patient, resulting in both economic and logistical benefits. One disadvantage is that substitution with plasma-free and thrombocyte-depleted RBCs may lead to coagulopathies at an earlier stage compared to the substitution of whole blood. For example, an analysis from the Vietnam War showed that platelet counts did not fall below 10×10^9/L, despite massive transfusions of 6 L of whole blood [82].

RBC transfusions are used to treat hemorrhage and anemia and to improve oxygen delivery to tissues. Erythrocytes also contribute to hemostasis by influencing the biochemical and functional responsiveness of activated platelets via the rheological effect on platelet margination and by supporting thrombin generation [83]. To date, no prospective randomized trials are available that have determined the optimal transfusion trigger in the resuscitation of traumatized patients. Reanalyzed data from the Transfusion Requirements in Critical Care trial showed that critically ill patients could tolerate hemoglobin levels as low as 7 g/dL [84, 85]. The restrictive transfusion regimen (transfusion trigger <7 g/dL) resulted in a reduced number of RBCs transfused compared to a liberal regimen (transfusion trigger <10 g/dL). Although the analysis did not show a beneficial effect of a restrictive transfusion approach, as reflected by similar incidences of multiorgan failure (MOF) and post-traumatic infections, the approach may still have provided benefits because the study was not primarily designed or powered to answer this question. In contrast, an observational study with 15,534 patients by Malone et al. revealed different results; in this trial, 1,703 trauma victims received on average 6.8 ± 6.7 units of RBCs [86]. After controlling for potential confounders—injury severity score (ISS), Glasgow coma score (GCS), shock variables, age, and race—RBC transfusion was associated with increased mortality, admission to ICU, and ICU length of stay. Until further RCTs adequately address these issues, it is generally agreed that hemoglobin levels in bleeding patients should be maintained at 7–8 g/dL. Although many centers transfuse patients with traumatic brain injury (TBI) to achieve hemoglobin levels of 10 g/dL, there is no strong evidence for this approach. Thus, targeting a higher hemoglobin level in patients with TBI compared to other critically ill patients is not recommended. In multiple studies, transfusions of RBCs have been shown to be associated with increased mortality, acute lung injury, incidence of post-traumatic infections, and renal failure (Table 2.8) [87, 88]. These adverse events may

Table 2.8 Acute and delayed complications of transfusion

Acute complications
Acute hemolytic transfusion reaction
Febrile non-hemolytic transfusion reactions
Transfusion-related acute lung injury
Allergic reactions
Bacterial sepsis
Hypocalcemia
Hyperkalemia
Acidosisa
Hypothermia
Dilutional coagulopathy
Delayed complications
Delayed hemolytic transfusion reactions
Transfusion-related immmunomodulation; Post-traumatic infections
Multi organ failure
Transfusion-transmitted diseases
Post-transfusion graft-versus-host disease
Post-transfusion purpura

be particularly important with RBC transfusions stored for more than 14 days [89].

Permissive Hypotension in Traumatized Patients

Traditional concepts of volume resuscitation in the actively hemorrhaging trauma patient emphasize maintenance of a normal systolic blood pressure. This approach may increase the risk of the dissolution of blood clots from the wound by increasing the hydrostatic pressure. The strategy to avoid this negative impact on early aggressive volume resuscitation while maintaining sufficient organ perfusion is called "permissive hypotension". Although the evidence from large RCTs is still missing, studies have shown a positive impact of permissive hypotension in patients with penetrating trauma [90, 91]. Aside, a Cochrane meta-analysis showed no negative effects of this regime as compared to early or larger volume resuscitation [92]. In patients with proven or signs of traumatic brain injury and spinal injuries, the low-volume approach is contraindicated. Lower blood pressures bear the risk of insufficient perfusion pressure, which is necessary to ensure tissue oxygenation of the injured central nervous system.

Conclusively, a systolic blood pressure of 80–100 mmHg should be maintained until major bleeding has been terminated in the initial phase of treatment of injured patients without injuries of the central nervous system.

Use of Vasopressors

Although fluid resuscitation is the first approach to restore sufficient mean arterial pressure in hemorrhagic shock, the use of vasopressor therapy may be required as adjunctive therapy to maintain tissue perfusion and sustain life. Aside, some severely injured patients may be nonresponsive to fluid resuscitation. Although larger studies from humans in the field of hemorrhagic shock are missing, current evidence from experimental studies suggests the use of norepinephrine vasopressor therapy [93]. Norepinephrine is a potent α1[alpha1]-adrenergic receptor agonist with modest β[beta]-agonist activity. The stimulation of α1[alpha1]-receptors predominantly exhibits vasoconstriction and less direct inotropic properties. Thus, norepinephrine primarily increases systolic, diastolic, and pulse pressure and with minimal impact on net cardiac output and chronotropic effects. The increase of the sympathetic tone may also be favorable in the face of the potential negative sympathetic tone effects of medications used for sedation. In cases of poor response to fluid resuscitation or any signs of cardiac trauma, a close evaluation of cardiac function is needed. To maintain cerebral perfusion in patients with TBI, the mean arterial pressure should be maintained at 80 mmHg, whereas a systolic pressure of 80–100 mmHg is advised in traumatized patients without TBI [29].

Disability

The assessment of the patient's mental status or cerebral function may be used in the early evaluation as a surrogate parameter for cerebral perfusion. The brain is very sensitive to an acute undersupply of oxygen. Therefore, any acute changes in the patient's level of consciousness or behavior must be noted and monitored carefully while searching for the underlying cause. Neurological alterations are often an early, if not the first, sign of severe deterioration (e.g., the initiation of shock or hypoxia). Changes in mental status must to be interpreted based on the initial neurological findings. This assessment begins with a critical look at the patient's behavior; for example, an aggressive, combative trauma patient who is reluctant to cooperate in any way is highly susceptible to an acute hypoxemia or a traumatic brain injury. In addition to observation, neurological scoring systems such as the Glasgow Coma Scale (GCS; Table 2.9) or the simple mnemonic AVPU (alert, verbal, pain, unresponsive; Table 2.10) [94] offer quantifiable scoring rates. To detect any changes over time, the neurological examination should be performed repeatedly. Both assessment tools rate the patient's response to external stimuli such as verbal commands and pain. The evaluation of mental status using the AVPU scheme is simple but also less detailed than that using the GCS. The AVPU focuses on

Table 2.9 Glasgow Coma Scale

Parameter	Score	Response
Eye-opening	4	Spontaneous
	3	To speech
	2	To pain
	1	None
Best verbal response	5	Oriented
	4	Confused
	3	Inappropriate words
	2	Incomprehensible sounds
	1	None
Best motor response	6	Obeys commands
	5	Localizes pain
	4	Withdraws from painful stimuli
	3	Abnormal flexion (decorticate posturing)
	2	Abnormal extension (decerebrate posturing)
	1	None

Table 2.10 AVPU

AVPU level	Response and assessment findings	
Alert	Spontaneous	
	Alert and oriented × 4	Person, place, time, and event
	Alert and oriented × 3	Person, place, and time
	Alert and oriented × 2	Person and place
	Alert and oriented × 1	Person only
Verbal	Responds to verbal stimuli	
Pain	Responds to painful stimuli	
Unresponsive	No response	

Modified according to Aehlert [94]

awareness as demonstrated by orientation to person, place, and time. For example, a patient who is aware of his own person as well as time and place is considered to be alert and oriented ×3. An APVU grade lower than three characterizes a patient who is confused or disoriented. The GCS is a more comprehensive and effective tool to assess neurological function. A score for each patient's response to eye opening (*E*) and best verbal (*V*) and motor response (*M*) is documented. The scores sum to a total score ranging from 3 (poorest) to 15 (best), for example, $E=2$, $V=3$, and $M=5$, total GCS score of 10. Significant changes in mental status (beginning in the prehospital setting; see Table 2.1) are reflected in changes in the documented scores. A score of 8 or less is usually considered an indication for endotracheal intubation. Both neurological assessment tools help to determine the patient's neurological status, but they share one major limitation: they do not account for pupillomotoric responses. Adequately functioning pupils are equal in size, round, and promptly reactive to stimulation with light. Unilateral dilatation and/or unequal reactivity to light in an unconscious trauma patient may be a consequence of brain herniation (i.e., pressure on cranial nerve III, oculomotor).

In summary, there are four principal reasons for a diminished state of consciousness in trauma patients:

- Reduced cerebral oxygenation (caused by hypoxemia and hypoperfusion)
- Injury to the central nervous system
- Drug or alcohol abuse
- Metabolic and neurological deviation (diabetes mellitus, epilepsy)

These causes may be present in any combination and may interrelate. They may have caused the trauma itself (e.g., a road accident due to drug intoxication). Every patient with a conspicuously altered mental state should receive a thorough neurological examination. A cranial computer tomogram should be considered in any unconscious (or intubated) patient who cannot be examined and/or who arouses suspicion of a brain injury.

Exposure

The final step in the initial assessment is the complete exposure of the patient. All clothing is removed to allow for a physical examination. Apparent injuries tend to attract attention by their graphic appearance, with the risk of underdiagnosing less obvious but sometimes more threatening issues. Therefore, a rapid head-to-toe or a more focused exam depending on the acute situation of the patient is necessary to detect any further injuries or deformities. This examination

must also include the patient's back. To avoid hypothermia and for ethical reasons, the patient is covered with blankets once the physical examination and the complete initial assessment are finished.

Key Points

- The perioperative management of trauma patients can be challenging, especially in the initial phase of care, when multiple diagnostic procedures and urgent treatment are necessary to be performed in parallel. The multidisciplinary approach of the initial care can be coordinated best by standardized, algorithm-based procedures that are practiced by the entire team.
- Airway management is crucial in the treatment of emergency patients because a lack of oxygen is the most urgent threat to life. An insufficient oxygenation puts the patient at imminent risk and will inevitably inhibit an effective treatment in the further course. Therefore, every care provider must be proficient in standard techniques for securing the airway.
- Airway management can be difficult, particularly in trauma patients. Because a difficult airway cannot always be anticipated, the trauma team must have a predesigned strategy to cope with difficulties in securing the airway, and alternative airway devices must be immediately available.
- Bleeding control at the earliest possible moment is fundamental to prevent circulatory collapse/shock and avoid secondary damage to the patient. Serial measurements of serum lactate and base excess are sensitive markers to estimate the extent of bleeding.
- The best volume resuscitation regime is still under debate, but currently the use of crystalloids in bleeding patients for initial therapy is advised.
- RBC transfusions are used to treat hemorrhage and anemia and to improve oxygen delivery to tissues, whereas the optimal transfusion trigger in the resuscitation of traumatized patients has still to be determined.
- Neurological alterations are often an early, if not the first, sign of severe deterioration (e.g., initiation of shock or hypoxia) and may be used as a surrogate parameter for cerebral perfusion during the very early evaluation of the patient.
- Hypothermia increases mortality and morbidity in injured patients and must be avoided/corrected by any means.

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The Pediatric Patient

3

Theddy F. Slongo

Introduction

"In childhood, fractures always heal" and "in childhood fractures the best treatment is conservative, not surgical treatment". These two statements are found at the beginning of practically every textbook about treating fractures in children, as though they were set in stone.

Whilst the first statement is undoubtedly correct even today, the second assertion must be questioned strongly. The overall expectation of adults and children increasingly demands an approach to injury that provides the best possible outcomes. Correspondingly accidents must be treated in such a manner that the child returns again to daily activity without long term disability or deformity.

Fortunately, over the past 15 years there have been significant advances in pediatric fracture care, including recommendations for more precise surgical treatment. Treatments have evolved to provide improved quality of life in the short and long term for both patients and families. We know that a great number of conservative fracture treatments, particularly in the forearm and femur area, lead to poor functional results. The fact that children adapt to functional losses does not absolve us from adopting better treatment strategies.

Underlying a comprehensive treatment strategy for pediatric fractures is the basic knowledge of the available treatment options and techniques:

- Closed and conservative fracture management including adequate and high quality plaster cast technique
- Knowledge of appropriate pediatric specific implants and surgical techniques (in respect to size and age as well as healing behavior)
- Specific knowledge of modern pediatric specific implants such as Elastic Stable Intramedullary Nails (ESIN), External fixators, locking plates for Minimally Invasive Osteosynthesis (MIO)

Regardless of the therapeutic procedure, whether conservative or surgical, one must have precise knowledge of the healing behavior, the potential for correction depending upon the age, and the respective fracture region or fractured segment. Ideally, only those who have this knowledge should treat fractures in children.

Training in and knowledge of the relevant literature must be demanded increasingly in the paediatric area too. At the same time, the healing behavior of childhood fractures depending on different treatment strategies must be subject to prospective and multi-center investigation and documentation. For this, a comprehensive fracture classification system as well as a good documentation system is available and must be used.

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Fracture Assessment According to the Morphology and Bone Segment of the Fracture and the Age of the Child

Injury Patterns

Children are sustaining injuries at an increased rate compared to past years. Because of the new variety of sports and sporting equipment introduced over the past years even younger children are sustaining injuries and fractures that normally older children were incurring. The new equipment allows children to drive and run at higher speeds and hence, the fractures that occur are becoming more complex. Because of this we have had to reconsider earlier classifications of childhood injury to account for higher energy and more severe fractures.

Too often these new types of fractures are not treated correctly. Too often people think that children are only small adults and that children's fractures always heal properly. However specific injuries to the growth plate and the joints as well as special fractures like the Greenstick and Bowing fracture are too often assessed wrongly with respect to their healing behavior.

Factors Influencing the Treatment

In childhood, especially with the skeleton growing, the following parameters have to be taken into consideration:

- Age according to bone size and physeal growth
- Healing time according to age
- Weight
- Type of fracture
- Bone and segment
- Adequate, correct diagnosis
- Option of treatment: Non-operative or operative
- If surgical treatment: Kind of equipment
- Own experience
- Practicability of postoperative management
- Cost effectiveness

These factors are not to be regarded individually but in sum. Thus not only has the x-ray image to be studied, but also the individual condition and surroundings. If, for example, the infant already goes to school, a cast may be a handicap.

Age According to Bone Size and Physeal Growth

Age is one of the most important aspects. Although treatment generally does not differ in adults between 20 and 60 years, in childhood there are large differences to consider. For example, the treatment of a fracture in a 5 year old child does not have to correspond to that for, and may even be inappropriate in one for, a 12–14 year old child.

The main difference relates to the difference in the potential for adaptive growth at various ages. In early infancy, the potential for spontaneous correction of displacement is extremely high but reduces considerably at 12–14 years. Depending on the age of the patient, fractures can stimulate or inhibit epiphyseal growth. It is absolutely necessary to take into account the sex of the patient also (Fig. 3.1).

In addition, healing time varies with age. Consequently, the period of immobilisation differs. Below the age of 10 years, 3–4 weeks of wearing a cast is sufficient in almost all cases. However, above 10 years, 5–6 weeks of immobilisation is often necessary.

In addition to that the above, fractures-types are age-related. This phenomenon is because of the typical physical properties of the bone, periost, and cartilage at that age.

Development and Growth

The immature bone is not only more capable of reaction and adaptation, but also more vulnerable than the mature bone. A fracture in an immature bone can cause growth to speed up or slow down, superimposing the problems of deformity on the complications of the fracture itself.

On the other hand, children's fractures heal very rapidly and, depending on the age of the child and direction of the deformity one can remodel by correcting the most angular malunion. The most important area of injury in the immature skeleton is the growth plate or physis.

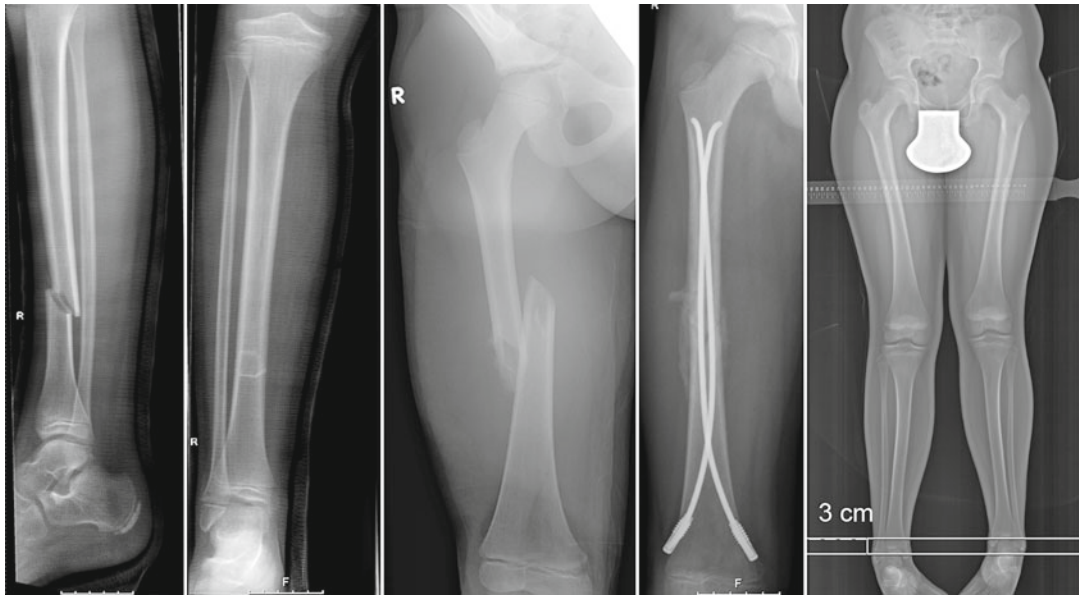

Fig. 3.1 A 9-year-old child sustained a lower leg and femur fracture on the right side; both fractures were aligned and the original leg length was achieved. Despite a short healing time and no remodeling it came to an over growth of 3 cm over the next 5 years

Regulation of Epiphyseal Growth

The physis is the primary center for growth in most bones and may be divided into two zones according to their function: the zone of growth and the zone of matrix formation. The zone of growth is involved with both longitudinal and circumferential growth of bone. The physis is capable of responding to different stimuli, either compression or tension.

Any kind of stimulation of the circulation leads inevitably to a stimulation of the growth zone, and therefore, of the growth. It was believed earlier that fractures would heal with shortening so that the bone has the same length at the end of the treatment. This shortening was inaccurate as it was not possible to calculate it correctly. On the other hand an anatomical reduction and osteosynthesis in most cases leads to a lengthening (Fig. 3.1). We know today that the stimulation of growth is a *result of the healing time and remodeling time*. We achieve the best results if we have a biological treatment and fixation, which anatomically adapts the bone fragments, has a short healing time, and no remodeling needs. This biological principle is put into effect with the ESIN technique as the best one.

Growth and Remodeling of the Metaphyseal and Diaphyseal Bone

The metaphysis is the site of the most rapid changes in bone structures as the deeper physeal zones mature and the physis produces primary trabecular bone.

The circumferential growth of the diaphysis is a function of appositional bone formation by the periostium, together with osteoclastic resorption by the endosteum so as to enlarge the medullary cavity. As growth continues, the bone is capable of reducing, or even correcting, angular deformity by selective resorption and apposition, possibly driven by forces of compression and tension (Table 3.1).

Healing Time based on Patient Age

In addition, healing time varies with age. Consequently, the period of immobilisation differs. Below an age of 10 years, 3–4 weeks of wearing a cast is sufficient in nearly all cases. However, above 10 years, 5–6 weeks of immobilisation are often indispensable. But we must also consider the weight of the child.

Table 3.1 Place of correction, factors and possibilities of the growing skeleton

Displacement/deformity			
Side to side	Axial (frontal–/sagittal plane)	Shortening/lengthening	Rotational failures
Periosteal–endostal	epiphyseal		
Direct correction			Indirect correction

Further, fracture-types are age-related. This phenomenon is because of the typical physical properties of the bone, periost, and cartilage in children.

The factors mentioned above determine the degree of tolerable dislocation and the choice of treatment. With rising age the option of conservative treatment declines and that of osteosynthesis increases.

In childhood especially, K-wires with an additional cast, cannulated screws, intramedullary elastic nails, and adapted external fixators are suitable implants. Plate osteosynthesis is exclusively used in adolescents, as also in adults. Solid intramedullary nails should be avoided in children (Table 3.2).

Patient's Body Weight

In childhood, absolute stability of the fracture is not necessary, neither following surgical treatment nor after conservative treatment. The reasons for this have been discussed in the previous section. In most cases, relative stability is sufficient for immobilisation and progressive weight bearing. However with increasing age, biomechanical limits are noticed. The key influence is not age itself, but the tendency toward increased body weight and size that can be observed. Frequently, 12–14 year old children are as tall and have the same body weight as adults, which is often associated with overweight, today. Little attention has been paid to this observable fact in the current literature on pediatric traumatology. Nevertheless, therapy planning has to integrate these aspects as the clinical impact is of major concern. Therefore, in older children who are also heavier, therapy should be provided as provided to adults after fracture- treatment, particularly in shaft-fractures (Fig. 3.2).

Fracture Pattern

Fracture morphology has a decisive influence on therapy, independent of the age or physical development of the child. The stability of the fracture is of primary interest. Then the completeness of the fracture is evaluated.

The recognition of a so-called "bowing-fracture" is of particular importance. This type of injury is subject to laws of its own. Bowing fractures show progressive angulation and possess only little potential for remodelling. In particular, at the forearm, where this fracture is frequent in young children, there is a good chance of ending up with bad functional results after conservative therapy (Fig. 3.3). The current treatment of choice is ESIN (elastic stable intramedullary nailing). The inserted nail equalizes the angulation of the bone with its own opposite curvature, which was applied to the nail before insertion. Further angulation is thereby prevented.

Nonetheless, for the most part, *stable fractures* are treated conservatively with a cast independent of the patients age. Healing of the bone occurs quickly and without problems. Immobilization between 3 and 5 weeks is nearly always sufficient. An exception is the fracture of the femoral shaft which is treated with ESIN or external fixator in most centres.

In the case of *unstable fractures* one should always be prepared for an operation. Even if the initial reposition of an unstable, dislocated fracture is successful, secondary dislocation may require a change from conservative to surgical therapy (Table 3.3).

Repetitive reductions or manipulations in the treatment are to be avoided. Considering the type of the fracture, the following rules should be taken into consideration:

Table 3.2 Guidelines for fracture immobilization in childhood (weeks)

	<5 years	5–10 years	>10 years
Clavicle	1	2	2–3
Humerus			
Proximal stable	1	2–4	3–4
Proximal unstable	1	2–4	3–4
Humerus shaft	2–3	3–4	4–6
Supracondylar	2–3	3–4	4–5
Radial condyle	3	3–4	4
Ulnar condyle/Y fracture	2–3	3–4	3–4
Ulnar epicondyle (+ dislocation of the elbow)	2–3	2–3	4
Radius proximal	1–2	2–3	3–4
Olecranon	2	2–3	3–4
Forearm shaft incl. Greenstick fracture	3	4	4–6
Radius (+ radius + ulna) distal	2–3	3–4	4–5
Salter Harris I radius distal	2	2–3	3–4
Carpus		4–6	5–8
Metacarpal proximal and distal		2–3	3–4
Metacarpal shaft		3–4	4–6
Fingers proximal and distal	1–2	2–3	3–4
Fingers shaft	2–3	3–4	4–6
Femur			
Neck of the femur		4–6	6–8
Subtrochanteric fractures	2–4	4–5	5–6
Shaft	2–3	4–5	4–6
Distal	2–3	3–4	4–5
Tibia and lower leg			
Tibial spine fracture		3–4	4–6
Proximal metaphysis	2–3	3–4	4–5
Shaft	2–3	3–5	4–6
Distal and malleolar	2–3	3–4	4–5
Hindfoot and calcaneus		4–6	5–10
Midfoot and toes distal	2–3	3–4	4
Toes	1–2	2–3	3–4
Fibulo—talar ligaments/osseous avulsion		3–4	4–6

Metaphyseal fractures heals in a half time of diaphyseal fractures
Diaphyseal transverse fractures heals more slowly than diaphyseal oblique fractures

- If general anaesthesia for reduction of a fracture in childhood is indicated, we aim at a definite, stable, and safe fixation; i.e. potentially unstable fractures will be reduced in the operation theatre with the option of an eventual operative treatment.
- Independent of the age of the child, totally unstable fractures are operated primarily.
- If general anaesthesia is required, the first treatment has to be the definite treatment; no second anaesthesia.

Because the fracture type has a direct influence on the therapy, it is important to classify the fractures correctly (Table 3.4). One of the most recognized fracture classifications is the AO/OTA today (Pediatric Comprehensive Fracture Classification of Long Bones; PCCF). See chapter appendix (below) and Table 3.5.

On the other hand we have to take into account different factors of the fractures as well as the localization/displacement and stability for a correct therapeutic decision.

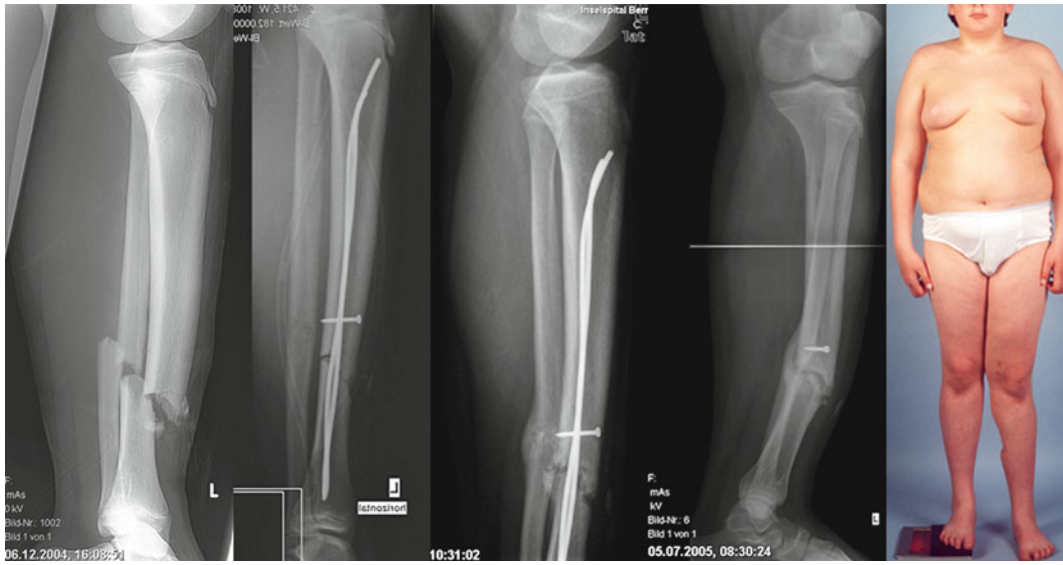

Fig. 3.2 A 13-year-old boy, overweight with unstable fracture of the lower leg. Osteosynthesis with ESIN, insufficient stability because of fracture type and the weight despite additional stability by an interlocking screw; so called "miss–a–nail" technique

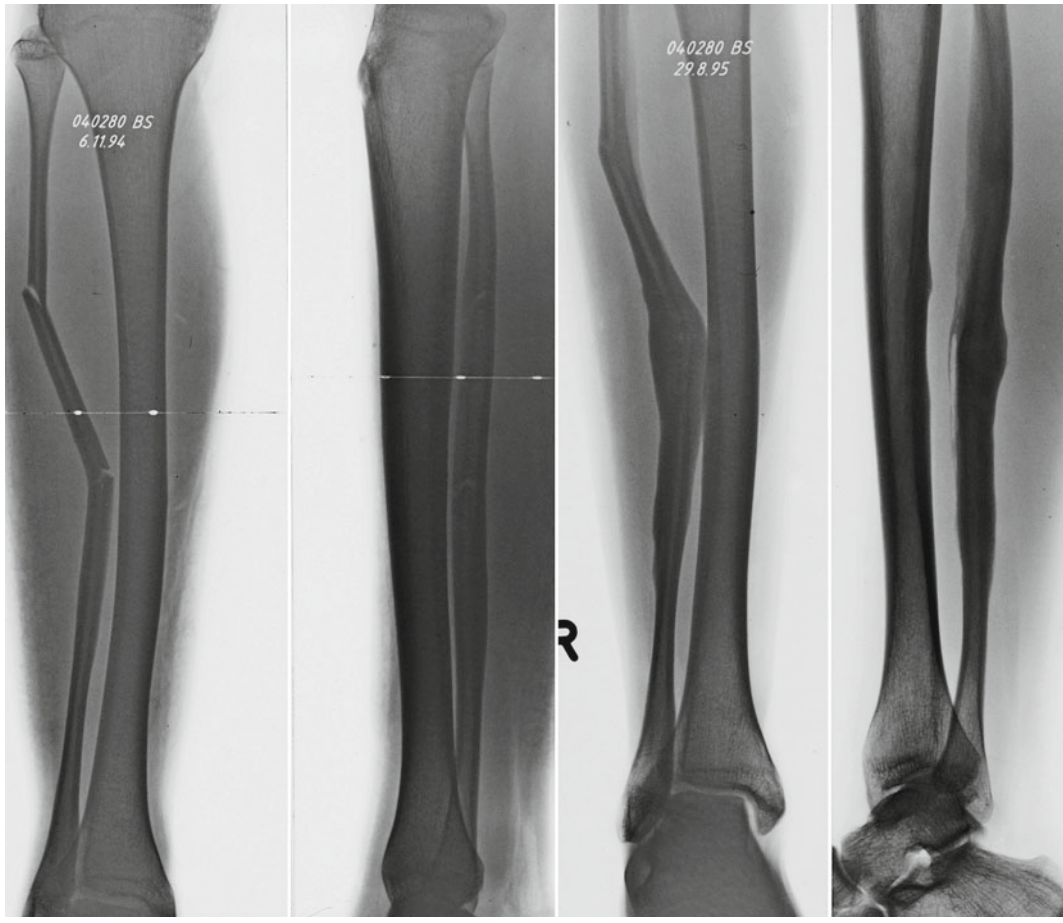

Fig. 3.3 Direct lower leg trauma in a 15-year-old boy with "bowing" fracture of the tibia. The x-rays show a malunion 8 months after this "bowing fracture" of the tibia (still remaining bowing)

Table 3.3 Overview of the influence of these three factors (localization/displacement/stability) regarding therapeutic options

Stability	Bone/segment	Therapy
Sufficiently stable for safe reduction and initial retention	Diaphyseal/metaphyseal	Immobilization with plaster of Paris, e.g. in combination with cast wedging
	Transverse fracture with tolerance limits depending the age	Fiber-glass cast
	Or	Standardized produced splints
	Oblique or spiral fractures only of one bone of the lower leg or forearm	
	Articular	
	Non or minimal (<2 mm) displaced articular fractures	
Unstable fractures (not reducible and not to stabilize with non-operative treatment)	Diaphyseal/metaphyseal	Reduction under anaesthesia is indicated
	All fully displaced fractures	Conservative (plaster cast) or operative stabilization is necessary
	Articular	
	Articular fractures with a gap over 2 mm	

Table 3.4 Overview of the influence of localization and the involvement of the physis regarding therapeutic options

Fracture localization		Stability	Morphology
Shaft fracture	Diaphyseal fracture	Stable fracture	Not displaced fractures without shortening
		Unstable fracture	Displaced fractures with shortening or the tendency for shortening or angulation
		Greenstick fracture	Bowing fractures with complete fracture of one cortex and incomplete facture of the opposite cortex
	Metaphyseal fracture	Buckle fracture	Compression of the metaphyseal cortex of one side
		Bowing fracture	Greenstick fracture in the metaphysic
		Supracondylar fracture	Ligament avulsion
		Ligament avulsion	
Articular fracture	Epiphyseal fracture	Aitken I	See below
		Salter I, II	See below in the puberty, partial closed physis bony or cartilage avulsion "normally" in combination with joint dislocation
		Aitken II + III	
		Salter III + IV	
		Fractures of Tillaux or two planes fractures	
		Lig. Avulsion	
		Flake fracture	

Bone and Segment

In addition, localization influences the choice of the treatment. The management is different if the lower or the upper extremity is involved. In childhood, even more important is the localisation of the fractured segment: diaphysis/metaphysis or epiphysis.

Although in diaphyseal fractures, stability has a decisive influence on therapy, in epiphyseal and some metaphyseal fractures, the extent of displacement of the fragments is crucial.

Epiphyseal fractures are special in two ways. On the one hand it's an injury of the growth plate with a possible impairment of the growth; on the other hand these are always injuries of the joint with all the difficulties of this type of injury. In principle, articular incongruency has to be redressed, and interfragmentary gaps up to 2 mm can be accepted. This proceeding has not been established by scientific evidence, but justified following observations and evidence.

In these fractures, the suitable implants are cannulated screws and K-wires of all sizes. In some articular fractures, as for example the two-plane fracture of the tibia, screw fixation allows a closed procedure.

The potential for remodelling of a facture is strongly influenced by the segment involved. Knowledge of the prevailing potential for remodelling is an indispensable requirement for treating pediatric fractures (Fig. 3.4).

Metaphyseal fractures of the proximal humerus and the distal forearm have to be emphasized. As the corresponding epiphyseal plates are responsible for a major part of the length growth of these bones, remodeling has the highest capacity at this place. For this reason, fractures involving the proximal humerus or distal forearm are treated conservatively for the most part, even in older children.

Independent of the localisation of the fracture, side-to-side (so called bayonet displacement) is corrected better than axial deviation.

Adequate, correct diagnostic: Skeleton standard x-rays and special radiological examinations (Ultrasound/CT/MRI)

Even in an emergency the correct AP and lateral (at least +/– perpendicular each-other) x-rays should always be taken.

General notes to the fracture types:

- Not every fracture is visible on the x-ray in childhood
- An x-ray is always indicated if the patient history/local signs or injury is unclear.
- If the decision for an x-ray has been made, then it should also provide the desired information; in other words we need good quality.
- It is absolutely necessary to take x-rays in two planes (perpendicular to each other). Moreover the proximal and distal joint of the broken segment should be visible on the x-ray.
- The x-ray of the opposite extremity is unnecessary as no new knowledge will be obtained.
- Remember, you can see only what you know.

Table 3.5 The Salter-Harris classification in comparison to the Aiken classification

Salter		Aitken
I	(a) Epiphysiolysis	I
II	(b) Epiphysiolysis with metaphyseal wedge	
III	(c) Epiphyseal fracture	II
IV	(d) Epiphyseal fracture with metaphyseal wedge	III
V	(e) Epiphyseal compression (only seen “post festum”)	

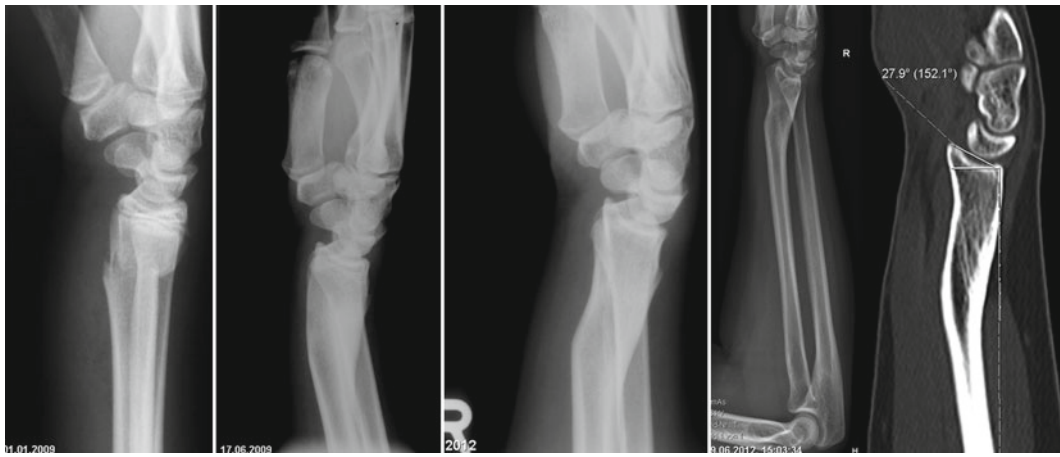

Fig. 3.4 Sometimes the “expected/normal” healing does not following the natural laws. Simple metaphyseal, nearly not angulated radial fracture fixed in a plaster cast. Six months later well healed but increasing angulation and deformation is growing proximal. Because of wrist pain the child came back 3 years later. A severe dorsal tilt of the wrist (35°) and “bayonet” malunion is clearly seen on the normal x-ray and CT scan

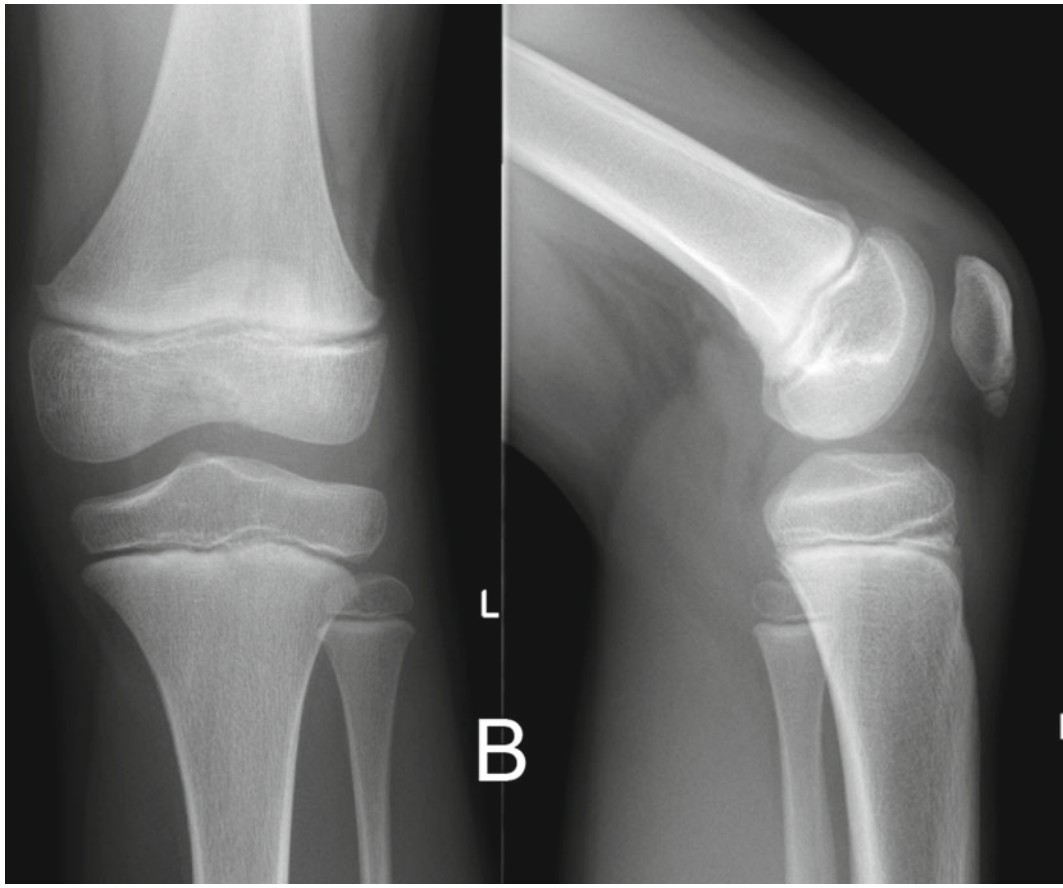

Fig. 3.5 X-ray shows typical radiological situation of knee joint in a 6-year-old girl

The evaluation and correct assessment of an x- ray is the prerequisite for correct diagnosis. The diagnosis, therefore, represents the base of the therapy decision. The phenotype of the childlike skeleton changes from year to year, sometimes from month to month. Therefore, the exact age-related development of the child's skeleton is important. Growth plates are very often missing—interpreted as a fracture. This often leads to non-indicated treatment. The most frequent and typical example of this is the misinterpretation of the three-dimensional form of the proximal physis of the humerus. The following table gives a summary overview of the age dependent skeleton development of the most frequent miss-interpreted skeletal regions.

On x-ray, children's joints seem much larger than they really are (Fig. 3.5). The reason for this is the thick cartilage around the epiphysis.

Treatment Options: Non-operative Versus Operative

To decide between conservative and surgical therapy, the four previously discussed criteria are to be taken into consideration. Experience shows that quite often all aspects of a fracture are not considered in the decision for the therapy.

The choice of operative or conservative therapy has to be based on the main goal of the treatment. Despite the fact that the treatment should be definite, an initial cast-immobilisation of a potentially stable fracture is legitimate if general anaesthesia is avoided. Radiological follow-up

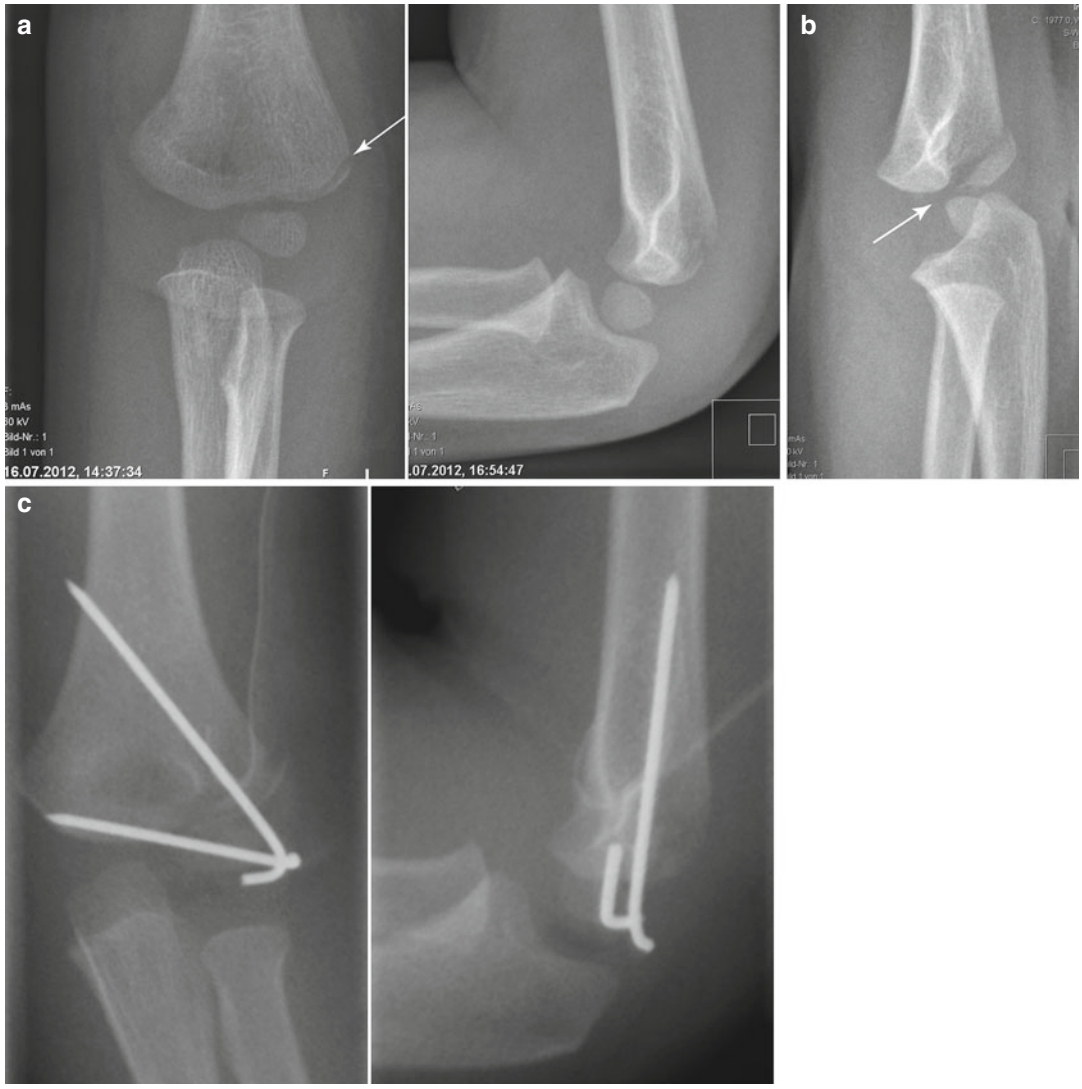

Fig. 3.6 Lateral condyle fracture of the humerus with displacement below 2 mm treated by cast immobilization (**a**). According to our recommendation after 5 days a cast free x-ray was made and shows a secondary displacement more than 2 mm (unstable situation). This is the indication for operative fixation (**b**). Correct K-wire fixation one parallel to the joint, one in the lateral column (**c**)

after 5–7 days is imperative. If progressive displacement is observed, definitive treatment is still possible, in most cases, by surgery. Outcome is not influenced negatively by a brief delay to definitive surgery.

For example, in lateral condyle fractures of the humerus, the arm is primarily immobilized if the interfragmentary gap is equal or below 2 mm. After 5–6 days, a radiological control without cast is performed. If the position of the fragments remains unchanged, cast immobilisation is continued. In the case of displacement above the tolerable limit, the radial condyle is fixed operatively (Fig. 3.6).

Modality of Surgical Treatment

In addition to the theoretical knowledge required for the treatment of pediatric fractures, certain technical prerequisites are necessary, especially with regard to surgical therapy.

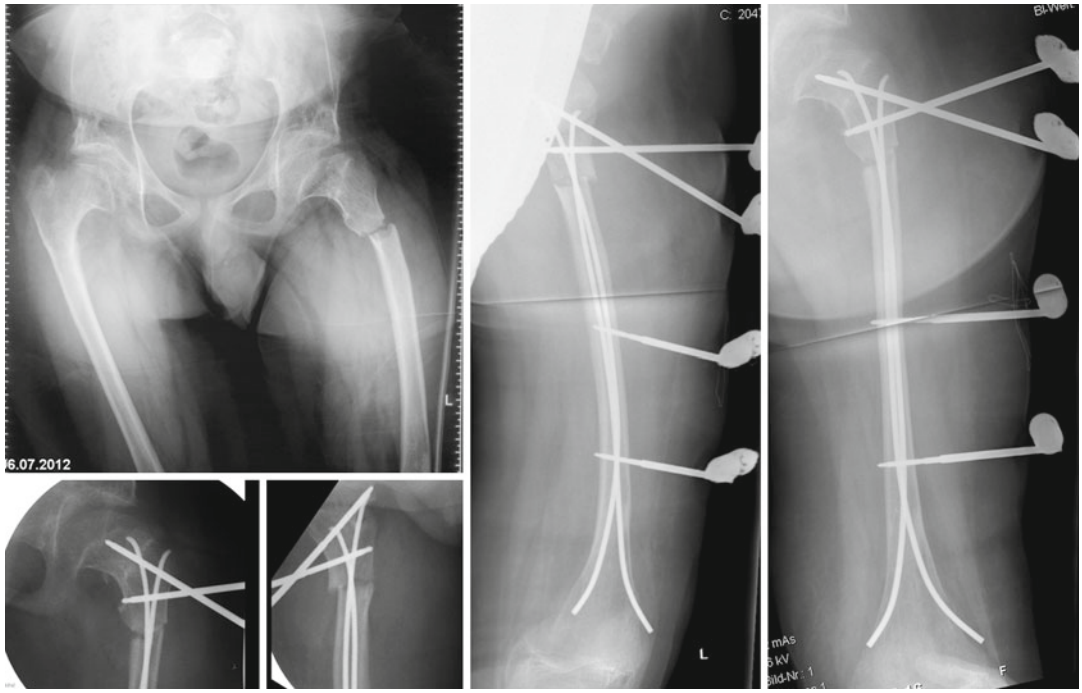

Fig. 3.7 A 14-year-old boy with cerebral palsy, previous hip and proximal femur osteotomies, osteoporotic bone sustained in a subtrochanteric transverse fracture: for alignment a closed reduction and fixation with two thin elastic nails was performed; to prevent any axial deviation and to manage the child immediately in the wheel-chair a medium external fixator was applied; removal of Ex Fix is planned when callus formation is visible

In childhood, implants should be adapted to body size. As stability does not have to be as high as in adults and as additional cast-immobilisation is well tolerated, very small implants can be used, especially in epiphyseal and metaphyseal fractures. K-wires and cannulated screws are particularly suitable.

For surgical therapy of shaft fractures, the various implants should be disposable, and alternatively, the surgeon should be experienced in several techniques.

An adequate surgical setting enables changes in therapy during the operation. The following essential guidelines are commonly accepted for the surgical management of shaft fractures.

- The therapy of choice for transverse, oblique, and short spiral fractures between the age of 4(/5)—14(/15) years is ESIN.
- Extremely unstable fractures of the lower limbs are preferably treated with external fixators. According to the experience of the surgeon, ESIN may be suitable or a combination of both ESIN and external fixators (Fig. 3.7).
- Today, osteosynthesis with plates in childhood get more and more rarity value and we see this type of fixation as a “special indication”. The main indication to use plates is older teenager and overweight children.

Personal Experience

A factor not to be underestimated is experience in the treatment of fractures in children. A retrospective analysis at our clinic of all femoral fractures treated with ESIN has shown that 75 % of all problems and complications have been caused by surgeons who had treated less than four fractures with this technique. This also points to the fact that the importance of the learning curve should not be underestimated. This also applies to smaller clinics, especially those that seldom

treat children or see only few, individual special fractures per annum.

Unfortunately, this statement does not only apply to operative therapy but also applies to handling the plaster cast and to conservative therapy. Being able to make an adequate plaster cast is still a part of the high art of conservative therapy.

The analysis of the malunions and complications to be treated in the cases assigned to this clinic within the last 5 years, shows that 90 % of these cases came from hospitals which that only sporadically treat children.

Consequently, treatment of pediatric fractures should only be carried out by experienced pediatric surgeons who:

- Are competent
- Treat pediatric fractures regularly
- Dispose of adequate equipment
- Are trained in the use of the implants

Practicability of Postoperative Management

It is in the nature of children to be always moving. It represents a basic need in childhood. Infants hardly support being tied up. Respecting this condition, a worldwide change in therapy planning has taken place.

Children do not like bed rest for 2 to 3 days or weeks, or restriction using the upper extremity as for writing in school or everyday activities. Therefore such treatments (as plaster cast) should be things of the past for children. Absence from school is a major strain for children today. Apart from this, taking care of ill or disabled children is a difficult charge for young and working parents and the society (Fig. 3.8).

Altogether, if possible, therapy planning should take these factors into consideration. During the initial setting up of the treatment with the parents, the postoperative and post-hospital care, follow up consultations, and the effort necessary for each therapy should be mentioned.

Cost Effectiveness

Financial factors increasingly influence medical treatment, including pediatric traumatology. A trend towards surgical treatment approaches reflects this tendency in pediatric bone trauma. Today, an operative treatment with ESIN for a femur fracture for a 12-year-old child (including out-patient nail removal) at this clinic is around 1/3 of the cost of a conservative therapy by means of extension and spica cast.

The Choice of the Method of Treatment According to the Type and Location of the Fracture and the Age of the Child

Conservative Immobilization Techniques

Plaster Immobilization

As mentioned earlier, plaster treatment is at the base of the fracture treatment in children. It is therefore indispensable that for a doctor who treats children has the skills of modern plaster technology.

Plaster Splint

Indications

- Fractures
- Distortions
- Pain after bone bruises

Technique

- The extremity should be covered with a cotton tube.
- It should be wrapped with thin half elastic cushion cotton.
- It should be wrapped with a paper bandage (not necessary if fibrecast is used.)
- Plaster gauzes should be placed longitudinally along both sides of the extremity with a 3-cm plaster-free gap between them
- After oedema regression, the cast can be closed circularly.

Circular Plaster Cast

Indications

- Functional treatment
- Secondary post-plaster splint (after oedema regression)

Fig. 3.8 Two girls, a 10-year old and a 11-year-old, with femoral fractures. *Right*: treatment with a modern scotch-cast/spica-cast; *Left*: treatment was performed with ESIN and mobilisation on crutches on the second postoperative day

Technique
- Same steps as above
- The extremity is enclosed in a circular plaster.
- Drying time for plaster ~1.5 h; for Scotch cast ~30 min.
- A primarily applied circular plaster should be opened longitudinally, especially when the tissue is swollen after fresh injuries.
- When swelling subsides, the plaster can be closed again.

Plaster Cast Windows

Indications
- Open wounds
- Pins
- K-wires

Technique
- The earliest that windows can be made in the plaster is after drying.
- The window in the plaster must always be covered with the piece that was taken out in order to prevent oedema in this region.
- The window in the plaster must not impair the stability.

Plaster Cast Wedging

Indication
- Remaining angulation of an undisplaced fracture, after fracture stabilization without reduction
- Secondary angulation following plaster cast stabilization of an undisplaced fracture

Technique
- The earliest the wedging of the plaster can be made is after one week after swelling and pain have completely subsided.
- The cut for the wedge must be at the deepest point of the concavity of the deformity.
- The more peripheral the fracture, the more proximal should be the point for the wedge.
- The child should not have any pain during this procedure.

Principles of Therapy (Non-operative and Operative)

One takes into account all considerations before choosing from the following options for the therapy so:
- Fixation without any manipulation
- Skin (skeletal should be avoided) traction
- Closed reduction and non-invasive fixation
- K-wire fixation
- Lag-screw fixation
- Reduction and fixation by "external fixator"
- Intramedullary elastic stable nail fixation (ESIN)
- Intramedullary rigid fixation
- Plate osteosynthesis

Fixation Without Manipulation or Fracture Reduction

Indications
- Fractures without any dislocation, angulation (in principle, stable) or distortions
- Stable fractures, which are within the age-related range, acceptably angulated

Traction

Indications
- Nowadays, less acceptable for children over 5–6 years (acceptable up to 3 years depending on the child's weight)
- Used as an overhead skin traction (skeletal traction should be avoided)
- More used for lower extremity (femur fracture) (Fig. 3.9)
- Advantage: can be applied without anaesthesia (only sedation)
- Fixation of the extension on the overhead arch so that the child's buttocks are raised (there should be space for a flat hand to move freely under the buttockswithout touching them)
- Possible for out-patient treatment

Closed Reduction and Non-invasive Fixation

Indications
- Carry out every fracture reduction under anaesthesia (plexus block, general anaesthesia).
- Extend the fingers, hang a weight at the humerus and maintain the upper extremity with 90° flexion at the elbow for 20–30 min
- Reduce the fracture using a reduction manoeuvre that mimics the movement that led to the fracture in the first place
- Apply the plaster in the hanging position
- Immobilize the fracture with a dorso-volar plaster splint
- Prepare and use help to fix the lower leg
- For plaster cast application ensure the following crucial point : one person is holds the leg, another prepares the plaster cast or fibre cast, and a third makes the cast.

Osteosynthesis with K-Wires

Indications
- Closed or open reduced metaphyseal fractures
- Fractures of the hand and foot

Contraindication
- Diaphyseal fractures.

Technique
- Whenever possible, the K-wires should be placed percutaneously so that they can be removed without anaesthesia.
- The crossing points of the K-wires should be proximal to the fracture line.
- If the epiphysis must be crossed, repeated attempts to fix it should be avoided.
- In cases such as this, thin K-wires should be used instead.
- K-wires should penetrate the opposite cortex.
- Daily care of the pins reduces the risk of infection.
- Normally K-wires can be removed after 3 – 4 weeks.

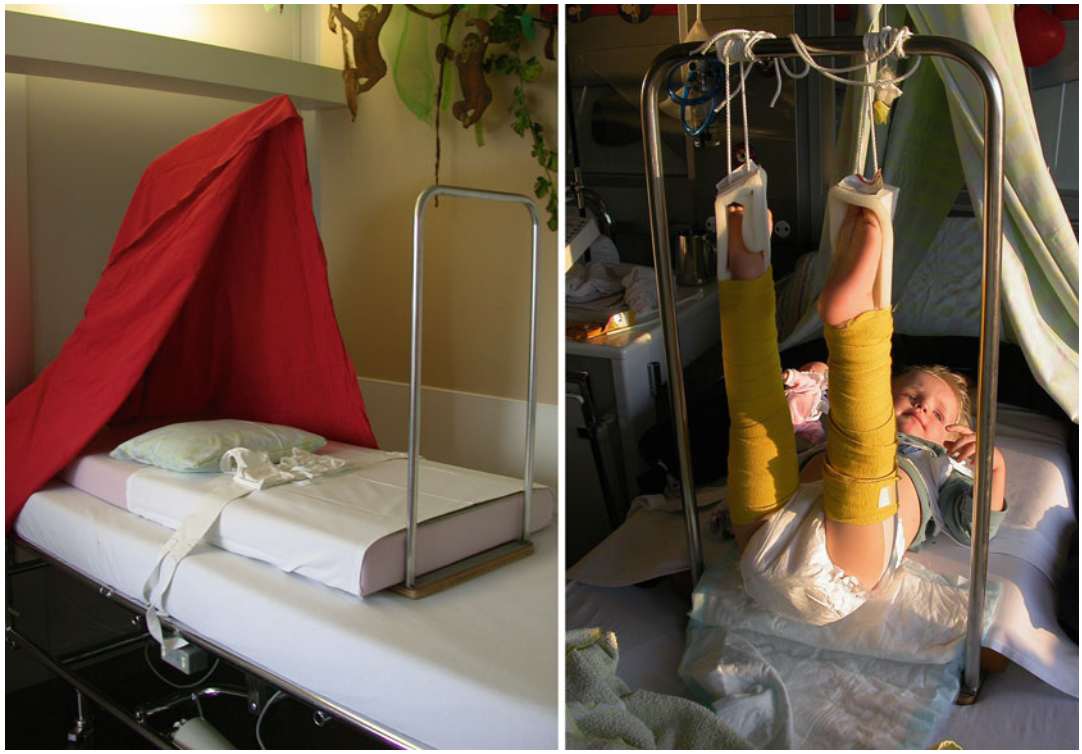

Fig. 3.9 Over-head traction for femur fractures in children between 2 and 4 years. *Left*: preparation of the equipment and the bed; *Right*: the child is fixed on the frame and applies the traction using the boy's weight. Within 2 days the parents learn the technique and the child can be treated at home for 3 weeks.

Lag Screw Fixation

Indications

- Articular and peri-articular fractures, Salter–Harris II fractures, mainly in the distal tibia and femur, femoral neck fractures

Technique

- Use mainly self-drilling and taping cannulated screws (dimensions 3.0/3.5/4.0/4.5/6.5 mm); if these types of screws are not approved in a region, use a normal cannulated cortex screw.
- Position the extremity on the intensifier to facilitate the correct view.
- Rotate the extremity so that the fracture line is visible in a proper a.p. view.
- Put the guide wire on the fragment parallel to the table of the intensifier.
- Drill the guide wire into the bone to the contralateral cortex.
- Measure the length.
- Put the correct-sized cannulated screw over the guide wire.
- Tighten the screws until the fracture is closed.

Closed Reduction and External Fixation

Note: Due to good healing and associated short healing time, the child is the ideal patient to be treated with an external fixator. To make post-operative management easier and to prevent complications, it is recommended that good patient information is always available.

Indications

- Comminuted fractures in older children (femur, tibia, forearm)
- Polytrauma
- Long spiral fractures, e.g. spiral wedge of the femur in older children

Technique
- The technique used is normally closed reduction with or without extension table
- The types to be used are the Monotube system, tubular system, circular frame, TSF
- The Schanz screws should be placed under imaging intensifier (take care of the bone size.)
- In Monotube systems, the distances between the entry points are predefined.
- In "frame systems", one entry point should be near the fracture, the other further away from it.
- All clamps must be open for reduction.
- After sufficient reduction, all clamps have to be closed and secured.
- Daily care of the pins reduces infection risk.

Intramedullary Elastic Stable Nail Fixation (ESIN)

Indications
- Transverse, oblique, short and long spiral, diaphyseal fractures of the long bones in children from (3) 4–14 (15) depending on the child's weight
- Metaphyseal fracture up to 2–3 cm from the growth plate distal and proximal femur, subcapital humerus fracture, supracondylar fractures of the humerus
- Radial neck fractures
- Nowadays a more complex fracture can be treated with elastic nails when so called Endcaps are used.
- There is a special indication for finger and clavicle fractures.
- ESIN is a minimally invasive, minimally traumatic, and sufficiently stable for movement and partial weight bearing, biologically and child friendly osteosynthesis using special elastic nails.

Technique
- Operation technique: see section on femoral fractures.

Intramedullary Rigid Fixation with Adolescence Lateral Femoral Nail (ALFN)

Indications
- Femur shaft fractures in children over 13–14 years when ESIN is not stable enough, especially with over-weight

> Note: Obese children, even if they have a body-weight of 100 kg do not have weight-related bone/medullary canal diameters.

Technique
- Supine or lateral position
- Preparation of the lateral aspect of the greater trochanter
- Opening of the proximal femur according to the OP-technique
- Indirect or direct (open) reduction of the fracture
- Insertion of the nail over the nail guide wire
- Rotation check on the not fractured side before operation; in critical cases, draping both legs to compare rotation during operation
- Proximal and distal locking of the nail

Plate Osteosynthesis

Indications
- Comminuted fractures of the diaphysis (femur, tibia, humerus, radius and ulna) mainly in older children
- Complex metaphyseal fractures

Technique
- Usage of plates is reserved to exceptions and special cases.
- When an osteosynthesis with a plate is indicated, the application of new types of plates such as LC-DCP and LCP plates is recommended. If possible, these types of plates can be applied using a minimally invasive technique (MIPO).

Joint Aspiration

Knee Joint Arthrocentesis

Indications
- Posttraumatic haemarthros (best time after 24 h)
- Signs or suspicion of infection

Technique/Procedure
- Apply local anaesthesia or anaesthetic cream well before the puncture.
- Puncture in the lateral/proximal recess.

Hip Joint Arthrocentesis

Indication

- Posttraumatic haemarthros as soon as possible to decompress the joint (lower limit 4–6 mm distance in the anterior recess in hip ultrasound.)
- Signs or suspicion of infection

Technique/Procedure

- From lateral approach, or
- From anterior approach (be careful of artery and nerve) or
- Ludloff approach (author's preferred approach)

Special Injuries

Spinal Injuries

General Considerations

- Spinal injuries in childhood are not as rare as one assumes.
- The most common injuries are simple fractures after anterior crush injuries or compression fractures of the vertebral body.
- In injuries involving the atlas and axis, rotatory subluxation or dislocation is one of the most common lesions in children, rather than fractures of the atlanto-axial articulation.
- Injuries of the thoracic and lumbar spine are more common in childhood than in adulthood. The majority of these fractures result from traffic accidents. Child abuse should also be considered.
- Obstetric fractures involve mostly the cervical spine (with high mortality.)
- End-plate fractures in younger children are followed by scoliosis.
- Fractures of vertebral bodies have good remodelling capacity, depending on the child's age.
- Diagnosis can be difficult owing to congenital malformations (congenital non-union, hemi vertebra, congenital vertebral fusions) or the following diseases (post-traumatic malformations or Scheuermann's disease).
- The normal spine in children differs considerably from that in adults, especially in the cervical region.

Classification

- Stable spine fractures with compression of the vertebral body or "end-plate" injuries
- Unstable fractures with involvement of the vertebral arch and the pedicles
- Ligament avulsion
- Fractures involving end-plates with growth disturbance
- Fractures not involving end-plates

Diagnostic Studies

- Diagnosis includes accurate evaluation of the level and extent of injuries to both chondro-osseous and nervous system tissues.
- Radiographic evaluation must be carried out paying due attention to potentially severe unstable injuries and must include prior adequate immobilization of the spine.
- Osseous injuries can be seen on an adequate a.p. and lateral view.
- Oblique views may be necessary.
- CT scan or MRI may be indicated.

Management

- A number of unique problems can be encountered in the treatment of infants, children, and adolescents with spine injuries.
- In any closed, nonoperative treatment regime, the spinal deformity must be reduced and adequately stabilized and protected from redisplacement during the healing process.

Treatment Guidelines

For stable/non-displaced fractures, simple bed rest is indicated, because most children with stable (compression) fractures are asymptomatic within a few days or weeks. External support may be necessary. Stable/displaced and unstable fractures require surgical stabilization with dorsal fusion.

Prognosis

The prognosis for undisplaced stable fractures is good, depending on the type of injury and the number of involved levels (growth plate/end-plates, wedge compression).

The prognosis for displaced fractures depends on the accompanying neurological problems (Fig. 3.10).

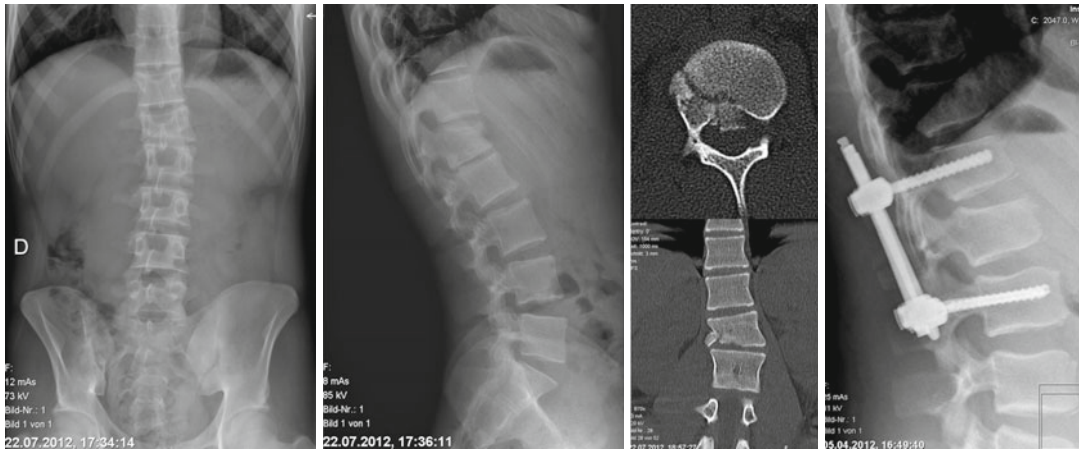

Fig. 3.10 Typical compression fracture in an older child (14 years). CT scan shows the dorsal wall fragment in the spinal canal; fortunately in childhood in 95 % cases there are no neurological problems. Stabilization is mandatory, in this case with an internal fixator system

Clavicle and Pectoral Girdle

Considerations

- Fractures of the clavicle are very frequent and occur most frequently in children during the first 12 years.
- Fracture in the first 2 years may be a "sign" of non-accidental injury.
- Fractures of the scapula in childhood are extremely rare and are mostly a result of a strong direct trauma because the scapula is well protected by muscles and is very mobile.
- Table 3.6 gives an overview of the sign/treatment and outcome of clavicle fractures.

Table 3.6 Overview of the sign/treatment and outcome of clavicle fractures

Morphology	Fractures of the mid-shaft are most common and range from greenstick to complete fractures
Signs	Pain, swelling, painful movement of the arm
Diagnosis	Clinically and radiologically
Correction potential	Good
Complications	Nerve problems, non-union, cosmetics
Nonoperative therapy	In principle non-surgical – sternal brace or "figure-of-eight" bandage pulling the shoulder backwards (pain management)
Operative treatment	Only fully displaced, comminuted fractures in adults (author's preferred method is ESIN)
Immobilization	2–3 weeks
X-ray control	After 3 weeks
Follow-up	Only clinically

Glenohumeral Joint Dislocation

- Subluxation and dislocation of the shoulder (Table 3.7) are rare in infants or young children.
- The capsule of the shoulder joint has some intrinsic laxity that allows some displacement during stress.
- "Dislocation" of the shoulder has also been described as a birth injury. However, great care should be taken before making such a diagnosis as the proximal humerus is most likely to be fractured through to the epiphysis.

Prognosis

- For clavicle fractures, the prognosis is very good
- Prognosis for the dislocation of the glenohumeral joint depends on the time lapsed since the incident and the type of injury.

Table 3.7 Overview of Gleno-Humeral joint dislocation

Morphology	Fractures of the mid-shaft are most common and range from greenstick to complete fractures
Signs	Pain, swelling, painful movement of the arm
Diagnosis	Clinically and radiologically
Correction potential	Good
Complications	Nerve problems, non-union, cosmetics
Nonoperative therapy	In principle non-surgical – sternal brace or "figure-of-eight" bandage pulling the shoulder backwards
Operative treatment	Only fully displaced, comminuted fractures in adults (author's preferred method is ESIN)
Immobilization	2–3 weeks
X-ray control	After 3 weeks
Follow-up	Only clinically

Humeral Fractures

General considerations:

- 80 % of humeral fractures occur on the distal segment.
- Proximal and diaphyseal fractures are rare.
- It is mostly direct trauma or so called "wring-injuries" or in Baby's obstetric injury.
- Don't forget the possibility of a non-accidental injury.
- Most of them are transverse or oblique fractures; an exception is wring-injury (spiral wedge fracture).

Fractures of the Proximal Humerus

Proximal humeral fractures and their management are detailed in Table 3.8.

Operative Treatment

- Author's preferred method
- ESIN from a monolateral, radial approach in an ascending technique
- Alternative: percutaneous (2.5-mm threaded) K-wire fixation
- Open reduction, a rare exception (Fig. 3.11)

Humerus Shaft (Diaphyseal) Fractures

See Table 3.9 for an overview of diaphyseal fractures.

Table 3.8 Morphology/diagnosis and treatment of proximal humerus fractures

Morphology	About 60 % are subcapital fractures; 38 % are Salter–Harris II fractures; pure epiphysiolysis are rare
Signs	Deformation, pain
Diagnosis	X-ray; interpretation is often difficult in undisplaced fractures, displacement of the epiphyseal line is interpreted as a fracture Note the three ossification centers
Correction potential	Great potential, angulation in the sagittal and frontal plane is tolerated up to 60° in children <12 years old and up to 30° >12 years
Complications	Practically unknown; in neonates premature close of the growth plate is possible
Nonoperative therapy	Stable, undisplaced fracture, any age Stable fracture with angulation <60°: <10 years <30°: >10 years Or Stable fracture with tolerable displacement, any age Immobilization for 3–4 weeks in a Desault or Gilchrist dressing Or Reduction, definitive treatment with stable fixation is recommended
Operative treatment	Children >10–12 years of age Unstable fracture if a reduction under anesthesia is necessary Major displacement after nonoperative treatment Author's preferred method: ESIN No additional immobilization is needed Nail removal after 3–4 months
Immobilization	3 weeks (see above)
X-ray control	Nonoperative therapy: days 3–4 and weeks 3–4 Operative treatment: postoperatively and week 4
Follow-up	Week 3 or 4 radiological and clinical

- It is mostly direct trauma or so called "wring-injuries" or in baby's obstetric injury
- Non accidental injury is also possible.
- One must be aware of radial nerve lesions.
- Rotational failures are not of such a high importance as on the lower extremity.

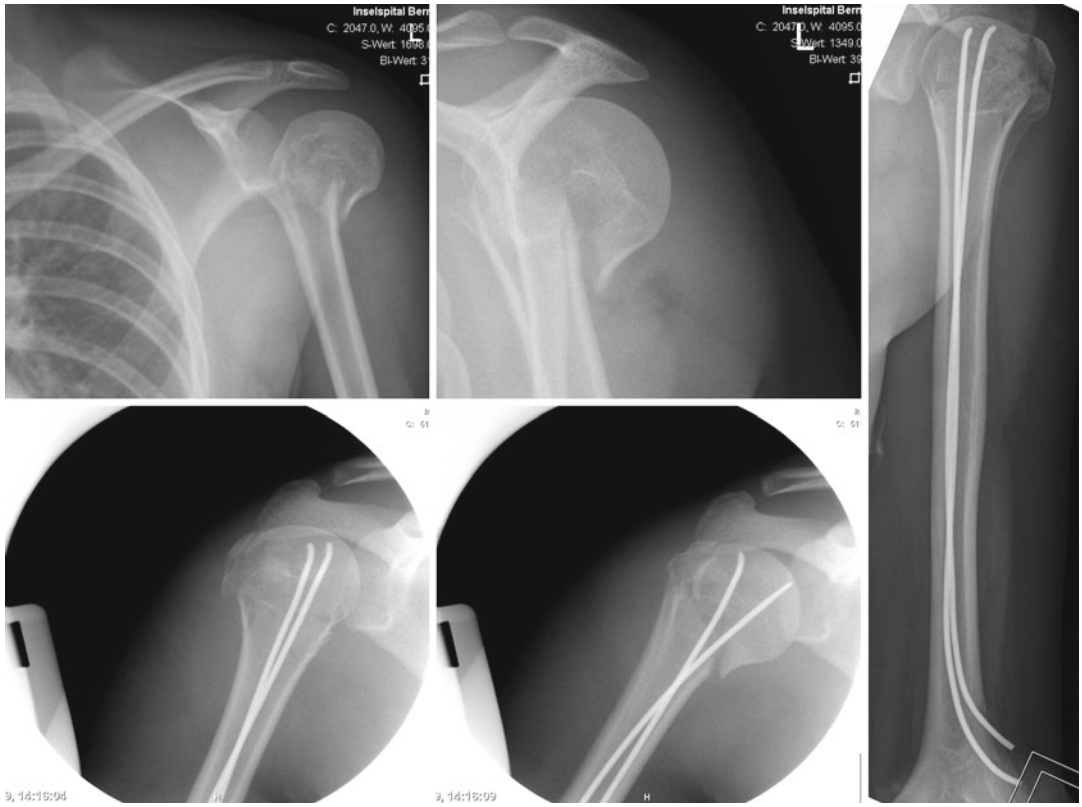

Fig. 3.11 A case example of the successful management of a displaced proximal humerus fracture in a skeletally immature patient. After a closed reduction maneuver, the fracture is stabilized by two retrograde elastic titanium nails inserted through a unilateral radial approach in ascending technique. Adequate reduction is ascertained by the "ice-cream on a cone" principle under fluoroscopy in antero-posterior and lateral planes

Table 3.9 Overview of the morphology, diagnosis and treatment of diaphyseal humerus fracture

Morphology	Rare fractures
Signs	Deformity, pain
Diagnosis	X-ray (two images taken at 90° to one another)
Correction potential	There is a great potential in all planes
Complications	Damage to the radial nerve (long spiral fractures of the distal third)
Non operative therapy	Stable undisplaced fracture, any age
	Stable fracture with angulation <30°
	Or
	Stable fracture with tolerable displacement, any age
	Immobilization for 3–4 weeks in a Desault or Gilchrist dressing
	Or
	If anesthesia is needed for reduction, definitive treatment with stable fixation is recommended
Operative treatment	Children >10–12 years of age
	Radial nerve irritation is not an indication for surgical intervention
	Unstable fracture if a reduction under anesthesia is necessary
	Major displacement after nonoperative treatment
	Author's preferred method: ESIN
	No additional immobilization is needed
	Nail removal after 3–4 months
Immobilization	3–4 weeks
X-ray control	Nonoperative therapy: days 3–4 and weeks 3–4
	Operative treatment: postoperatively and week 4
Follow-up	Week 3 or 4 radiologically and clinically

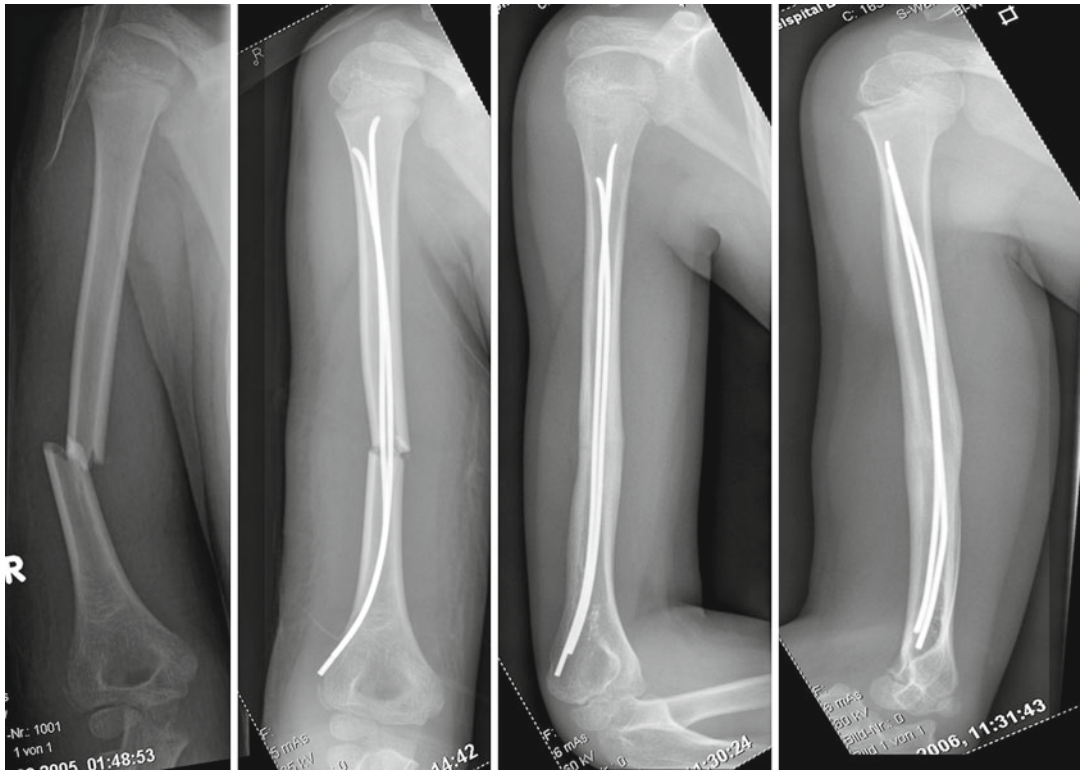

Fig. 3.12 One of today's most popular fixation modalities for humerus shaft fractures. Nowadays, displaced and unstable fractures (10 %) are preferably treated with elastic nails (ESIN). The major indications in our hand are children with head injury (advantage for rehabilitation)

Prognosis

- Very good, independent of age (Fig. 3.12).

Supracondylar Humerus Fractures

General Considerations (Table 3.10; Fig. 3.13):

- These constitute 80 % of all fractures of the humerus.
- They can happen from the first year of life up to adolescence.
- One must be aware of radial and medial nerve injury.
- The absence of the radial pulse is not an absolute indication for a revision of the cubital artery.
- We have to distinguish between pulseless pink and pulseless white hand.
- A compartment syndrome is the result of too tight a plaster cast; a huge swelling in addition to a tight plaster cast or the swelling of the soft tissue and muscles following the direct trauma.

Note:

1. Poor quality reduction leads to poor/insufficient stabilization.
2. Insufficient/poor stabilization frequently leads to poor functional and cosmetic (cubitusvarus) results.

Prognosis

If reduction and stabilization is adequate and sufficient, a perfect or at least a good functional and cosmetic result can be expected.

Most nerve injuries are temporary; revision is indicated if within 4–5 months there is no visible recovery.

Table 3.10 Overview of the morphology, diagnosis and treatment of the supracondylar humerus fracture

Morphology	See Fig. 3.13
Signs	Swelling, pain, visible deformation
Diagnosis	X-ray in two planes
Classification:	Incomplete fracture; no displacement (Type I)
According AO pediatric	Incomplete fracture with more 2 mm gap (Type II)
Long bone classification	Complete fracture with bone contact (Type III)
	Complete fracture no bone contact (Type IV)
Correction potential	Practically does not exist at this place
Complications	Radial (medial) nerve injury (deep branch)
	Premature closure of the growth plate after repeated drilling
	Varus deformity as a consequence of a rotational failure
Nonoperative therapy	Classification: type I and II
	Blount loop
	Dorso-volar plaster splint in 90° position
Operative treatment	Classification: type III and IV
	Closed reduction (in 90–95 % is possible)
	Percutaneous K-wire fixation (ascending crossed bilateral, parallel radial, ascending or descending monolateral radial)
	Small external radial fixator (method preferred by author)
	ESIN
Immobilization	Operative and nonoperative treatment; 3–4 weeks of plaster fixation
	Removal of the percutaneous K-wires at this time
X-ray control	Nonoperative treatment: after a few days and at week 3–4, depending of the child's age
	Operative treatment: after 3–4 weeks
Follow-up	2–3 months after injury functional, clinical examination
	No physiotherapy

If a cubital artery repair was performed after 4–6 h after injury, a prophylactic fasciotomy is mandatory (Fig. 3.14).

Fractures Around the Elbow Region

General considerations:

- Mostly children between 3 and 10 years old sustain these fractures.
- One must be aware of non-accidental injury.
- Good knowledge of the child's anatomy of the distal humerus and the proximal forearm is imperative (Tables 3.11 and 3.12).
- The x-ray is often difficult to interpret; nevertheless an x-ray of the contralateral, uninjured site is unnecessary and no longer required.

> Note: The knowledge of the development of the different ossification centers around the elbow region is "key" for adequate assessment of injuries.

- Correct diagnosis should always be achieved before starting treatment; at least, when the child is under anesthesia, the elbow region should be analysed under the image intensifier to look for special fractures and nondisplaced fractures of the lateral condyle.
- Isolated fractures of the radial neck
- Monteggia fractures
- Rotation failures in supracondylar humerus fractures due to varus or valgus deformity resulting from the fracture

Epiphysiolysis of the Distal Humerus

General Considerations:

- Epiphysiolysis of the distal humerus is a rare injury; occurs in babies and children under 1 year of age; more seldom in older children (Table 3.13).

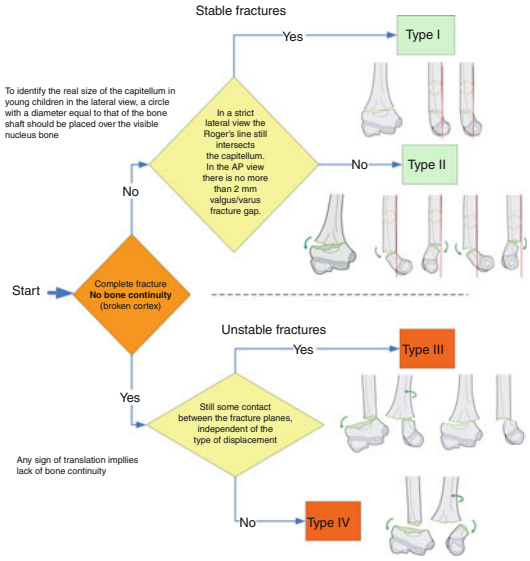

Fig. 3.13 Classification regarding the direction of displacement of the distal fragment according the AO Pediatric Comprehensive Classification for long bone Fractures (PCCF)

- So called pseudo-paralysis of the arm is an indirect sign in new-born children.
  - One must be aware of non-accidental injury.

Transcondylar/Intercondylar Fractures of the Distal Humerus

General Considerations:

The trans- and intercondylar fractures of the distal humerus are the most frequent intra-articular fractures in childhood (Table 3.14).

Note: every Salter-Harris III and IV fracture is always an intra-articular fracture.

Regarding the stability we have to distinguish between the so-called "hanging" fractures (potential stable) and the complete fractures with interruption of the articular cartilage (unstable fracture)

Regarding the displacement we have to differentiate between "step and gap". The limit of 2 mm traditionally implies we proceed to surgical treatment (Fig. 3.15).

Fractures of the Proximal Radius (Neck and Radial Epiphysiolysis)

General Considerations:

The most frequent fracture is a metaphyseal radial neck fracture (Table 3.15).

In childhood real "radial head" fractures are absolutely rare.

The annular ligament worked as so-called "hypomochlion".

Comminuted fractures are rare (Fig. 3.16).

Elbow Dislocation

General Considerations:

- In the majority of the cases an elbow dislocation is accompanied by a medial epicondyle fracture (Table 3.16).
- We must also be aware of a radial instability.
- Functional stability test in critical cases is mandatory.
- The interposed completely displaced epicondyle in the elbow joint (especially after spontaneous reduction) can be overlooked very easily.

Fracture of the Medial and Lateral Epicondyle of the Distal Humerus

General Considerations:

- Injuries of the lateral or medial epicondyle are mostly combined with elbow instability (Table 3.17).
- The stability must be checked absolutely.
- The medial epicondyle injury is an extra-articular lesion while the radial epicondyle osteochondral lesion is an intra-articular lesion.

Olecranon Fracture

General Considerations (Table 3.18):

- Three different lesions:
  - Extra-articular; avulsion of the olecranon apophysis

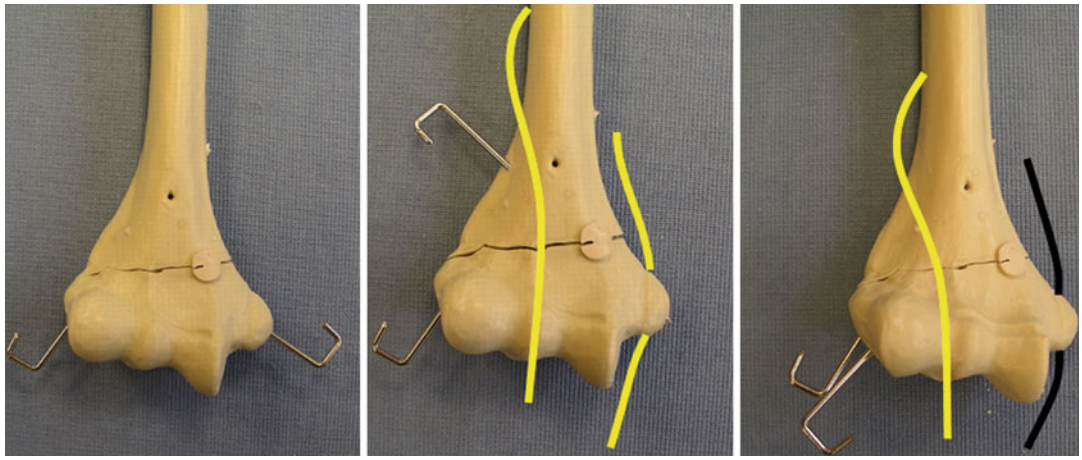

Fig. 3.14 Different treatment/fixation modalities for supracondylar fractures; the most popular and used K-wire fixation, crossed bilateral, crossed only from radial and divergent from radial

Table 3.11 Classification regarding the joint

Articular	Fractures of the lateral condyle
	Transcondylar fractures of the humerus
Extra-articular	Supracondylar fracture of the humerus
	Epicondylar fractures

Table 3.12 Classification regarding the direction of displacement of the distal fragment

Fractures in extension	In 95 % of all cases, the distal fragment is displaced dorsally
Fractures in flexion	The distal fragment is displaced ventrally only in 5 %

  - Intra-articular: real fracture of the olecranon involving the articular cartilage
  - Metaphyseal fracture of the proximal ulna (Be aware of Monteggia lesion.)
- In undisplaced fractures the triceps tendon and the periostium stabilize the fracture in hyper-flexion as a tension-band.
- Even in fractures with intra-articular gap this tension-band effect adapts and closes the fracture-gap.

Table 3.13 Epiphysiolysis of the distal humerus

Morphology	Very rare fracture; *most occur in new-born children*
Signs	Pain, deformity/swelling
Diagnosis	X-ray
	Ultrasound (Baby)
Correction potential	Very high (especially in new-born babies)
Complications	Same as for supracondylar fractures (see Table xx)
Nonoperative therapy	Dorso-volar plaster splints; in new-born children fixation of the arm to the body
Operative treatment	If a reduction is indicated: fixation with crossed K-wires
Immobilization	3 weeks
X-ray control	Day 7 (nonoperative) 3–4 weeks end control
Follow-up	2 months up to 1 year

Subluxation of the Radial Head ("Chassaignac" Pronation Douloureuse)

General Considerations (Table 3.19):

- This occurs in young children when they start to walk.
- This is a typical trauma mechanism (report of the patient).

Table 3.14 Morphology, diagnosis and treatment of trans- and intercondylar humerus fractures

Morphology	The entire distal epiphysis of the humerus is displaced posteriorly, laterally or forwards, depending on the injury mechanism. The most frequent fracture is that of the lateral condyle
Signs	Pain, swelling
Diagnosis	X-ray in two planes. Sometimes only the oblique view will disclose either displacement or evidence of the undisplaced fracture line
Correction potential	None
Complications	Delayed healing and blocked union with varus deformity, late ulnar nerve irritation, avascular necrosis of the capitulum Pseudarthrosis of the lateral/radial condyle (*the most frequent lactation for pseudarthrosis in Childhood*)
Nonoperative therapy	Initial undisplaced /stable fractures (long arm cast) followed by a cast-free X-ray control on day 4–5 Secondary displacement over 2 mm needs surgical intervention
Operative treatment	Initial displacement over 2 mm (open reduction and K-wire or screw fixation) Implant removal after 8–12 weeks
Immobilization	4 weeks
X-ray control	Undisplaced fractures day 4–5, cast free Consolidation is visible after 4–5 weeks
Follow-up	6 months and 1 year

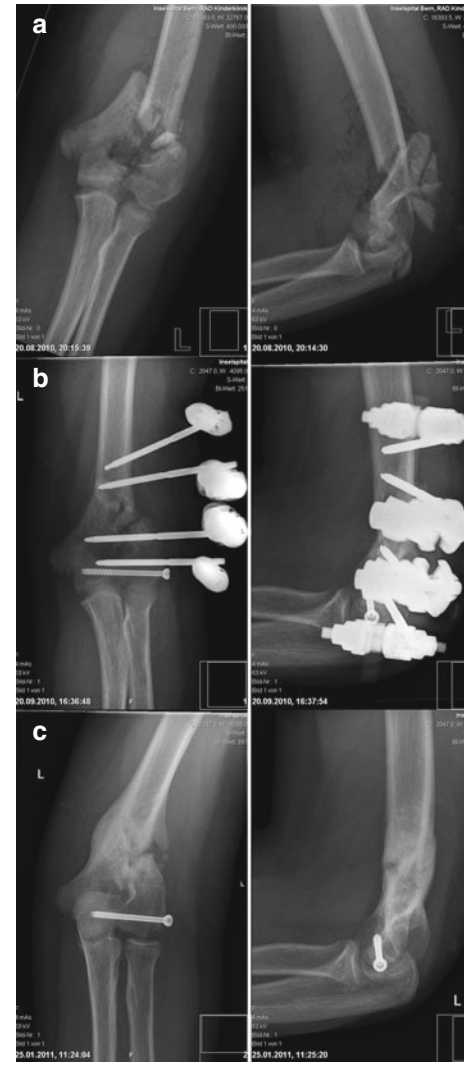

Fig. 3.15 Comminuted transcondylar distal humerus fracture in a 14-year-old boy. Injury film AP and lateral shows the different fragments (**a**). A new approach to this fracture; instead of the traditional dorsal approach and bi-condylar plate osteosynthesis a little lateral incision was chosen. Stabilization was by means of small radial external fixator (**b**). Uneventful healing after 3 months with correct anatomical axis, cosmetical result, and full function (**c**)

These Are Forearm—Shaft Fractures (Fractures of the Diaphysis of Ulna and Radius)

General Considerations:

- Diaphyseal injuries of the ulna or radius or both are common in children.
- The morphology may vary from pure bowing (Table 3.20) to greenstick (Table 3.21) or even to a complete fracture (Table 3.22) with or without displacement.
- The level of the fracture varies.
- Regarding the type of therapy it is important to recognize and distinguish between stable, potential unstable and complete unstable fractures.

Table 3.15 Radial neck fractures: morphology, diagnosis, and treatment

Morphology	65 % subcapital = metaphyseal fractures of the radial neck *35 % Salter–Harris II fractures*
Signs	Pain, blockage of pronation and supination
Diagnosis	X-ray in two planes, sonography
Correction potential	None in lateral displacement Good in the sagittal and frontal plane up to 45° in children under 8–10 years
Complications	Avascular necrosis, malunion or non-union, premature fusion of the growth plate, ectopic calcification, limited pronation and supination
Nonoperative therapy	<10 years of life up to 45° (long arm cast)
Operative treatment	If anesthesia is needed for reduction Closed reduction by indirect manipulation Fixation with ESIN *Trick*: the fully displaced radial head can be manipulated by a percutaneous K-wire, and thereby moved to the right location (Joy-stick technique) for ESIN fixation
Immobilization	Nonoperative therapy: 2–3 weeks, then functional therapy Note: *Operative treatment: no immobilization is required*
X-ray control	Nonoperative therapy: day 4 and 8 and after 3 weeks Operative treatment: only after 4 weeks
Follow-up	Clinical controls for 2 years after accident

- The following are the signs of instability (Fig. 3.17):
  - Fracture on the same levels/oblique fracture of one of the bones or both. No bone contact
- In children, the tendency for the radius and ulna fractures to be aligned greater than in adults
- Great fracture variability
  - Same fracture type in both bones, but not aligned
  - Isolated fracture of the radius
  - Bowing of radius and ulna (Table 3.20)
  - Fracture of the ulna and bowing of the radius or the reverse
- A Galeazzi fracture is the name given to a fracture of the distal radius with dislocation of the distal radio-ulnar joint; this fracture is very rare in childhood and occurs as a so called Galeazzi-like lesion with spontaneous reduction after shaft alignment.
- Factors which have a bad influence on the functional result:
  - Remaining deformity in the proximal third of the shaft
  - Fragments healed in a convergent position (Fig. 3.17)

Bowing Fracture of the Forearm

General Considerations

- Because of the bad functional outcome, even in younger children, and less angulation there is an increasing tendency to opt for operative treatment of those fractures.
- These fractures occur only in the shaft area and in younger children.
- We should not accept a bad functional result because a child will adapt to a limited function (children always adapt to a "mal-function").

"Greenstick" Fracture of the Forearm Shaft

General Considerations:

- Typical fracture of the pre-school child
- Seldom in children over 12 years (depending on the child's evolution)
- Sometimes combined, one bone bowing, other greenstick or one bone complete and other bone greenstick (Fig. 3.18)

Complete Diaphyseal Fracture of the Forearm

General Considerations

- High risk of instability and, even after perfect reduction, high risk for secondary dislocation

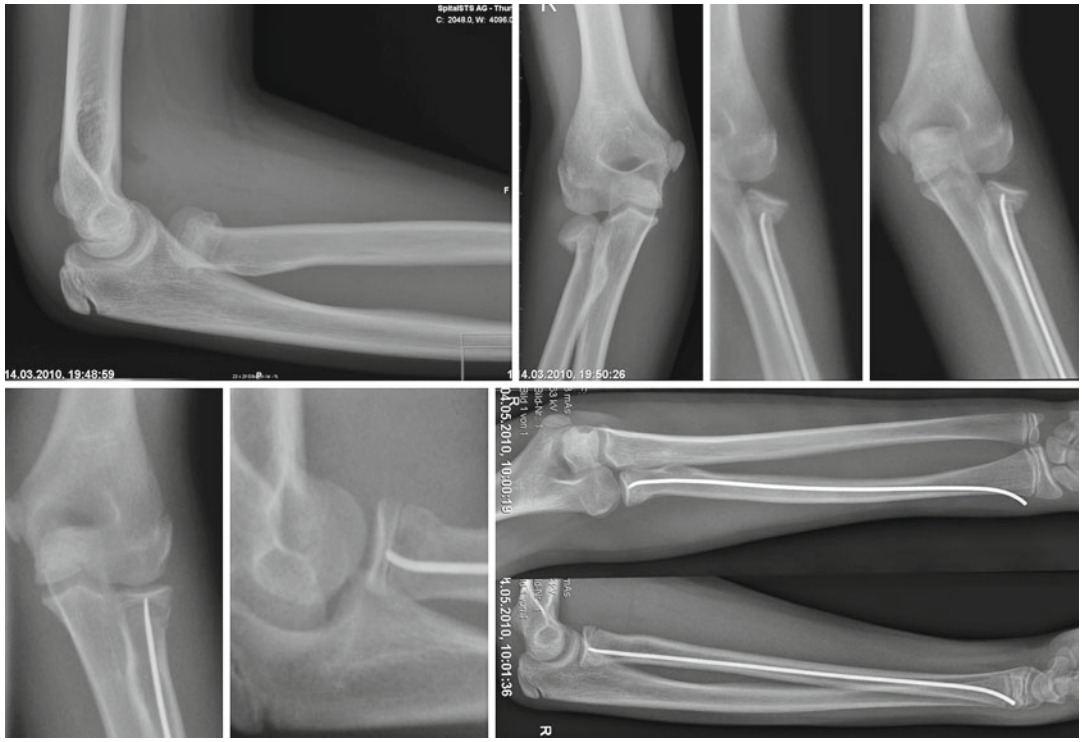

Fig. 3.16 "Gold Standard" of the treatment of radial neck fractures with closed reduction and intramedullary manipulation and fixation of the fracture with elastic nail accepted worldwide; cast free postoperative treatment is recommended

Table 3.16 Dislocation of the elbow

Morphology	Mostly in children over 8 year of age Displacement direction correlates with deforming force direction
Signs	Deformity, pain, swelling, ulnar nerve irritation
Diagnosis	Clinically and X-ray The differential diagnosis of an elbow dislocation basically consists of distinguishing a dislocation from a supracondylar fracture, a lateral condylar fracture, or a trans-condylar fracture
Correction potential	None
Complications	Medial ligament avulsion, fracture of the medial epicondyle Vascular and nerve complications
Nonoperative therapy	Immediate reduction of an acute posterior dislocation may often be accomplished without general anaesthesia Dislocation of the radial head during this manoeuvre can occur and must be realized. Long arm cast for 3 weeks, then functional therapy
Operative treatment	Re-fixation of the medial epicondyle with K-wire or screw Reconstruction of the medial and lateral ligaments if the elbow is unstable
Immobilization	3–4 weeks
X-ray control	Only after surgery in week 4
Follow-up	6 weeks and 6 months

Table 3.17 Medial and lateral epicondyle fracture of the distal humerus

Morphology	Fracture of the medial or lateral epicondyle (nearly always as a result of an elbow dislocation)
Signs	Swelling, local pain (lateral or medial) Persistent elbow dislocation.
Diagnosis	X-ray in two planes
Correction potential	None
Complications	Non-union
Nonoperative therapy	Only undisplaced fractures: Look for secondary displacement on day 3–4 without cast (x-ray) Operative treatment Displaced (>2 mm) fractures: Open reduction and K-wires or screw fixation Secondary displaced fractures after nonoperative therapy in the control
Immobilization	3–4 weeks, long arm cast
X-ray control	Undisplaced, cast free on day 3–4 operated fractures in week 4
Follow-up	2–3 months after injury

Table 3.18 Fracture of the olecranon

Morphology	Olecranon fractures are usually undisplaced and incomplete, particularly in younger children *Often seen in combination with other injuries*
Signs	Swelling and pain, elbow in flexion
Diagnosis	X-ray in two planes, sometimes very difficult, especially in young children (absence of ossification centers)
Correction potential	None
Complications	Restricted movement
Nonoperative therapy	Long arm cast for undisplaced fractures
Operative treatment	Longitudinal pinning and tension band wire fixation in dislocated fractures
Immobilization	Long arm cast for 4 weeks
X-ray control	Nonoperative therapy: days 5–6 and at week 4 Operative treatment: at week 4
Follow-up	Clinical control at week 8

- Risk factors for bad outcome
  - Oblique fractures
  - Fractures at the same level in both bones
  - Convergent malunion in the proximal third
- Better outcome after operative treatment followed by conservative management (Fig. 3.19)

Table 3.19 Radial head subluxation

Morphology	Mostly due to traction on the forearm in 1- to 3-year-old children Subluxation of the radial head
Signs	Painful pronation, elbow in extension
Diagnosis	Clinically, history
Correction potential	Good
Complications	Neglected fracture of the radial neck, persistent dislocation
Nonoperative therapy	Elbow in flexion – fast supination and extension – the click is noticeable
Operative treatment	Indicated only in neglected cases
Immobilization	None
X-ray control	None
Follow-up	None

Metaphyseal Fractures of the Distal Third of the Forearm

General Considerations (Table 3.23):

- Most frequent fractures in childhood
- Most "benign" fractures
- Use of the square of the AO Pediatric Fracture Classification to identify the real metaphyseal segment recommended.

Treatment recommendation for distal forearm fractures without anaesthesia

- Place child in supine position (Fig. 3.20).
- Place upper extremity in 90° abduction on the edge of the table with the elbow in 90° flexion.
- Cover the whole arm with a stocking.
- Elevate the forearm using finger traps.
- Attach a counterweight across the upper arm with the elbow at 90° flexion, to the extent that the child can tolerate.
- Maintain this position for 15–20 min.
- Reduce the fracture by pressing both hands together to stretch the interosseous membrane.

Table 3.20 Bowing fracture of the forearm—shaft

Morphology	Plastic deformity of the shaft without fracture of the cortex® microfractures
Signs	Pain, deformity, restricted movement
Diagnosis	X-ray, two planes
Correction potential	None
Complications	Restricted movement (pronation and supination), re-fracture
Nonoperative therapy	<20° bending – long arm cast, cast wedging (only if necessary), no anaesthesia
Operative treatment	>20° bending – closed indirect reduction and stabilization with ESIN technique
Immobilization	Nonoperative therapy – 4 weeks Operative treatment – immobilization is not required
X-ray control	At weeks 4
Follow-up	Over 1 year

Table 3.21 Greenstick fractures of the forearm shaft

Morphology	Plastic deformity of the shaft with one-sided cortex fracture
Signs	Pain, deformity, restricted movement
Diagnosis	X-ray, two planes
Correction potential	None
Complications	Restricted movement (pronation and supination), re-fracture
Nonoperative therapy	<20° bending – long arm cast, eventually cast wedging, no anaesthesia
Operative treatment	>20° bending – closed indirect reduction and completion of the fracture – stabilization with ESIN technique
Immobilization	Nonoperative therapy – 4 weeks Immobilization is not required after operative treatment
X-ray control	At week 4
Follow-up	Over 1 year

- X-ray control
- Apply a well-padded dressing.
- Apply a well molded long arm cast (must be opened after drying) or dorso-volar long arm splint (author's preferred method).

Table 3.22 Complete diaphyseal fractures of the forearm

Morphology	Both cortexes are fractured, with or without displacement
Signs	Pain, swelling, deformity
Diagnosis	X-ray in two planes
Correction potential	Partial, 10°–15° depending the age and the location
Complications	Mal-union with restricted movement, re-fracture
Nonoperative therapy	Only non-displaced, stable fractures bone contact), using a well-molded cast with three-point fixation Angulation <15° can be treated by cast wedging
Operative treatment	All displaced unstable fractures at any age Failure of retention in nonoperative therapy (ESIN with the possibility of reducing the fracture by making a small incision at the level of the fracture in about 10 % of cases)
Immobilization	5–6 weeks for nonoperative therapy No immobilization when ESIN is used
X-ray control	Nonoperative therapy: days 6–7 and weeks 5–6 ESIN: weeks 5–6 and before nail removal (nail removal not before complete re-modelation (all cortexes must be completely healed)
Follow-up	8–10 months

Wrist and Hand Fractures

General Considerations:

- Whereas carpal injuries and multiple unstable fractures of the metacarpals are rare in children, other hand and finger fractures are frequent in children, especially phalangeal fractures and inter-phalangeal joint dislocations.
- Fractures of the scaphoid are rare in children under 12 years of age. The treatment is nonoperative with a scaphoid cast for 6 weeks.
- Displaced fractures are treated operatively, similar to fractures in adults.
- Diagnosis can be difficult and often requires special x-ray techniques.
- Fractures of the metacarpals are the most frequent hand fractures.

- Each metacarpal has only one epiphysis; for MC I it is proximal (Table 3.24), for MC II–V, distal (Table 3.25).
- Hence, more proximal fractures can be found on the MC I and more distal fractures on the MC II–V.

Pelvic Ring Injuries

General Considerations:

- Fractures of the pelvic ring are rare in childhood, and usually the result of high-energy trauma.
- Most of them appear in combination with other severe injuries or as multiple trauma.
- The majority are simple peripheral fractures but the degree of severity ranges from simple fractures to complete and complex unstable fractures.
- Acetabular fractures are mostly the result of an isolated axial trauma as we see frequently in snowboarding, ski-cross events, and high jumps.

Classification

- We can classify pelvic fractures after two points of view:
  - Classic classification as shown in Table 3.26
  - Classification according to the outcome
    - Fractures without expected severe negative effects or malunion
    - Fractures with possible severe negative effects or malunion

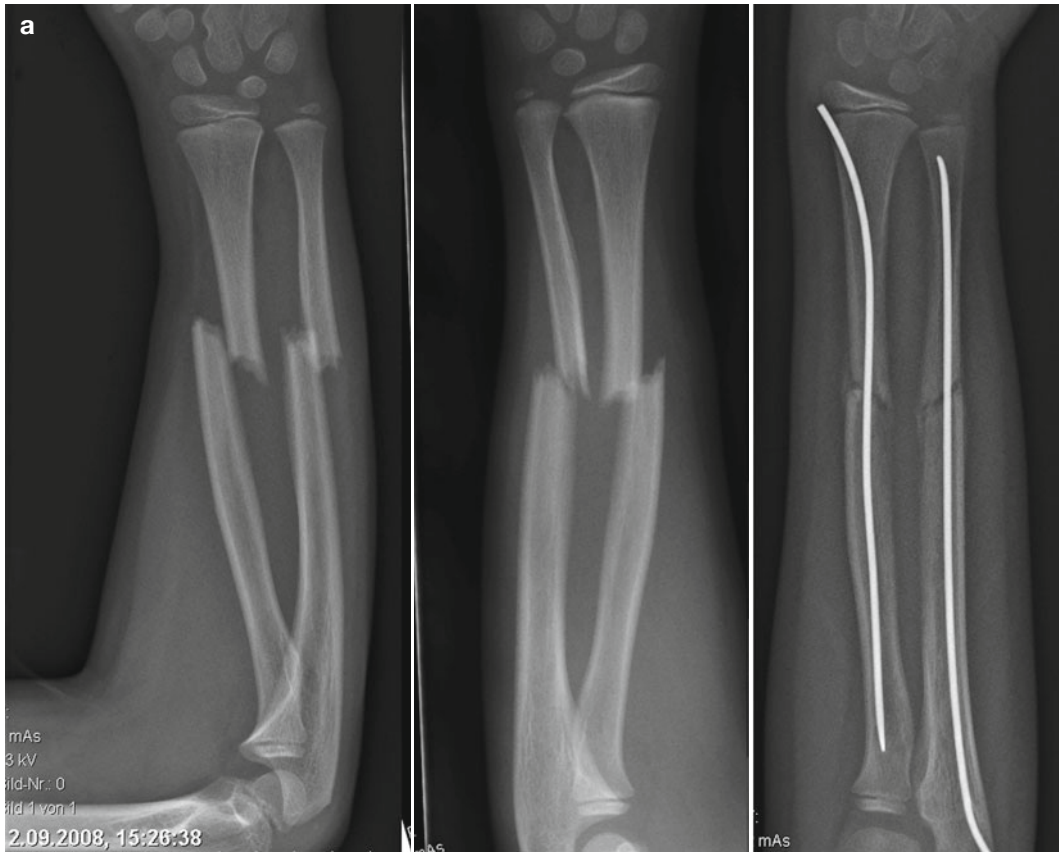

Fig. 3.17 Stable or unstable fracture? Signs of instability: fracture at the same level, oblique fractures, complete displaced, mid-shaft. Therefore this fracture needs stabilization (ESIN) (**a**). Signs of stability (never absolute): transverse fracture, fracture plane bone contact, no displacement. In this situation a long arm cast can be sufficient (**b**)

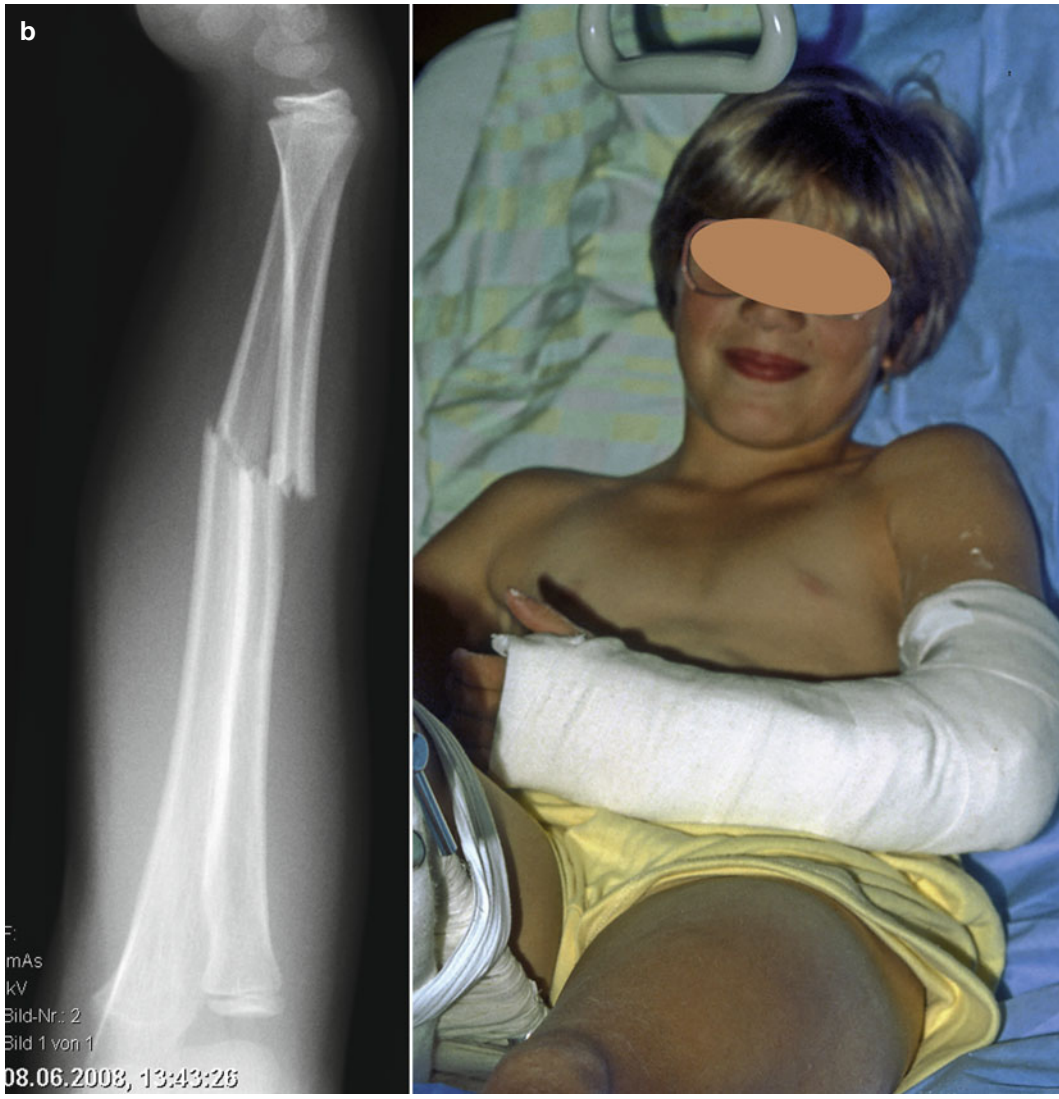

Fig. 3.17 (continued)

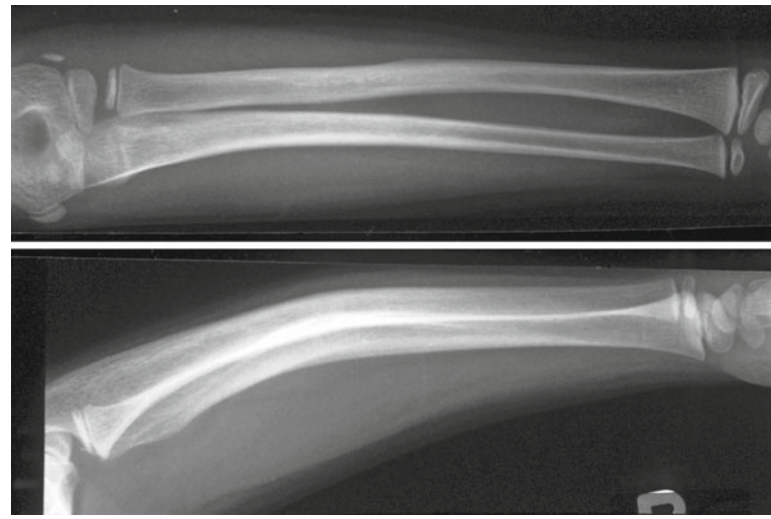

Fig. 3.18 "Complication" of an unproblematic bowing/greenstick forearm fractures; because of the low remodeling stimulation and capacity very often an increasing deformity is visible with a high rate of functional deficit

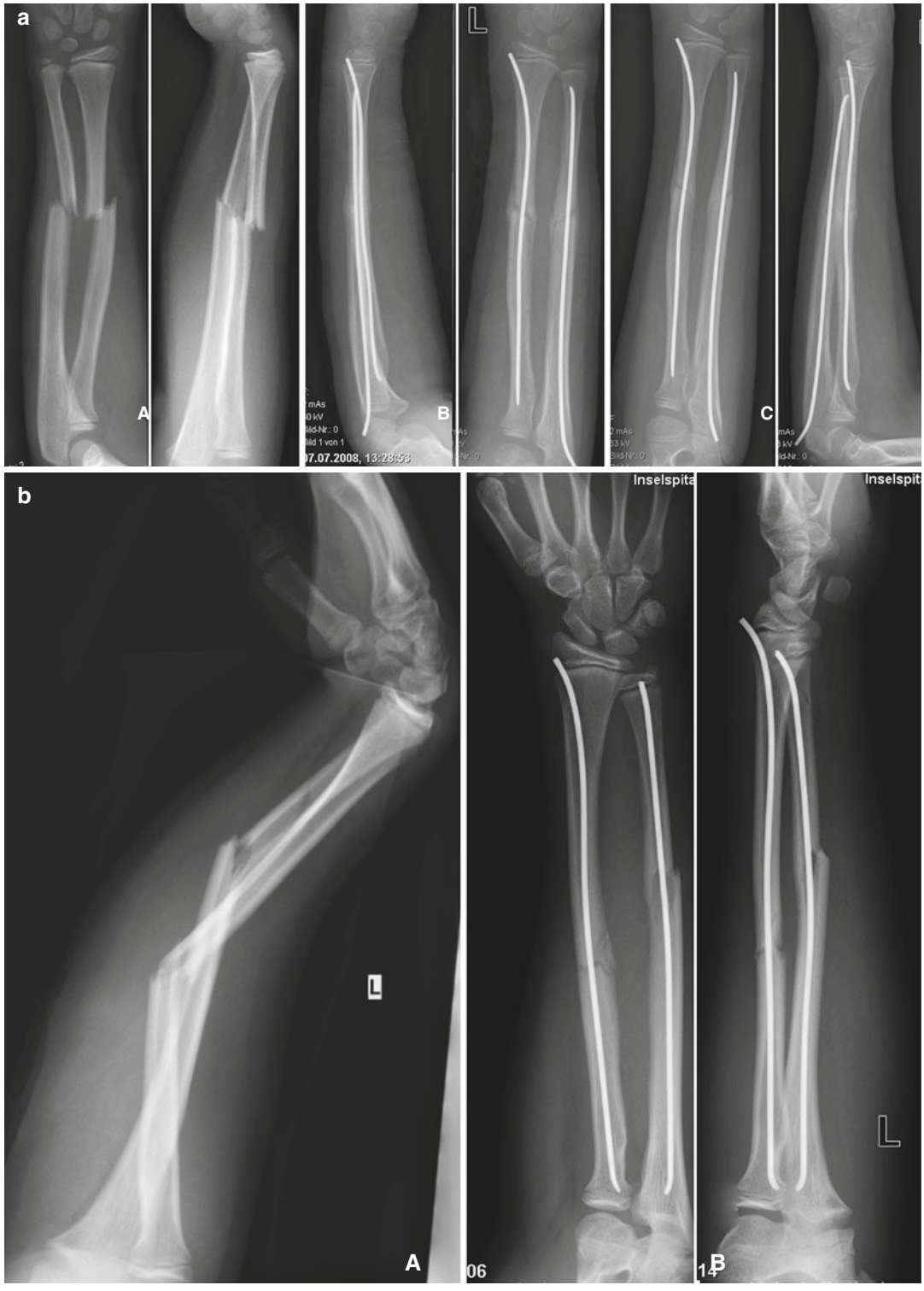

Fig. 3.19 Unstable forearm mid-shaft fractures treated with elastic nails; in both cases postoperative management without any cast fixation; this is an important prerequisite for a good functional healing. So called traditional technique; ulna from radial/proximal to distal, radius from distal radial to proximal (**a**); new technique, both nails from distal and the radial approach from the Lister's tubercle (**b**)

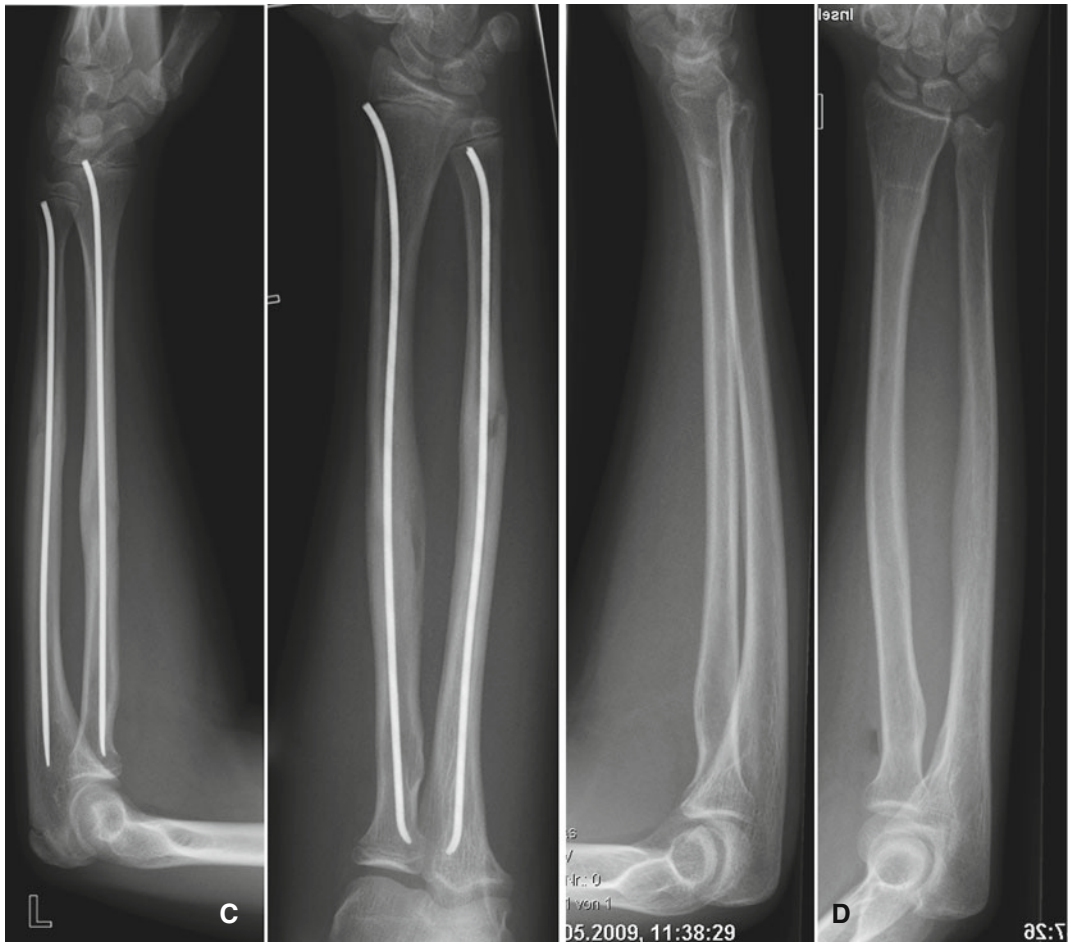

Fig. 3.19 (continued)

Table 3.23 Forearm fractures—distal third (metaphyseal fractures)

Morphology	Metaphyseal torus or buckle fractures
	Metaphyseal bowing and greenstick fractures Complete metaphyseal fractures with or without displacement
	Salter–Harris I and II fractures
Signs	Pain, swelling, deformity (medial nerve irritation)
Diagnosis	X-ray in two planes
Correction potential	Extremely good, children in <10 years, up to 50°
Complications	Correctly treated, practically none
	However:
	Be aware of growth arrest
	Avoid pin fixation trough the growth plate (can cause growth arrest.)
	Overgrowth of the radius is possible
Nonoperative therapy	Long arm cast immobilization for torus, greenstick and bowing fractures without reduction
	Complete fractures should be reduced under general anesthesia since muscle relaxation is an essential part of the reduction
	In the hands of an experienced surgeon 95 % of all fractures can be reduced and stabilized non-operatively with good outcome

(continued)

Table 3.23 (continued)

Operative treatment	Only complete unstable fractures of the distal radius in older or adolescent children need surgical stabilization (K-wires or external fixator) especially when the fracture is located on the proximal line of the "metaphyseal square". Plate fixation is an exception
Immobilization	3–4 weeks for the majority No immobilization for external fixator or plating
X-ray control	Days 6–7 and weeks 3–4
Follow-up	If there is a malunion at consolidation

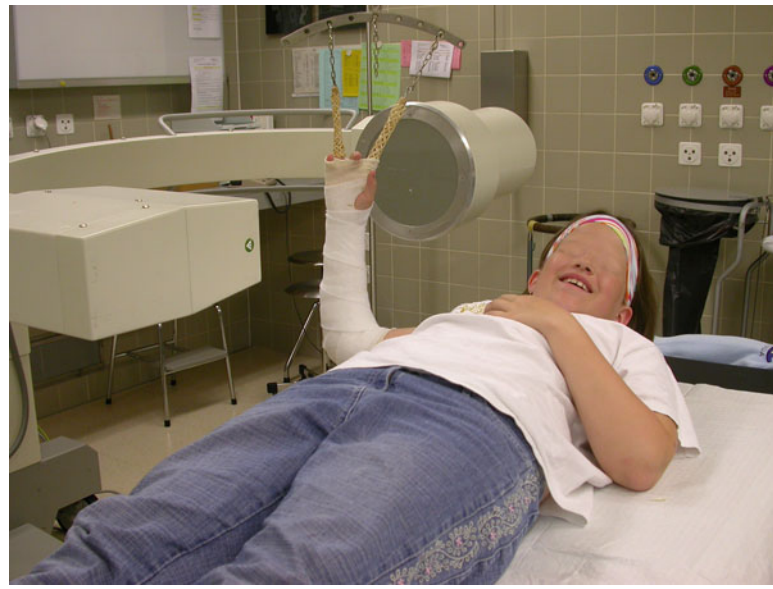

Fig. 3.20 Our treatment method of forearm fractures without anaesthesia and hanging in finger traps. The C-arm is in a horizontal position possible for rotating around the wrist (note: The patient should not rotate around the C-arm)

Therapy
- Table 3.27 gives a general overview/recommendation of the pelvic ring treatment.

Hip dislocation
- The management of hip dislocation is discussed in Table 3.28.

Lower Limb Fractures

Femoral Neck Fractures

- The capital femoral and trochanteric epiphysis has a unique growth plate along the posterior superior femoral neck due to embryonal development.
- Very often this unique physis does not separate into the head physis and the greater trochanter physis.
- Damage of this cartilaginous physis, as in a femoral neck fracture, may seriously impair normal development of the neck (coxavalga).
- Femoral head necrosis (AVN) results from vessel damage in this region, especially when the dorso-lateral reticular flap is damaged.

Classification:
- Different classifications of femoral neck fractures are used around the world; the most frequently used and well known one is the Delbet Classification.
- We actually use the new AO Pediatric Classification (PCCF)

Femoral Shaft Fractures

General Considerations:
- Femoral shaft fractures occur at any age.
- They are most frequent in school age and older children.
- There are two mechanisms: direct trauma produces more transverse fractures while indirect trauma produces oblique and spiral fractures.
- In babies and younger children one should be aware of nonaccidental injury.

Table 3.24 Fracture of the first metacarpal

Morphology	Mostly metaphyseal torus fractures or Salter–Harris I and II fractures; shaft fractures are rare
Signs	Pain, deformity
Diagnosis	X-ray in two planes
Correction potential	Possible in all planes, exception the frontal plane
Complications	Premature closure of the growth plate
Nonoperative therapy	Undisplaced metaphyseal and diaphyseal fractures treated with a forearm cast without reduction
Operative treatment	Displaced metaphyseal fractures, closed reduction and ± K-wire fixation
	Displaced diaphyseal fractures, closed reduction ± osteosynthesis (mini-ESIN, author's preferred method)
Immobilization	Proximal fractures: 2–3 weeks
	Shaft fractures: 3–4 weeks, independent of the fixation
X-ray control	Non operative therapy: day 4 and week 3
	Operative treatment: week 4
Follow-up	End of treatment

- Nowadays, because of better care and management of the child, around the world, for children of the age of 3 or 4 years, the operative treatment has become more and more popular.
- The reason for such an active treatment lies in the fact that today more child-friendly implants and methods are available (e.g. Elastic Stable Intramedullary Nailing; short ESIN)
- The ESIN method is the most popular osteosynthesis technique for all shaft fractures (humerus/forearm/femur and tibia)

As a worldwide accepted standard, the osteosynthesis method ESIN technique is described here in more detail.

For a variation of this technique and for the different bone segments corresponding operation brochures are available and must be respected.

Only the correct operation and respecting all the described operation steps guarantee a good stability and result.

Table 3.25 Fractures of metacarpals II—V

Morphology	Proximal fractures are rare and mostly undisplaced
	Subcapital fractures are more frequent, especially metacarpal V
Signs	Pain, deformity
	When the metacarpal or phalanx bones are involved, the uniform plane of the fingernails is disrupted, and the finger affected overlaps the others
Diagnosis	X-ray in two planes
Correction potential	Very good
	Remodeling is never capable of correcting a rotational deformity of the fingers
Complications	Axial deviations
Nonoperative therapy	Undisplaced basal fractures and fractures of the shaft
	Well-fitting plaster cast or splint or "Iselin splint" without reduction
Operative treatment	Displaced proximal fractures: closed reduction ± K-wire fixation and plaster cast
	Displaced shaft fractures: closed reduction ± plaster cast or mini-ESIN
Immobilization	Proximal fractures: 2–3 weeks
	Shaft fractures 3–4 weeks, independent of the fixation
X-ray control	Nonoperative: day 3 or 4 and weeks 3–4
	Operative: weeks 3–4
Follow-up	End of treatment

Table 3.26 The traditional classification of pelvic ring fractures

Fractures of the pelvic ring without delayed deformities (stable fractures)
Avulsion of the apophysis of
The inferior iliac spine
The superior iliac spine
The ischial tuberosity
Peripheral Iliac wing fractures
Pubic arch fractures
(Pubis and ischium)
Complete iliac bone fractures
Ilio-sacral joint loosening
Fractures with severe delayed deformities (unstable fractures)
Symphysis separation
Sacroiliac joint disruption
Acetabular fractures

Table 3.27 Treatment of pelvic ring fractures

Undisplaced/stable	Displaced/un-stable	
Avulsion of apophysis	Crutches/analgesia	Same
	<10 days	
Iliac wing fracture	Crutches until pain free	Open reduction with screw or K-wire fixation
Pubic arch fracture (pubis and ischium)	Crutches until pain free	Same
Complete fracture of the ilium	Crutches until pain free	Same
Symphysis loosening	Crutches until pain free	Same
Complete unstable symphysis separation	Crutches for 3–4 weeks	External fixator or Recco-plate fixation in older children
Sacro-iliac joint disruption	External fixator	Reduction, external fixator ± transarticular screw or 4-hole plate
Acetabular fractures	Spica cast 5–6 weeks	Open reduction and screw/plate fixation Nowadays recommendation – surgical hip dislocation approach.

Table 3.28 Hip dislocation and its management

Morphology	*Different types: superior-iliac, posterior-iliac, anterior-pubic, fracture dislocation* *Very rare in childhood*
Signs	Pain, the involved limb is shorter and is held in flexion, adduction, and internal rotation
Diagnosis	Clinical, X-ray, CT
Correction potential	None
Complications	Femoral head necrosis, re-dislocation, secondary hip dysplasia
Nonoperative therapy	Within the first 8 h, aspiration of the joint; if there is any sign of incongruence, open reduction is indicated
Operative treatment	Incongruence after reduction Combined with fracture of the acetabular rim
Immobilization	Depending on the injury and treatment: 1–6 weeks
X-ray control	In weeks 4–6 Scintigraphy or MRI if indicated additionally
Follow-up	When necrosis is suspected: every 6 months

Operative Technique with Elastic Stable Intramedullary Nailing (ESIN)

- See Fig. 3.21 for operative technique.
- Place child in supine position.
- Free position of the child, fixed with towels on the standard table.
- For transverse or complex fractures in older children the fracture table is recommended.
- Decide on the direction of the nailing.
  - The nail insertion in the standard technique is from distal to proximal (middle and proximal third of the femur).
  - For the distal third the so called descending, monolateral technique isrecommended.
- Determine the nail diameter; this is normally 35–40 % of the isthmus of the medullary canal on the x-ray image.
- Make a preliminary reduction using an image intensifier.
- Pre-bend the nails for a better and sufficient three point contact at the inner cortex. It is recommended to pre-bend the nail over the length of the bone three times the diameter of the diaphyseal segment.
- The nail entry point is normally one finger's breadth about the proximal tip of the patella, which corresponds to 2–3 cm proximal to the epiphyseal line.
- Make a skin incision about 3–4 cm in the distal direction from the planned entry point in the bone.
- Create the nail entry point by penetrating the near cortex with the awl or drill bit.
- Insert the awl primarily vertically down to the bone then lower the awl to an angle of 45° in

relation to the shaft axis whilst rotating it so that the bone cortex is perforated in an upwards showing angle.
- With a rotating motion, continue to penetrate the cortical bone at an upward angle.
- The nail diameter should be one-third of the narrowest diameter of the medullary canal and as long as the bone when bent. Both nails must be bent in the same way.

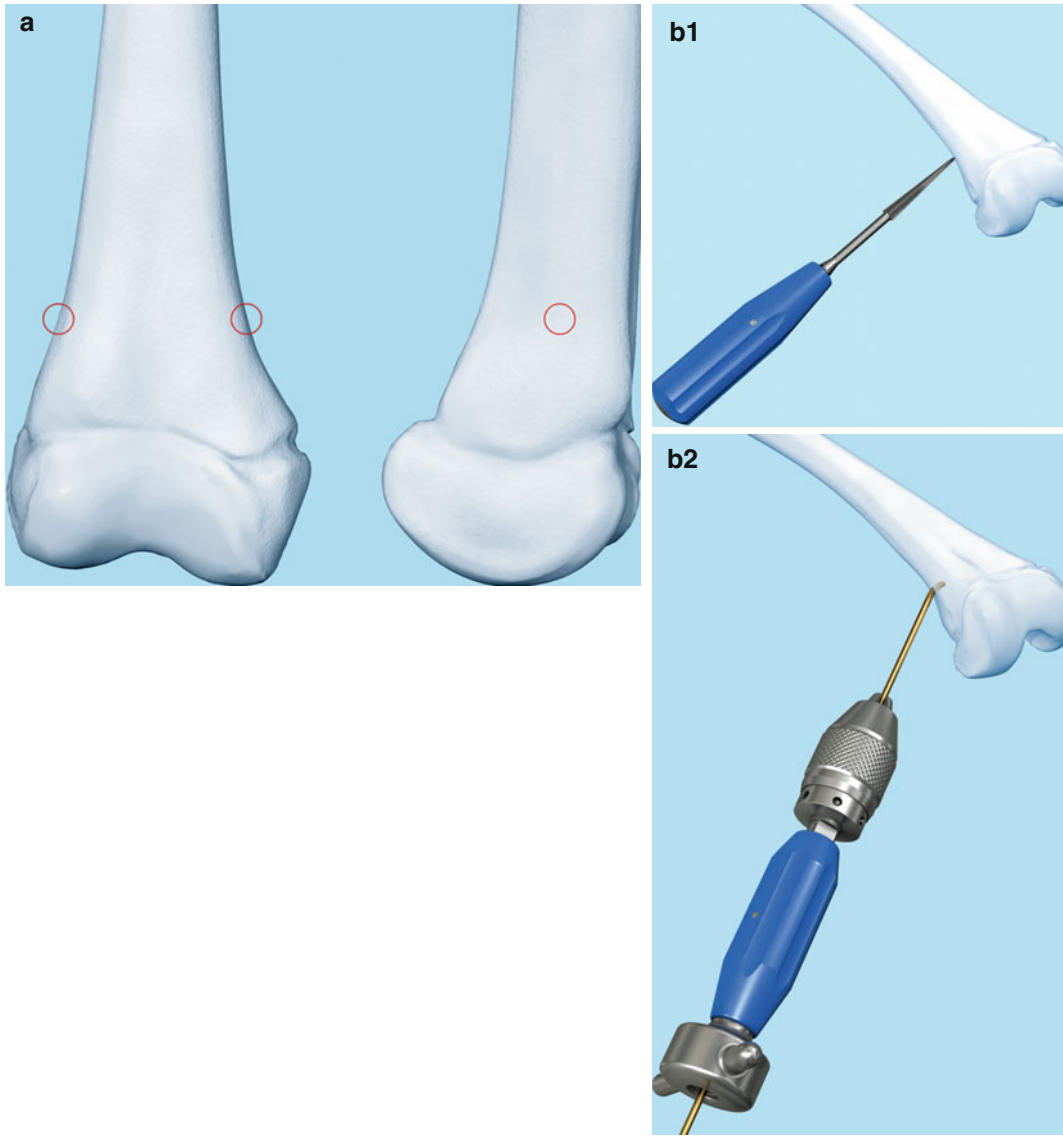

Fig. 3.21 The principle steps of the ESIN technique for a femur fracture. (**a**) Entry-point on the distal femur – >2 cm proximal to the growth plate. (**b**) Perforation of the cortex medial lateral and medial on the same level; the awl must be lowered up to 45°. (**c**) The first nail is introduced up to the fracture. (**d**) The second nail is inserted in the same way up to the fracture line. (**e**) Indirect reduction with the first (lateral) nail; depending on the morphology of the fracture the reduction is primarily made with the medial nail first. (**f**) After reduction, the second nail passes the fracture and both nails are advanced to the proximal metaphysis; at this point the rotation must be checked. (**g**) Then the nails are cut with the special nail cutter or a standard cutting device. You must pay attention to the ends of the nails that are under the facia

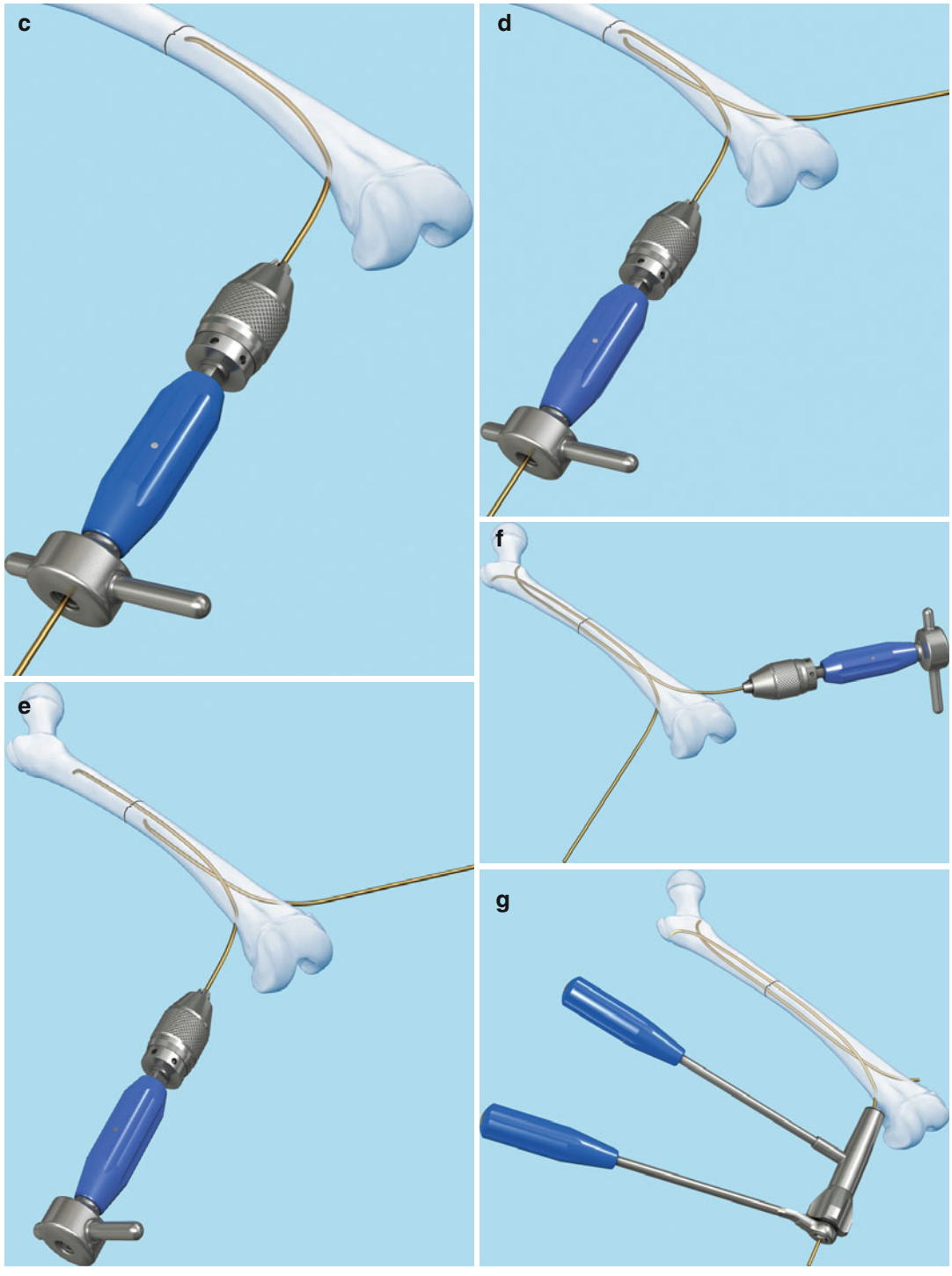

Fig. 3.21 (continued)

- Insert the first nail into the medullary canal with the nail tip at right angles to the shaft. Rotate the nail through 180° and align the nail tip with the axis of the medullary canal.
- In a manner similar to that previously described, open the femur on the opposite side.
- Advance the second nail up to the level of the fracture.
- Visualize the fracture with fluoroscopy and decide which nail will be easier to pass across the fracture and will most effectively pull the proximal fragment into alignment.
- Advance this nail across the fracture, monitoring its position with fluoroscopy.
- Advance this nail into the proximal fragment only so far as to ensure that the reduction is maintained
- Position the second nail in the same manner.
- Both nails can now be advanced to the proximal epiphysis.
- Cut the nails to the right length outside of the skin.
- Control the rotation of the leg before the definitive fixation of the nail tips in the proximal metaphysis.
- The final position of the nails is achieved when the end points are placed in the proximal fragment.
- If the nail tips in the proximal metaphysis are correctly located, then the nails can be shortened to the required length with a special or normal nail cutter.
- The distal ends of the nails should poke out at least 8 mm from the cortex in order to facilitate easy removal, whilst the low profile minimizes soft-tissue irritation.
- The correct protrusion of the ends of the nails is important when the so called end-caps are used.
- In axial unstable or critical situation, nowadays the end-caps can prevent a shortening and push out of the nails; so it is today possible to fix even comminuted fractures in older children with a higher body weight also (Fig. 3.22).
- If the fracture is distracted, release traction and impact the patient's heel.
- Close the skin
- Do not remove the nail before complete consolidation; wait at least 4–5 months
- For fractures of the distal third of the femur, up to 2–3 cm proximal to the supracondylar growth plate the so called anterograde or descending/monolateral technique must be used (Fig. 3.23).

Fractures Around the Knee

Distal Femur and Proximal Tibia Fractures

General considerations:

- These are very rare fractures in childhood, following high-energy trauma or traffic accidents.
- The radiological diagnosis is not easy; however, it is not as difficult as for the elbow.
- Therefore, a CT scan imaging is recommended and useful for a better understanding of the fracture morphology.
- Haemarthros indicates a severe trauma.
- Osteochondral fragments, "flake fractures," must be looked for.

Classification:

- Distal femur and proximal tibia fractures are described in Table 3.29.
- Supracondylar and condylar fractures of the femur and their management are described in Table 3.30.

Patella Fractures and Dislocations

General Considerations:

Patellar dislocations (Table 3.31) are common when considering the entire spectrum of acute and chronic subluxation and dislocation injuries.

More frequently, chronic subluxation mimics actual dislocation.

Dislocation of the patella is frequent in young girls.

Prerequisites are "genuavalga," being overweight, and "patella alta".

In addition to this the hypoplasia of the M. vastusmedialis as well the flat trochlea of the femur are the most important factors for chronic patella dislocation.

Fractures of the patella (Table 3.32) result from direct trauma and high-energy extension trauma (such as high jump).

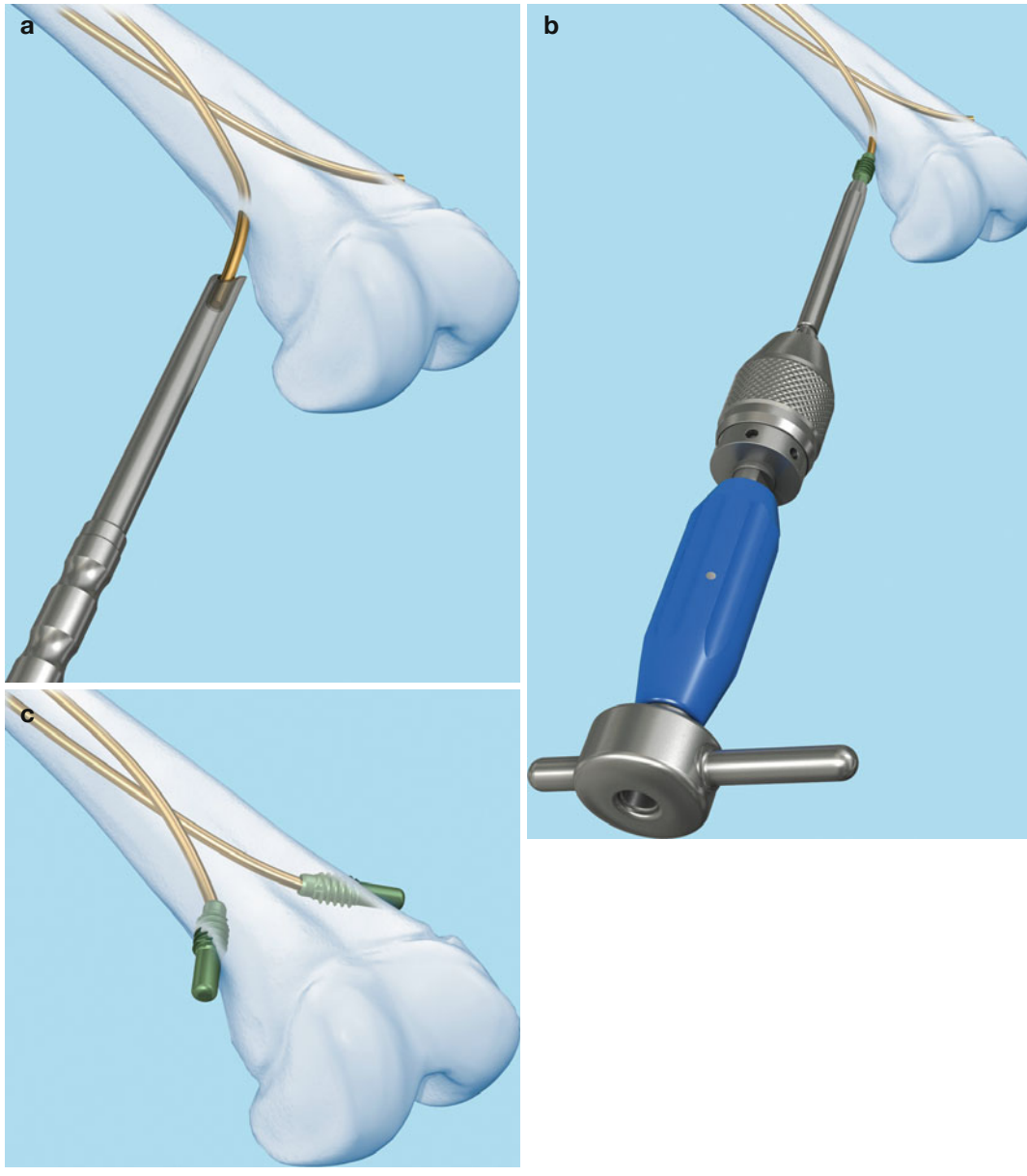

Fig. 3.22 The principle steps of the ESIN technique for comminuted, unstable femur fracture: In case of axial instability, nowadays with a simple additional implant, the so called end-caps, the axial stability can be dramatically improved. (**a**) After appropriate shortening of the nails, the definitive impaction must be done with a special "bevelled" impactor. This guarantees a correct length of the nail end to bring the end-caps over the nails. (**b**) With the help of the inserter, connected with the special "screwdriver" the end-cap is screwed in the bone; the threaded part of this self – drilling, self-taping canceller bone screw must have good contact to the bone only at the distal part. (**c**) Final positioning of the end-caps

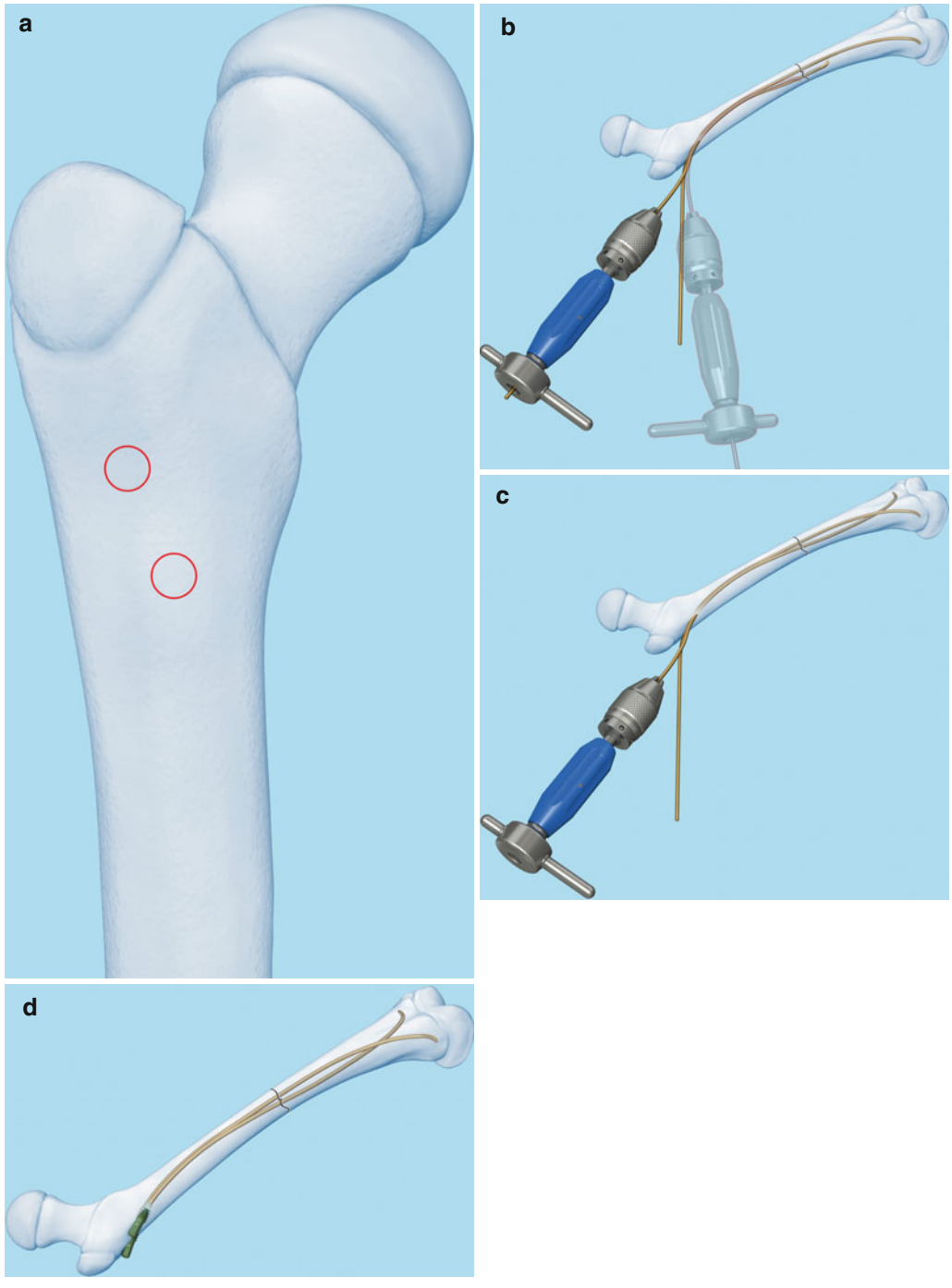

Fig. 3.23 The principle steps of the ESIN technique for distal femoral fractures; so called descending/monolateral technique: (**a**) Entry points are lateral, subtrochanteric region; it is recommended to make a small (5–7 cm) skin incision and to prepare the lateral aspect of the femur. (**b**) The first nail is inserted in a C-shape pre-bended manner up to the fracture; then with this nail the fracture can be reduced. (**c**) The second nail is pre-bended also in a C-shape but only in the proximal third of the nail. Then the nail is in inserted in the same way as the first one. In the distal, metaphyseal part but before passing the fracture, the nail is turned 180° so that both the tips are divergent; then the fracture is also passed. (**d**) Definitive positioning of the nails, in this picture in addition with end-caps

Table 3.29 Femoral shaft fractures and their management

Morphology	Transverse/oblique/short and long spiral and comminuted fractures Subtrochanteric fractures Fractures (transverse or oblique) of the proximal and middle third
Signs	Pain, deformity, restricted movement, blood loss, shock Note: also a small child can have a blood loss of 200–300 ml in the muscles
Diagnosis	X-ray in two planes, including both hip and knee joint
Correction potential	Very good, depending of the child's age Frontal plane better then sagittal plane
Complications	Leg length discrepancy, rotation failure, deviation of the axis
Nonoperative therapy	Children <3–4 years – outpatient overhead traction or for stable fractures initial hip spica cast (not for children with multiple injuries)
Operative treatment	Children 4–13/14 years, depending on their weight – closed reduction and ESIN as the first method of choice Unstable, complex fractures – external fixation or minimal invasive plating (MIPO) Solid intramedullary Nail; nowadays special for young adolescences – ALFN (Adolescence Lateral Femoral entry Nail)
Immobilization	3–4 weeks No immobilization after ESIN, MIPO, Ex-Fix treatment
X-ray control	Overhead traction or spica cast: weeks 3–4 Operative treatment: weeks 5–6, before implant removal
Follow-up	Children >10 years until growth stops

Table 3.30 Morphology/clinic and management of supracondylar and condylar fractures of the femur

Morphology	Supracondylar buckle fractures Complete transverse or oblique fractures, metaphyseal or physeal (Salter–Harris I+II) Uni- or bicondylar (Salter–Harris III+IV) fractures (very rare in childhood)
Signs	Pain, knee stiffness, swelling, pulseless lower leg
Diagnosis	X-ray in two planes
Correction potential	Good, depending on the child's age
Complications	None for supracondylar fractures Varus or valgus angulation following premature partial closure of the growth plate, limitation of knee motion, leg length discrepancy
Nonoperative therapy	All undisplaced fractures independent of the age Long leg cast immobilization in 10° flexed
Operative treatment	All displaced fractures, which need reduction Closed (extra-articular fractures) reduction and K-wires or Ex-Fix stabilization Open (articular fractures) reduction and K-wires or screw fixation Any fracture that needs a reduction under anesthesia should be treated with a stable, definitive fixation
Immobilization	4–6 weeks
X-ray control	Nonoperative: days 4, 10 and week 4 Operative: postoperative and weeks 4–6
Follow-up	Up to 2 years, depending on the fracture type

Lower Leg Fractures

General Considerations:

- Fracture of the tibial shaft (Table 3.33) is one of the most frequent fractures of the lower leg in childhood.
- In infants and children, the typical injury is a spiral tibial fracture with an intact fibula.
- Isolated tibial shaft fractures have the tendency to varus malalignment.
- On the other hand because of the expected overgrowth of the isolated fractured tibia a slight shortening (varus) is desired.
- One must be aware of the bowing of the fibula in isolated tibial fractures.
- This circumstance influences the kind of treatment and the outcome.
- Complete fractures of the tibia and fibula (Table 3.34) are unstable, and often shortened with rotational failures.

Table 3.31 Patellar dislocation and its management

Morphology	Nearly always dislocations in the lateral direction The dislocation may be complete or incomplete
Signs	Pain, swelling, blocking of the knee, hemorrhage in the joint
Diagnosis	Clinic signs, X-ray in two planes
Correction potential	None
Complications	Overlooked "flake fracture" Repeated dislocations
Nonoperative therapy	Reduction and extension of the knee while the hip is flexed ± aspiration of the haemarthros if the knee is painful Intensive physiotherapy with special attention on the vastus medialis exercises
Operative treatment	If there are any signs of osteochondral fractures arthroscopy is indicated Re-fixation or removal of the fragment Only in this cases when a conservative, intensive physiotherapy has no effect Our preferred treatment: Open lateral release/medial vastus advancement/medialization of 40 % of patellar tendon under the medial collateral ligament (so called modified Slocum procedure)
Immobilization	Long term immobilization is contra-productive. Short time Immobilization for pain in a cylindrical cast or cast splint or brace After operation (patella stabilization) 6 weeks but never complete; means daily CPM up to 60° of flexion
X-ray control	After reduction
Follow-up	3–6 months to check the physiotherapy if habitual dislocation is suspected 1 year if an operation was performed

Table 3.32 Patellar fractures and their management

Morphology	Incomplete and complete fractures Inferior and superior fractures Longitudinal and transverse fractures "Sleeve" fracture Variations of the norm – bipartite patella
Signs	Pain, swelling, blocking of the knee, haemorrhage in the joint
Diagnosis	Clinic, X-ray in two planes Sometimes MRI may be necessary
Correction potential	Partial, a cartilaginous gap is always filled out with fibrous cartilage
Complications	Non-union, pre-arthrosis
Nonoperative therapy	Fissures, undisplaced, stable fractures – cylindrical cast or brace
Operative treatment	All displaced fractures – traditional tension wiring of the patella Implant removal after 4–5 months
Immobilization	Permanent for 4–5 weeks in conservative therapy Passive ROM with physiotherapy or CPM after operation
X-ray control	Nonoperative therapy: day 6 and week 5 Operative treatment: postoperative and week 5
Follow-up	6 months after implant removal

- Some special types are toddler fractures or bowing of the fibula or (rarely) of the tibia.
- Transverse fractures of the lower leg can be managed in all ages conservatively.
- Oblique and spiral fractures are unstable and need a safe and stable fixation (nowadays preferably with ESIN or MIOP in older children).

Classification:

- The most practical classification we use today is the PCCF*.

Distal Tibia and Ankle Joint Fractures

General Considerations:

- Like the proximal region, the distal metaphysis may sustain injury in patterns of varying severity.
- Because of the microstructural differences between the thick diaphysis and the thinner metaphysis, so called metaphyseal greenstick and torus fractures are common.
- Because of the high energy transmitted to the distal physis of the tibia, damages of the physeal structures are not uncommon, resulting in an asymmetrical growth arrest.
- Malalignment of more than 5° varus or valgus is high risk for an early arthrosis.

Table 3.33 Tibial shaft fractures

Morphology	Fractures of the middle and distal third Spiral fractures are more frequent than transverse fractures
Signs	Pain, swelling, angulation
Diagnosis	X-ray in two planes including the knee and ankle joint
Correction potential	Good, depending on the age No correction for rotation deformities
Complications	Different rotation of the feet, remaining angulation, shortening
Nonoperative therapy	Undisplaced and stable fractures with angulation <10° (open long leg cast and if necessary cast wedging on days 4–5)
Operative treatment	The indication for operative therapy is rare Shortening of the tibia with bowing of the fibula (Ex-Fix or MIPO) ESIN can produce nonunion because of the blocking fibula
Immobilization	4–5 weeks for conservative treatment No immobilization is required after operative treatment
X-ray control	Nonoperative therapy: days 4 and 10 and weeks 4–5 Operative treatment: postoperative, week 5
Follow-up	Every 6 months, up to 2 years after the procedure

- Fractures of the ankle joint are classified according to the maturation of the epiphysis and the child's age (PCCF).
- The epiphysis begins to close from the age of 12 in girls and 14 in boys.
- Note: the physeal closure starts posterior-medial and ends anterior-lateral; this leads to typical fracture patterns in older children.
- Fracture types vary depending on epiphyseal maturation.
- Extra-articular and intra-articular fractures and their management are discussed in Table 3.35 and Table 3.36.

General Considerations:

These are two plane "Tillaux fractures" and Tri-plane fractures.

- This particular type of injury affects a part of the anterolateral tibial epiphysis.
- The segment may extrude anteriorly and laterally.

Table 3.34 Complete fracture of the tibia and fibula; lower leg fracture

Morphology	Fractures of the middle and distal third Spiral fractures are more frequent than transverse fractures Very often fully displaced as the stabilizing effect of the fibula is missing
Signs	Pain, swelling, angulation
Diagnosis	X-ray in two planes including the knee and ankle joint
Correction potential	Good, depending on the child's age No correction for rotation deformity
Complications	Rotational failure, remaining angulation, leg length discrepancy Be aware of a compartment syndrome especially following direct trauma (traffic accident)
Nonoperative therapy	Undisplaced and stable fractures with angulation <10° if at least 2–3 years of remaining growth time (open long leg cast and if necessary cast wedging on days 4–5)
Operative treatment	The indication for surgery is rare Displaced/unstable fractures (oblique and spiral) Method of choice – ESIN or Ex-Fix (MIPO in older children)
Immobilization	4–5 weeks for conservative treatment No immobilization required after operative treatment
X-ray control	Nonoperative therapy: days 4 and 10 and weeks 4–5 Operative treatment: postoperative, week 5
Follow-up	Every 6 months, for up to 2 years after the initial treatment

- Ankle congruity is of concern because juvenile two-plane fractures involve the weight-bearing articular surface.
- The fracture may be accompanied by a posterior metaphyseal fragment and in this case the fracture is called a "tri-plane fracture".
- The exact diagnosis can often be difficult when only based on standard a.p. x-ray projection of the distal tibia and fibula.
- Exact examination under the image intensifier can clarify the diagnosis.

Table 3.35 Extra-articular fractures of the distal tibia and their management

Morphology	Metaphyseal torus fracture Metaphyseal bowing fracture Complete metaphyseal fracture Epiphysiolysis (Salter–Harris I) Epiphysiolysis with metaphyseal wedge (*SH II*)
Signs	Pain, deformation, restricted movement
Diagnosis	X-rays in two planes Look for accessory ossification Documentation under image intensifier may be necessary
Correction potential	Very good
Complications	Alteration of leg length, valgus deformity, premature closure of the growth plate, fibulo-tibial synostosis
Nonoperative therapy	Metaphyseal torus and bowing fracture (plaster cast immobilization and cast wedging if necessary) Metaphyseal complete fracture (stable reduction, plaster cast) Stable, Salter–Harris I + II fractures (reduction, plaster cast)
Operative treatment	Complete displaced, unstable metaphyseal fractures (closed or open reduction and Ex-Fix or K-wires or (ESIN) fixation when distal fragment >4 cm) Displaced, unstable Salter–Harris I fractures (closed or open reduction and minimal invasive cross K-wire fixation) Displaced, unstable Salter–Harris II fractures (closed or open reduction and minimal invasive cross K-wire fixation or cannulated screw fixation)
Immobilization	4–6 weeks
X-ray control	Nonoperative therapy: day 6, weeks 4–5 Operative treatment: postoperative, week 5
Follow-up	Every 6 months, for up to 2 years after the procedure

- However, CT may exhibit far greater accuracy than plain radiographs in delineating the degree of joint displacement and fragment separation.
- This fracture type occurs especially in adolescents.

Table 3.36 Articular fractures (Salter—Harris III and IV) of the distal tibia and their management

Morphology	Epiphyseal fractures (Salter–Harris III) Epiphyseal fractures with metaphyseal wedge (Salter–Harris IV) "two-plane" or "tri-plane" fractures
Signs	Pain, deformation, restricted movement
Diagnosis	X-ray in two planes Look for accessory ossification Documentation under image intensifier may be necessary
Correction potential	Moderate
Complications	Alteration of leg length, valgus deformity, premature closure of the epiphysis, non-union Joint incongruency
Nonoperative therapy	Cast or splint for undisplaced fractures with an articulargap <2 mm
Operative treatment	Displaced unstable Salter–Harris III + IV fractures (closed or open reduction and minimally invasive screw fixation under image intensifier with cannulated self—drilling, self—tapping screws or K-wire fixation)
Immobilization	4–6 weeks
X-ray control	Nonoperative therapy: day 6, weeks 4–5 Operative treatment: postoperative, week 5
Follow-up	Every 6 months for up to 2 years after the procedure

- Therefore growth arrest is not of importance.
- A schematic and model illustration of a tri-plane fracture at the ankle joint is shown in Fig. 3.24.

Therapy:

- The aim of therapy is an exact reconstruction of the joint surface.
- Undisplaced (<2-mm gap) two-plane and tri-plane fractures are treated non-operatively by a well-padded compression dressing and posterior splint. After the swelling has disappeared the fixation is changed to a Sarmiento type cast.
- Displaced two- and tri-plane fractures are treated surgically (gap or step >2 mm).

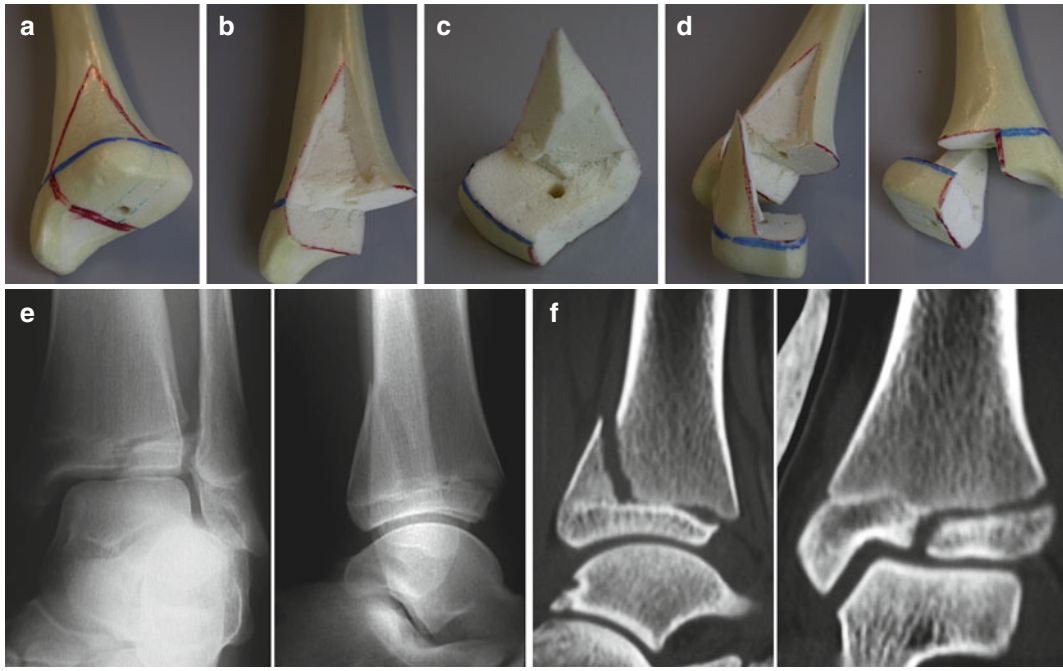

Fig. 3.24 Model illustration and radiological signs of the tri-plane fracture. (**a**) Schematic drawing on the bone model; (**b**) Proximal fragment from posterior; (**c**) Isolated distal fragment from anterior; (**d**) Relation of both fragments from anterior and posterior; (**e**) X-ray; (**f**) 2D CT scan reconstruction shows the fracture better than a 3D image

- Reduction of the fracture and retention are achieved using a MIP such as that using cannulated, self-drilling, self-tapping screws.
- The extremity is placed directly on the image intensifier.
- The leg must be turned so that the fracture can be seen exactly in an a.p. view.
- The screws must be placed exactly perpendicular to this plane. The adaptation of the fracture can easily be observed with the intensifier.

Foot Fractures

General considerations
- Fractures of the talus and the calcaneus are rare in childhood.
- The etiologies for these fractures are direct or axial injuries.
- Fractures of the metatarsals are frequent; the first and fifth rays in particular are involved.
- The first metatarsal may be injured proximally, either in the metaphysis or the proximal growth plate.
- Solitary fractures of the metatarsal diaphysis are usually undisplaced.
- Symptomatic accessory bones make diagnosis difficult.

Therapy
- The treatment of foot fractures is detailed in Table 3.37 and complications arising are listed in Table 3.38.

Appendix

Classification

There are numerous systems for classifying bone fractures: according to localization, displacement, and stability (Table 3.39); according to localization and involvement of the growth plate (Table 3.40); and the Salter–Harris/Aitken

system, which is one of the most popular. A summary of all this "sub–classification" for long bones represents the AO Pediatric Comprehensive Classification of Long-Bone Fractures (PCCF) (Figs. 3.25, 3.26, 3.27, and 3.28). Actual this is the best validated and comprehensive classification for paediatric long bones accepted worldwide. Behind this classification exists a so called Automatic Classifier System (AO-COIAC) with a database that allows documenting all fractures (including x-rays) with patient history/injury/treatment/complication and follow-up.

Table 3.37 The treatment of foot fractures

Fracture type	Therapy	Immobilization
Calcaneus	Undisplaced – non-weight-bearing cast Displaced – open reduction and screw or plate stabilization, non-weight-bearing cast	6–8 weeks
Talus	Undisplaced – non-weight-bearing cast Displaced – open reduction and screw or K-wire fixation, non-weight-bearing cast	6–8 weeks
Metatarsals	Undisplaced – non-weight-bearing cast Displaced – open reduction and screw or K-wire fixation, non-weight-bearing cast	6–8 weeks

Table 3.38 The complication of foot fractures

Fracture type	Complication
Calcaneus	Arthrosis, stiffness in the subtalar joint, pain
Talus	Necrosis, arthrosis of the ankle joint, stiffness in the ankle joints
Metatarsals	Non-union, pain

The AO Paediatric Comprehensive Classification of Long-Bone Fractures (PCCF)

"Research into the healing patterns of paediatric fractures assumes a common language that must be the prerequisite for comprehensive documentation as the basis for treatment and research".

The overall structure of the classification system is based on fracture location and morphology. The fracture localization is related to the four long bones and their respective segments and sub-segments. The morphology of the fracture is documented by a type-specific child code, a severity code, and an additional code for displacement of specific fractures.

Localization

The bones, and the segments within the bones follow a coding scheme similar to that in adults, but the identification of segments differs from that in adults. Malleolar fractures are coded as distal tibia fractures.

Table 3.39 Fracture classification according to bone segment, displacement, and stability

Stability	Localization		Therapy
	Diaphyseal	Articular	
	Metaphyseal		
Sufficiently stable for initial retention	Transverse fractures, with tolerance limits depending on age Or Oblique/spiral fractures of one bone of the lower leg or forearm	Non-displaced or minimally (<2 mm) displaced articular fractures	Immobilization with plaster in combination with cast wedging if necessary
Unstable fractures	All fully displaced fractures	Articular fractures with a gap >2 mm.	Reduction under anesthesia with either conservative (plaster) or operative stabilization

Table 3.40 Classification according to bone segment and growth plate involvement

Shaft fractures	*Diaphyseal*	*Stable*	Non displaced fractures without shortening
		Unstable	Displaced fractures with shortening or having the tendency for shortening
		Greenstick	Bowing fractures with complete fracture of one cortex and incomplete facture of the cortex of the contra lateral side
	Metaphyseal	Buckle	Compression of the metaphyseal cortex of one side
		Bowing	Greenstick fracture in the metaphysis
		Lig. avulsion	Ligament avulsion
Articular fracture	*Epiphyseal*	Aitken I	
		Salter–Harris I + II	
		Aitken II + III	
		Salter–Harris III + IV	
		Tillaux or two-plane fracture	In puberty by partially closed growth plate
	Metaphyseal	Flake fracture	"Normally" in combination with joint dislocation
		Bowing	Bony or cartilage avulsion

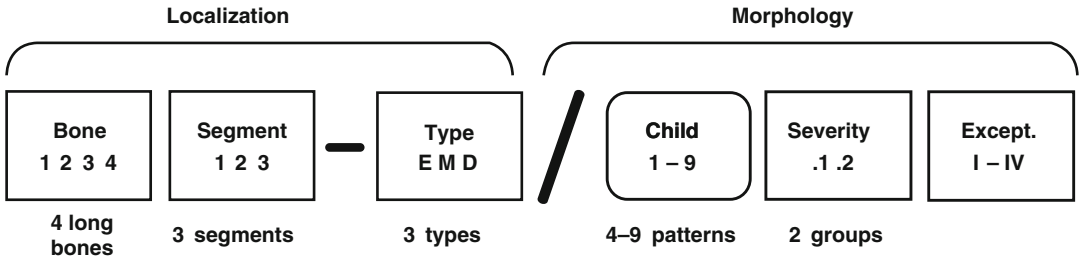

Fig. 3.25 Overall code of PCCF

For paediatric long bone fractures, the metaphysis is identified by a square whose side has the same length as the widest part of the physis in question. For the pairs of bones radius/ulna and tibia/fibula, both bones must be included in the square. Consequently, the three segments can be defined as:

Segment 1: Proximal: including sub segments epiphysis (E) and metaphysis (M)

Segment 2: Diaphysis (D)

Segment 3: Distal: including sub segments metaphysis (M) and epiphysis (E)

Epiphyseal fractures (E) involve the epiphysis and respective growth plates (physis), whereas the metaphyseal fractures (M) are identified through the position of the square (the centre of the fracture lines must be located in the square). This square definition is not applied to the proximal femur where metaphyseal fractures are located between the physis of the head and the intertrochanteric line.

Morphology

Child Code

Relevant paediatric fracture patterns, transformed into a "child code", are specific to one of the fracture localizations E, M, or D, and hence grouped accordingly. Internationally known and accepted child patterns are considered.

Severity

A grade of fracture severity distinguishes between simple (.1), and wedge or complex (.2).

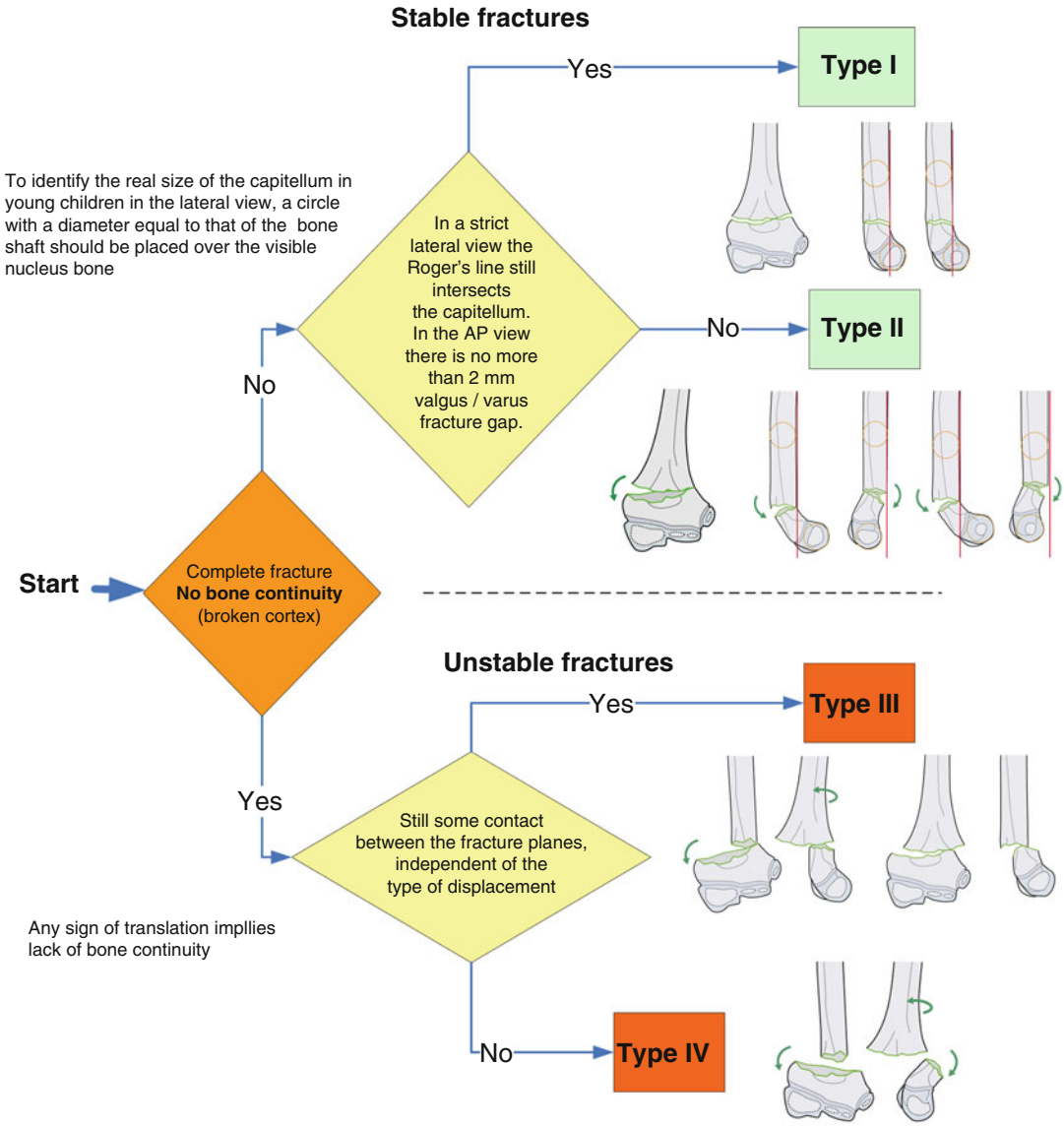

Fig. 3.26 Algorithm for supracondylar fractures

Fracture Displacement for Specific Fracture Localization

Supracondylar humerus fractures (code 13—M/3) are given an additional code regarding the grade of displacement at 4 levels (I to IV). The proposed algorithm is recommended.

Radial head fractures (21—M /2 or /3, or 21—E /1 or /2) are given an additional code (I–III) regarding the axial deviation and level of displacement.

Paired Bones

Except for the known Monteggia and Galeazzi lesions, when paired bones radius/ulna or tibia/fibula are fractured with the same child pattern (see child code in a next section), a single classification code should be used with the severity code being the worst of the two bones. When a single bone is fractured, a small letter describing that bone (i.e. "r", "u", "t" or "f") should be added after the segment code (e.g. a code "22u"

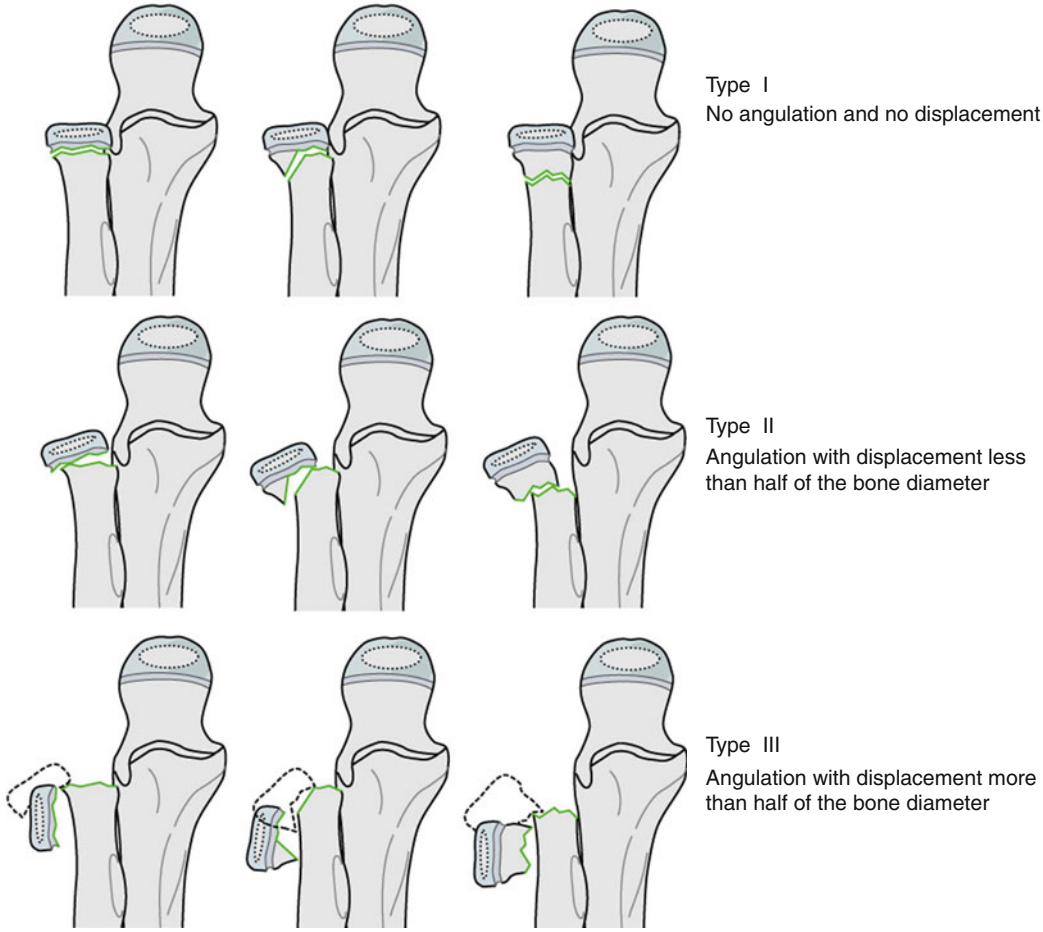

Fig. 3.27 Exception code for radial head/neck fractures

identifies an isolated diaphyseal fracture of the ulna).

When paired bones radius/ulna or tibia/fibula are fractured with different child patterns (e.g. a complete fracture of the radius and a bowing fracture of the ulna), each bone must be coded separately including the corresponding small letter (22r—D/5.1 & 22u—D/1.1).

Some further rules:

- Fractures of the apophysis are recognized as metaphyseal injuries.
- Transitional fractures with or without metaphyseal wedge are classified as epiphyseal fractures.

Ligament Avulsions

Intra- and extra-articular ligament avulsions are epiphyseal and metaphyseal injuries, respectively. The side of ligament avulsion fractures of the distal humerus and distal femur is indicated by the small letter "u" (ulnar/medial) or "r" (radial/lateral) for the humerus and by "t" (tibial/medial) or "f" (fibular/lateral) for the femur.

Femoral Neck Fractures

Epiphysiolysis and epiphysiolysis with a metaphyseal wedge are coded as normal Type E epiphyseal SH I and II fractures E /1 and E /2. Fractures of the femoral neck are coded as normal type M

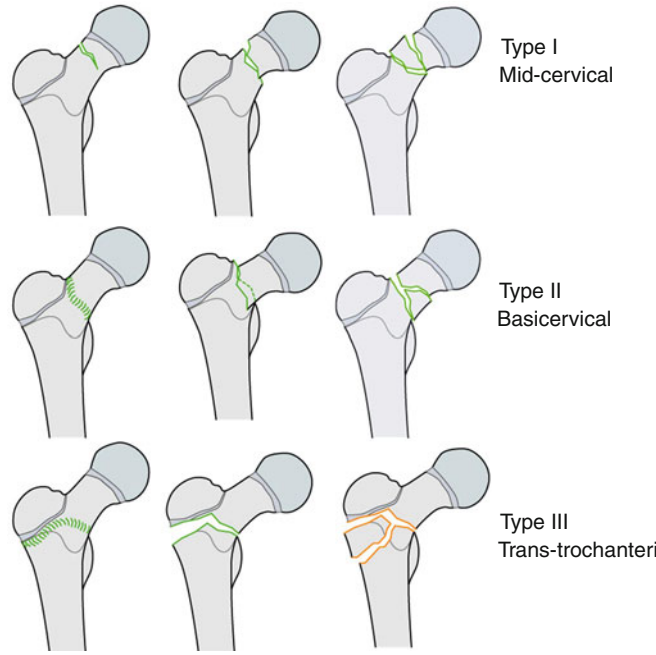

Fig. 3.28 Exception code for femoral neck fracture

metaphyseal fractures code I to III. The intertrochanteric line limits the metaphysis.

Discussion

Although fracture-healing in Childhood practically never represents (or better is) a problem we must nevertheless carefully plan every treatment. The seldom seen healing problems (including non-unions) are usually the result of an inadequate assessment and treatment. Therefore the considerations mentioned again and again in the article must be taken always into account.

The morphology, the localization, and the patients age are the main criteria for decision-making. Moreover socio-political and socio-economic aspects are growing influences on therapy.

As a consequence of that, besides the attempt at minimal restriction of mobility, therapy implies more and more surgical repair of fractures.

This trend toward surgical treatment in therapeutic strategy is based on the following considerations:

- Improved implant technology corresponding to the age of the patient
- The request for short hospitalisation
- The demand for early mobilisation and mobility
- Easier home care
- Less complications and better functional results
- Fewer costs

Furthermore, the same attention and carefulness in the treatment should be paid to children as to adults.

Our therapy should always be led by the following principle: The first treatment should be the definitive treatment; no second anaesthesia, manipulations, or therapy attempts.

Further Reading

Introduction

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The Geriatric Patient

4

Richard D. Southgate and Stephen L. Kates

Introduction

As the population ages, older adults will be involved in trauma. The number of geriatric trauma patients is rising as the elderly remain active later in life. While there is some lack of consensus about at what age a person becomes elderly, most studies define a patient as geriatric if his or her age is ≥65 years old [1]. According to the United States Census Bureau, 12.9 % of the population was older than 65 in 2009 and that is projected to grow to 20.1 % in 2050 (Fig. 4.1) [2]. The very elderly (>80 years of age) is the fastest growing segment of the US population, rising from 3 % at present to approximately 9 % 25 years from now [3].

Not only are the elderly becoming a larger part of the population, but they are often involved in trauma, which is the fifth leading cause of death in that age group [4]. Patients over the age of 65 represent 12 % of the population but 28 % of all fatal injuries in the United States [5]. In addition, they sustain a disproportionate number of fractures [6]. The lifetime prevalence of hip fractures is 1 in 3 in women and 1 in 12 in men [7]. In a study of a Medicare database, it was determined that between 1986 and 2005, the annual mean number of hip fractures was 957 per 100,000 in females and 414 per 100,000 for males [8]. One problem encountered in treating elderly trauma patients is that most traditional treatment protocols are designed for younger patients. The elderly differ in important ways from young and middle-aged adults. When caring for them, providers should take into account their special characteristics, such as increased risk of mortality, physiologic changes, and preexisting medical conditions/comorbidities [9].

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Mortality

The most important factor for the orthopaedic surgeon to consider when caring for elderly patients is their increased risk of mortality compared to younger age-cohorts. Increased mortality in elderly patients has been correlated with higher Injury Severity Score (ISS), lower Glasgow Coma Scale (GCS) score, as well as greater transfusion and fluid resuscitation requirements (Table 4.1) [10]. One review of 100 trauma patients aged ≥65 compared with 100 younger controls found that geriatric trauma patients were six times as likely to die as their younger counterparts despite having similar ISS scores. Mortality in the group aged 65 and older was 17 % compared to 3 % in controls [11]. The presence of shock was found to increase mortality significantly among older patients [11]. Hospital and the intensive care unit (ICU) length of stay as well as costs were also higher in the elderly group. The mechanisms of injury in the geriatric

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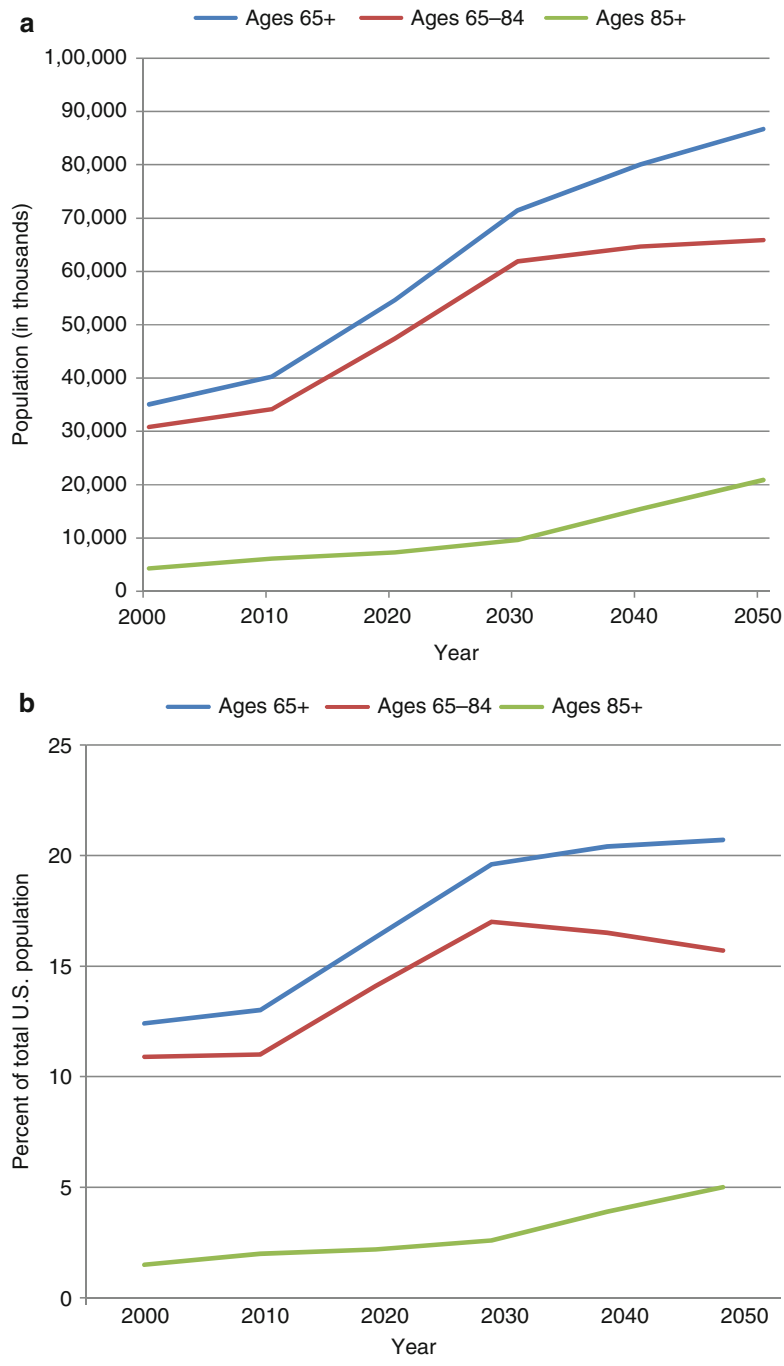

Fig. 4.1 (**a**) Absolute growth of elderly population in the United States, subdivided by age group. From 2000 to 2050, the population of persons over the age of 65 in the United States is expected to increase from 35 million to 87 million (Source of data: United States Census Bureau). (**b**) Growth of proportion of total US population classified as elderly 2000–2050. The population of patients over age 85 is the fastest growing age group in the United States (Source of data: United States Census Bureau)

Table 4.1 Factors associated with increased mortality in geriatric trauma patients

Higher ISS
Lower GCS
Greater transfusion and fluid resuscitation requirements
More advanced age
Increased number of preexisting medical conditions

patients were more likely to be a simple fall or pedestrian-car injury versus motor vehicle accidents (MVA), gunshot wounds, or crush injuries for younger patients [11]. Similar findings have been reported found in other studies, including one that demonstrated 42 % mortality in patients > [greater than] 65 years of age compared to

20 % in younger patients admitted to a level one trauma center. The same study also reported a mortality rate of nearly 50 % in patients >75 years of age [9].

In a review of 7,798 trauma patients, the effect of comorbidities was studied. Despite having similar ISS and GCS, those with preexisting comorbidities had higher mortality; 9.2 % mortality was seen in those with comorbidities compared to 3.2 % in those without [12]. Mortality increased with the number of preexisting diseases: 15.5 % in those with ≥2 and 24.9 % in those with ≥3 comorbidities. The highest mortality was seen in those with renal disease, malignancy, and cardiac disease [12]. In a review of the Pennsylvania Trauma Systems Foundation database of 33,781 geriatric patients, an overall mortality rate of 7.6 % was reported [13]. The investigators also found that, for each 1 year increase in age beyond age 65, the odds of dying after geriatric trauma increased by 6.8 % [13]. Furthermore, the same investigators demonstrated that when controlling for vital signs, GCS, and ISS, comorbidities (hepatic disease, renal disease, cancer, congestive heart failure, chronic obstructive pulmonary disease, and chronic steroid in descending order of association with mortality) had a significant effect on mortality on these older patients [13]. Notably, warfarin use had no effect on odds of death. Preexisting medical conditions increase complications and contribute to both early and late mortality in the geriatric trauma patient [2].

Clement et al. demonstrated that early mortality is correlated with more severe injuries (higher ISS). Late mortality, occurring more than 13 days after injury seen in patients with lower ISS, was most often due to medical complications [14]. Patients over the age of 65 and those with ISS less than 16 were at higher risk for late mortality: 33.3 % versus 12 % in younger patients with similar injury scores [14].

Increased mortality is also seen when looking at specific injuries in the elderly. In a review of 234 pelvic fractures treated at a single trauma center, investigators found that older patients were 2.8 times more likely to receive blood and required more blood transfusions (7.5 vs. 5.0 units) than younger pelvic trauma patients [15]. Lateral compression fractures (LC) occurred 4.6 times more frequently than anterior-posterior compression (APC) fractures in the elderly. In addition, the lateral compression fractures were minor in older patients (98 % were grades LC 1 or 2), but these patients were four times more likely to require blood. Overall, older patients had a higher mortality rate even after adjusting for ISS [15].

Most published data on mortality after geriatric fractures pertains to hip fractures. The mortality after hip fractures that are surgically treated is 9 % at 30 days, 19 % at 90 days, and 30 % at 12 months [16]. Other studies have demonstrated a 20 % or greater mortality within 1 year of sustaining a hip fracture in geriatric patients [17, 18]. The risk of mortality is highest in the first year after the fracture [19]. This increase in risk of mortality decreases over the first 2 years but never returns to the baseline rate [20]. Surgical treatment of hip fractures is associated with a 4 % mortality risk [21]. Age at the time of fracture is known to be a significant risk factor. In one prospective series of 1,109 patients with hip fracture, mortality risk was found to increase 4 % for each additional year of age [22].

In those over age 60, mortality is also increased after other major types of osteoporotic fractures, including those of the vertebrae, pelvis, distal femur, multiple ribs, and proximal humerus [23]. Even seemingly benign injuries can have significant effects on geriatric patients. One study found that, in this population, each additional rib fracture increases the risk of pneumonia by 27 % and the risk of mortality by 19 % [24]. It is likely that deaths from traumatic injury are underreported as these patients often die from complications that are recorded as the cause of death instead of the true cause, trauma [6].

Initial Triage

Considering the increased mortality associated with trauma in older patients, extra vigilance should be maintained when treating them, even prior to arrival at the hospital. Under-triage is common and harmful to geriatric patients. A review of the Florida trauma system found that

under-triage of patients older than 55 years of age occurs 71 % of the time [25]. When triaging patients in the field, paramedics and emergency medical technicians used a trauma scorecard consisting of physiologic criteria (systolic blood pressure less than 90 mmHg, respiratory rate less than 10 or greater than 29 bpm, GCS less than 12) as well as anatomic and mechanistic criteria (second- and third-degree burns greater than 15 % body surface area, paralysis, ejection from vehicle, amputation proximate to wrist or ankle, penetrating injury to the head, neck, chest, abdomen, or groin) to determine whether a patient should be classified as a trauma alert, which occurred if any of the eight criteria were met. Patients classified in this way were taken to a trauma center for immediate care by a dedicated trauma team. This under-triage is thought to occur because older patients often do not exhibit hypotension or tachycardia in response to a significant trauma.

When initially evaluating geriatric trauma patients, it is important to recognize that normal presenting vital signs may not accurately reflect injury severity [2, 26]. There are two reasons for this situation. Polypharmacy can significantly alter the elderly patient's response to injury: beta blockers, for example, can mask hypotension and tachycardia [27]. The second reason is the frequent presence of comorbidities. Preexisting hypertension in geriatric patients should be considered. A normal blood pressure for a younger adult may represent hypotension in the older geriatric patient [1].

Another potential reason for under-triage is the mechanism of injury in the elderly. Triage criteria often fail to identify cases of major trauma from falls [1]. Treating physicians may also fail to recognize the significant trauma that may result from a fall in a geriatric patient. A review of 26,025 patients from a single metropolitan area found being elderly or female to be two of the most significant risk factors associated with being under-triaged [28].

Because of high under-triage rates, some centers have added old age as a criterion for trauma team activation. In an effort to avoid missing patients who might benefit from activation of the trauma system, Demetriades et al. suggested trauma team activation for all patients more than 70 years old [29]. In a follow-up study, the same group created a new protocol for trauma team activation: age less than 70 years, systolic blood pressure less than 90 mmHg, heart rate less than 120 bpm, respiratory rate less than 10 or greater than 29 bpm, unresponsive, or emergency department physician judgment. In addition to the new trauma activation protocol, the patients received early invasive monitoring and resuscitation, as well as early ICU admission. The authors studied 336 patients with ISS >15 and found a decrease in mortality from 53.8 to 34.2 % [30]. Another group uses patient age >55 as a criterion for considering transportation to a trauma center [3]. Further support for the move to consider age as an indication to activate a trauma team in addition to standard hemodynamic and mechanistic criteria is research which has shown that older patients still have a relatively high risk of death even in the absence of physiologic abnormality and should thus be treated more aggressively [9, 26]. Further research in this area is needed to establish the inclusion of advanced age as a criterion for trauma team activation.

The sentiment that trauma centers have significantly better outcomes than community care hospitals in treating older injured patients has been borne out in the literature. In a retrospective review of the very elderly (≥80), Meldon et al. found that patients taken to level II trauma centers experienced less mortality (5.2 %) than those taken to community care hospitals (9.9 %) [3]. Mortality at level I centers was higher (24 %), but these hospitals also cared for patients who were injured more seriously than the other groups. For very old patients, location of treatment for geriatric trauma patients is an important factor.

Mechanism of Injury

One of the largest and most comprehensive studies examining the mechanisms and outcomes of serious injury in geriatric trauma patients was performed by Richmond et al. [31]. They queried the Pennsylvania Trauma Systems Foundation database for seriously injured patients and excluded

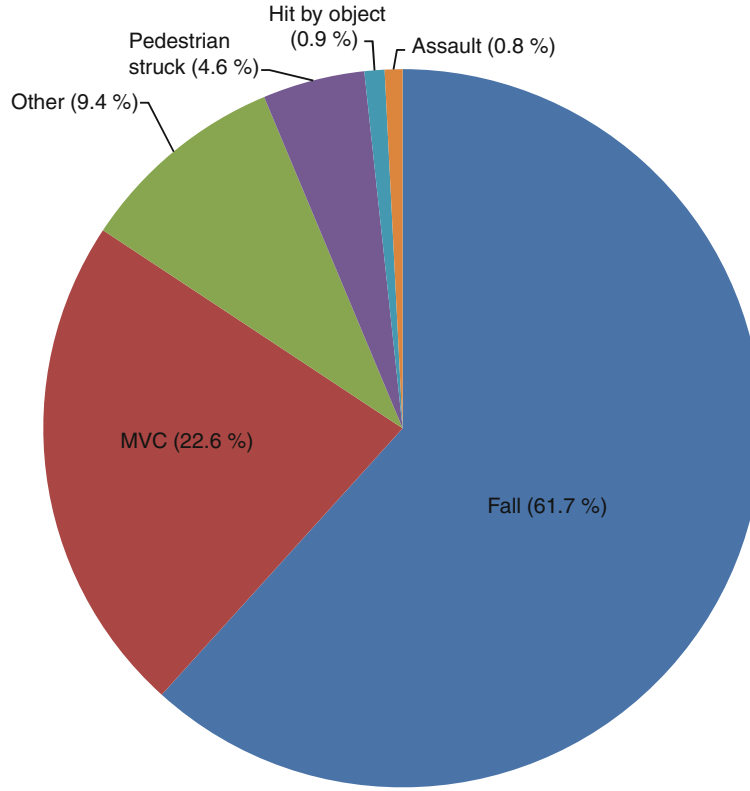

Fig. 4.2 Mechanisms of injury in trauma patients older than 65 years of age. Falls and motor vehicle collisions (MVC) are the most common mechanisms of traumatic injury in elderly patients, accounting for more than 80 % of trauma cases in one series [31]

Table 4.2 Relative frequency and fatality rate of various mechanisms of traumatic injury in geriatric patients [32]

Mechanism	Relative frequency (%)	Fatality rate (%)
Fall	40.6	11.7
MVC	28.2	20.7
Pedestrian struck	10.0	32.6
Other	7.0	13.8
GSW	5.5	52.1
Stab wound	2.6	17.3
MCC	0.4	11.8
Unknown	0.3	19.0

Falls and motor vehicle collisions (MVC) are the most common mechanisms of injury, but gunshot wounds (GSW) and pedestrian struck by motor vehicle are the most fatal injuries. Other abbreviations: *MCC* motorcycle collision

those who sustained isolated hip fractures after falls from standing height. A total of 38,707 patients over the age of 65 years over a 10-year period were included in the study. Mechanisms of injury were, in descending order of rank, as follows: falls (61.7 %), motor vehicle collision (22.6 %), others (9.4 %), pedestrian (4.6 %), hit by object (0.9 %), and assault (0.8 %) (Fig. 4.2) [31]. As patients get older, falls become the most common mechanism, responsible for 49.2 % of traumas in the 65–74 age group and 81.1 % in the above 85 age group. Extremities and the pelvic girdle were the most injured body regions in 47.4 % of cases [31]. Another multicenter study found the cause of injury in patients ≥65 years of age to be falls (40.6 %), motor vehicle collision (20.2 %), pedestrian struck (10 %), others (7 %), gunshot wound (5.5 %), stab wound (2.6 %), motorcycle collision (0.4 %), and unknown (0.3 %) (Table 4.2) [32]. Physicians should also be cognizant of elder abuse, which has an estimated prevalence of 32 cases per 1,000 persons [33]. Unlike child abuse, there are no fracture patterns that are considered pathognomonic for elder abuse. It should be suspected when there are ambiguous, inconsistent stories that do not match with the presenting injury.

Even though falls from a standing height often are relatively benign in other population groups, they are significant in the elderly, as they can cause multiple injuries which may result in an ICU admission similar to a higher-energy mechanism. Older adults can sustain multiple injuries from low-energy trauma [34]. The reason for the increased incidence of falls in the elderly is multifactorial. Physiologic changes take place with aging, including decreased visual, auditory, proprioceptive, and vestibular inputs, which combine with diminished reaction times, unsteady gait, and loss of strength and coordination to contribute to the increased likelihood of falls. This may be compounded by cardiac dysrhythmias and orthostatic hypotension of various etiologies, including polypharmacy [1].

Finally, it is important not only to think about the mechanism of injury but also the reason that the patient became injured. An older individual injured in a motor vehicle collision may have had a transient ischemic attack, stroke, or arrhythmia. Other causes to consider include hypoglycemia or dementia (which interferes with the ability to recognize and avoid road hazards). The underlying reason could be more benign, such as presbyacusis, presbyopia, or slowed reaction time. The cause of the trauma can not only significantly alter the patient's immediately course of care but could also prevent future injuries.

In summary, there are a number of possible mechanisms for injury in a geriatric patient with falls being the most common. However, because the older segment of the population is staying active, an increasing number of injuries from all mechanisms are expected [2].

Physiology

When considering physiology, there are several important differences between old and young adults. Older adults have reduced physiologic reserves and diminished compensatory mechanisms and are therefore less able to deal with the added stress presented by trauma. This narrow physiologic tolerance and the restricted reserves should be expected when managing geriatric trauma patients [1]. A patient's chronologic age may not equal their physiologic age, which is modulated by their preexisting conditions (Table 4.3). What makes management of these patients difficult is that their comorbidities may be unknown at the time of presentation.

Table 4.3 Common comorbidities, or preexisting conditions, that are frequently encountered in geriatric patients

Cardiovascular	Coronary artery disease, congestive heart failure, hypertension, peripheral vascular disease
Pulmonary	Shunting, chronic obstructive pulmonary disease
Renal	Decreased renal function
Gastrointestinal	Malnutrition
Central nervous system	Cerebral atrophy, dementia, cervical stenosis
Dermatologic	Thinner subcutaneous tissue
Endocrine	Diabetes mellitus
Musculoskeletal	Osteoporosis, previous orthopaedic hardware, or implants

A thorough understanding of these will assist in management of these patients and can potentially avoid or minimize the effects of certain complications

Aging of the cardiovascular and respiratory systems reduces the older patient's ability to respond to hypoxia and shock [1]. The heart of a geriatric patient, often affected by coronary artery disease and congestive heart failure, accommodates poorly to hypovolemia. A low cardiac rate aggravates decreased preload from hypovolemia, resulting in decreased cardiac output. This in turn results in myocardial ischemia and an additional drop in cardiac output. Older patients have increases in resting ventilation/perfusion mismatch resulting in pulmonary shunting, which make them more susceptible to developing hypoxia. Many also have chronic pulmonary obstructive disease, which can complicate their perioperative management.

Aging is accompanied by diminished renal function and decreased creatinine clearance, which is masked by a serum creatinine that is falsely normal because their muscle mass is lost. It is not uncommon for older patients to have some degree of malnutrition, which can impair healing and recovery. Their dura mater becomes adherent to the cranium, eliminating the epidural

space [1]. With concomitant age-related brain atrophy, the bridging veins are more susceptible to injury. Consequently, epidural bleeds are less frequent but subdural bleeds are more common in the older population. Preexisting cervical stenosis places the patient at significant risk for central cord syndrome. Their integument has impaired thermoregulation and loss of subcutaneous cushion, the latter of which can lead to more degloving injuries. Both diabetes and peripheral vascular disease complicate wound healing and predispose to infection and nonunion [6]

The musculoskeletal system, affected by muscle atrophy and osteoporosis, is often subject to more severe injuries even when less kinetic energy is imparted on the limb. When a previous orthopaedic implant is present such as a joint replacement or other implant, it almost always alters the fracture pattern and treatment for a given injury. The effect of osteoporosis in the care of the geriatric patient cannot be overemphasized.

Osteoporosis

Osteoporosis is a condition of decreased bone mineral density resulting from an imbalance of bone formation and resorption. Osteoporosis is divided into primary and secondary osteoporosis. Primary osteoporosis is the loss of bone mass associated with the aging process, since peak bone density is attained in young adulthood and decreases steadily thereafter. Secondary osteoporosis is due to a variety of causes, including insufficient intake of calcium or vitamin D, gastrointestinal malabsorption, metabolic derangements (hyperparathyroidism, hypo- or hyperthyroidism, Cushing's syndrome, renal pathology), medications (anticonvulsant drugs, prednisone), and deficiencies of gonadal hormone (estrogen deficiency, low testosterone levels) (Table 4.4) [35]. The majority of these causes can be detected with laboratory tests [35]. Recommended tests include a basic metabolic panel with serum calcium levels, a 24-h urine calcium measurement, a 25-hydroxy-vitamin D level, as well as thyroid-stimulating hormone and parathyroid hormone levels. Other tests can be ordered as indicated based on history and physical examination. In addition, all patients with fragility fractures should be considered for dual-energy X-ray absorptiometry (DXA) scans and prescriptions for calcium and vitamin D supplements [35]. Bukata et al. stated that, when caring for patients with geriatric fractures, the identification of potentially correctable etiologies of impaired bone quality should be sought [35]. Failure to do so may impair recovery and places the patient at increased risk for future fractures.

Table 4.4 Common causes of secondary osteoporosis that should be considered in evaluating patients with fragility fractures [35]

Insufficient intake of calcium
Insufficient intake of vitamin D
Gastrointestinal malabsorption
Hyperparathyroidism
Hypothyroidism
Hyperthyroidism
Cushing's syndrome
Renal pathology
Medications (anticonvulsant drugs, prednisone)
Estrogen or testosterone deficiency

Osteoporosis is common, affecting 45 % of women and 15 % of men over the age of 50 [36, 37]. The lifetime prevalence of osteoporosis is 13–18 % in women and 3–6 % in men [38]. In postmenopausal women with fragility fractures, the prevalence is 30 % [39]. This number is higher for lower-risk patients such as men and premenopausal women [40]. Known risk factors for osteoporosis include female gender, multiparity, BMI < [less than] 18.5 kg/m^2, smoking, excessive alcohol consumption, certain medications, and northern European or Asian ancestry.

Since the architecture of the bone is compromised by osteoporosis, fractures in osteoporotic bone may display a high-energy pattern and be challenging to treat in spite of being a low-energy mechanism [35]. In addition, the altered mechanics of osteoporotic bone make fixation difficult. Failure of hardware in osteoporotic bone typically occurs at the bone-implant interface, resulting in cutout, fracture subsidence, or pull off of the plate. Failure happens when the load transmitted at the bone-implant interface exceeds the

diminished strain tolerance of osteoporotic bone [37]. Based on a cadaveric study on pullout strength in human tibiae whose bone density was assessed with CT scanning, Seebeck et al. demonstrated that decreased cortical thickness and loss of trabecular bone make it difficult to obtain good purchase with standard hardware in osteoporotic patients [41]. Consequently, constructs that maximize surface contact area between the hardware and the bone are preferred [35]. Examples include hardware with locking screws, screws with larger diameter, and bicortical screw purchase. Load-bearing, as opposed to load sharing, devices are preferred in these patients [35]. In addition to proper implant selection, the treating surgeon should perform thorough preoperative planning and accurate fracture reduction when treating these patients.

A number of studies have shown that orthopaedic surgeons have not been proactive in identifying patients who may benefit from treatment of their osteoporosis. In a study of 1,162 older female patients who sustained distal radius fractures, only 24 % of patients underwent either diagnostic evaluation or treatment for osteoporosis. In addition, those who were older were significantly less likely to be treated appropriately with antiresorptive agents [36]. A similar study was performed to assess management of osteoporosis in older women who sustained low-energy femoral neck fractures and found calcium supplements and antiresorptive medications to be under-prescribed at the time of discharge from the hospital [38]. A similar, more recent study found a 20 % treatment rate with osteoporosis in older patients who sustained distal radius fractures [40]. Orthopaedic surgeons are often the first to see the patient with fragility fractures. This pattern, combined with the fact that treatment options exist that are effective in reducing the rate of additional fractures, provides an additional reason for the orthopaedist to initiate the evaluation for osteoporosis or metabolic bone disorders and either commence treatment or refer the patient to a provider who can do so [6]. It has been suggested to assess and treat patients with fragility fractures or provide referral for osteoporosis care. One center reported a >95 % rate in successful diagnosis, treatment, or referral after implementation of such a program, which involved a team consisting of a dedicated coordinator supported by surgeons, residents, allied health-care professionals, and administrative staff [42]. Administration of bisphosphonates, which are first-line agents in the treatment of osteoporosis, is important because they have been shown to decrease fracture rates [6].

Secondary Fracture Prevention

In addition to selecting the appropriate procedure to perform, the orthopaedic surgeon should also start patients on calcium and vitamin D supplementation to correct any preexisting vitamin D deficiency and optimize fracture healing in osteoporotic patients. In a study of 954 patients at metabolic bone clinics, 73–89 % were found to have levels of vitamin D below the normal range (32 ng/mL or 80 nmol/L). In those with hip fractures, this figure was 84–96 % [43]. When vitamin D levels fall below 10 ng/mL (25 nmol/L), the patient is at risk for secondary hyperparathyroidism, which can further complicate bone metabolism [44]. There is mounting evidence that all patients with fragility fractures should have their vitamin D levels normalized, but current recommendations are under review [35]. One group of authors made the recommendation that orthopaedists should correct 25-hydroxy-vitamin D levels to more than 32 ng/mL, as this is the level at which PTH secretion normalizes [45].

There are two forms of vitamin D supplementation: ergocalciferol (vitamin D2), which is derived from plant and yeast sources, and cholecalciferol (vitamin D3), which is derived from animal sources and produced in the skin [35]. Numerous protocols exist for vitamin D supplementation. Bukata et al. suggest administering 50,000 IU ergocalciferol weekly for a duration that depends on baseline vitamin D levels: over 5–8 weeks if 20–30 ng/mL, 16 doses if 10–19 ng/mL, and 24 doses if less than 10 ng/mL [35]. This is followed by daily cholecalciferol (2,000 IU) in addition to vitamin D contained in their multivitamin or calcium supplements as long-term

therapy [35]. With normalized vitamin D levels and bone metabolism optimized, the patient is at a reduced risk for fragility fractures.

Prevention of falls represents the other aspect of secondary prevention of fractures. Falls cause 90 % of fractures of the forearm, hip, and pelvis in geriatric patients [6]. Risk factors for falling include the use of sedatives (benzodiazepines, phenothiazines, antidepressants), cognitive or visual impairment, lower extremity disability, foot problems, balance or gait abnormalities, neurologic conditions, and use of an assistive device for ambulation [46, 47]. Strategies to reduce the risk of falls include ensuring that the patient has vision correction, proper shoe wear, discontinuation of excessively sedating medications, and modification of the home environment [48]. Examples of the latter include providing good lighting throughout the home, lowering beds, installing carpet over hard floors, eliminating throw rugs and thick carpets, and providing grab bars in the bathroom [49]. Unfortunately, even with the best efforts at prevention, fragility fractures still occur. When they present to the hospital emergency department, there are several steps that can be taken to provide care that is both appropriate and expedient.

Interdisciplinary Comanagement

Advances in medical and anesthetic management have permitted less healthy geriatric patients to undergo orthopaedic surgical procedures successfully that may have been contraindicated in the past because of preexisting conditions [6]. When interviewing these patients, it is important to determine not just their past medical histories but to inquire about which medications they are currently taking, including antihypertensives and anticoagulants. Beta blockers should not be discontinued, as this can trigger rebound hypertension. The management of anticoagulants should be discussed with the consulting medicine provider. Reversal of warfarin (Coumadin) should be done with oral vitamin K or fresh-frozen plasma or while waiting for hepatic synthesis of clotting factors; the international normalized ratio (INR) should be ≤ [less than or equal to] 1.5 before the patient is taken to the OR [35]. Patients on clopidogrel (Plavix) or other platelet inhibitors should not receive spinal or epidural anesthesia but may undergo early surgery under general anesthesia [35]. Many patients who are on clopidogrel after placement of drug-eluting stents are at increased risk for stent thrombosis if they discontinue the drug. Therefore, risks and benefits of operating on a patient under the effect of clopidogrel must be balanced with platelet transfusions. Similar considerations should be made with respect to newer anticoagulants such as dabigatran (Pradaxa) and rivaroxaban (Xarelto), as these medications have no direct reversal agents and take 1–2 days to be eliminated from the body. Medications to avoid include nonsteroidal anti-inflammatory drugs (NSAIDs, as they impair bone healing and kidney function) centrally acting antihistamines, meperidine, most antiemetics, benzodiazepines, H2 (histamine) receptor antagonists, and anticholinergics [35, 50].

Most patients benefit from medical evaluation and optimization prior to surgery, as doing so will ultimately lead to decreased time to surgery and length of stay [50, 51]. Most have comorbidities and are at increased risk for adverse outcomes and postoperative complications. Comorbidities increase the risk of functional decline and death [19, 50]. Geriatricians are trained to address these comorbidities and manage potentially adverse outcomes and complications, thereby helping to maximize outcomes [50]. The medicine specialists should coordinate overall medical care of these older patients, both preoperatively and postoperatively [50].

The comanagement model of orthopaedics and geriatrics, characterized by the comanagement of the patient by geriatricians and orthopaedists who share responsibility throughout the patient's hospital stay, was developed in England in the 1950s [50, 52]. It has been shown to reduce complications, length of stay, readmission rate, cost of care, and mortality, as well as lead to better function, higher levels of patient, and provider satisfaction [50]. In-hospital mortality rates for comanaged hip fractures have been shown to range from 0.6 to 11 % [50]. Under this model,

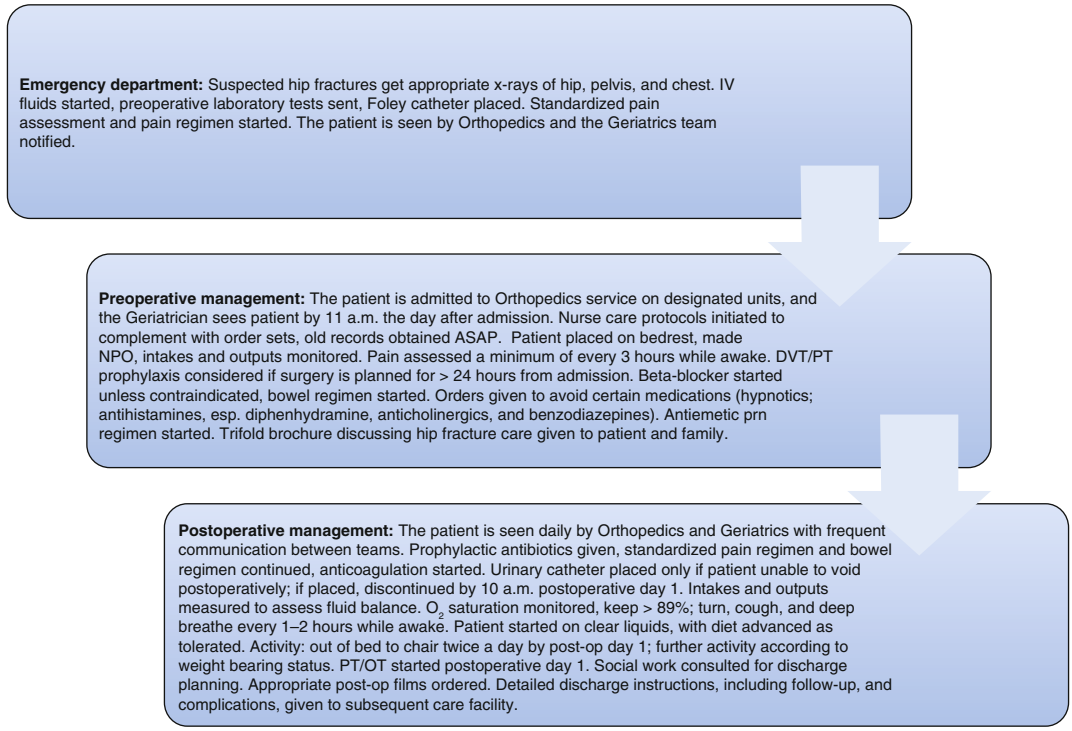

Fig. 4.3 Standardized protocol for comanagement of hip fractures patients in the Geriatric Fracture Center at Highland Hospital, University of Rochester Medical Center, Rochester, NY [50]

Table 4.5 Five principles of management at the geriatric fracture center [50]

1.	Most patients will benefit from surgical management of their fracture
2.	The shorter the delay to surgery, the less time to develop iatrogenic complications
3.	Comanagement with frequent communication avoids iatrogenesis
4.	Standardized protocols decrease unwarranted variability in patient care
5.	Discharge planning begins immediately, at the time of admission

Highland Hospital; University of Rochester Medical Center; Rochester, NY

each of the two specialists sees the patient, writes his or her own orders, and communicates with the other specialist on a daily basis (Fig. 4.3, Table 4.5) [50]. Frequent communication between medical and surgical providers reinforces the rationale between treatment decisions for the patient and provides continuous education for the providers [50]. An interdisciplinary team of health-care professionals should provide support for the patient and the admitting physicians [52].

In general, this model has resulted in reduced short-term mortality and complications and increased 1-year survival compared to traditional models where only orthopaedic surgeons are responsible for the management of the patient [52]. However, implementing such a program requires considerable effort on the part of the physicians as well as administrative support and strong leadership for continuous monitoring and improvement of the model once it is implemented. Additional studies are needed to evaluate the model's cost-effectiveness and long-term outcomes as well as its applicability to lower volume hospitals. It is believed that approximately 100 cases per year are needed to develop sufficient expertise in managing these patients [53]. The limited number of geriatricians combined with good availability of hospitalists implies that hospitalist comanagement

will become an important variation of the geriatric comanagement model.

Another point regarding consultation is important—the need for a cardiology consultation. This decision should be made by a geriatrician or hospitalist. In the case of a complex cardiac condition, these consultations represent a common reason for delay of fixation of fractures. Some authors feel that these consultations are usually unnecessary in patients with geriatric fractures [35].

Other salient points on perioperative management of geriatric fracture patients are worth mentioning. Dehydration is almost always present on admission, so immediate hydration with normal saline and administration of red blood cells as indicated preoperatively will help minimize the risk of hypotension upon induction of anesthesia [35]. Polypharmacy is common in these patients. Harmful or unneeded medications should be discontinued by the consulting medical provider while the patient is admitted [35].

Familiarity with geriatric patients on the anesthesiologist's part is important to ensure safe and efficient anesthetic care. The American Society of Anesthesiologists classification of surgical risk is useful to assess preoperative risk secondary to comorbidities [6]. Special considerations that anesthesiologists must take into account when caring for geriatric patients is that they are at significant risk for aspiration pneumonia and they do not tolerate excessive hypotension. Most orthopaedic procedures may be performed with either regional or general anesthesia. Studies have shown no difference in short-term or long-term mortality or functional treatment in hip fractures performed under spinal or general anesthesia [54, 55]. Efficacy of regional anesthesia (spinal/epidural) as prophylaxis against deep vein thrombosis and pulmonary embolism has been previously demonstrated [56].

An important principle in preoperative management of geriatric fractures is to provide the patient with a prompt work-up so as to minimize delays in surgery. This minimizes the length of bed rest for the patient, which is associated with venous thromboembolism, skin breakdown, pulmonary decompensation, delirium, and infection [50]. Additionally, surgical delays have been shown to affect the mortality. Delays of more than 48 h have been shown to increase 30-day mortality by 17 % in an analysis of 18,209 Medicare patients treated for hip fractures [57]. In a published model of geriatric fracture care, the authors suggest that surgical cases should be treated as urgent but not emergent and should only proceed to the operating room once the patient is optimized for surgery [50]. In a subsequent publication by the same group, the average time from admission to the operating room was <24 h [19]. Another way to expedite care is admission directly from a nursing home or assisted-living center to the hospital floor. This can eliminate delays in the emergency department and may reduce time to surgery [50]. Other authors have found that having a standardized pathway for geriatric fractures that are either directly admitted or present to the emergency department leads to a shorter length of stay, lower mortality, both in-hospital and 1-year mortality [58].

Surgical Management Strategies

When determining how a patient's orthopaedic injuries are to be treated, it is important to obtain a detailed history of his or her functional status, including what if any assistive devices are used for ambulation at baseline, how independent the patient is with activities of daily living, where the patient lives, with whom, how many floors he or she has they have in his or her their residence and how many steps to enter, and whether a first-floor living situation can be arranged. The patient's medical and cognitive histories are also important because the information contained in these histories has an impact on surgical options and potential for rehabilitation [6]. Given the multitude of factors and complexity of decision-making related to patient care, treatment recommendations should be made by the surgeon after an informed discussion with the patient, his or her family members, and other consulting providers. These are summarized in Table 4.6 and described in more detail later in this section as well as in their respective chapters throughout the rest of this text.

Table 4.6 Management options for common osteoporotic fractures [35]

Fracture location	Character	Options	Comments
Femoral neck	Nondisplaced	Three cannulated screws	Allow impaction with weight-bearing, stabilizing the fracture, expect some shortening to occur
		Sliding hip screw with antirotation pin	
	Displaced	Hemi or total arthroplasty	Physiologically younger, active patients without dementia may qualify for total hip arthroplasty
			Physiologically older, low-demand patients and all dementia patients receive hip hemiarthroplasty
			Controversy as to whether cemented or press-fit stems are better; limited evidence that cemented stems are a slightly better choice
Pertrochanteric	Stable	Sliding hip screw	Maintain tip-apex distance of less than 27 mm to prevent cutout
	Unstable[a]	Trochanteric entry nail	Position implant in the center of femoral head to avoid failure of fixation
			Use long nail for subtrochanteric fractures
			Plating associated with increased rate of mechanical failure and nonunion
Distal femur		95° condylar blade plate	Inexpensive but unforgiving implant
		Distal femoral locking plate	Can be placed percutaneously, expensive, requires intraoperative fluoroscopy
		Retrograde intramedullary nailing	Limited distal fixation, can be used in periprosthetic fractures if the implant has an open box, expensive, requires intraoperative fluoroscopy
Proximal humerus	Simple, minimally displaced	Nonoperative management	Satisfactory outcomes in most cases but radiographs less appealing
	Simple, displaced	Locked plate	Lack of medial buttress can result in varus collapse and screw penetration of humeral head, requires intraoperative fluoroscopy, high complication rate
	3 and 4 part	Total or reverse shoulder arthroplasty	Inconsistent levels of functional recovery, limited long-term results with reverse shoulder arthroplasty
Distal radius	Nondisplaced	Nonoperative management	Shown to have equal outcomes in elderly to plating
	Displaced	Volar locked plating	Long screws risk dorsal penetration and attritional rupture of extensor tendons
Vertebral compression		Nonoperative management	Pain control, activity modifications, bracing for 1–2 months
		Kyphoplasty or vertebroplasty with PMMA	Some studies have shown no difference compared to nonoperative management at 1 year

[a]Reverse obliquity, subtrochanteric extension, lateral wall comminution, loss of posteromedial cortex

Surgical intervention for hip fractures is warranted in vast majority of patients, even those who are nonambulatory, as they benefit from pain relief [50]. Only patients who have extremely limited life expectancies should be offered nonoperative care but only after a discussion between the providers and the patient. While there are numerous considerations that must be taken into account when treating elderly patients with fractures, they are all important and merit

extensive discussion with the patient and the patient's family so that informed decisions can be made about the plan of care.

In general, early stabilization of orthopaedic injuries permits early mobilization and reduces complications associated with prolonged immobility. However, there are some exceptions to this when dealing with the injured geriatric patient, as described in the next section.

Damage Control Orthopaedics

As recently as the middle of the twentieth century, early definitive fracture stabilization was not commonly performed, as physicians believed that polytraumatized patients were too unstable to survive surgical intervention [59]. Estimates of a patient's ability to withstand surgery were made on a clinical basis alone, as advanced laboratory tests and ICU monitoring equipment were not available. During the 1980s, however, a paradigm shift occurred and early total care (ETC) was performed, as multiply injured patients were then believed to be too unstable not to have fracture stabilization. This usually entailed fracture management within 24 h of presentation. Several benefits of ETC were touted, including pain reduction, early mobilization of the patient, and a reduction in thromboembolic complications [59]. However, because early total care was associated with an unacceptably high number of complications in those with a very ISS, the universal doctrine of ETC for every patient began to be questioned.

In severely injured patients, treatment according to the principles of "damage control orthopaedics" (DCO) became the standard of care in the 1990s. DCO employs temporizing methods of fracture treatment, such as application of external fixators, allowing for delayed definitive fixation once the patient was stabilized (Fig. 4.4). Under the principles of DCO, primary procedures with duration greater than 6 h and major surgical procedures on post-injury days 2 through 4 should be avoided, if at all possible [59]. Damage control orthopaedics consists of three stages [60]. The first stage is temporary stabilization of unstable fractures and control of hemorrhage, followed by resuscitation in the ICU and then delayed definitive management of the fracture as the patient's condition allows.

Treatment according to the principles of damage control orthopaedics can be applied in the case of trauma patients who are unstable or in extremis [61]. Its indications include inability to achieve hemostasis due to coagulopathy, inaccessible major venous injury, time-consuming procedure in a patient with suboptimal response to resuscitation, management of extra-abdominal life-threatening injury, reassessment of intra-abdominal contents, and inability to re-approximate abdominal fascia due to visceral edema [59]. By avoiding a "second hit," DCO avoids the additional physiologic burden of prolonged and more invasive procedure [60]. There is evidence that DCO may control the lethal triad of hypothermia, acidosis, and coagulopathy and also regulates the evolving systemic inflammatory response [61]. Studies have shown mortality in patients treated using DCO to be in the range of 1.8–7.2 % [61].

The concept of DCO may have broader indications in the elderly than in younger adults, as it reduces complications such as acute respiratory distress syndrome (ARDS) and multiple organ dysfunction [1]. It could reduce mortality and improve outcomes, since complications as a whole are less well-tolerated in older trauma patients. Unfortunately, there is little published literature on the applicability and benefits of DCO in the elderly. The need to manage according to DCO principles needs to be balanced against ability to perform single surgery in the frail elderly patient, as external fixators may cut out of osteoporotic bone. Definitive fixation in some instances can be preferred if the patient's condition allows it. The topic of DCO in the elderly requires future research to further explore its benefits and risks.

Early Total Care

For those patients stable enough to undergo early total care, their primary surgery becomes the definitive treatment of their fracture. The treatment of long bone fractures in the elderly may be

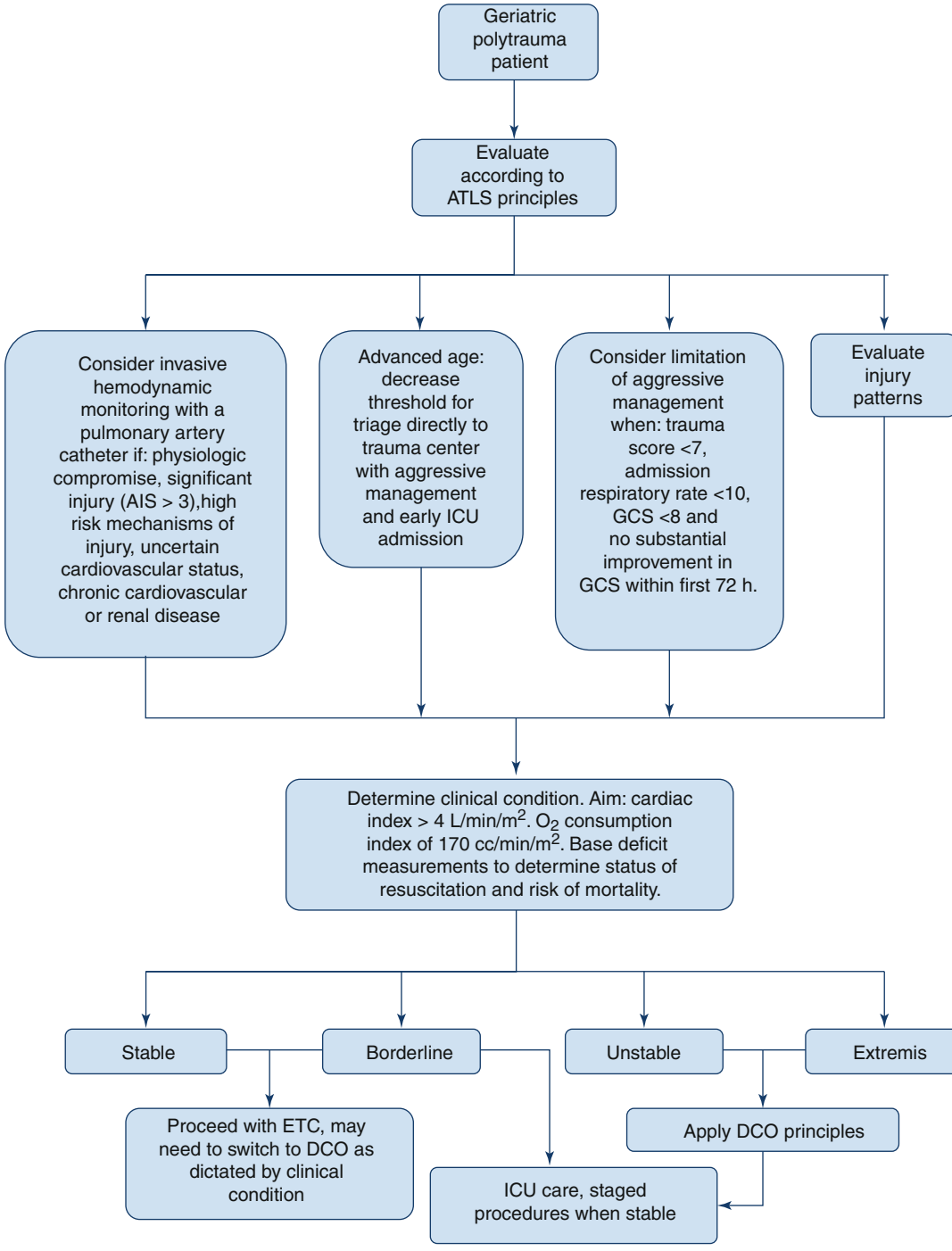

Fig. 4.4 Algorithm for management of the geriatric polytrauma patient and applications of damage control orthopaedic (*DCO*) and early total care (*ETC*) principles [1]. Abbreviations: *AIS* Abbreviated Injury Scale, *ATLS* Advanced Trauma Life Support, *GCS* Glasgow Coma Scale, *ICU* intensive care unit

Table 4.7 Four basic principles in the management of patients with fragility fractures [35]

Impaction at the fracture site to increase stability
Functional fracture restoration
Splinting of the fracture over a long area
Use of materials to augment the strength of bone where needed

complicated and altered by the presence of implants or prostheses, arthritis, decreased bone density, poor soft tissue quality, specific rehabilitation conditions, and delayed surgical intervention [6]. In patients with osteopenic bone, screw purchase is often poor, increasing the risk of screw pullout and subsequent fixation failure [6]. As a result, older patients are occasionally subjected to prolonged immobilization, which further decreases bone density. This reduction in density leads to "disuse osteopenia," which further complicates treatment of geriatric fractures [62].

There are four basic principles that guide the management of patients with osteoporotic fractures: impaction at the fracture site to increase stability, functional fracture restoration, splinting of the fracture over a long area, and use of materials to augment the strength of bone where needed (Table 4.7) [35]. Obtaining stable fixation is sometimes difficult in osteopenic bone, thereby complicating early range of motion and weight-bearing, which are required to help the patient regain function after a fracture. Osteopenia leads to poor screw purchase and increased risk of screw pullout, leading to fixation failure [6]. In a study of intertrochanteric hip fractures treated with sliding hip screw, osteoporotic patients with unstable fracture patterns were found to have a 53 % failure rate, compared to 14 % in those patients with normal bone density and similar fracture patterns [63]. Another study looked at transcervical hip fractures and found four potential risk factors for failure of repair: presence of osteoporosis, comminution, fracture angle, and fracture level. Of these, only osteoporosis placed the patient at risk for failure of fixation and need for additional surgery within 12 months of the index procedure [64].

Open Fractures

Treatment of open fractures in geriatric patients follows the same principles of treatment as those for patients of any other age: debridement of soft tissues and fracture ends, fracture stabilization, and adequate soft tissue coverage. Open fractures place the patient at risk for many complications. One review of 28 open tibia fractures found that, when compared with closed fractures, those that were open had a significantly increased rate of surgery, reoperation, and length of admission [65]. Furthermore, type IIIB open fractures were significantly more likely to require flap coverage, have complications, be admitted to the ICU, and require longer hospital stays [65]. Even with higher-grade open fractures, patients can have good outcomes. A series of ten type IIIB open fractures treated with free tissue transfer in patients older than 60 years of age found a 0 % infection and amputation rate at an average follow-up of 43 months; these patients were also able to walk without assistive devices 80 % of the time [66]. That being said, open fractures in the elderly should be treated as limb-threatening conditions.

Periarticular Fractures

The goals of treatment for periarticular fractures are anatomic restoration of the articular surface and mechanical axis with enough stability to allow for early range of motion. Risks associated with these fractures include delayed union, nonunion, loss of fixation, and osteonecrosis. Outcomes in elderly patients with periarticular fractures are more variable than in younger patients. In a retrospective review of 40 individuals over 50 years of age with a tibial plateau fracture treated with plate fixation, 72 % of patients reported unsatisfactory results with no significant differences in satisfaction between different Schatzker or AO types of fractures [67]. A similar review of 72 patients over the age of 70 years

who underwent ankle internal fixation found a 9 % risk for development of wound edge necrosis and delayed wound healing. 85 % of patients regained their pre-injury mobility and residential status and clearly benefitted from the operation [68]. Open reduction and internal fixation with plates and screws undoubtedly plays a primary role in treating periarticular fractures in the elderly, especially with the advent of locking plate technology.

Primary prosthetic replacement may be considered in certain fracture types when there is substantial destruction of an osteoporotic joint [35]. Examples of this include displaced, comminuted fractures of the femoral neck, proximal humerus, and elbow.

Total elbow arthroplasty for a displaced distal humerus fracture in elderly patients with low physical demands has gained momentum over the past decade. One series of ten patients reported high patient satisfaction and mean range of motion of 24–125° [69]. A larger review of 49 total elbow surgeries performed in cases which were not amenable to fixation found mean range of motion to be 24–131° with a 29 % complication rate; only ten cases required reoperation, of which five were revisions [70]. When advising patients about this procedure, they must be informed that they will have a permanent five-pound weight-lifting restriction on that extremity after the operation. Primary prosthetic replacement for distal femur fractures is less well studied, and its role has been defined in treating this fracture pattern. The anatomy of the fracture and associated loss of bone stock and ligamentous support require use of custom or modular constrained implants, making this treatment option more difficult and less appealing. Some complications have been described with treating distal femoral fractures in this manner, including prosthetic dislocation and loosening [6]. Additional studies need to be performed on indications and techniques before this becomes a more widespread surgical technique.

Intra-articular fractures of an arthritic joint can also be treated with primary prosthetic replacement. Primary total hip arthroplasty is indicated in certain displaced femoral neck fractures, and total elbow arthroplasty can be performed for elbow fracture-dislocations. Comminuted proximal humerus fractures may be treated with arthroplasty. Oftentimes, these elderly patients either have preexisting rotator cuff-tear arthropathy or there are associated massive rotator cuff tears. In this case, reverse total shoulder arthroplasty may be indicated. One study reported that outcomes are more predictable than with hemiarthroplasty and it consistently produces a greater amount of anterior elevation: 91° compared with 60° in hemiarthroplasty [71]. Similar results have been found in other studies, and complication rates are reported to be 10–28 % [72, 73]. Some caution must be exercised in interpreting this literature, as reverse total shoulder arthroplasty is relatively new and little information is available about its intermediate and long-term outcomes and revision rates.

Periprosthetic Fractures

Fractures around previous joint replacements or orthopaedic implants are challenging fractures to treat. Fortunately, they are uncommon. In a review of 108,595 arthroplasties, Meek et al. found 5-year periprosthetic fracture rates to be as follows: 0.9 % after primary THA, 4.2 % after revision THA, 0.6 % after primary TKA, and 1.7 % after revision TKA [74]. Risk factors are female gender, age >70 years, osteopenia, malaligned prosthesis, and previous revision surgery. Complications encountered with these fractures are not uncommon and include nonunion, malunion, and infection. These complications are thought to arise from a loss of endosteal blood supply from the initial joint replacement and the loss of periosteal blood supply from the fracture and associated surgery [6].

Priorities in the treatment of periprosthetic fractures are early mobilization of the patient, preservation of the mechanical axis of the limb, and stability of the bone-implant-cement interface [6]. If treated nonoperatively, these fractures may heal with malalignment of the mechanical axis. When operative treatment is undertaken, difficulties that may be encountered include the

presence of the prosthesis or a cement mantle as well as peri-implant bone loss [6].

With periprosthetic femur fractures after total hip arthroplasty, the surgeon needs to determine whether the stem is stable or loose. If the stem is stable, then open reduction internal fixation can be performed using a plate and/or cortical strut allograft. Fixation options include plates with a combination of unicortical and bicortical screws or cerclage wires or cortical strut allografts [6]. On the other hand, if the stem is loose, then it is advised that the most distal fracture line be bypassed with a longer stem prosthesis by at least two femoral diameters [75].

For periprosthetic distal femur fractures after total knee arthroplasty, retrograde femoral nailing is an option if the implant has an open box design. The main advantages of this option are that it only requires a small incision and does not require as much soft tissue dissection around the fracture, preserving blood supply in the area that needs it most. In patients treated with retrograde femoral nailing, weight-bearing is resumed at 2–3 months postoperative; one series of seven patients reported no complications, with all but one patient achieving greater than 90° of knee flexion [76]. If the patient does not have an implant with an open box design, then the patient can be treated with periarticular locking plates.

Implant Choice

Many implant options are available to the orthopaedic surgeon when dealing with the geriatric patient. Historically, the implant of choice for osteopenic patients was an intramedullary device, which is less likely to fail because of fatigue than plates because it is positioned closer to the mechanical axis of the bone [6]. Traditional plate and screw-constructs function as load-bearing devices when there is a gap between fracture fragments. They also depend on a stable bone-screw interface, which is most affected by bone density. Elderly osteopenic patients are susceptible to failure due to screw pullout. Pullout of these locking plate and screw constructs is much less likely because failure has to occur at each of the locking screws. The introduction of locking plates has improved treatment options in treating geriatric patients, as it allows for more secure fracture fixation in their osteopenic bone. Minimally invasive plate osteosynthesis (MIPO) is another new development in the treatment of fractures. Since traditional osteosynthesis requires periosteal stripping, there are concerns regarding bone devitalization as well as disturbances to the surrounding soft tissues. This has led to the development of plates which can be applied with submuscular techniques through much smaller incisions. One cadaveric study demonstrated that MIPO disrupts the distal femoral blood supply less than conventional plate osteosynthesis. An intimate familiarity with all the implant options available for treating fractures in the elderly will improve their care in the operating room.

Augmentation of fixation in osteoporotic bone can include use of PMMA or calcium bone cements [6, 35, 77]. None of the available bone cements are FDA approved for use as fixation augmentation agents. Their use is under exploration by many investigators as a way to improve implant fixation in porotic bone.

Soft Tissue Considerations

The status of soft tissues should be considered when performing surgery on geriatric patients. The skin of elderly patients is more fragile and intolerant of surgical insults [6]. The use of plates and screws in patients with substantial muscle atrophy and thinner skin can result in prominent hardware that is symptomatic and is more likely to require removal once the fracture has healed [6]. Pressure ulcers at or near the fracture site require alterations to the surgical approach. Those that are actively infected and untreated may require deferral of surgical treatment [6]. If the patient is immobilized, strict turning every 2 h will help avoid pressure ulcerations in dependent areas.

Treatment that allows weight-bearing should almost invariably be chosen for geriatric hip fracture patients. It is physically difficult for the elderly to ambulate with a weight-bearing

limitation on one extremity, and in the presence of cognitive impairment, it is almost impossible for them to adhere to this restriction.

Aftercare protocols which allow weight-bearing should almost invariably be chosen for geriatric hip fracture patients.

Timing of Surgery

Most evidence showing detrimental effects of delay in surgical fixation of fractures comes from literature on femoral neck fractures [2]. In a prospective study of 2,660 hip fractures, one group found that patients with acute medical comorbidities requiring treatment and delayed surgery had 2.5 times the risk of 30-day mortality [16]. The authors also found that a delay of up to 4 days in surgical care without acute medical comorbidities did not increase morbidity or mortality in hip fracture patients. They were also clear to state that they do not advocate such delays, but when surgical facilities are overwhelmed, a short delay does not place the patient at increased risk [16]. Another group of authors found that delay in surgical treatment of hip fractures not only increases mortality but also increases the rate of infections, length of stay, and total cost of care [78]. Those treated within 24 h had a 27 % chance of developing infectious complications compared to 81 % in those whose surgery was delayed more than 72 h. The difference in noninfectious complications was 7 % and 63 %, respectively. Similarly, the length of stay was increased by 7 days in those treated more than 72 h after admission [78].

In a prospective series of 367 patients at a single center, 267 (73 %) had surgery within 2 days of admission. The authors found that those treated within this time frame had half the 1-year mortality than those who waited more than 2 days for surgical treatment [79]. A another study found a decreased 1-year mortality in those relatively healthy patients (those with one or two comorbidities) treated with surgery within 24 h of admission compared to those who faced a surgical delay. Those with three or more preexisting conditions actually had an increase in mortality if treated within 24 h [80]. This highlights the balance between preoperative optimization and surgical management in the treatment of hip fractures in geriatric patient. Despite facing an increased risk of mortality in the first post-injury year, elderly patients with proximal femoral fractures who survive 1 year after their injury have survival approaching that of the normal population [34].

In one of the few studies assessing the timing or surgical management and mortality in multiply injured elderly trauma patients, Tornetta et al. found no difference in mortality between early and late fixation of fractures. Mortality was 11 % in those treated with early surgery (<24 h from admission) compared with 18 % in those who had late surgery [10]. This difference was not significant, but there were only 30 patients in the latter group. Therefore, the study was not adequately powered for subgroup analysis. In addition, those patients whose surgery was performed more than 24 h from the time of injury were often delayed because of medical issues [2].

Regardless of the fracture, earlier fracture fixation is beneficial and permits early healing and rehabilitation. The one exception to this, as described above, is when the patient is so seriously injured that the principle of DCO needs to be applied.

Perioperative Management

Because cardiac function is reduced as a person ages, excessive fluid boluses should be avoided. If hemodynamic status is ever in question, there should be a low threshold for placement of central lines to monitor the patient's hemodynamics. In their review of the multiply injured geriatric trauma patient, Soles and Tornetta suggested early invasive cardiac monitoring in the emergency department to identify occult shock, limit end-organ hypoperfusion, prevent multiple organ failure, and ultimately improve survival [2]. For the intubated patient, the ventilator should be

managed by the ICU team. Every attempt should be made to wean the patient from the ventilator as soon as possible to prevent pneumonia, barotrauma, tracheal stenosis, and ventilator dependence. After being extubated, vigorous pulmonary toileting and use of incentive spirometry should be encouraged to minimize atelectasis and prevent pneumonia.

All medications should be renally dosed, and fluid status must be monitored so as to preventing hypo- or hypervolemia. Urinary catheters, if used, should be removed as soon as possible to minimize unnecessary impediments to mobility and also prevent urinary tract infections. As geriatric patients are oftentimes malnourished to some degree, a protein-rich diet should be encouraged to help maximize healing potential during the initial posttraumatic and postoperative catabolic period. Vitamin D supplementation should also be pursued if necessary, as described above. In order to avoid venous thromboembolism, patients should be placed on chemoprophylaxis and mobilized early in the postoperative period.

Adequate analgesia should be provided, again with adjusted dosing. Around-the-clock acetaminophen with low-dose oral oxycodone as needed or low-dose intravenous hydromorphone for breakthrough pain is an effective regimen.

Should the patient have significant trauma, early transfer from the emergency department to the intensive care unit should be considered to prevent deterioration. It is important to bear in mind that the traditional markers for resuscitation, such as heart rate, blood pressure, and urine output, can be unreliable clinical end-points because of beta-blockade, hypertension, and preexisting organ dysfunction [2]. Instead, base deficit and serum lactate are better markers of tissue perfusion [2]. In a study examining the association between base deficit and serum lactate levels with mortality, Callaway et al. studied 558 elderly patients age 65 and above who were normotensive (systolic blood pressure ≥90 mmHg) after blunt trauma [27]. The overall mortality was 20 %, and mean lactate was higher among non-survivors. As lactate level at the time of presentation increased, so did mortality; levels of 0–2.4 mmol/L were associated with 15.4 % mortality, 2.5–4.0 mmol/L with 23.4 %, and greater than 4.0 mmol/L with 39.6 %. Similar trends were seen with base deficits. Base deficits greater than 0 mEq/L were associated with a 13.7 % mortality, 0 to −6 with 27.2 %, and less than −6 with 39.5 % [27]. One should keep in mind, however, that even with normal lactate and base deficits at presentation, the elderly still face a significant risk of mortality from trauma.

It is prudent to seek the help of a geriatrician with complicated geriatric fracture patients. The geriatrician can help manage many of the issues described above, including analgesia, fluid management, antibiotics, pulmonary issues, and nutrition. In addition, geriatricians can help manage other issues that may arise, such as how to handle dual antiplatelet therapy (clopidogrel/Plavix and aspirin) after cardiac stenting, cognitive impairment and dementia, delirium, and fall prevention. The role of a geriatric comanagement consultation service could be crucial to the proper care of both seriously injured and less seriously injured geriatric patients in the intensive care units and on the regular inpatient floors; geriatricians are an important part of the interdisciplinary trauma team [31].

Postoperative Complications

Complications that develop during the course of treatment in the geriatric trauma patient negatively affect outcomes, and every effort should be taken to avoid them (Table 4.8). A review of the Pennsylvania Trauma Systems Foundation database found that risk factors for developing complications include the presence of comorbidities, injuries, more body systems injured, and higher ISS [31]. In addition, complications significantly increase the odds of mortality in elderly trauma patients [31]. Since complications were found to increase mortality and comorbidities were found to correlate with complication rates, the authors recommended that great attention be paid to the immediate identification and comprehensive management of comorbid conditions. This should permit prompt management of these preexisting

Table 4.8 Common medical complications in geriatric patients

Heart	Myocardial infarction, congestive heart failure exacerbation, cardiac arrhythmias
Pulmonary	Difficulties weaning from mechanical ventilation, atelectasis, aspiration
Kidney	Fluid or electrolyte imbalance (including fluid overload), acute renal failure, increase in adverse effects of medications
Liver	Coagulopathy from hepatic dysfunction
Brain	Delirium
Infection	Urinary tract infection, pneumonia, surgical site infections, sepsis
Soft tissues	Wound dehiscence, decubitus ulcers

Complications are often relating to altered physiology and preexisting conditions in the elderly population. Perioperative management should focus on avoiding or mitigating the effects of these complications

conditions and may help decrease complication rates [31].

Delirium

Older patients often become delirious while in the hospital. Types of delirium include hyperactive delirium and hypoactive delirium. The hyperactive delirium can be recognized by agitation, crying out, pulling at intravenous lines, and confusion. Hypoactive delirium may be more easily missed. Patients with hypoactive delirium are quiet and somnolent. They may respond to verbal questions and then fall immediately back to sleep during the conversation. The hypoactive form of delirium carries a worse prognosis. There is no effective treatment for delirium. Avoidance is the best approach. Older adults should retain their glasses and hearing aids to avoid sensory deprivation. The presence of tethers such as Foley catheters and intravenous lines should be avoided or minimized. The delirious patient requires frequent reorientation from family, staff, or a bedside sitter., The judicious use of 0.5 mg of haloperidol is reserved for those who fail conservative management options [50]. Delirium is associated with increased length of stay, poor patient, and family satisfaction as well as poor clinical outcomes. Additionally, the delirious patient cannot effectively participate in postoperative rehabilitation and are often left in their bed or chair by their therapist.

Soft Tissue Complications

Elderly patients have both muscle atrophy and diminished bone mineral density. Wound healing issues are also more common in the elderly, as they have a less robust subcutaneous layer and are often malnourished, placing them at increased risk for wound dehiscence. Their more delicate skin places them at increased risk for decubitus ulcers. Meticulous multilayer closure should be performed to minimize complications such as wound dehiscence and seroma or hematoma formation, which predisposes to infection. Monofilament, nonabsorbable sutures are better, and caution should be exercised when handling frail geriatric subcutaneous tissues and skin. Sutures should be kept in place for longer periods of time in elderly patients, taking into account the slower healing that they experience.

Cardiovascular and Pulmonary Complications

Cardiovascular complications that may arise during the care of elderly patients include myocardial infarction and congestive heart failure exacerbation. Congestive heart failure if untreated carries a poor prognosis and increases the likelihood of hospital readmission. Cardiac arrhythmias are common issues including atrial fibrillation. Assistance from medical colleagues is essential to prevent harm from arrhythmias.

The pulmonary system is subject to difficulties weaning from mechanical ventilation as well as atelectasis, aspiration, and ARDS because of elderly patients' loss of protective reflexes, impaired coughing, decreased thoracic elasticity, impaired ciliary clearance, and osteoporotic ribs, which are prone to fractures. Aspiration pneumonia is the most common pulmonary complication following geriatric trauma. It carries a grim prognosis, so avoidance is the best approach.

Elevation of the head of the bed 30° at all times can offer some benefits as can a swallowing evaluation done by a speech therapist.

Hepato-Renal Failure

The aged kidney places the patient at higher risk of developing a fluid or electrolyte imbalance, including fluid overload, or acute renal failure. In addition, there is an increased chance of developing adverse effects from medications secondary to reduced drug metabolism. Hypotension, hypovolemia, and fat embolism are common post-trauma issues that can result in an acute kidney injury. Correction of the cause and waiting are the only options for treatment.

Hepatic dysfunction can put the patient at risk for coagulopathy. Monitoring of coagulation parameters is important to avoid both bleeding complications and thrombosis. The daily total acetaminophen dosage given should be kept at ≤3 g to avoid hepatic injury.

Postoperative Infections

Elderly patients are more susceptible than younger individuals to nosocomial infections, such as urinary tract infections, pneumonia, surgical site infections, and sepsis. Nosocomial infections result in longer stays in the hospital and ICU and increased mortality [4]. OIder infected patients were shown to have a 22 times greater relative risk of mortality when compared to younger noninfected patients [4]. It goes without saying that patients that develop complications have worse outcomes as a whole than those who remain free of complications.

Outcomes

Most studies on geriatric fracture outcomes tend to focus on hip fractures, given their morbidity in this population. There are few published studies on mortality in critically ill and multiply injured elderly trauma patients. In elderly patients who are critically injured, aggressive resuscitation is the initial step in stabilization. In the landmark randomized trial on early goal-directed therapy in patients of all ages with sepsis and septic shock mortality, Rivers et al. found that such management leads to improved outcomes [81]. Early goal-directed therapy consisted of placement of a central venous catheter capable of measuring venous oxygen saturation, administration of crystalloid, blood products, vasopressors, vasodilators, and intubation according to a standardized protocol, while the patient was monitored in a unit contained within the emergency department. When admitted to a regular inpatient bed, the continuous monitoring was discontinued. Patients were randomized to receive one of two forms of treatment in the ED: 6 h of goal-directed therapy versus standard care, and the admitting clinicians were blinded to ED care [81]. In-hospital (30.5 % vs. 46.5 %) and 60-day (44.3 % vs. 56.9 %) mortality were both significantly lower in the treatment group compared to the control group [81]. Another study was performed on old and young trauma patients who were given a standardized resuscitation protocol to attain and maintain an oxygen delivery index of 600 mL/min × m^2 or greater for their first 24 h in a trauma ICU [82]. The group found that patients older than 65 years of age had similar 7-day survival compared to younger patients (92 % vs. 94 %) [82]. Even though they had a significant decline in 30-day survival (42 % vs. 89 %), this study showed that resuscitation efforts were effective in older patients.

For less seriously injured patients, several studies have been performed, almost exclusively on hip fracture patients. Schnell et al. found that independent predictors of mortality after hip fracture include age, male gender, low Parker mobility score, dependent with activities of daily living, and Charlson score ≥4 [19]. Patients with dementia also had a significantly higher 1-year mortality: 29.3 % versus 13.9 % in those without dementia (Table 4.9) [19]. This finding is important, as 47 % of patients in the study carried a diagnosis of dementia prior to their fracture. These findings were similar to other studies, which also found higher rates of mortality in

Table 4.9 Predictors of increased risk for mortality in hip fracture patients [19]

Increasing age
Male gender
Decreased mobility
Dependence with activities of daily living
Increasing number of comorbidities
Dementia

Knowledge of these risk factors and their presence or absence in hip fracture patients will help counsel patients and families, as well as guide management

demented patients [83, 84]. With regard to age, Schnell et al. found that mortality was 2 % in patients under age 70 and 27 % in those older than 90 years of age [19]. Berry et al. had similar findings: for every 5 years of advancing age, there was a 30 % increase in mortality [85]. It is apparent that reducing mortality after hip fracture should be one of the primary goals of future studies in the realm of geriatric orthopaedics.

Improved outcomes have been reported with a comanaged approach to geriatric fractures [50]. One group found a decreased time to surgery, fewer postoperative infections, decreased rate of complications, as well as shorter lengths of stay, 30-day readmission rates, and decreased in-hospital mortality after implementation of their comanaged geriatric fracture program [50]. In a later study, the same authors found that pre-injury residence was an important risk factor for mortality in hip fractures [19]. Mortality was increased if preadmission residence was a nursing home or assisted-living facility compared to a community dweller (30.7 % and 23.7 % vs. 13.2 %, respectively) [19]. Another study showed that 1-year mortality for hip fracture patients residing in nursing homes at the time of the injury was 36 % for women and 54 % for men [85]. Pre-injury residence was not an independent predictor of 1-year mortality after adjusting for other characteristics such as comorbidities and function, mostly because nursing home residents tend to be the more physically ill and more debilitated [19]. Following a fragility fracture, few patients will recover to their pre-injury levels of function [35]. These are truly life-changing diagnoses, as only 50 % of patients regain their pre-fracture level of mobility [21]. With geriatric patients, immobilization of an extremity or dependence on assistive devices for ambulation will often change disposition status such as the need for institutional care after discharge from the hospital [6]. Many patients end up in long-term care facilities. Others become dependent on other family members for help with their activities of daily living [21]. One study found that approximately one fourth of individuals who were living independently at the time of sustaining a hip fracture subsequently required long-term nursing home care [86].

Discharge destination is important outcome in geriatric fracture patients, as they can be discharged to a nursing home, skilled nursing facility (SNF), or to their home, with or without nursing and therapy services. In an examination of approximately 38,000 patients from a single state trauma database, authors found that 25 % of all patients were discharged to a SNF or nursing home. They also found that age significantly increased the odds of being discharged to a SNF or nursing home, with an 11 % increase in the odds for each additional year in age after 65 [31]. There are some measures that can be taken to minimize the need for post-discharge institutionalization. By improving surgical treatment of hip fractures in community dwelling patients, Ceder et al. allowed for immediate postoperative weight-bearing and commencement of an intensive rehabilitation program, while the patient was in the hospital followed by a home program. They showed that this program not only decreased the length of stay but also decreased the need for discharge to SNF or nursing home [87]. Zuckerman et al. reported similar results after instituting an interdisciplinary hospital care program; the authors also reported a decreased complication rate [88]. These studies emphasize the need to weight bear as tolerated after hip fracture surgery.

Readmission is burdensome for the patient, family, and the physicians. It has been shown that with the comanaged geriatric fracture model, providers have been able to achieve a lower readmission rate. In one study, Friedman et al. found a 9.7 % 30-day readmission rate, half of which

(5.1 % of the total study population) had readmissions related to their fractures. This was much lower than the 19.4 % expected readmission rate [50]. Reducing readmission rates is beneficial not only to the patient and the providers but to society as well, as these readmission events are typically complicated cases that require significant medical resources and healthcare dollars to treat.

Conclusions

Both high-energy and low-energy trauma are an important causes of morbidity in the elderly, the fastest growing segment of the population in the United States and many other countries. Both are associated with an increased risk of mortality with falls representing the most common mechanism of injury. Trauma increases the risk of developing significant complications. Osteoporosis is common and it increases the difficulty of treatment of musculoskeletal injuries. Orthopaedic and geriatric comanagement offers many benefits in the perioperative period to reduce occurrence of adverse events in older patients. The treating surgeon should be familiar with numerous orthopaedic and medical management considerations that accompany care of geriatric trauma patients. Despite the increased risk of complications, older patients can have good outcomes when they are provided with the proper surgery and proper care.

The fields of geriatric trauma and geriatric orthopaedics are still in their infancy. As with the rest of orthopaedic surgery, few randomized prospective clinical studies have been published to guide management. Therefore, it is difficult to provide level I evidence to guide treatment of patients [35]. Much more research on this rapidly growing population is needed. The ideal method of dealing with fragility fractures is prevention, mostly by preventing falls and treating osteoporosis [6]. When injured, the patient needs to be thought of as a whole, not just a specific injury. In addition, a systematic approach to patient care will improve outcomes [35].

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The Multiply Injured Patient

5

Philipp Lichte and Hans-Christoph Pape

Initial Assessment

In patients with severe injuries, fatal outcome continues to be a major concern [1]. Trunkey described a trimodal pattern of death which characterizes the treatable conditions.

Within the first hours, severe hemorrhage and brain injury are the most common reasons of death. In patients who succumb within several hours, death is usually a result of airway, breathing, or cardiovascular issues and has been identified to be potentially preventable. The third mortality peak appears more than 1 week after trauma and is caused by sepsis and multisystem organ failure [2].

To quantify the severity of multiple injuries, several trauma scores have been described. The most common ones are the Injury Severity Score (ISS) and the New Injury Severity Score (NISS), both of which are based on the Abbreviated Injury Scale (AIS). Injuries of six body regions (head, face, chest, abdomen, extremities (including pelvis), and external structures) are classified from 1 (mild) to 6 (usually fatal). Based on the ISS, a polytraumatized patient is considered if the ISS exceeds 15 points.

The initial systematic assessment is performed to immediately identify potentially life-threatening conditions. Therefore in addition to the extent of the anatomic injuries, assessment of the pulmonary and hemodynamic status is required as described in the Advanced Trauma Life Support (ATLS) algorithm. ATLS requires ruling out major causes of acute decompensation such as tension pneumothorax, cardiac tamponade, and herniation. It is very important to remember that the clinical scenario can change during the initial assessment as hemorrhage can become more severe or improvement can be achieved by volume therapy.

Hemorrhagic Shock

Severe hemorrhage should be identified and controlled as early as possible. Alterations in pulse and blood pressure are late signs, especially among patients younger than 40 years. Due to the cardiovascular reserve of these patients, the extent of hypovolemia may be underestimated. Therefore capillary refill and, as a secondary parameter, urine output, along with arterial pH, base excess, and plasma lactate levels, are more valid.

In multiply injured patients, there are four major sources of bleeding: external, thoracic, abdominal, and pelvic.

External blood loss may be difficult to quantify, especially in cases of prolonged extrication. Initial treatment before rushing the patient to the operating room may be the use of a compressing towel or a tourniquet. Thoracic and abdominal

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sources of hemorrhage can be identified by clinical examination, chest X-ray, abdominal ultrasound, or computed tomography (CT) scan.

From the musculoskeletal point of view, unstable pelvic fractures are the most common source of massive hemorrhage, requiring immediate treatment [3, 4].

Grading the Patient After the Initial Assessment

After completion of the initial assessment and urgent interventions, patients can be matched into four different groups (stable, borderline, unstable, in extremis) [5]. The graduation is based on the overall injury severity, the presence of specific injuries, and the current hemodynamic status at the endpoint of resuscitation. Grading the patient is an important step to determine the further therapy strategy.

Stable

Stable patients have no immediate life-threatening injuries, respond to initial interventions, and are hemodynamically stable. They are normothermic and show no other major signs of physiological disturbance.

Stable patients should undergo early total care of their major fractures due to their physiological reserve to endure prolonged operative interventions [6].

Borderline

Patients of this category respond to the initial resuscitative procedures but there may be additional sources of occult bleeding. Several criteria had been identified to classify a patient as borderline condition (Table 5.1) [7].

These patients have a higher risk of rapid deterioration. Nevertheless, if these patients are stabilized appropriately, early definitive care can be used safely in the treatment of their major fractures [8]. In the appearance of deterioration, conversion to "damage control" techniques has to be considered. Some authors consider damage control nailing to minimize the duration of initial surgery. In these cases, an unreamed, unlocked nail is used initially, and locking and/or further reduction is performed secondarily.

Table 5.1 Criteria for borderline condition [7]

ISS > 40
Body temperature below 95 °F
Multiple injuries (ISS > 20) in combination with thorax trauma
Multiple injuries in combination with severe abdominal or pelvic injury and hemorrhagic shock in the moment of administration
Radiographic evidence of pulmonary contusion
Patients with bilateral femur fractures
Patients with moderate or severe head trauma

Unstable

Patients who do not respond to the initial procedures and remain hemodynamically unstable have a high risk of rapid deterioration, multiple organ failure, and death in the course. Therefore surgical treatment consists of lifesaving surgery followed by temporary stabilization of major fractures [9]. Afterwards the patient should be stabilized on an intensive care unit.

Essential operations are:

- Hemorrhage control
- Exteriorization of gastrointestinal injuries
- Temporary fixation of unstable fractures using external fixation

Complex reconstruction procedures should be postponed until stability is achieved and the acute immunoinflammatory response has subsided.

In Extremis

These patients are very close to death because of severe injuries and ongoing uncontrolled bleeding. They show an inadequate response to continuous resuscitation maneuver and are suffering from the effects of hypothermia, acidosis, and coagulopathy which are known as the "deadly triad." Thus only lifesaving procedures are indicated. Reconstructive operations can be done in course, if the patient survives [10].

The Concept of "Damage Control Orthopedics"

Fracture stabilization is important to reduce pain, minimize fat embolization, and allow for early patient mobilization. The optimal way to achieve this goal is primary definitive osteosynthesis. Usually, temporary external fixation, splints, or casts should be avoided. Exceptions may apply according to the status of the patient.

The concept of "damage control" has been established for the treatment of borderline patients and patients in an unstable or extremis condition. The intent is to control but not to definitively repair injuries early after trauma in the sequence of their urgency.

The treatment is separated in three stages:

1. The first step involves the early temporary stabilization of unstable fractures and stopping the hemorrhage.
2. The second step is to optimize the patient's condition in the ICU.
3. In the third stage, delayed definitive fracture reconstruction is indicated if the patient's condition allows.

The goal is to minimize the additional biological stress ("second hit") due to prolonged surgical procedures in the initial phase [11]. Application of an external fixator can achieve sufficient stabilization of unstable long bone and pelvis fractures with minimal invasiveness and without prolonged operation time [9, 12, 13].

The optimal timing to perform the definitive reconstruction is an individual decision based on clinical judgement in combination with laboratory tests. It has been shown that major surgical procedures should be avoided in days 2–4.

The conversation of an external fixator to a definitive internal osteosynthesis should be done within the first 2 weeks to minimize the risk of infection.

Priorities in Fracture Care

The sequence of fracture treatment in multiply injured patient is a crucial part of the management concept. Due to their anatomy, some body sections are more vulnerable for progressive soft tissue damage. Therefore, in hemodynamically stable patients, the generally recommended sequence of treatment is tibia, femur, pelvis, spine, and upper extremity.

In multiply injured patients, the simultaneous approach to different extremity injured should be considered if certain logistic requirements are fulfilled.

Tibial Fractures

Especially in tibial fractures, the treatment strategy depends not only on the fracture type and the patient's condition but also on the status of the soft tissue. Unstable fractures in multiply injured patients should be stabilized initially. Primary definitive internal osteosynthesis is preferable in stable patients; in unstable patients, fracture stabilization can be reached by an external fixator. Early secondary conversion to a definitive osteosynthesis can be performed after stabilization of the patient's condition.

Bilateral Tibial Fractures

Simultaneous treatment can be a useful concept for the treatment of bilateral fractures. In bilateral tibia factures, both legs can be prepped and draped simultaneously. Because of the handling of the fluoroscope, fixation should be performed sequentially.

Compartment Syndrome

An increasing intrafascial pressure induces a compartment syndrome which can lead to irreversible damage of muscles, vessels, and especially nerves. A manifest compartment syndrome is defined with a pressure difference of <20 mmHg between the subfascial pressure and diastolic blood pressure. Due to the decreased blood pressure in patients in hemorrhagic shock in combination with the limited possibility to communicate (intubation, sedation, brain injuries), the risk for development of a compartment syndrome is increased in multiply injured patients [14]. Therefore prophylactic fasciotomy is recommended with a generous range of indication.

Table 5.2 Staged approach for ipsilateral femoral and tibial fractures

		Stable	Borderline	Unstable	In extremis
Initial treatment	Femur	Nailing	Resuscitation successful: nail Resuscitation difficult: ex fix, consider damage control nailing	Ex fix/traction	Traction
	Tibia	Nailing	Nailing	Ex fix/traction	Traction
Staged treatment	Femur	–	Nailing	Nailing	Nailing
	Tibia	–		Nailing	Nailing

Femoral Fractures

In multiply injured patients, stabilization of the femur should be performed before admission to the ICU. An early fixation of the femur declines morbidity and mortality due to reduction of fat embolism and pneumonia, thromboembolic complications, MODS, and sepsis. Also nursing and positioning of the patients will be facilitated.

Primary definitive osteosynthesis is the method of choice in stable and stabilized borderline patients. In patients in an unstable condition, we recommend closed reduction and application of an external fixator.

Several studies could show that the intramedullary pressure increases during the insertion of the nail, and thereby proinflammatory mediators and fatty particles could be released. In patients with multiple fractures and especially in patients with pulmonary impairment, this could lead to rapid deterioration of the lung function [7]. Therefore we recommend primary intramedullary nailing in multiply injured patients only in the absence of severe thoracical injuries and if the ISS is below 25 points.

Bilateral Femoral Fractures

In case of bilateral femoral fractures, a higher kinetic energy has occurred. Additional injuries imply a higher risk of acute respiratory distress syndrome (ARDS) and multiple organ dysfunction syndrome (MODS) [15, 16]. Therefore we recommend to consider external fixation in these patients if resuscitation is difficult or the patient's condition deteriorates during operation.

Ipsilateral Femoral and Tibial Fractures

For the management of ipsilateral femoral and tibial fractures, a staged management is advised as shown in Table 5.2.

Unstable Pelvic Injuries

Unstable pelvic injuries with active bleeding in multiply injured patients are acute life-threatening situations which require immediate therapy. Therefore unstable pelvic injuries should be excluded as fast as possible within the first minutes after arrival in the ED.

Pelvic injuries can be classified roughly on the basis of the clinical and radiological examination results under consideration of the history of the trauma. A useful classification has to be practicable and should offer a guideline for further therapy. The following reduced and simple classification by the AO A B C system fulfills these requirements.

Type A fractures include stable fractures of the anterior pelvic ring with intact integrity of the dorsal structures which do not require operative treatment.

Type B injuries are characterized by partially intact dorsal structures. Rotational instability is possible. Especially open-book-type fractures with external rotated alae have an increased risk of hemorrhage complications. On the other hand, type B injuries may initially be in internal rotation (closed book fractures) which results in bony compression and self stabilization of the pelvis. Type B injuries require osteosynthesis only of the anterior pelvic ring.

Type C fractures are characterized by a translational instability of the dorsal pelvic rim due to completely destroyed posterior stabilizing structures. This results in a 3-dimensional instability of the pelvic ring and is associated with an extremely high risk of hemorrhagic complications and concomitant injuries of pelvic organs as urogenital lesions. The differentiation of type B and C fractures may often be difficult. A CT scan can give important additional information on stable

patients. Type C injuries require a stabilization of the anterior and posterior pelvic ring.

Goal of the initial treatment of unstable pelvic injuries among multiply injured patients is an adequate stabilization and bony compression to avoid massive bleeding which predominantly is from venous vessels. In cases of arterial bleeding, selective angiography and embolization of the source of bleeding are becoming more common [17].

Stabilization techniques for a supine position of the patient are preferred during the primary period. Despite of the usage of pelvic slings, operative procedures as an external fixator or a pelvic C-clamp are the most common opportunities [18]. Internal stabilization techniques are normally time-consuming and technically difficult procedures which require stable patients. Therefore internal procedures in the initial phase are commonly only recommended in special cases. In the literature, for example, plate osteosynthesis of the symphysis or ventral plate osteosynthesis of the SI joint after laparotomy was described. In the last years, some authors recommend primary definitive osteosynthesis of pelvic fractures also in severely injured patients [13, 14, 19, 20].

In exceptional situations with exact closed reduction of the dorsal ring, initial percutaneous screw fixation of the SI joint is possible [12, 21].

In the secondary phase, we recommend the earliest possible stabilization of fractures of the pelvic ring to make possible early mobilization and to simplify intensive care maneuvers. External fixators should be replaced not later than within the 18th to 21st day. Later definitive osteosyntheses are correlated with worse reduction results.

Unstable Spine Injuries

If the clinical examination gives a hint of a spine injury, X-rays should be performed. Multiply injured patients are often not able to cooperate, and therefore X-rays of the complete spine are necessary to rule out spinal injuries. If there is a suspicion of a complex injury (protruding fragment in the spinal canal, rotational injuries, ligamentous participations), further diagnostics (CT, MRI) are indicated.

Operative treatment of unstable spine injuries is obligatory to allow appropriate nursing and early mobilization of the patient. Nonoperative methods (jacket, halo fixator) are often inappropriate for multiply injured patients due to an increasing risk of complications caused by immobilization. Internal stabilization of spinal fractures even without neurological symptoms has been performed more often in the last years because it can significantly reduce the length of immobilization and ICU stay [15, 22].

Closed reduction of unstable spine injuries without neurologic symptoms is indicated in fractures of the cervical spine and rotational injuries of the lower thoracic or lumbar spine (AO classification type C injury). In multiply injured patients, closed reduction may be difficult because of injuries of the extremities. In these cases a proper correction of rotation and axis needs surgical treatment.

If bony fragments or an intervertebral disc is interposed or dislocated into the spinal canal, open reduction and extraction of the fragment should always be performed to avoid spinal cord injury.

The standard approach to operative management of the cervical spine is the ventral approach. During the operation, the head is fixed in a special reduction system using the ring of the halo fixator. Injuries of the thoracic or lumbar spine which need dorsal and ventral stabilization should be treated with two operations depending on the general status of the patient. In the initial phase, dorsal stabilization should be performed through dorsal instrumentation with an internal fixator [16, 23]. Intrathoracical or intra-abdominal injuries are not necessarily a contraindication for the required prone position. The ventral stabilization may be done secondarily.

Upper Extremity Fractures

As already described for injuries of the lower extremities also injuries of the upper extremities were treated according to the “DCO” principles.

Dislocated joints should be reduced as fast as possible. In unstable patients only humeral shaft

fractures and unstable dislocated distal humeral fractures as well as dislocated forearm fractures and open fractures with join exposure should be operated. In the primary phase, the external fixator offers a good possibility for sufficient stabilization. In younger patients with dislocated fractures of the humeral head, initial operative treatment should be considered due to the risk of humeral head necrosis [24].

If it is predictable that the intensive care period takes longer, fractures of the humerus should be splinted with the elbow in extension to minimize the tension of the muscles which pull the elbow in a varus position.

It is often advisable to operate fractures of the clavicle and proximal humerus in multiply injured patients which could also be treated conservatively in monotraumatized patients due to the importance of early mobilization.

Soft Tissue Management

Open fracture care is an essential part of primary management. The primary surgical therapy should include radical debridement, extensive irrigation, assessment of the damage, and stable fixation of the fracture. Especially open fractures due to high-energy traumata with severely damaged soft tissue need a large debridement during the initial assessment. In cases of minor soft tissue damage in low-energy fractures, the fractures often can be treated like closed fractures after the initial debridement.

The generalized tissue hypoxia, acidosis, and hypoperfusion due to hemorrhagic shock have a large impact on the prognosis of the soft tissue. Therefore especially in patients with extensive tissue destruction combined with massive bone destruction after high-energy traumata, overall injury severity, extent of shock, and initial blood loss should be considered.

High-energy traumata often cause extensive soft tissue destruction combined with massive bone destruction. In these cases an individual, sophisticated treatment plan should be established considering overall injury severity, extent of the shock, and initial blood loss. Among multiply injured patients, the generalized tissue hypoxia and acidosis and the general hypoperfusion of the extremities due to the hemorrhagic shock have a large impact on the prognosis of the soft tissue damage. Therefore forced primary wound closure is often not indicated.

Soft Tissue Reconstruction

Smaller wounds can be temporarily covered with the vacuum-assisted closure (VAC) until the swelling decreases. It can be used to prepare the wound for the following definitive closure. Advantages are low infection rates and a proper granulation of the wound. Afterwards the definitive closure of the wound or a mesh graft transplantation can be performed.

If there are implants, bones, joints, or tendons on the ground of the wound, they have to be covered with vital and well-perfused tissue. Otherwise there is a clearly increased risk of infections and nonunions of the bone or joint which can necessitate secondary amputation of the extremity. In these cases, cooperation between the orthopedic and a plastic surgeon is recommended for an optimal overall result.

Medium-sized wounds can often be closed by local transposition of the surrounding tissue after mobilization (local flaps). This secondary covering procedure should be performed in the period of 72 h after trauma. Among multiply injured patients, it can be difficult to find enough healthy tissue to perform a local flap due to serial injuries. Therefore distant flaps are often required in the treatment of these patients. Distant flaps are also indicated if the defect zone is too large to be covered with a rotational flap. Free Microvascular flaps are progressively used, but it has to be kept in mind that prolonged surgical procedures stress the general condition of the patient. If a distant flap is indicated, the timing of the operation depends on the one hand on the general condition and on the other hand on the need for an urgent covering of the defect.

Amputation vs. Salvage

After the assessment of the fracture severity, it has to be decided whether a reconstruction of the extremity is possible or a primary amputation is indicated. In multiply injured patients, the general condition plays a crucial role in this decision-making process.

From the surgeons point of view, the attempt to preserve the extremity often seems to be the best choice for the patient. The possibilities to save an extremity, especially the lower limb, increase due to new microsurgical techniques [25]. On the other hand, bone and soft tissue reconstruction often requires repeated operations which cause prolonged hospital treatment [26].

The decision has to be differentiated between different anatomic regions: The sensibility of the foot, continuity of all large nerves, and maintenance of proper length are less important in the treatment of the arm. On the other hand, the prosthetic care for the upper extremity is much more sophisticated than that for the lower extremity, especially in below-knee amputations. The reduced function of a reconstructed upper limb is often better than the function of an artificial limb.

Especially among multiply injured patients, a prolonged reconstruction or replantation procedure may further harm the patient and put him into a life-threatening condition. In general, the expected result of a reconstruction or limb-saving strategy should outweigh the result of an amputation and fitting of a good prosthesis.

In multiply injured patients with an ISS up to 25 points and grade III soft tissue injures, generally there is an indication for reconstruction attempt (Table 5.3).

Complete or incomplete amputations with multiply injured patients with an ISS below 26 points should be managed very similar. In these cases replantation has to be considered with referral to a specialized center. Hemorrhage may be temporarily stopped by elevation and application of a pressure bandage or a tourniquet before further treatment.

Amputation injuries in children always have to be considered for replantation. Children show better functional outcome than similarly injured adults due to their better tissue regenerative ability.

Table 5.3 The sequence of grade III soft tissue injury treatment

1. Extensive debridement
2. Vascular reconstruction
3. Fracture stabilization (external fixator)
4. Temporary covering (vacuum-assisted wound closure)
5. Early secondary reconstruction

Severely traumatized extremities in multiply injured patients with a high overall injury severity (ISS > 25) often require secondary amputation. Therefore in this subgroup the reconstruction of the extremity should only be attempted in a few selected cases. The principle "life before limb" should absolutely hold true, and the indication for amputation may be widened. In summary:

- ISS < 25: Reconstruction/replantation are recommended analogous to monoinjured patients.
- ISS > 25: Reconstruction is only recommended in cases of minor soft tissue injury, replantation only in selected cases after successful resuscitation.

Long-Term Outcome

Due to improvements in the treatment of polytrauma patients during the last decades, the long-term outcome regarding functional aspects, quality of life, and patients' satisfaction is getting more and more in the focus of interest [27].

Large long-term studies have been recently completed in order to evaluate the outcome of severely injured patients and define the influencing factors for individual outcome. They revealed that not only injury-related factors but also the individual character and the socioeconomic have a strong impact on the long-term outcome and patients' satisfaction. Especially posttraumatic stress disorder should be more considered in the interdisciplinary care of multiply injured patients.

Focussed on orthopedic injuries, lower extremity injuries have been shown to be associates with a significant loss of long-term function [28]. Patients with pelvic injuries frequently suffer of chronic pain. Concerning the upper extremities especially the combination of fractures and vascular and nerval injuries predisposes for worse outcome.

In summary, many patients after multiply injuries suffer of chronic pain and functional deficits. Therefore, the improvement of the long-term function and patients' satisfaction will be the next great challenge in the development of new treatment strategies for multiply injured patients.

Summary

The treatment of multiply injured needs an individually adapted approach. In the decision-making process, the overall injury severity and the fracture distribution play the crucial role.

Patients can be categorized into the four subgroups "stable," "borderline," "unstable," and "in extremis" based on the patient's physiological status, the ISS, and specific injury combinations.

Stable patients should be treated by initial definitive osteosynthesis. The generally recommended sequence of treatment is tibia, femur, pelvis, spine, and upper extremity.

In borderline patients the fracture treatment is always an individual decision. Therefore we recommend a staged approach according to the abovementioned sequence with periodically reassessment of the patient. In case of deterioration, DCO maneuvers should be performed before the patient should be transferred to the ICU.

In unstable patients, stopping the bleeding and stabilization of tibial and femoral fractures as well as unstable pelvic fractures is prioritized. Definitive osteosynthesis should follow after stabilization of the patient on the ICU.

The treatment of in extremis patients should be limited to lifesaving procedures.

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Part II

Management of Specific Injuries

Femur Fractures

6

David C. Teague and Melissa A. Gorman

Introduction

Fractures of the diaphysis of the femur are relatively common in the adult population and are typically a result of high-energy trauma. The femoral diaphysis comprises the shaft segment extending from approximately 5 cm below the level of the lesser trochanter to approximately 9–10 cm above the knee joint. Substantial force must be applied directly or indirectly to this largest and strongest bone in the body to cause a fracture of the diaphysis of the femur. Mortality from this injury has substantially decreased with modern treatment protocols and methods, although the mortality following bilateral femoral diaphyseal fractures can be as high as 30 % [1]. Each femoral shaft fracture can lose two to three units of blood [1], resulting in hemodynamic instability in even the healthiest of patients. However, the associated injuries that typically occur with a femoral shaft fracture are a much more likely cause of both morbidity and mortality in these patients. Thus, femoral shaft fractures are typically markers of significant trauma, and appropriate management of these injuries is a crucial component of the overall management of polytraumatized patients.

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Epidemiology

The incidence in the United States has been estimated at 1–1.3 fractures per 10,000 people/year. There is a typical bimodal distribution of femoral shaft fractures with an initial peak in the mid-20s and a second peak in the mid-60s. The younger population typically sustains the injury as a result of high-energy trauma, while the older population is usually the result of a lower-energy mechanism. Motor vehicle collision is the most common cause of femoral shaft fractures, followed by auto versus pedestrian accidents, falls from height, and gunshot wounds [2]. Less frequent causes include direct blows, sports injuries, and falls from standing height.

Diagnosis

Signs and Symptoms

Fractures of the femoral shaft are very rarely missed in conscious patients. Significant pain in the extremity will be present, and the deformity is usually obvious. Soft tissue swelling is also typically present very early following the injury. A thorough history and physical should be obtained from the patient to determine the cause of injury and help establish any other concurrent

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injuries. Even in an unconscious patient, deformity is typically obvious, and the affected limb will be accompanied by swelling. A subtle finding may simply be that the foot is either extremely externally or internally rotated and the limb is short. Femurs should always be carefully examined on all unconscious patients, especially if the patient's mechanism of injury is high energy.

A complete physical exam should also be performed according to standard Advanced Trauma Life Support (ATLS) guidelines. The fractured extremity should be examined closely to assess for any open wounds and for the severity of the soft tissue injury. The compartments should be assessed, and a detailed neurovascular exam should be performed. The ipsilateral hip joint should also be examined closely to rule out a concurrent hip dislocation or hip fracture. Normal pulses do not necessarily rule out a vascular injury, so repeat clinical exams are of paramount importance.

Radiographic Studies

Anteroposterior and lateral radiographs are typically all that are required to characterize a femoral shaft fracture. It is important to image the entire bone, and it is imperative that both the hip and knee are included in the radiographic series. Inadequate radiographs should not be accepted as associated injuries can be easily missed (Fig. 6.1). A number of features should be assessed on these radiographs, including the location of the fracture, the presence of comminution, amount of displacement, any bony defects, as well as any air in the tissues suggestive of an open fracture. Many institutions routinely obtain CT scans with thin cuts through the femoral neck as a protocol-driven effort to avoid missed ipsilateral femoral neck fracture. Tornetta et al. were able to reduce by 91 % the delay in diagnosis of ipsilateral femoral neck fractures with femoral shaft fractures by using a standard protocol that consisted of a dedicated anteroposterior internal rotation plain hip radiograph, a fine (2 mm) cut CT scan through the femoral neck, and intraoperative fluoroscopic lateral radiographs of the hip in the operating room prior to awakening the patient [3]. With this protocol, the authors in this series only had one delayed diagnosis of a femoral neck fracture.

Associated Injuries

Multiple injuries are commonly associated with femoral shaft fractures and include not only other musculoskeletal injuries but also systemic injuries to the chest, head, and abdomen. Clearly any other extremity can be involved, but particular care should be given to the affected leg, specifically the hip and knee joints. The most common injury around the hip is a concurrent femoral neck fracture which has been reported to occur in approximately 2.5 % of femoral diaphyseal fractures [4]. As noted it is critical to assess for a femoral neck fracture as this injury can easily be missed in both conscious and unconscious patients. However a concurrent hip dislocation, pelvic ring injuries, and acetabulum fractures can also be present depending on the mechanism of injury.

About the knee, the most commonly associated injury is a ligamentous injury, specifically injury to the PCL. This instability can be difficult to detect before the fracture is stabilized, so the knee should be thoroughly examined after fixation. Surgeons should also assess the limb for an ipsilateral tibia fracture known as a "floating knee." These patients tend to be very seriously injured, and almost 30 % of patients with a "floating knee" have other significant musculoskeletal injuries in the same limb [5] (Fig. 6.2).

The most commonly associated systemic injuries that occur in patients with femoral shaft fractures are significant head or thoracic injuries. Abdominal injuries tend to be less common and, if present, often signify a concurrent pelvic ring injury. The severity of head and chest trauma can significantly influence the manner of treatment of femoral shaft fractures, especially as it relates to timing of fixation [6]. This matter will be discussed in a later section.

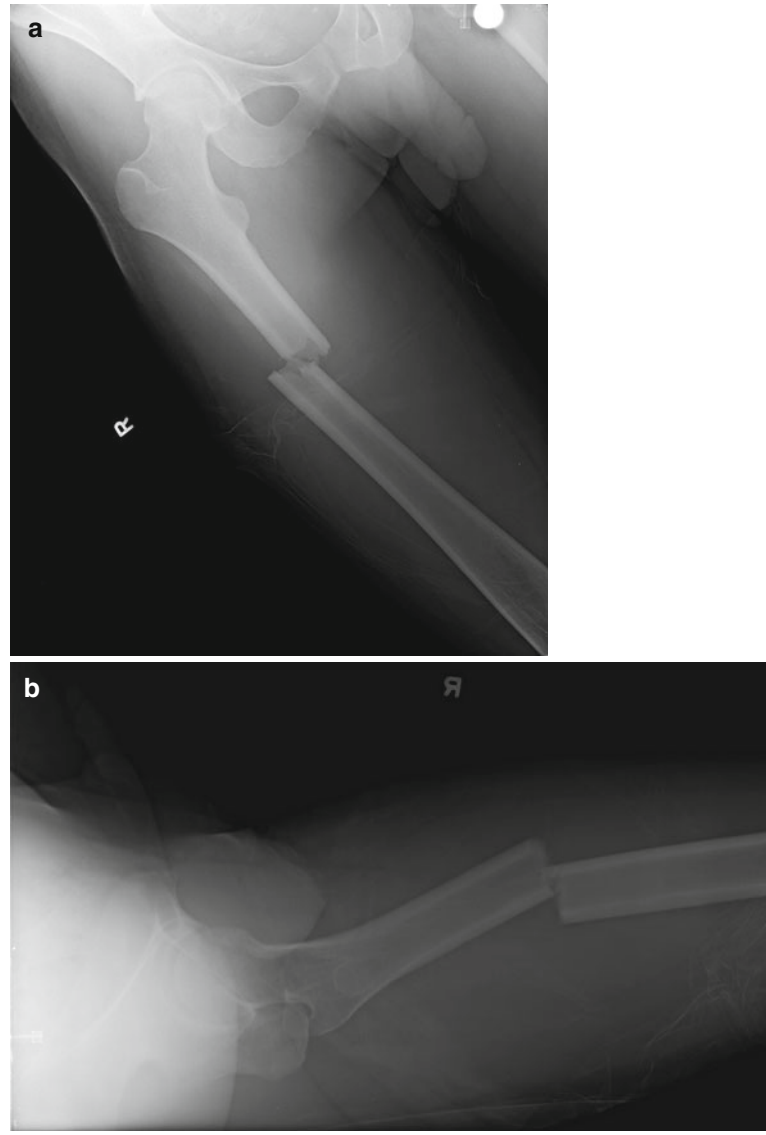


Fig. 6.1 These are examples of inadequate plain radiographs of a femur fracture. (**a**) AP radiograph; (**b**) lateral radiograph. As the knee joint is not included on the radiograph, a distal femur fracture could be easily missed. It is imperative that the entire bone be included in the radiographic series

Initial Management

Nonoperative management of femoral shaft fractures in adults is not appropriate except where adequate equipment, technology, and personnel are not available. Indeed, surgical stabilization is established as the standard of care for almost all femoral shaft fractures. Timing of surgical stabilization has become a more significant variable, even if definitive stabilization has to be delayed secondary to associated injuries. Stabilization should be performed as soon as the patient has been appropriately resuscitated and is felt to be stable from any concomitant systemic traumatic injuries. Some controversy exists in regard to timing of treatment in polytrauma patients with concurrent chest and/or head injuries, and this matter will be examined in a later section.

Traction

Placement of a distal femoral traction pin is a good temporizing measure and can always be

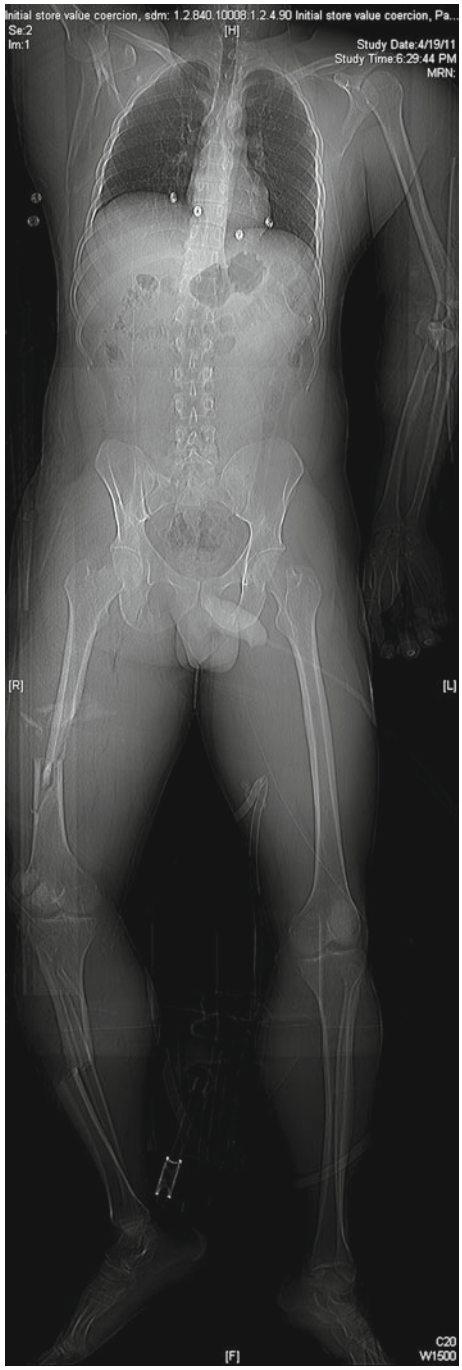


Fig. 6.2 This is a scout film from a CT scan performed on a polytrauma patient. Notice the ipsilateral femoral shaft fracture and tibia fracture or "floating knee." Also noted on this scout film is a left elbow dislocation

considered in patients that are too unstable to go to the operating room within the first several hours after injury. This procedure can be done in the emergency department or in the ICU and will help stabilize the bone and soft tissue envelope while holding the fracture out to length prior to definitive fixation. A pin placed 3–4 cm proximal to the superior pole of the patella and midlateral to slightly anterior in the femur usually avoids an intra-articular path, which is important if the pin must be utilized more than temporarily (Fig. 6.3). Twenty-five pounds of traction is usually sufficient. The goal is not to reduce the femur fracture but to keep the limb from shortening and control the muscle spasms and further fracture instability that accompany a femoral shaft fracture. The traction pin can also be used during nailing if prepped into the field or can be removed prior to the procedure. Patients can remain in skeletal traction for as long as necessary for appropriate resuscitation and while awaiting clearance from other services. Placement of proximal tibial traction pins for femur fractures is less utilized, as traction must be pulled through a possibly injured knee joint. There have been no studies to show an increased rate of infection in femurs that were treated initially with distal femoral traction and converted to intramedullary nails.

External Fixation

Application of a uniplanar external fixator to femoral shaft fractures can be a very good temporizing step in a patient who may be too unstable for definitive operative management. The best indication for external fixation is in the polytraumatized, under-resuscitated patient already in the operating room (e.g., following an exploratory laparotomy), although it should be considered in patients with a large open, contaminated soft tissue injury or in an extremity with a vascular injury [7]. A simple external fixator should be placed with two pins proximal and two pins distal to the fracture site, typically with pins oriented from anterior to posterior or medial to lateral or somewhere in between those corridors. This quick procedure should leave the limb out to length and stable even for an extended stay in the critical care unit.

Placement of a temporizing spanning external fixator presents no barrier to definitive fixation

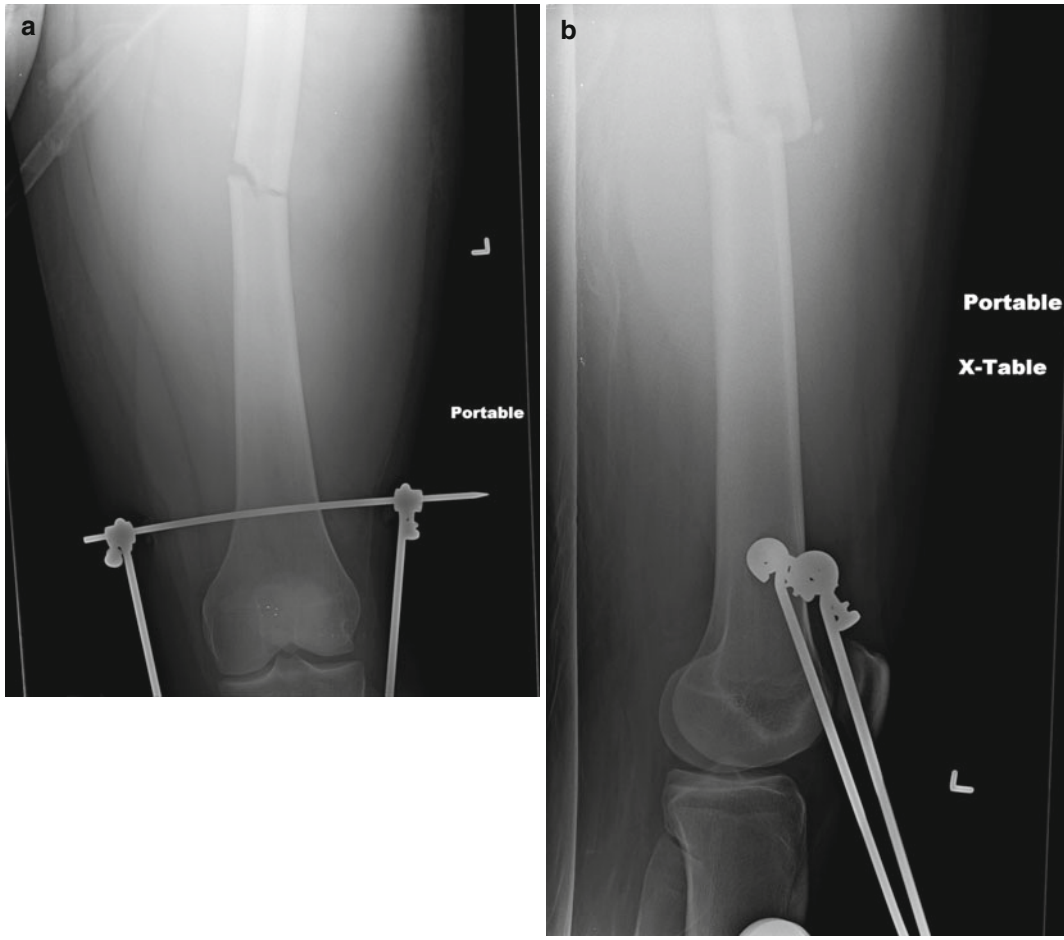


Fig. 6.3 An AP (**a**) and lateral (**b**) radiograph of the knee following placement of a distal femoral traction pin. Note the position of the pin approximately 3 cm proximal to the superior pole of the patella and slightly anterior. This pin position can facilitate nailing as a guidewire, and intramedullary nail can pass posterior to this traction pin during operative stabilization

with an intramedullary nail. Nowotarski et al. [8] examined the role of initial external fixation with early conversion to intramedullary nail in multiply injured patients. They examined 1,507 patients treated with nailing, and 59 (4 %) of those were treated with early external fixation in patients who were deemed to be critically ill and poor candidates for an immediate procedure. These patients had a mean Injury Severity Score of 29 on initial exam. All fractures were stabilized with a unilateral external fixator within the first 24 h after the injury. The average operative time was 30 min for placement of the external fixator. The average duration before a conversion to an intramedullary nail was 7 days and the infection rate was 1.7 %, which is comparable to the infection rate in immediate intramedullary nail placement. They therefore concluded that immediate external fixation followed by early closed intramedullary nailing is a safe treatment method for fractures of the shaft of the femur in selected multiply injured patients [8]. Many centers routinely perform conversion to nail up to 2–3 weeks post-injury provided no pin tract problems are evident.

Immediate fixation

Immediate definitive treatment is an option in a patient with an isolated femur fracture or in a polytrauma patient that has been sufficiently

resuscitated. A stable patient will be more comfortable following stabilization of this long-bone fracture, and fixation should be done as soon as is reasonable for patient comfort as well as to lower the risk of further local and systemic injury due to the unstabilized fracture. Fixation within 24 h is generally endorsed, indicating that while emergent night work is not typically required, expeditious scheduling should be undertaken.

Treatment

Plating: Compression Versus Bridge

Indications for plate fixation of a femoral shaft fracture are limited with the advent of modern nailing techniques. However, plating can be considered in a patient with large open wounds, if the instrumentation, expertise, or imaging capability for intramedullary nailing is not available; in periprosthetic femur fractures; and in some select pediatric femur fractures. Plating can be accomplished through a subvastus lateral incision, or submuscular plating can be performed using a percutaneous technique. Wenda et al. demonstrated that closed plating techniques merely increase the difficulty of the operation without imparting any real benefit [9]. When performing a lateral incision, however, surgeons must control bleeding from the perforating vessels. Plating is not recommended for most femoral shaft fractures if interlocked intramedullary nailing is available. Also, as plating represents a load-bearing type of construct as opposed to the load-sharing construct of an intramedullary nail, patients often require protected weight bearing for 12–14 weeks, which may negate some of the anticipated benefits of early fixation and mobilization.

Compression Plating

Standard compression plating can be done in any simple fracture pattern (transverse, spiral, short oblique) through a direct lateral approach or through a traumatic wound. Anatomic reduction of the fracture in these simple patterns can be performed directly with reduction clamps. Interfragmentary lag screws should be placed if possible, and then a large fragment compression plate can be placed laterally to be used as a neutralization plate. Current recommendations suggest three to four well-spaced bicortical screws proximal and distal to the fracture site through a 10–14-hole plate to provide long, balanced fixation.

Bridge Plating

If there is a significant amount of comminution present, bridge plating can be considered. Reduction of the fracture mostly occurs indirectly in these fractures. Direct manipulation of fragments can be undertaken, but care should be taken to avoid stripping the soft tissues from any pieces. Correct length, alignment, and rotation of the femur must be restored. Longer plates are typically required and should ideally have a bow in order to restore the patient's normal anatomic femoral bow. Comparison radiographs of the uninjured leg can be a helpful adjunct in significantly comminuted fractures or in open fractures with a significant amount of bone loss. One can also consider prepping the nonoperative limb into the operative field for direct comparison.

Percutaneous bridge plating is also an option. A small incision is made through the tensor fascia lata at the level of the epicondyle distally, and a counter incision is made laterally at the level of the vastus ridge. The plate can then be slid submuscularly in a retrograde fashion. Typically the plate is then attached to the bone with a single bicortical screw at one of the incisions, and the leg is then manipulated to gain a reduction prior to fixation through the opposite incision. Care must be taken as mentioned above to assure that the correct length, alignment, and rotation has been accomplished prior to fixation. Additional screws can then be placed percutaneously into the plate with the goal of four well-spaced bicortical screws on both sides of the fracture site.

Intramedullary Nail

Where available, reamed, locked, intramedullary nailing represents the standard of care for femoral

diaphyseal fractures. Outcomes following treatment of femoral diaphyseal fractures with intramedullary nailing are excellent, and the complications are minimal. Non-reamed nails have been associated with a higher incidence of nonunion and hardware failure and should not be used. In addition to benefitting union rates, limited reaming does not increase the incidence of malunion, infection, pulmonary embolism, or compartment syndrome [10].

Antegrade Nailing

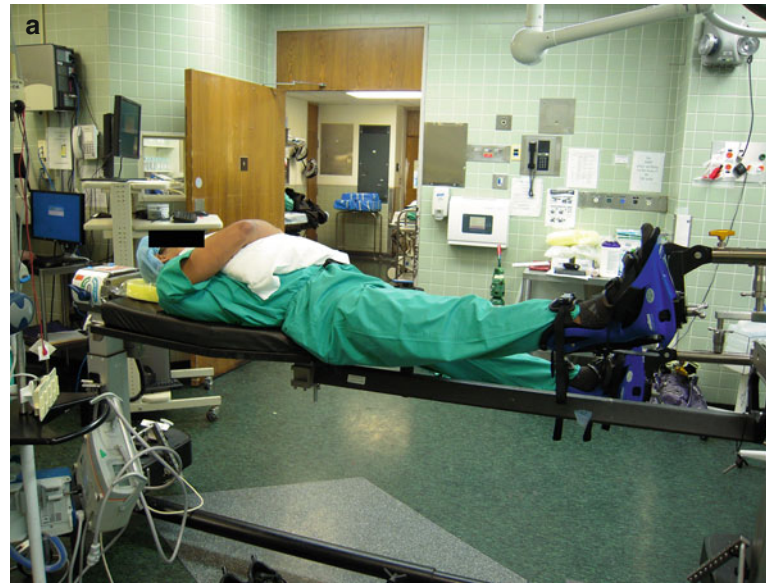
Most femoral diaphyseal fractures can be managed with an antegrade femoral nail, and antegrade nailing is the preferred method of treatment of these authors. Common positioning strategies include supine on the fracture table with the limb in a traction boot or skeletal traction, supine on a radiolucent table with a large bump underneath the patient's injured hip and the entire limb draped free into the field, or lateral either on a traction table or with the limb draped free. Each position has its advantages and disadvantages. Use of a fracture table may be helpful when a surgeon does not have any assistants as the fracture position is maintained by the traction (Fig. 6.4). The fracture tends to sag on the fracture table, however, and reaching the piriformis fossa can sometimes be a challenge, especially in obese patients. The supine position on a radiolucent table is preferentially employed at many centers where ample help available. Setup time is minimal. The limb can be manipulated during the procedure to assist in obtaining an appropriate starting point in the piriformis fossa or greater trochanter, depending on the type of nail selected. Assistants are necessary to manipulate the limb, or a distal femoral or proximal tibial traction pin may be placed to facilitate regaining length and achieving reduction (Figs. 6.5 and 6.6). Finally, the lateral position is extremely useful when nailing morbidly obese patients in an antegrade fashion, but setup time can be quite extensive. This position provides easier access to the piriformis or trochanteric starting point. It can also be advantageous in very proximal femoral shaft fractures in order to counteract the usual flexion deformity in these types of fractures. However, fluoroscopic visualization of the proximal femur is difficult in the lateral position, and care must be taken to avoid allowing the distal fragment to drift into valgus because of gravity.

As noted, modern nail instrumentation allows the surgeon to choose nails designed to be inserted either through the piriformis fossa in line with the canal or through the greater trochanter slightly lateral to the femoral canal. No compelling data demonstrate superiority of one approach over the other. Both nail types contain an anterior bow, and the trochanteric nails also have a lateral bend proximally to account for the off-axis starting point. Modern straight femoral nails should not be placed through a greater trochanteric insertion point because the femoral neck is at risk of being damaged by the stiff nail, and this tends to malreduce the fracture. Similarly, one must be very meticulous with reduction of the fracture when using trochanteric start nails to assure that the implant does not force the femur into varus upon insertion.

It is also critical to ream the canal with the fracture site well reduced. Fluoroscopy should be used during reaming to assure that the canal is not eccentrically reamed at the fracture site. Passage of the nail will facilitate improved alignment when the fracture is in the isthmus, the narrowest portion of the femoral diaphysis. However, if the fracture is proximal or distal to the isthmus and the canal eccentrically reamed, reduction may not be affected by nail passage. Most sources now recommend a ream to fit approach, and most manufacturers recommend over-reaming 1 mm larger than the selected nail size. In straightforward diaphyseal femur fractures, a smaller nail (typically an 11 or 12 mm nail) is sufficient in most adult femur fractures.

Retrograde Nailing

Multiple established indications for retrograde femoral nailing include but are not limited to ipsilateral femoral neck fractures identified prior to nailing, ipsilateral tibia fracture ("floating knee" injuries), ipsilateral acetabulum fractures, bilateral femur fractures, morbid obesity, and pregnancy. Some centers preferentially treat virtually all femoral shaft fractures with a retrograde reamed nailing technique, citing ease of



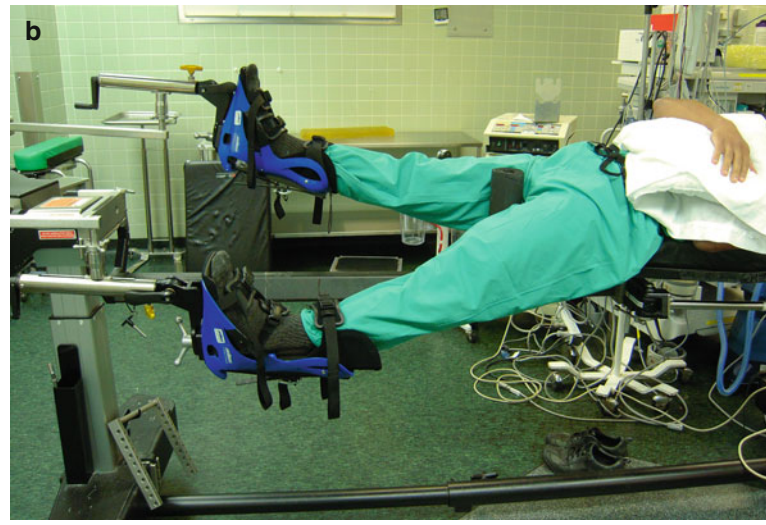


Fig. 6.4 This is a demonstration of positioning a patient supine on a fracture table for intramedullary nailing of a right femur fracture. (**a**) Positioning with both legs into traction boots and the arm on the affected side brought across the body to facilitate nailing. (**b**) Note that the non-affected limb is also placed in a traction boot and dropped down to facilitate fluoroscopic evaluation of the affected limb. The nonoperative limb can also be placed into a well-leg holder if the surgeon prefers. (**c**) The patient from the end of the table, with the well leg dropped out of the operative field. It should be noted that with this technique, the femur fracture tends to sag on the fracture table; this deforming force will have to be overcome in order to reduce the fracture. A crutch placed under the drapes can be useful to help counteract this sag

positioning, relative rapidity of the procedure, and equivalent functional results as support. As in antegrade nailing, the starting point is critical in retrograde nailing and must be in line with the somewhat more distant canal to avoid creating angular malalignment. The knee joint and proximal femur must both be adequately visualized using fluoroscopy. The nail is inserted through an entry point in the knee joint at the level of Blumensaat's line on the lateral view and just anterior to the posterior cruciate ligament fibers.

Controversies

Multiply Injured Patient/ Damage Control

The dogma of early total orthopedic care for multiply injured patients has undergone a dramatic shift over the last decade. With the advent of widespread ATLS protocols and critical care advances, more patients are surviving injury constellations that were frequently fatal in the past.

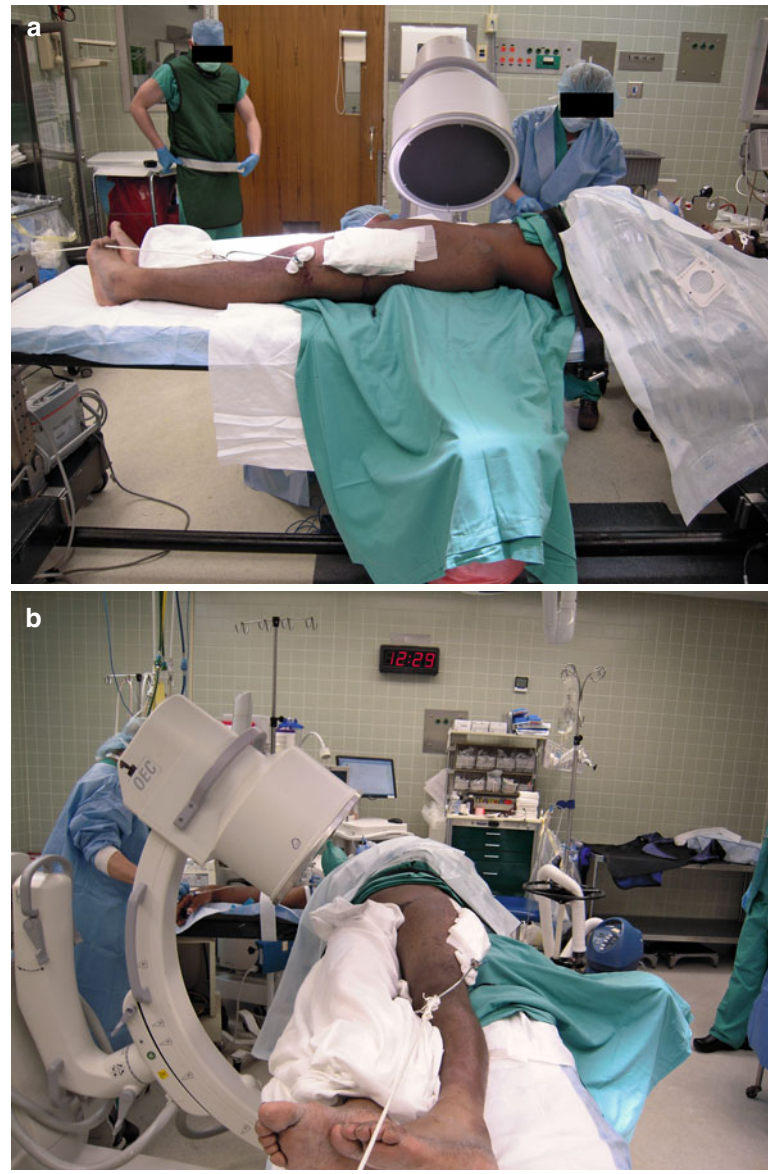
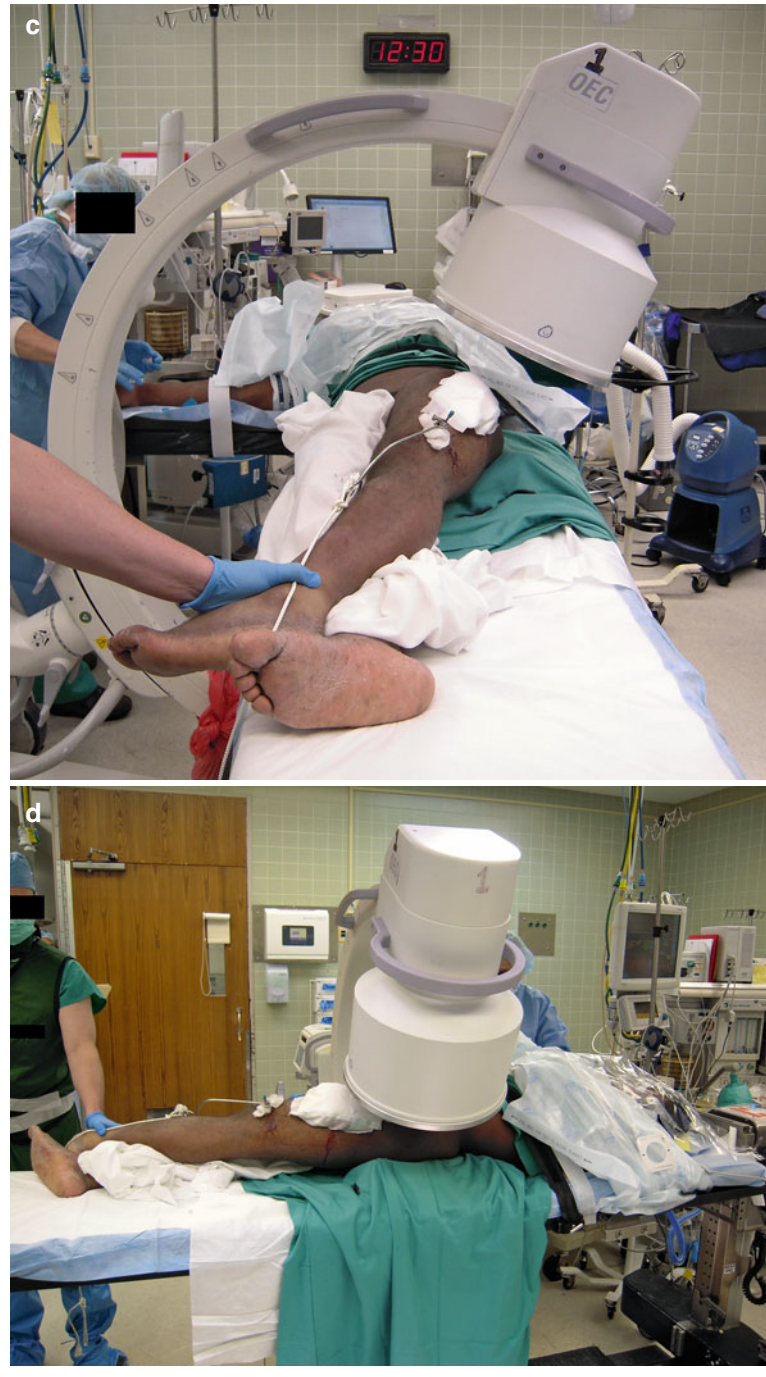


Fig. 6.5 An example of the positioning of a patient with a left femur fracture supine on a radiolucent table. The patient had a previously placed distal femoral traction pin during prolonged resuscitation following multiple gunshot wounds. (**a**) The bump placed under the fractured limb, allowing easier access to the piriform fossa. (**b**, **c**) Position of the image intensifier for obtaining an AP radiograph. (**d**, **e**) Position of the image intensifier for obtaining a lateral radiograph of the proximal femur. Occasionally the limb has to be manipulated from the foot in order to obtain a good lateral radiograph. The traction pin may also be removed prior to the procedure or prepped into the field with use of a sterile traction bow and sterile rope. If this is to be utilized, the traction should be taken off the opposite side of the distal end of the table, pulling traction medially and adducting the limb

Fig. 6.5 (continued)



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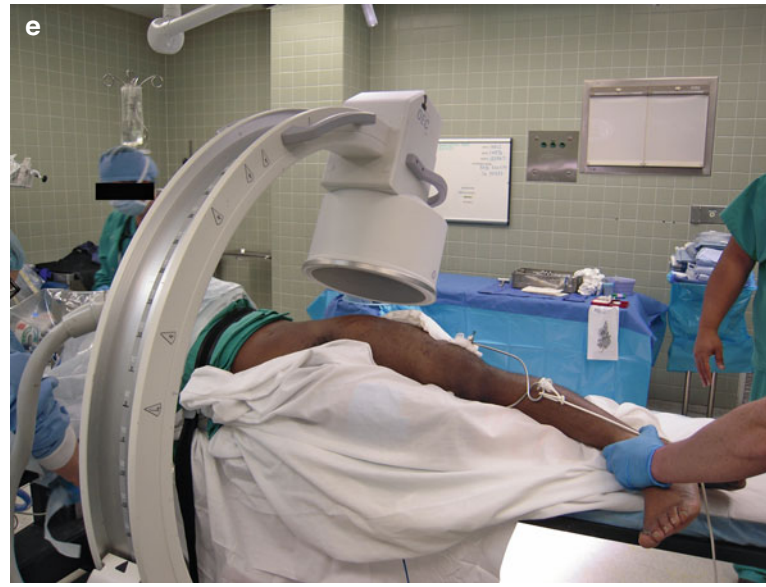
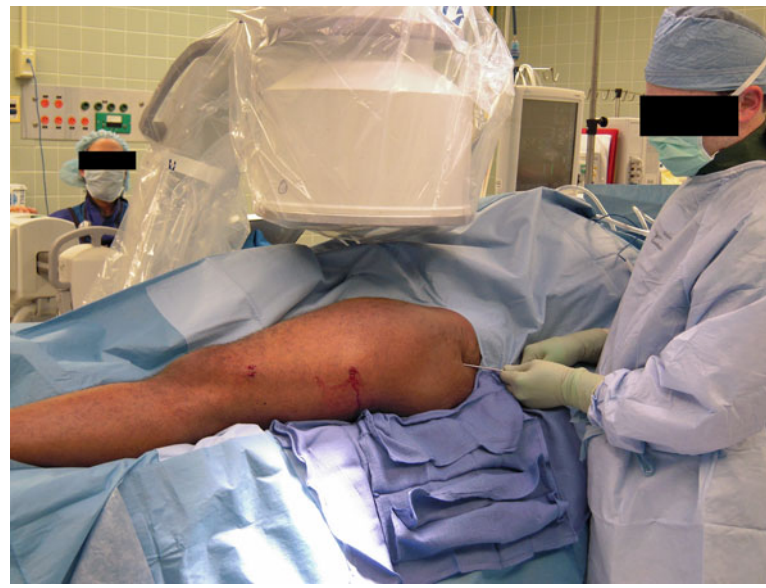


Fig. 6.6 An example of placement of a guidewire with the patient in a supine position. In a thin patient, this can be done with relative ease in this position



Critical care and orthopedic surgeons, in response to evidence supportive of appropriate, targeted early intervention for the most seriously injured patients, have adapted their practices to incorporate damage control measures when early total care is not appropriate. This practice involves early often temporary stabilization of injuries via brief procedures with limited blood loss in order to assist in the resuscitation of multiply injured patients.

Laboratory values as well as vital signs and urinary output are all good measures of a patient's level of resuscitation and provide guidelines for the timing of any sort of operative intervention on an injured femoral shaft. The lethal triad in polytrauma patients includes hypothermia, metabolic acidosis, and coagulopathy. Serum lactate levels of less than 2.5 mmol/L, a base excess of greater than 8 mmol/L, a temperature of less than 35 °C, and a pH of less than 7.24 are all indicators

of hypoperfusion and under resuscitation in a polytrauma patient [11]. Crowl et al. [12] demonstrated a significantly higher proportion of postoperative complications in patients who were not completely resuscitated (50 %) when compared to patients that were completely resuscitated (20 %) as evidenced by a serum lactate level of greater than 2.5. Patients whose resuscitation fails to correct these parameters likely are not yet safe candidates for intramedullary definitive stabilization of the femur and should be considered candidates for damage control measures like external fixation or skeletal traction.

The other phenomenon that must be considered is the idea of a "first hit" and "second hit" that are often sustained by trauma patients. The first hit includes anything that occurs during the initial trauma. This includes the initial organ, bony and soft tissue injuries, as well as any initial hypotension, hypoxia, or hypothermia. The second hit occurs at the hospital and includes any surgical procedures that can lead to a secondary time of blood loss, hypoperfusion, hypoxia, ischemia, reperfusion, and tissue damage [6]. All of these things must be taken into consideration when determining the surgical timing for all long-bone injuries and specifically for femoral diaphyseal fractures.

Brain-Injured Patient

There is some controversy regarding the timing of intramedullary nail placement in patients that have a concurrent significant head injury. The concern has been that intraoperative hypoxia and hypotension that commonly occur during intramedullary nail stabilization of femur fractures may lead to aggravation of the patient's brain injury [11]. Other studies, however, have shown little to no effect on the patient's brain injury whether or not definitive fixation was delayed.

Poole et al. reported a series of 114 patients with fractures of either the femoral or tibial shaft who also had head injuries [13]. Adverse cerebral effects in their series were unrelated to the time of fracture fixation of the lower extremity and seemed to be more affected by the severity of the initial injury. The authors of this study recommended early fracture stabilization as it simplified patient care.

Starr et al. also performed a retrospective review of 32 femur fractures in brain-injured patients, 14 of which underwent immediate intramedullary nailing and 18 underwent delayed or no stabilization of their fractures [14]. They found no increase in prevalence of CNS complications with immediate stabilization of femur fractures.

An additional concern has been raised in regard to the high rate of fat embolism syndrome that can be caused by intramedullary nailing. This can occur in up to 15 % of femoral shaft fractures, and the mental status changes that can occur in a patient with fat embolism syndrome can confound treatment in a patient with a concomitant head injury. Smith and Cunningham [15] presented a study that retrospectively reviewed the risk of adverse neurological complications in patients with traumatic brain injuries undergoing femoral intramedullary nailing. They were unable to demonstrate that early fracture fixation caused any increase in the severity of CNS complications.

A literature review was performed by Dunham et al. [16] that found in patients with brain injury, there appeared to be no advantage or disadvantage from a neurological standpoint with early versus delayed orthopedic stabilization. They found no difference in mortality, stay in the ICU, or overall hospital stay. With this data, it can be determined that femoral diaphyseal fracture fixation may proceed once the patient has been initially resuscitated and stabilized from a neurological standpoint, provided the critical care team and the neurosurgeons concur.

Chest-Injured Patient

Perhaps the most controversy when it comes to timing of intramedullary femoral nailing involves those patients with a significant concurrent chest and lung injury. Early research demonstrated that early intramedullary fracture fixation was important and advantageous in decreasing the

rate of ARDS and pulmonary complications in polytrauma patients. However, the Hannover study group challenged this view. Pape et al. [17] retrospectively examined 766 polytrauma patients. Those patients were divided into four groups depending on whether they had femoral stabilization within the first 24 h and whether they had a severe chest injury. In those with severe chest trauma, they found a higher incidence of ARDS and mortality in the group treated by early femoral nailing. A secondary study [18] by the same group found that lung function was unaffected by the use of small unreamed femoral nails but deteriorated with the use of reamed nails, only to improve 48 h later. They found an increase in pulmonary artery pressure during reaming and believed that this increase in pressure could trigger ARDS in at-risk patients.

Subsequently, an abundance of animal and clinical studies were performed to investigate the effects of reamed intramedullary nailing on multiply injured patients. Wolinsky et al. [19] used a sheep model to evaluate this effect. They created an ARDS-like state in these sheep prior to reaming in some sheep and compared them to sheep with no pulmonary dysfunction. They found no evidence that reamed intramedullary nailing in a sheep that had been appropriately resuscitated had any statistically significant effect on pulmonary dysfunction.

Brundage et al. [20] examined more specifically the timing of fracture fixation on patients with thoracic and head injuries. They retrospectively reviewed data from a Level I trauma center and identified 1,362 patients with a femoral shaft fracture over a 12-year period. Five groups were categorized based on timing of femur fracture fixation: Group 1 within 24 h, Group 2 within 24–48 h, Group 3 within 48–120 h, Group 4 >120 h, and Group 5 with no operative fixation. They then looked at morbidity (specifically pulmonary complications) and mortality, as well as ICU length of stay, hospital length of stay, and discharge Glasgow Coma Scale score. They found that ARDS, pneumonia, hospital length of stay, and ICU length of stay were the lowest in the group fixed within the first 24 h, even in patients with concomitant head or chest trauma. They discovered that fixation between 2 and 5 days was associated with a significantly increased incidence of pneumonia, ARDS, and fat embolism syndrome in patients with chest trauma. The highest discharge GCS score was also found in the patients fixed within 24 h. They were then able to conclude that early femur fracture fixation (<24 h) was associated with improved outcome in all patients, including those with coexisting head and/or chest trauma. However, surgeons should be aware that there was a significant increase in pulmonary complications in those patients treated within 2–5 days.

Elderly Patient

Femoral nailing in the elderly population is likely to increase as the number of elderly patients increases. Prolonged bed rest is not well tolerated in the elderly population. Plate fixation in osteoporotic bone is often biomechanically inferior, and many elderly patients do not possess the upper body strength to remain partial weight bearing for prolonged periods. Therefore, intramedullary nailing remains the preferred method of treatment for elderly diaphyseal fractures. Care must be taken preoperatively to assure that the fracture did not occur through a metastatic lesion, and consideration may be given to placing a cephalomedullary device in order to protect the femoral neck as well in a patient who has significant osteoporosis.

Moran et al. [21] demonstrated the problems and complications following intramedullary nailing in elderly patients. Their series of 24 fractures in patients with an average age of 77 years demonstrated a high complication rate (54 %) as well as a high mortality rate (17 %). They noted the difficulties of operating on osteoporotic bone, as well as operating on osteoporotic hips. They recommended lateral positioning in patients with concurrent ipsilateral hip arthritis with a flexion deformity. They also recommended the use of cement augmentation in severely osteoporotic bone. Despite the difficulty and high complication rate, however, intramedullary nailing remains the best treatment option in elderly patients.

Morbidly Obese

According to statistics available by the National Center for Health Statistics (2009), obesity rates have more than doubled in adults and children. Two-thirds of adults in the United States are now considered overweight or obese based on these statistics. Orthopedic surgeons in the United States already encounter more obese and morbidly obese patients in their practices. The challenges are numerous in these patients and begin with appropriate preoperative evaluation of all concurrent medical problems. Intraoperatively, positioning, adequately visualizing the limb (specifically the hip) under fluoroscopy, and obtaining an appropriate starting point are some of the biggest challenges. Postoperative complications are also much more numerous in these patients, including respiratory distress and deep vein thrombosis.

McKee and Waddell [22] looked at the role of intramedullary nail placement in the management of morbidly obese patients with femoral diaphyseal fractures. They reported on seven patients with an average weight of 300 lb. They drew attention to the considerable difficulty in establishing the correct insertion point for antegrade nailing, and this difficulty resulted in a fracture of the greater trochanter in two patients. They recommended use of the lateral position to assist with obtaining a starting point in these patients. They also noted a high incidence of deep vein thrombosis (4 patients) and pulmonary embolism (2 patients—1 of which was fatal). As stated, many centers prefer to manage morbidly obese patients with a retrograde nailing technique, although medical complications are not reported to be different with this technique.

Bone Loss

Bone loss can be a significant problem, especially in high-energy injuries. Treatment depends primarily on the volume of bone that will need to be replaced, coupled with an increased risk of infection as these injuries are typically generated by open fractures. Typically up to 5 cm of bone loss can be replaced by corticocancellous autograft placed around an intramedullary device. The Masquelet technique [23, 24] has produced promising results when dealing with this length of bone loss. During the definitive closure of the wound, an antibiotic cement spacer is placed circumferentially around the fixation within the defect. A biologically active pseudomembrane forms around this spacer over 6–8 weeks. When returning for spacer removal and grafting, this pseudomembrane provides a bed for placement of autograft and has been shown to secrete growth factors that could help stimulate bone regeneration [24].

When the bone loss exceeds 5–6 cm, it is often treated much like one would treat bone loss following debridement of an infected long bone. Depending on the amount of bone needed, one can consider massive autogenous bone grafting, vascularized bone grafts, shortening procedures, or bone transport.

Complications

General complications of femur fractures that are not specific to any method of fixation include nonunion, malunion, compartment syndrome, and infection. As these are common complications in any long-bone fracture, we will not specifically address these. Briefly, care must be taken to assure any open wound is appropriately debrided and that appropriate sterile technique is used. Also, the surgeon should always be meticulous in obtaining and maintaining a good reduction (especially rotational alignment) during fixation, as malunion rate following intramedullary nailing has been reported to be as high as 5–10 % [25] while the union rate is 98–99 %.

Plating

Complications following plating of femur fractures are numerous. Nonunion, malunion, infection, hardware failure, and refracture are some of the complications that can occur following plating of femoral diaphyseal fractures. Malunion is

by far the most common complication and can usually be avoided with meticulous surgical technique during the initial procedure. Delayed unions have been found to be slightly higher in femoral plating when compared to intramedullary nailing. Infection rates have not been shown to be any higher than with modern nailing techniques.

Hardware failure is the most problematic complication following femoral shaft plating. This complication has been reported to occur in 1–11 % of femur fractures treated with plate fixation. The increased risk of hardware failure results from the need to achieve early bone healing and stability prior to allowing weight-bearing activities. Complete healing of a femur fracture typically takes 16 weeks, so the typical need to protect these load-bearing implants for several months presents challenges to the patient's ability to comply and rehabilitate and the device's ability to maintain fixation. Late complications such as fractures at the ends of plate fixation of femoral shaft fractures can occur, especially in osteoporotic patients in whom the plate ends represent significant stress risers between the plate-supported segment and the unsupported native femur.

Antegrade/Retrograde Nailing

Ipsilateral Femoral Neck Fracture

Numerous complications can be associated with femoral nailing, the most detrimental of which is an unrecognized femoral neck fracture (not rare) or an iatrogenic femoral neck fracture (very rare) with an ipsilateral femoral diaphyseal fracture. Missed fracture of any kind following treatment of femoral shaft fracture is around 3–7 %, and ipsilateral fracture of the femoral neck can occur in as many as 1–9 % of femoral shaft fractures [26]. Good anteroposterior radiographs with the hip internally rotated must be performed following all femoral fixation procedures to assure that no occult femoral neck fracture is present. When available the bony windows of the femoral neck on the trauma abdomen/pelvis CT scans should also be reviewed preoperatively. If a fracture is identified intraoperatively, the femoral neck should be promptly reduced and stabilized. If the neck fracture is nondisplaced and the nail is already inserted, screw fixation anterior or posterior to the nail is usually sufficient. If the neck fracture is displaced at the time of intraoperative discovery, often the nail requires removal to facilitate anatomic reduction and fixation of the neck. Any patient complaining of hip pain and irritability early after fixation of a femoral shaft fracture should be carefully evaluated for a possibly missed femoral neck fracture.

Heterotopic Ossification

Ectopic bone formation is primarily a complication in antegrade nailing and can occur within the hip abductor musculature following the procedure. Some reports have shown an incidence of heterotopic ossification (HO) in up to 68 % of antegrade intramedullary nailing procedures [27]. Typically, this is limited to a small amount of ossification just proximal to the end of the nail but can occasionally be much more severe, causing decreased hip range of motion and even ankylosis.

There are several factors that may increase the likelihood of HO formation, both systemic and related to the procedure itself. The systemic factors include significant head injuries and severe burns, both have which have been shown to significantly increase the risk of a patient forming HO. Procedural factors that increase risk include the magnitude of muscle injury that occurs during the procedure as well as the amount of reaming debris that remains in the musculature following reaming. Because of this, it is important to use soft tissue protectors while reaming and to adequately irrigate the hip wound following nail placement prior to closure. Severe HO formation may cause significant hip dysfunction by limiting range of motion and causing significant pain, both of which can cause the patient to walk with a limp. Affected patients should be followed closely with sequential radiographs in order to assure that HO excision is not necessary to allow appropriate hip range of motion. Because this is typically a relatively minor complication, routine HO prophylaxis is not currently recommended.

Rotational Deformity

Assessment of rotational alignment can be difficult to perform in femoral diaphyseal fractures, especially with increasing amounts of comminution. Tornetta et al. [28] examined 22 patients treated with static intramedullary nailing and found the average malrotation to be approximately 16°. Ten of these were placed in external rotation, the more common of the rotational deformities. They found no difference between the results of nailing in the supine or lateral position. However, the patients in their study compensated well, and none underwent derotational osteotomy.

Several authors have published ways to use fluoroscopy intraoperatively to accurately assess femoral rotation intraoperatively. The various methods all involve assessing the rotation by facing the patella directly anterior in the supine position (parallel to the floor in the lateral position) and comparing the appearance of the femoral neck to the normal limb. This can be very difficult to accomplish accurately in the operating room. Fortunately, it appears that most patients tolerate a small rotational deformity, and unless the deformity is considerable, rarely do patients require a derotational procedure.

Knee Pain

Knee pain is the most common complication following retrograde femoral nailing and has been reported in up to 30 % of cases. This is likely related to the incision itself as knee pain is also reported as a primary complication in tibial nailing. Care must be taken in obtaining an appropriate starting point. One should also be diligent to use a soft tissue protector when reaming to protect not only the patella tendon but also the undersurface of the patella. It is also critical to assure that the nail is not left prominent within the knee joint. The nail should be buried deep to the subchondral bone.

Hardware Failure

Although intramedullary nailing is an extremely successful procedure, hardware failure can occur with these implants as well. The most commonly seen hardware failure is broken interlocking screws that occur with weight bearing through the construct. Screw breakage is typically of limited consequence provided the bone is length stable. Often screw failure allows slight compression through a slightly distracted fracture site and facilitates an otherwise slow-healing fracture to progress to union. The nail can experience fatigue failure at the fracture site in the event of a nonunion, especially if there is a large bony defect or segmental comminution, and fractures can occur at the tip of the implant as well (Fig. 6.7).

Guidelines

Isolated Femur Fracture

The isolated femur fracture should be treated with an antegrade reamed intramedullary nail as soon as the patient's condition and the schedule will allow. Retrograde nail management is also an acceptable alternative for some types of patients and centers. Typically these patients do not require a substantial amount of resuscitation, but any resuscitation should be addressed prior to taking any patient to the operating room. The nailing can be done at the surgeon's earliest convenience and need not necessarily occur in the middle of the night if the following day's schedule will allow for this to proceed in a timely manner the next day. Patients can typically be kept relatively comfortable with a long leg splint or Buck's traction until operative intervention can proceed. Serial exams should be performed on the limb if the case is delayed to check for any decrease in vascular status or development of a thigh compartment syndrome.

Multiply Injured Patient

Timing of fixation of femur fractures is the major controversy when addressing the multiply injured trauma patient. Fixation should proceed within the first 24 h after injury if the patient can be safely and successfully resuscitated in this time frame. Also, any life-threatening injuries should be addressed prior to consideration of

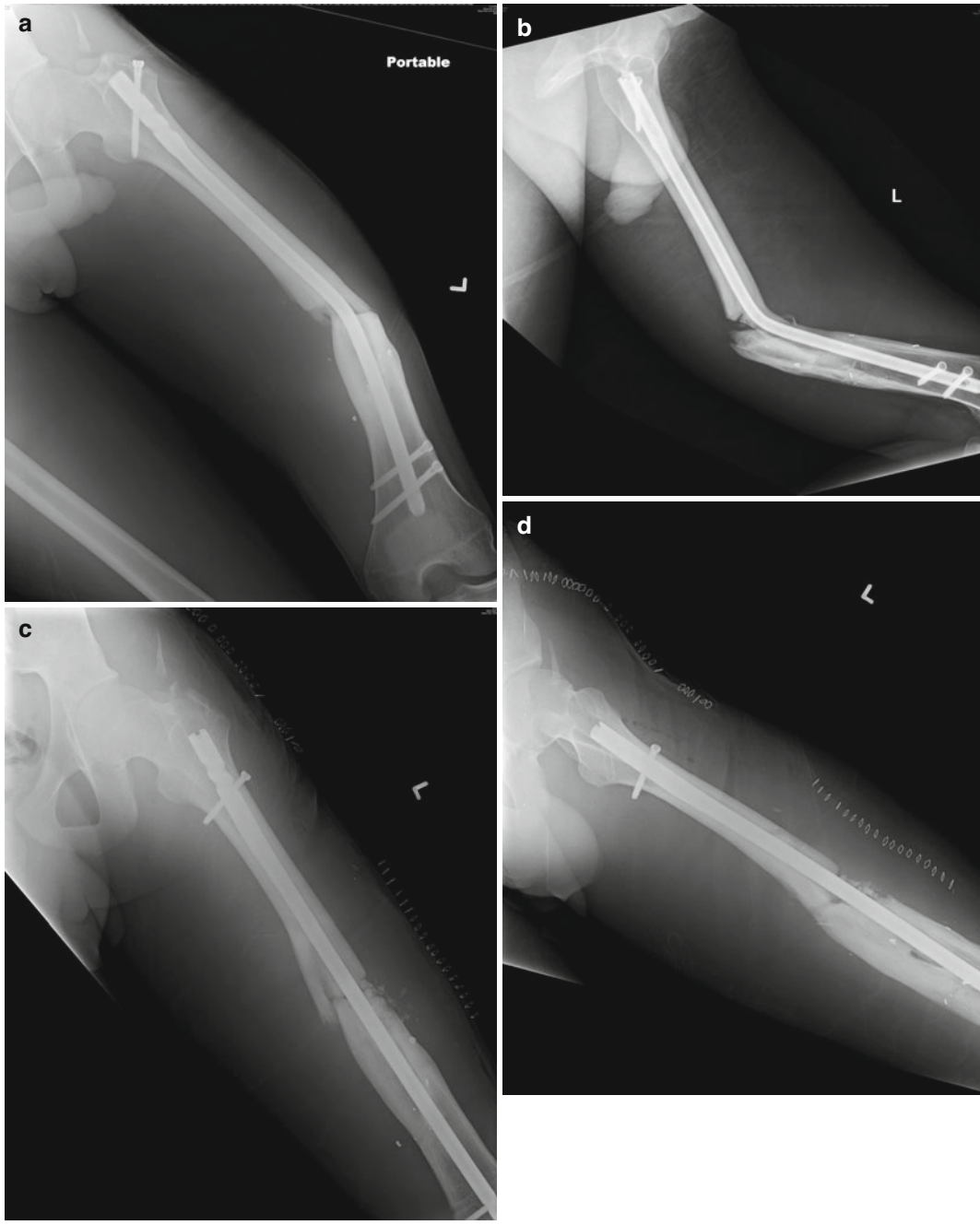


Fig. 6.7 (**a**, **b**) A novel complication following femoral nail placement. The patient had an uncomplicated femoral nailing performed at an outside facility following a gunshot wound approximately 5 years prior. This patient presented to our institution following a high-speed motor vehicle collision with a significant deformity to his lower extremity, and radiographs demonstrated this bent femoral nail through a suspected nonunion. However, the patient had no antecedent pain, so this may have represented a new fracture. (**c**, **d**) The stabilized fracture. The patient underwent removal of the bent femoral nail and subsequent antegrade femoral nailing. The patient went on to heal this fracture uneventfully

fixation of the extremity injuries. Early femoral nailing has been shown to have no detrimental effect on patients with concomitant traumatic brain injuries, and early fixation will often assist in the ICU care of these patients. However, if the patient has had significant chest trauma and the patient cannot undergo operative stabilization in the first 24 h, consideration should be given to avoiding operative intervention on hospital day 2–5 secondary to the increased rate of respiratory problems that have been shown to occur in this time frame. A temporary traction pin or uniplanar external fixation device can be very helpful adjuncts in patients who need more time for stabilization prior to definitive stabilization.

Open Fractures

As with all open fractures, open femoral diaphyseal fractures should proceed to the operating room in a timely manner. With small, non-contaminated wounds, definitive fixation can proceed at the same time as the irrigation and debridement of the open wound. With larger, contaminated wounds, placement of a spanning external fixator should be considered as a temporizing means of fixation until a clean wound bed can be obtained and arrangements made for soft tissue coverage if necessary.

Vascular Injuries

A femur fracture with a vascular injury is a surgical emergency. The patient should be taken to the operating room as quickly as possible in order to regain perfusion to the limb. Most orthopedists would agree that a timely fixation of the femur fracture should proceed prior to vascular repair in order to decrease the risk of disrupting the vascular repair with manipulation of the fractured limb. Stabilization can be performed with external fixation, but definitive fixation with either a nail or a plate is a reasonable method to avoid further manipulation at the fracture site following vascular repair, provided this work can be executed expeditiously without delaying revascularization. However, some vascular surgeons prefer to proceed with temporary bypass repair in order to regain perfusion to the limb during fixation, then following with definitive vascular repair. The order in which management will proceed is somewhat institution dependent, but there is no controversy that this is a surgical emergency and should occur as soon as the patient is stable for operative intervention.

Compartment Syndrome

Compartment syndrome can occur at the time of the injury secondary to soft tissue disruption and can also develop gradually secondary to bleeding from the fracture site into a closed muscle compartment. Serial extremity exams should be performed on anyone with a diaphyseal femur fracture both preoperatively and at least 24 h postoperatively, as a compartment syndrome can develop following fracture fixation. Clinical exam is typically sufficient in the conscious patient, but compartment measurements can be taken in obtunded patients. Compartments should be released as soon as a compartment syndrome is diagnosed in order to avoid permanent muscle damage.

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Pelvic Fractures

7

Navid M. Ziran and Wade R. Smith

Introduction

Pelvic fractures are traumatic injuries that may be caused from low- or high-energy trauma and are a leading cause of mortality from hemorrhage. A discussion of pelvic fractures would not be complete without a brief historical perspective of these injuries and the surgeons who studied them. Malgaigne first described fractures of the hemipelvis in 1847 [1]. In 1965, Peltier first proposed pelvic reduction, hemorrhage control via laparotomy, and resuscitation with blood products [2]. Huittinen and Slätis further correlated the mechanism of injury to pelvic ring and visceral damage [3]. They performed postmortem, latex injection studies of the pelvic vasculature and noted that 14 % of patients who died following a pelvic fracture had an arterial injury. Peltier's work would later be expanded upon by Pennal and Tile in the 1980s at which point they correlated disruption of the pelvic ring with direction and deforming forces [4–7]. Young and Burgess then furthered Pennal and Tile's work by (1) confirming the relation between the force vector and subsequent pelvic ring injury and (2) developing subgroup classifications based on the degree of disruption [8, 9]. In 1989, Matta published his principles of pelvic ring internal fixation [10]. Despite recent advancements, the majority of contemporary surgeons' knowledge and practice still originates from the work discovered over the past 50 years. Recent progress in early identification of shock, aggressive resuscitation methods, and operative stabilization offers orthopedic and general trauma surgeons more opportunity than ever to have a strong impact on patient mortality. This chapter will summarize the diagnosis, initial management, and recent controversies of pelvic ring injuries.

Background

The majority of pelvic fractures are mechanically and hemodynamically stable. However, a subgroup of pelvic fractures, usually associated with higher-energy forces, can result in significant bony and soft-tissue disruption with subsequent hemodynamic instability from blood loss. These fractures are the true "unstable" pelvic injuries. While often associated with a biomechanically "unstable" fracture, the key element is physiologic, and not mechanical, instability. In fact, numerous reports have documented that physiologic instability can occur from pelvic bleeding, despite fracture patterns with minimal displacement. Thus, the key definition of an unstable pelvic fracture must incorporate physiologic instability.

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Unstable pelvic fractures result in significant morbidity and mortality—usually from exsanguination (within the first 24 h), associated visceral or brain injury, resuscitation intervention, and/or multiple organ failure (after 24 h) [11]. Mortality rates for pelvic fractures vary widely in the literature. This discrepancy is due to the comparison of dissimilar cohorts and the absence of standardized definitions for stable and unstable injuries. When authors focus on cohorts of patients with physiologic evidence of instability, such as shock or the need for blood transfusion, mortality is high in every study and does not appear to have decreased significantly in the past 20 years. Cryer et al. [12] demonstrated that 50–69 % of unstable pelvic fractures will need 4 or more units of blood and 6–18 % will have an arterial injury. Starr et al. [13] found 57 % mortality in patients with a pelvic fracture who presented with shock. Smith et al. [14] found 40 % mortality in pelvic fracture patients requiring 6 or more units of red blood cell transfusion within 12 h of injury. According to Gililand [15], factors affecting mortality after a pelvic fracture include severity of the posterior pelvic ring injury, presence of head injury, hypotension on admission to the hospital, decreased level of hemoglobin, and an increased need for blood and blood products. Significantly displaced pelvic fractures also have an increased mortality risk as demonstrated by Rommens [16].

Given the potential mortality of these injuries, early diagnosis is paramount. A key first step in the emergency department (ED) is recognizing the clinical, radiographic, and physiologic indicators of an unstable pelvic ring injury. Clinical indicators include the patient age, mechanism of injury, open fractures, soft-tissue injuries about the pelvis, and hematuria. Radiographic indicators include a biomechanically unstable pelvis on an anteroposterior (AP) X-ray—one in which the anterior and posterior portions of the ring are disrupted; as mentioned before, though, hemodynamic instability can result from biomechanically stable pelvic fractures. Any fracture of the pelvis on the initial AP pelvis X-ray indicates that the pelvis is likely a source of bleeding and a contributor to shock. Physiologic instability is generally acknowledged as a patient with a systolic blood pressure (SBP) less than 90, a significant base deficit (>6), elevated lactate, or tachycardia. The Advanced Trauma Life Support (ATLS) classification for hemorrhage (classes I–IV) is useful to guide average blood loss but should be used in conjunction with more prognostic indicators such as base deficit [17], presence of hypotension, and initial hematocrit.

Patients with physiologic and biomechanically unstable pelvic ring injuries have relatively poor long-term outcomes. Significant long-term sequelae include leg-length discrepancy, neurologic dysfunction, and chronic pain, especially with sacroiliac joint fractures/dislocations [18]. Huittinen and Slätis [3], Monahan and Taylor [19], Pohlemann et al. [20], and Smith et al. [21] have all demonstrated significant long-term musculoskeletal sequelae associated with pelvic fractures in both adult and pediatric populations, including pain, gait disturbance, neurologic injury, and functional disability. Numerous authors have found a correlation between reduction quality and functional outcome [22–25], while others have found the presence of sacral fractures [25], neurologic injury [26], or pure SI joint dislocations [27] to correlate with an unsatisfactory functional result. Tornetta and Matta [28] reported good functional results with properly performed open reduction internal fixation of unstable posterior pelvic ring injuries. However, Lefaivre et al. [29], in a systematic review, found the existing literature inadequate to prognosticate the functional outcomes of pelvic ring injuries after fixation. Despite the controversy, the treating physician should be aware that (1) displaced posterior ring injuries, (2) the presence of neurologic injury, and (3) pure SI joint dislocations all correlate with significant long-term sequelae.

While there are multiple protocols designed to assess the unstable pelvis, few studies have quantitated the contribution of either clinical or radiographic indicators in diagnosing the specific pelvic ring injuries that lead to morbidity and mortality. In this chapter, we describe pelvic fracture patterns, initial assessment of these injuries, and the management of the physiologically unstable pelvic injury. The goals are (1) to improve early recognition of these potentially

fatal injuries and (2) to provide a rationale for aggressive treatment in order to (a) decrease mortality and (b) improve the long-term outcome in these severely injured patients.

Fracture Classification Systems

An effective classification system is accurate, providing reproducible intra- and interobserver reliability while also aiding the surgeon in determining treatment. The classification systems for pelvic fractures are based on the mechanism and degree of biomechanical disruption. Thus, the resulting fracture displacement and type of injury can contribute to the degree of severity. Nevertheless, the bony/ligamentous injury may be more severe than upon presentation since some of the structures may "recoil" from the initial trauma—giving the appearance of a more benign injury. Thus, some injuries may be underestimated if based on a static X-ray or CT scan.

The pelvis is a bony ring composed of two innominate bones and the sacrum—all stabilized by the symphysis pubis, anterior/posterior sacroiliac (SI), sacrospinous, and sacrotuberous ligaments. The anterior structures contribute 40 % of the stiffness to the ring, but the posterior SI ligaments are responsible for maintaining the majority of pelvic stability and are the strongest ligaments in the human body. The relative contributions of the ligamentous structures have been demonstrated from cadaveric studies [30]. Historically, it has been accepted that the anterior SI ligaments have been compromised with a pubic symphyseal diastasis of greater than 2.5 cm. More recently, Doro et al. [31] demonstrated, in cadaveric models, that anterior SI ligament injury is likely for a pubic diastasis greater than 4.5 cm and unlikely for values less than 1.8 cm, with the average at approximately 2.2 cm. Sacrospinous and sacrotuberous ligaments may not rupture simultaneously with the anterior SI ligaments. Damage to the pubic symphysis and anterior SI ligaments results in a rotationally unstable but vertically stable pelvis, whereas injury to the sacrotuberous and stronger posterior SI ligaments results in vertical and posterior instability. The sacrospinous and sacrotuberous ligaments are orthogonal to each other and together, with the anterior/posterior SI ligaments, resist most of the external deforming forces acting on the pelvis—external rotation and vertical shear. When the sacrospinous, sacrotuberous, and anterior/posterior SI ligaments are transected, the pelvis is rotationally, vertically, and posteriorly unstable. In addition to damage to these structures, injury in one part of the pelvic ring is nearly always accompanied by ligamentous or bony injury in another portion of the ring [32, 33]. Other clues of a vertically unstable pelvic ring injury include an avulsion fracture of either the L5 transverse process or ischial spine, since these sites are attachments of the iliolumbar and sacrospinous ligaments, respectively. The classification systems developed take these biomechanical principles into account. Letournel and Judet proposed a classification system based on anatomic fracture location: posterior ring, acetabulum, and/or anterior ring. Fractures were classified as follows: (A) the iliac wing, (B) ilium fractures with extension to the SI joint, (C) transsacral fractures, (D) unilateral sacral fractures, (E) SI joint fracture-dislocations, (F) acetabular fractures, (G) pubic ramus fractures, (H) ischial fractures, and (I) pubic symphysis separation. Letournel and Judet's system is one of the most comprehensive classification systems, although more conventional systems are currently used. In this chapter, we will review three commonly used classification systems that are based on the aforementioned biomechanical principles: (1) Young and Burgess, (2) the modified Tile classification system, and (3) the AO/OTA system. Figures 7.1, 7.2, and 7.3 demonstrate these three classifications systems.

Young and Burgess (Mechanism of Injury Classification)

The Young and Burgess classification is based on the force vector of injury to the pelvic ring. There are three types of injury patterns: (1) anterior/posterior compression (APC), (2) lateral compression (LC), and (3) vertical shear (VS).

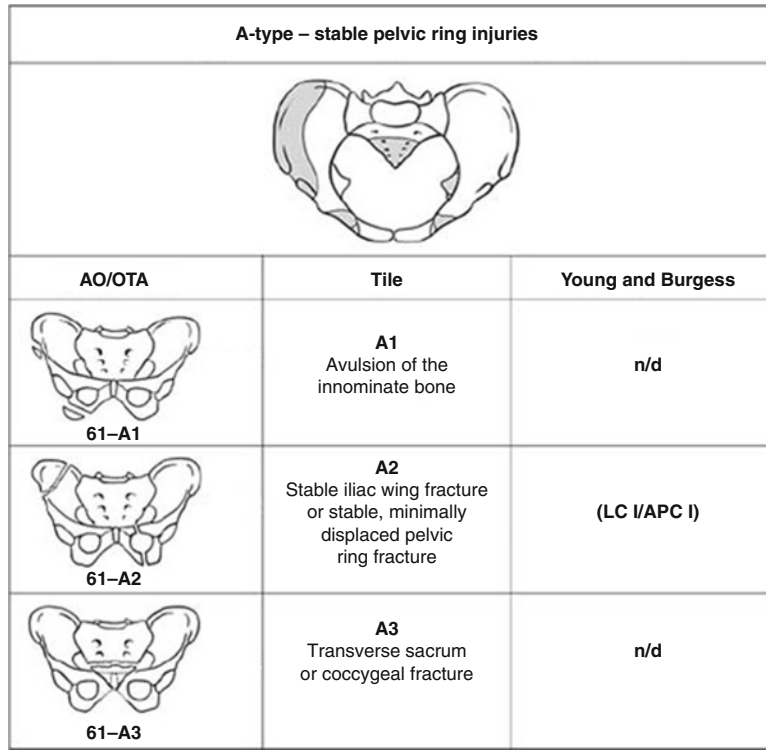

Fig. 7.1 Type A pelvic ring injuries. The pelvic ring is mechanically stable

The APC-type injuries are divided into three types. Type I injuries are due to an anteroposterior force that "opens" the pelvis like a book with intact posterior ligamentous structures. Type II injuries are a type I injury with disrupted sacrospinous +/− sacrotuberous ligaments as well as the anterior SI ligaments. A type III injury is complete disruption of all ligaments ("open book") and associated with retroperitoneal rather than intraperitoneal hemorrhage. *APC types II/III are unstable*.

The LC-type injury is divided into three types as shown. Type I caused by a posteriorly directed force resulting in a sacral crush injury with ipsilateral horizontal pubic rami fractures. Type II is a more anterior-directed force with a resulting anterior sacral crush and ipsilateral rami fractures and injury either to the ilium (i.e., "crescent" fracture) or SI joint. These injuries have a high incidence of associated head and/or intra-abdominal injuries. Type III injury is more severe than types I/II due to an external rotation component to the contralateral side and possible internal rotation component to the ipsilateral side (so-called windswept pelvis). This injury results in a similar fracture pattern to an LC II injury with additional disruption of the sacrospinous and sacrotuberous ligaments. As discussed before, due to injury to the posterior SI ligaments and sacrotuberous and sacrospinous ligaments, the pelvis is rotationally, vertically, and posteriorly unstable. Thus, an LC *III injury is a mechanically unstable pelvis* injury due to a more anterior, lateral-directed force.

Comprehensive Pelvic Disruption Classification (Modified After Tile)

This classification scheme is based on the mechanism of injury as well as the degree of pelvic stability. This system is based on whether the posterior arch of the pelvis is disrupted and is summarized per Figs. 7.1, 7.2, and 7.3. Type A injuries do not affect the mechanical integrity of the pelvic ring (avulsion of the innominate bone, iliac wing fracture, etc.). Type B injuries are rotationally unstable with partially stability of the posterior pelvic ring, while type C injuries are

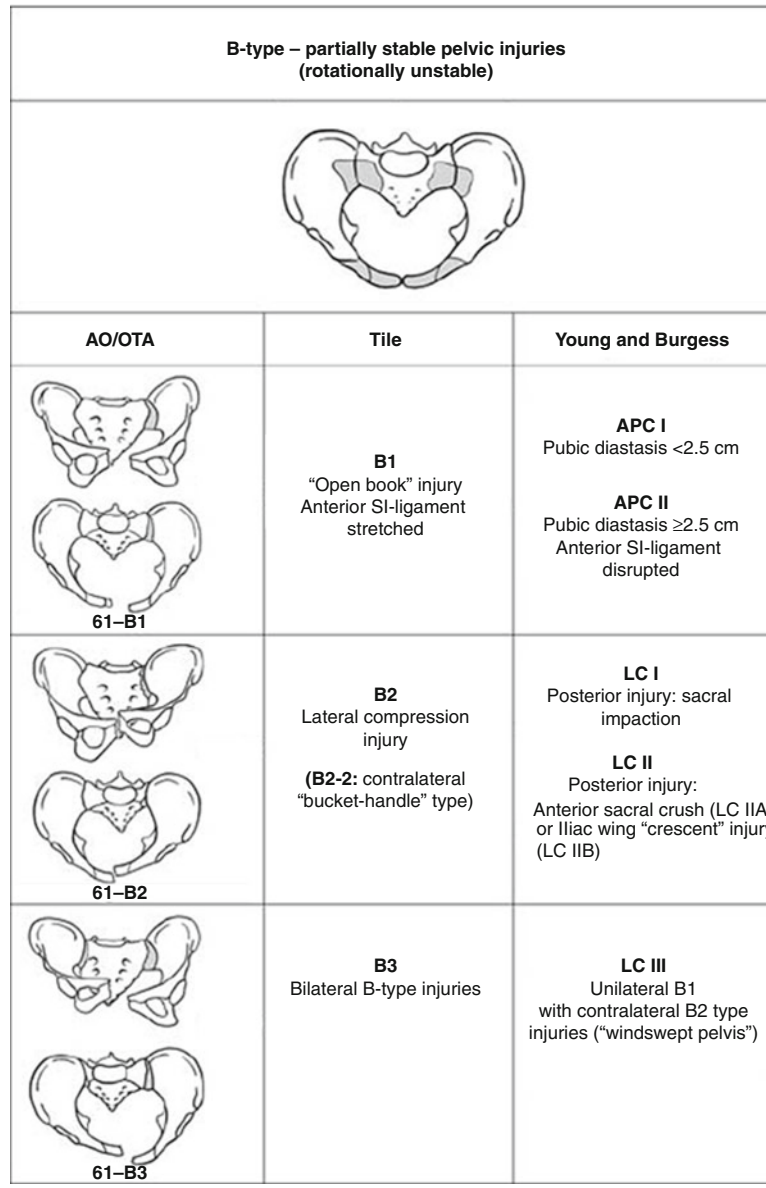

Fig. 7.2 Type B pelvic ring injuries. The pelvic ring is rotationally unstable but the posterior SI ligaments are intact

completely unstable anterior and posterior pelvic ring injuries. Figure 7.4 demonstrates the correlations between the Young and Burgess and Tile classification patterns.

AO/OTA Classification

This classification is similar to the Tile classification in that it is based on the stability of the posterior pelvic structures. Type A fractures are stable fractures with no involvement of the mechanical ring (50–70 % incidence). Type B fractures are partially unstable injuries with partial posterior, rotational instability after anteroposterior or lateral compression (incidence 20–30 %). Type C fractures are unstable injuries with combined anterior and posterior vertical instability (incidence 10–20 %).

Although these classification schemes are useful in deciding if treatment is necessary, it is important to realize that in many cases, patients will present with a combined mechanism (CM) fracture pattern. In these cases, the surgeon

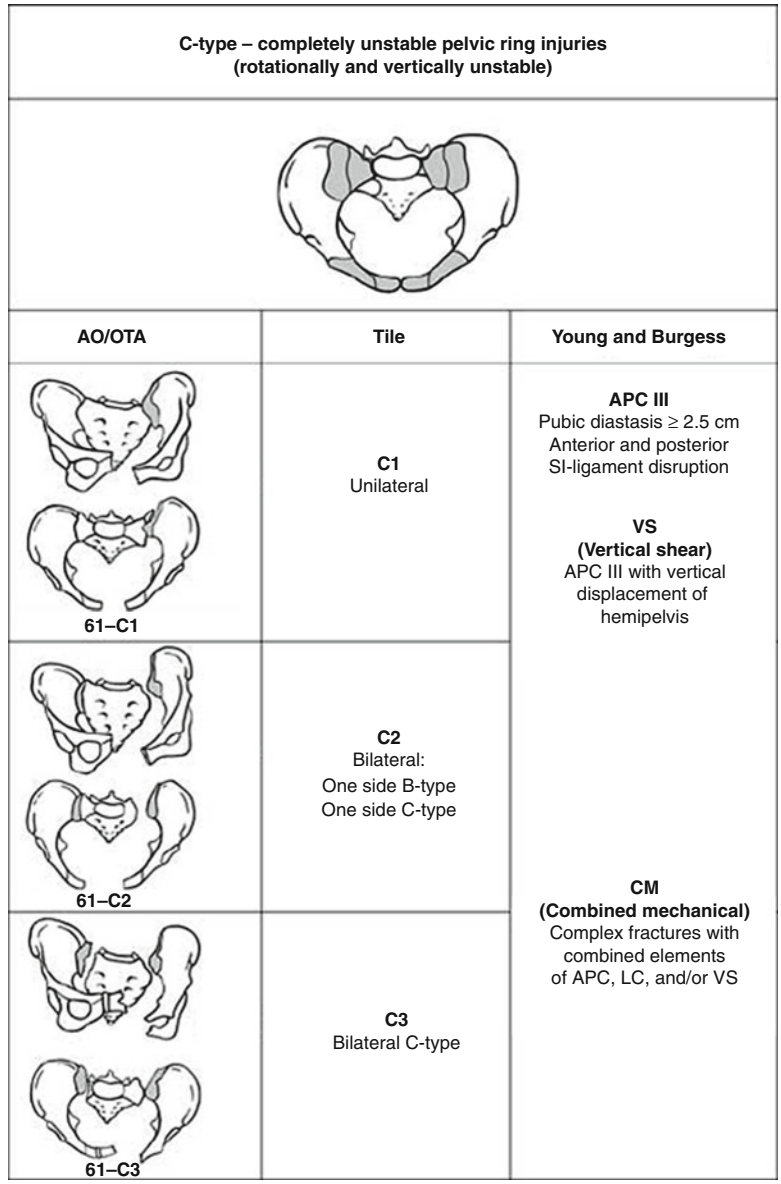

Fig. 7.3 Type C pelvic ring injuries. Rotationally and vertically unstable pelvic ring injuries with complete disruption of both anterior and posterior SI ligaments

should base his/her decision on the patient's clinical scenario and basic biomechanical principles. While fracture classification is important in identifying mechanically and potentially hemodynamically unstable pelvis fractures, minimal time should be spent deciding upon classification during the initial evaluation. Obtaining extra X-rays, special views, or CT scans in order to classify the fracture has no place in the acute setting of the unstable or borderline stable patient.

Assessment of Pelvis Fractures

History

In assessing pelvic injuries, the patient must be fully evaluated including history, physical exam, and appropriate radiographic studies. If the patient sustained a high-energy injury or is hemodynamically unstable, the ATLS protocol should be followed. This assessment is important since both the history and physical examination

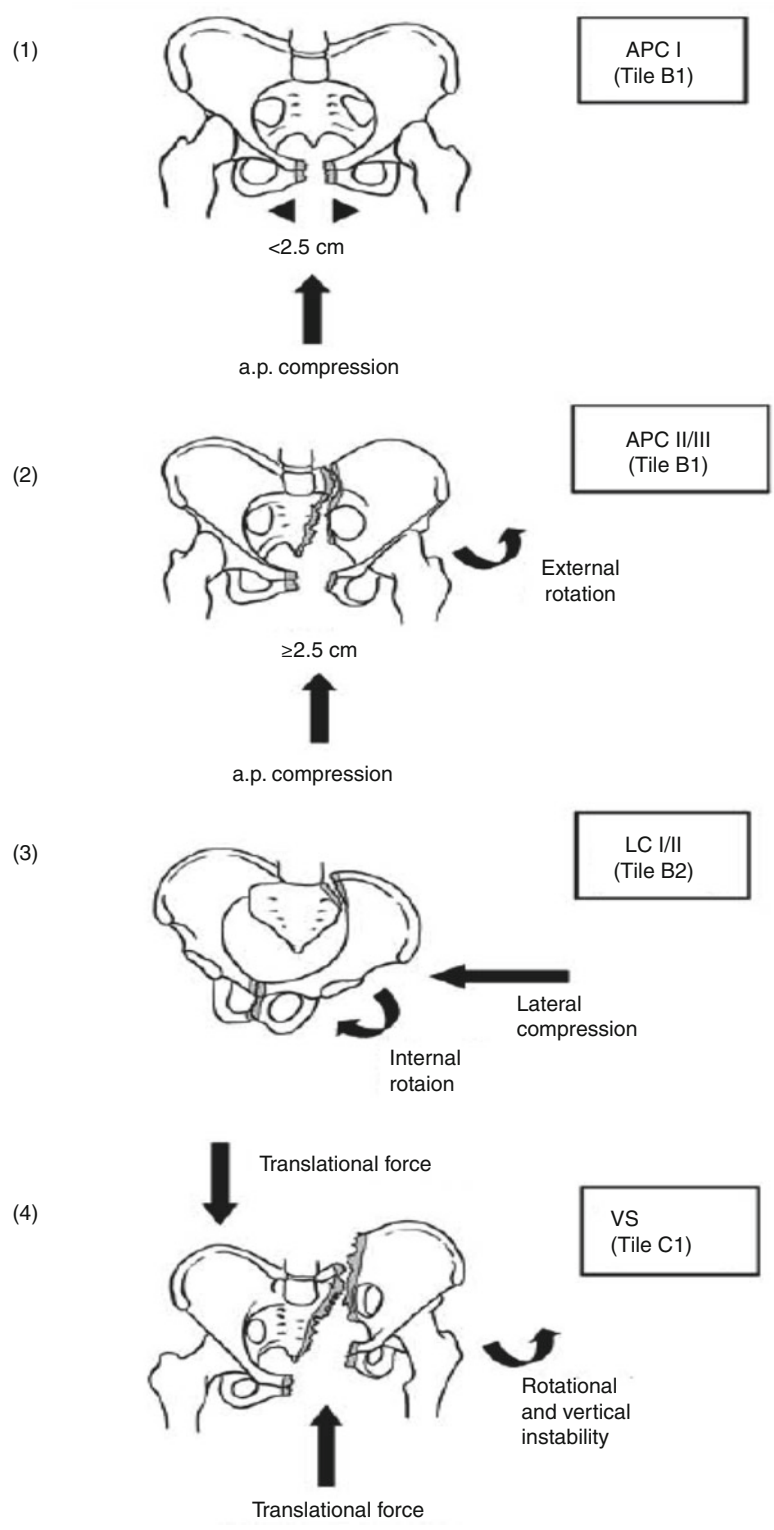

Fig. 7.4 A correlation between the Young/Burgess and Tile classification schemes

offer clues to fracture severity and potentially guide treatment. For example, a fall from a low height or ground level in an elderly individual may imply a stable fracture type, whereas a high-energy injury, i.e., motor-vehicle accident or fall from a large height, would suggest an unstable injury pattern. Nevertheless, it is important to take the entire patient's clinical scenario into consideration, since a seemingly low-energy pattern in an elderly individual may cause significant morbidity and mortality.

As mentioned before, high-energy injuries to the pelvis are associated with significant mortality due to hemorrhage. Associated injuries include urogenital injuries in 10–15 % of patients [34] with the bladder and the male urethra the most common sites of injury [35]. Sexual dysfunction is also a common, and often underreported, complication of pelvic fractures with an average rate of 35.9 and 39.6 % in men and women, respectively [36]. Lumbosacral plexopathy injury is present in 10–15 % of patients [37] with pelvic fractures. Risk factors for neurologic deficits include unstable pelvic and/or sacral fractures with the L5 and S1 nerve roots most at risk [38]. In addition to the mortality rates as discussed before, 60–80 % of these patients have associated musculoskeletal injuries.

Physical Examination

Physical examination should follow the ATLS principles of A (airway), B (breathing), C (circulation), D (disability), and E (extremity). In some cases, pelvic injuries may have dramatic presentations including open fractures and gross deformity. The evaluating trauma team must remain disciplined, however, and not skip initial evaluation steps—i.e., airway is more important than X-ray. Critical aspects of the standard ATLS workup of these patients include identifying the sources of hemorrhage. Part of the primary survey entails inspection for ecchymosis or compromised skin around the pelvis or genitalia. Blood at the urethral meatus, scrotal hematoma (Destot sign), and/or a high-riding prostate on rectal exam indicate pelvic disruption with possible bladder injury. Signs of pelvic instability include leg-length discrepancy with either shortening or external rotation on the involved side. The posterior aspect of the pelvis should be examined for palpable defects along the sacroiliac joint, hematoma, or ecchymosis. Studies have shown that palpation of the posterior pelvis in patients with pelvic fractures can accurately detect injuries of the posterior ring in the awake patient [39]. The absence of posterior sacral tenderness in a cooperative, alert patient can nearly rule out a posterior pelvic injury [40]. On the contrary, the presence of pelvic deformity or an unstable pelvic ring on physical examination has poor sensitivity for diagnosing a mechanically unstable pelvis in blunt trauma patients [40]. In awake, alert, and cooperative patients, a standard neurologic exam should be performed to assess the lumbosacral plexus since there can be significant neurologic damage, especially if the posterior injury is a sacral fracture [41, 42].

Radiographic Examination

According to Young and Burgess, 95 % of pelvis injuries can be diagnosed from an anterior/posterior (AP) pelvis film [8]. A standard AP pelvis radiograph along with inlet and outlet views as described by Pennal [4] should be obtained. Inlet and outlet views demonstrate injury to the pelvic ring and vertical instability of the pelvis, respectively. Finally, CT has proven to be invaluable in assessing injury to the posterior pelvic ring, and given the current technological advancements, CT can be done in an efficient and expeditious manner.

While the abovementioned studies are useful in understanding the fracture, it is important to emphasize, as mentioned earlier, that inlet/outlet views and CT scans have absolutely no role in the hemodynamically unstable pelvic injury patient, in terms of imaging the bony injuries, pelvic hematomas of fracture categorization. These studies do not in any way reduce shock or assist the surgeon in controlling blood loss. The AP pelvic X-ray, however, is critical in allowing the treatment team to decide whether the pelvis could be a source of shock. Bleeding in the pelvis can originate from damage to (1) the venous plexus anterior to either the posterior pelvic ring and/or the bladder, (2) the common/external/internal iliac arteries/veins, and/

or (3) the fracture bone surfaces. If the source of hemorrhage is arterial, the most common site of injury is the internal iliac artery or a branch thereof. Huittinen and Slätis [43], in a seminal autopsy study on patients with pelvic fractures, demonstrated that in 85 % of deaths, bleeding was from bone surfaces and veins; in only 15 % of mortality cases could they identify an arterial source of bleeding. Thus, management strategies must be directed primarily at controlling bone and venous bleeding and secondarily at arterial bleeding.

Initial Management

Initial management of the patient with a pelvic injury follows ATLS guidelines.

Standard 14 or 16 gauge peripheral intravenous (IV) cannulas should be used for resuscitation. A 2-L bolus of IV crystalloid should be given to hypotensive patients. If the blood pressure or urine output does not improve, another 2 L bolus followed by O-negative (non-crossmatched) blood should be administered promptly. Type-specific blood, crossmatched to ABO and Rh type, can be administered once available (approximately 30 min at most institutions). If blood transfusion is required, fresh frozen plasma (FFP) and platelets should be given in a 1:1 ratio with packed red blood cells. The 1:1 ratio of FFP to PRBC should be started immediately [44], and platelets should be coadministered due to the demonstrated platelet dysfunction accompanying traumatic coagulopathy [45]. This 1:1 ratio of FFP-PRBC administration has demonstrated improved survival to discharge by decreasing death from hemorrhage [46]. Although there is recent controversy regarding the ratio of FFP to PRBC, the amount of crystalloid administration, the use of fibrinogen with/without FFP, and recombinant coagulation factor usage, the authors recommend a 1:1 ratio of FFP-PRBC until further prospective randomized controlled trials are done. Despite the controversy, one of the most common errors in pelvic resuscitation is delaying the transfusion of clotting factors. If the definitions and markers of hemodynamic instability are not universally understood, the patient at greatest risk for under-resuscitation is the borderline patient, characterized by transient responses to fluid or blood. In these patients, FFP is often omitted initially. Once they become hypotensive, however, they have already received significant fluid and undergo a rapid dilutional coagulopathy that is difficult to reverse and often ends in death. Therefore, once the first unit of red blood cells is transfused, the authors recommend converting to a preplanned protocol of 1:1 transfusion of RBC-FFP. *This single strategy may be the most critical improvement in resuscitation that has resulted in decreased mortality*. Unfortunately, it is often neglected due to the lack of standardized protocol requirements at most trauma centers.

Core body temperature should also be measured and kept as close to 37 °C as possible during initial evaluation and resuscitation since hypothermia results in impaired coagulation. If there is suspected ongoing bleeding, 1 of the 5 sources must be identified: external (on the field), thoracic cavity, fracture (long bones), abdomen (intraperitoneal), and/or pelvis (retroperitoneal). Ongoing evaluation and reevaluation for hemorrhagic sources such as the Focused Abdominal Sonography for Trauma (FAST) exam, direct peritoneal lavage (DPL), high-speed CT, and/or exploratory laparotomy should be implemented based on protocol and surgical judgment. Pelvic angiography has an important role in nonresponders with negative FAST examinations but is not recommended as a primary diagnostic tool in the hemodynamically unstable patient. In fact, Hou et al. have demonstrated, in a small cohort of trauma patients, that primary angiography can be detrimental [47].

Initial stabilization of the “open-book” or APC-injured pelvis can be done by wrapping a sheet around the pelvis and closing down the retroperitoneal volume, thus helping to tamponade ongoing bleeding. This stabilization can also be accomplished by devices such as the Trauma Pelvic Orthotic Device (T-POD®, Pyng Medical), SAM Pelvic Sling® (SAM Medical Products), a C-clamp, or external fixation (Fig. 7.5). The physician should pay careful attention to the location of the binder. Frequently, the binder placement is too high and results in an inadequate pelvic reduction. Recent literature has demonstrated that accurate placement of a pelvic binder, at the

level of the greater trochanter, improves reduction of pelvic ring diastasis while permitting unobstructed access to the abdomen for laparotomy [48]. All these devices function as splints to decrease pelvic volume, stabilize the bone and soft tissues, decrease laceration of small blood vessels, protect intrapelvic clot formation, and decrease catecholamine release. In patients who are obese, internal rotation and taping of the lower extremities can be done if a pelvic binder cannot be placed [49]. Initial stabilization of VS-type injuries to the pelvis includes a traction pin, in addition to an external fixator or other device, placed in the vertically unstable pelvis to help pull the displaced hemipelvis into a more anatomic and stable position. Mechanically stable fractures and LC-type injury patterns do not require volume closure or splinting. However, patients with apparently stable fracture patterns who are taken urgently for laparotomy, thoracotomy or pelvic packing should be quickly and carefully reevaluated for mechanical instability in the operating room. A recent retrospective review of 68 patients demonstrated occult instability in 50 % of presumed APC I, 39 % of APC II, and 37 % of LC 1 pelvic injuries [50]. In some cases, stable appearing fractures are indeed unstable once stressed under anesthesia using fluoroscopy. APC-type injuries will demonstrate increased pubic diastasis after anteroposterior-directed force on the iliac crest. LC-type injuries will be exacerbated (i.e., more internal rotation or displacement) after a laterally directed force. Vertical shear injuries can be evaluated by the "push-pull" test in which longitudinal traction or compression is exerted on the affected lower extremity and displacement of the hemipelvis is assessed using dynamic fluoroscopy. For hemodynamic and/or mechanically unstable pelvic injuries assessed in the operating room, a simple form of external fixation device can be rapidly applied if the reexamination shows dynamic instability or is equivocal.

During the workup of these patients, if a urethral injury is suspected based on physical exam (i.e., blood at the urethral meatus or in the vagina), Foley catheterization should not be done since the catheter could potentially disrupt an already existing urethral tear. If a bladder injury is suspected and the patient is stable, a cystogram can be performed.

During the initial assessment of the patient with an unstable pelvic fracture (AO/OTA type B or C), particular attention should be paid to the patient's age, Revised Trauma Score (RTS), and blood transfusion requirements since all have been shown to be predictors of early mortality [13, 14]. Specifically, age >60 and Revised Trauma Score (RTS) are easily accessible during the assessment and suggest the severity of the injury and risk of mortality [13, 14]. The RTS is a physiological scoring system obtained from the sum of the Glasgow Coma Score (GCS), systolic blood pressure (SBP), and respiratory rate (RR)—with lower scores associated with increased mortality. Although ISS also correlates with early mortality, it is not easily determined upon initial assessment. As discussed before, fracture pattern has not always been shown to correlate with mortality nor can it be used to determine the need for angiographic embolization [51]. Despite numerous relevant indicators of potential mortality, the three most useful to the evaluating team are *age*, *shock*, and *transfusion requirements*. These require no calculation and are apparent to even minimally trained staff. Furthermore, each indicator has been shown by various investigators to be a predictor of high mortality. For example, Sathy et al., in a study of 63,000 trauma patients, found the odds of mortality in patients aged over 60 years with a pelvic fracture to be 4.5–8.8 times higher than patients less than 60 [52]. The same study also demonstrated that a systolic blood pressure <90 mmHg is a key predictor of mortality in pelvic fracture patients [52]. Therefore, when one of these risk factors is present, the treatment team should immediately presume the patient is "unstable" until clearly proven otherwise. The trauma team should also obtain an initial arterial blood gas and monitor the base deficit during management to assess physiologic stability. The cause of death for unstable pelvis fracture patients in the first 24 h is most commonly due to acute blood loss. After 24 h, the cause of death is usually from multiple organ failure; thus, the

cornerstone of treatment should be aggressive shock management, with the goal to decrease the required transfusion volume. Preplanned protocols involving a multidisciplinary management approach are essential and have been shown to lower mortality in these severe injuries. Therefore, if the patient is elderly, in shock, or requires a transfusion, the management team should initiate a preplanned protocol immediately. If the patient stabilizes quickly, then the protocol can be discontinued. Since the goal of most protocols is to avoid "under-triage," some "over-triage" is expected and necessary. However, if the surgeon neglects the borderline patient with the aforementioned warning signs due to a false sense of "stability," this is a grave error that may result in the patient's mortality.

Mechanical and Hemodynamic Instability

In the workup of patients with pelvic fractures, two aforementioned factors must be considered since they will guide both resuscitation and treatment: (1) mechanical instability and (2) hemodynamic instability. Rommens demonstrated higher mortality in higher-energy pelvis injuries in 122 patients with unstable B- and C-type injuries [16]. Burgess et al., in a series of 210 patients, correlated fracture type with transfusion requirements: LC, APC, VS, and CM injuries required 3.6, 14.8, 9.2, and 8.5 units of PRBC, respectively [9]. Mortality from LC and APC patterns was due to closed-head injury and combined pelvic/visceral organ injury, respectively [53]. Similarly, Magnussen et al., in a retrospective review of 382 patients with isolated pelvic and/or acetabular fractures, demonstrated that APC II/III, LC III, VS, or CM pelvic injuries required more frequent transfusion than other pelvic fractures [54]. These correlations of fracture pattern and mortality should serve as a guide, but not as a definite rule, since patients can still exsanguinate from more "benign" fracture patterns. For example, Smith and Starr demonstrated that specific fracture patterns were not predictive of mortality [13, 14]. Mechanical instability needs to be determined to reduce the risk of further bleeding, decrease pain, and to allow early mobilization. Hemodynamic instability needs to be addressed by standard ATLS protocols for the obvious reasons to decrease mortality. Thus, these two issues, while not necessarily predictive, should be considered related. For example, a lateral compression type I (LC I) pelvic fracture is not an "unstable" definition but can still result in significant retroperitoneal hemorrhage (venous > arterial) in an elderly patient with osteoporotic bone. Thus, hemodynamic instability is possible with a mechanically stable fracture pattern.

Mechanical instability of the pelvis can occur in three planes: rotational, translational, and vertical. As discussed before, the sacrotuberous and posterior SI ligaments contribute to vertical and posterior stability, whereas the sacrospinous and anterior SI ligaments contribute to rotational stability. It is difficult to assess specific damage to these ligaments, but the fracture patterns can clue the surgeon to the severity of the injury and thus the treatment needed.

The basic premise in all the fracture classification schemes is the importance of the posterior ligamentous structures in the pelvic ring. Partial or complete disruption of these structures contributes to an unstable pelvic ring injury, particularly with rotation. Specifically, the LC III, AP II/III, and vertical shear injuries all have disruption of the posterior ring and thus are unstable pelvic injuries. Clear indications for surgical stabilization of the pelvic ring are rare in type A fractures. Stabilization of the anterior ring is usually sufficient for type B fractures, while anterior/posterior stabilization is necessary for type C fractures.

As mentioned before, the history (i.e., patient age, high vs. low energy, exsanguination) can aid the surgeon in determining whether the injury is an unstable pelvic fracture pattern. Similarly, the physical exam can also clue the surgeon to the instability of the fracture pattern (i.e., lack of tenderness to palpation posteriorly). However, AP/lateral compression tests are not sensitive examinations and should only be performed once to avoid disrupting a pelvic hematoma.

Management of Unstable Pelvic Fractures

Once a patient is identified as unstable or potentially unstable, a series of preplanned steps should take place. Numerous protocols have been reported in the literature, and to some degree, all show improvement in diagnosis, management, and outcome compared to no protocol. Controversy exists regarding which is the "ideal" protocol, and it is clear from the experience of high-volume trauma centers that there is no single protocol that fits all centers. The rationale for each specific interventional methodology is relatively anecdotal and institution-dependent as these studies are ongoing. As noted, however, the literature has identified the key priorities required to reduce mortality: rapid identification of bleeding sources, graded resuscitation with clotting factors, and interventions to stop bleeding, including laparotomy, pelvic packing, and angiography. As improvements in early hemorrhage, source identification and stabilization have evolved, so have resuscitative methods for patients involved in major trauma. Damage-control resuscitation (DCR), which has been passed down from the military, has continued to evolve over the past 10 years. The key components to DCR include (1) transfusion protocols with fixed ratios of blood products (PRBC, FFP, and platelets), (2) permissive hypotension to minimize hemorrhage, (3) prevention and aggressive treatment of hypothermia, (4) temporizing acidosis correction with exogenous buffer agents, and (5) use of recombinant blood products [55]. The optimal ratios of blood products as well as the type of coagulation factors have yet to be determined as more randomized controlled trials are in process. The future may entail goal-directed resuscitation as newer techniques such as point-of-care rapid thromboelastography (TEG) provide more detailed assessment of trauma-induced coagulopathy [56]. Still, despite the evolution of DCR, mechanical means of hemorrhage control via pelvic packing have demonstrated increasing efficacy at stabilizing the patient. Pelvic packing has not been widely adopted across the United States of America, and the trauma team is ultimately left with designing protocols that fit the proven pathophysiology and the resources of a given institution. The current protocol recommended by the authors has been developed over a 27-year period and represents the fourth iteration since the original paper by Moreno et al. in 1976 [57]. Each iteration was based on an evaluation of a prospective registry for pelvic trauma and was published for critique and discussion in the peer-reviewed literature [14, 46, 57, 58]. Currently, the protocol emphasizes the common pathways for all trauma patients: rapid ATLS assessment, shock control with transfusion and blood products, and simple mechanical stabilization with a binder or sheet, if indicated. If more than 2 units of RBC transfusion are required, the patient is taken immediately to the OR for direct retroperitoneal pelvic packing, C-clamp or ex-fix, laparotomy, and damage-control external fixation of extremity fractures as needed. If there is subsequent, ongoing shock or hemodynamic instability, the patient then undergoes angiography and pelvic embolization if appropriate.

A similar approach to pelvic trauma, with an emphasis on pelvic packing (venous hemorrhage control) prior to angiography, has been used by European traumatologists. Since the majority of pelvic bleeding is either venous or from cancellous bone [43, 59], embolization may not address the source of instability. Indeed, because of the time for setup and procedure time, angiography may delay other urgent procedures such as laparotomy and external fixation of open fractures by 1–2 h. External fixation and retroperitoneal packing with three laparotomy sponges per side via a 6 cm midline incision can be completed within 1 h of arrival in an efficient trauma setting. The incision can be extended proximally so that an exploratory laparotomy can be performed with a general trauma surgeon simultaneously. The bladder is then gently retracted to the side. Visualization is sometimes difficult due to hemorrhage, and the surgeon should take great caution to avoid disrupting a stable retroperitoneal hematoma during initial exploration. The color of the blood is frequently a clue to the source of hemorrhage as large amounts of bright red blood is usually an arterial injury. After the bladder is

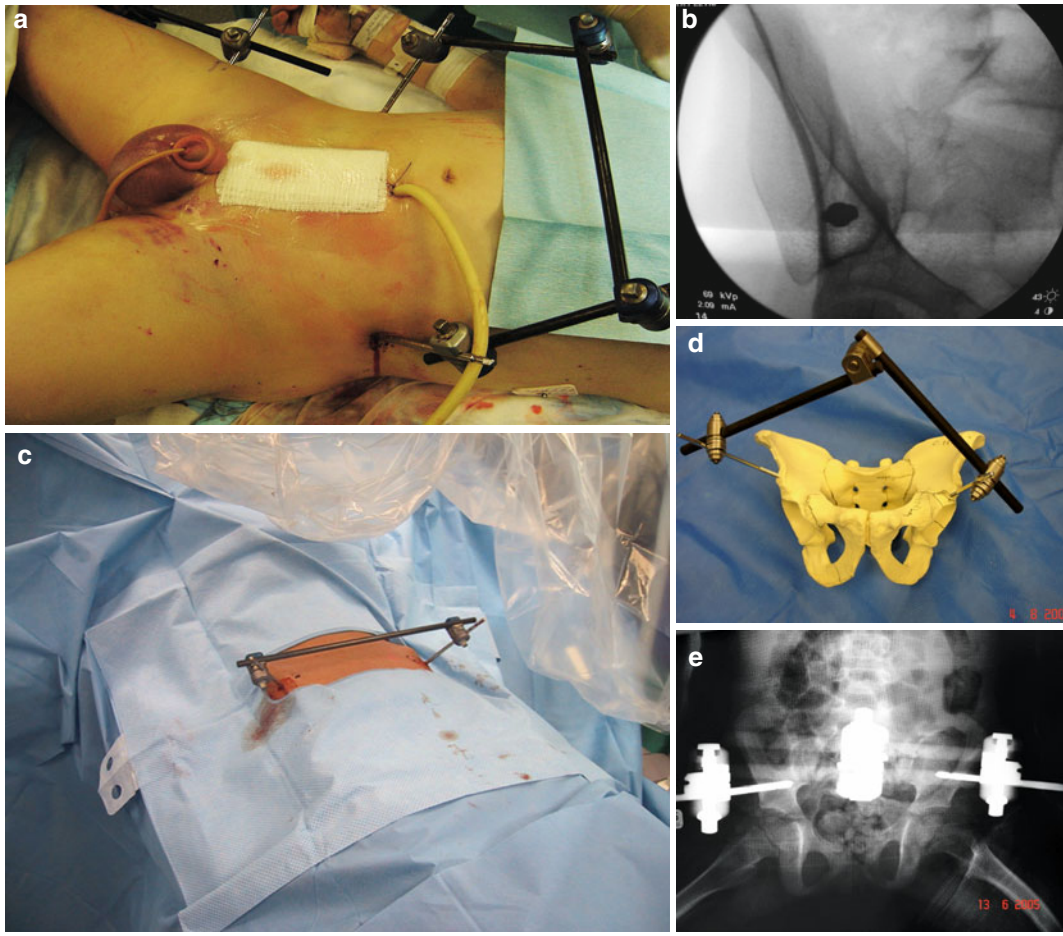

Fig. 7.5 Pelvic packing is performed after external fixation so that the surgeon can pack against a "stable" pelvis. The external fixator can be placed in the iliac crests (**a**) or in the anterior inferior iliac spine (AIIS)-between the two tables of the pelvis in the supra-acetabular region (**b**). In either case, the external fixator is placed to allow access to the abdomen and pelvis (**a**, **c**). The position of the AIIS pins is demonstrated in a bone model (**d**) with corresponding X-ray (**e**). Due to the strong bone stock in the supra-acetabular region, an anterior external fixator frame is more stable; however, fluoroscopy (**b**) is usually more necessary for accurate pin placement

retracted, the surgeon places three laparotomy sponges below the pelvic brim (in the true pelvis) on each side: the first is placed anterior to the SI joint, the second sponge is placed anterior to the first below the pelvic brim (medial to the quadrilateral plate), and the third sponge is placed in the space of Retzius (retropubic space) (Fig. 7.6). The bladder is then retracted in the opposite direction, and the procedure is repeated. Pelvic packing should only be performed after stabilization of the pelvis to allow the surgeon to pack "against" a stable pelvis. An anterior external fixator or C-clamp can be applied quickly for pelvic disruptions predominantly involving the anterior ring (i.e., APC injury), with subsequent reduction of pelvic volume and a tamponade effect. An anterior external fixator can be placed in the iliac crests (Fig. 7.5a) or, preferably, in the anterior inferior iliac spine (Fig. 7.5b–e). Posterior ring disruption can be addressed via a C-clamp placed against the lateral ilium to compress across the posterior pelvic ring (Fig. 7.7). C-clamp or anterior external fixation should ideally be performed under fluoroscopic guidance; the entire procedure, including placement of a C-clamp or external fixator

and packing, should take approximately 25 min. The pelvic incision should be closed even if the abdomen is left open. The surgical team can simultaneously address other injuries increasing the efficiency of the "golden hour." This technique has been shown effective at rapidly restoring hemodynamic instability and decreasing the need for transfusion and the accompanying risks such as multisystem organ failure. If the patient is stable after packing, the sponges can remain up to 24–48 h, at which point they are removed. Definitive fixation of the pelvis can be performed at that time. If patients continue to have hemodynamic instability after packing, then angiography and embolization should be considered. Branches of the internal iliac artery frequently are involved. Nonselective angiography appears to be a more rapid and effective means of gaining hemodynamic stability than super-selective embolization [60, 61]. However, cases in which bleeding is primarily from arterial injury are rare. In those patients with arterial bleeding, there is also significant associated venous and bone bleeding. Therefore, pelvic packing and angiography are not a "one or the other phenomena"; rather, these techniques should be considered complementary and sequential [62, 63]. Pelvic packing effectively serves as a triage tool for angiography. Since at most centers, pelvic packing can happen much faster than angiography—perhaps within minutes of arrival—no time is lost in those patients who require both venous and arterial control. Osborne et al., in a 2009 case-control study, compared a protocol

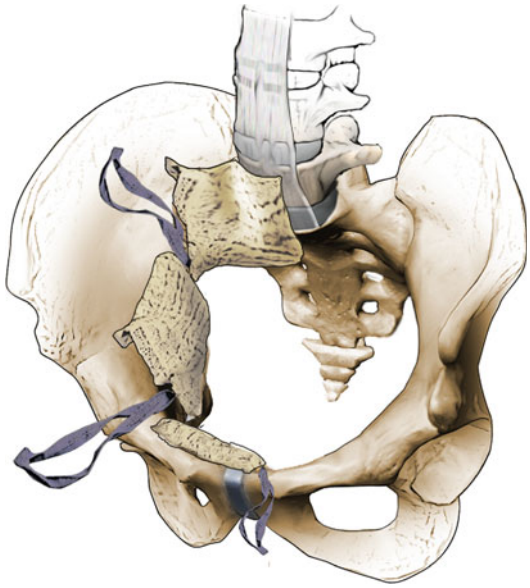

Fig. 7.6 Schematic drawing which demonstrates placement of laparotomy sponges for pelvic packing. All sponges are placed in the true pelvis below the pelvic brim. Three sponges are placed on each side of the pelvis (anterior to the sacrum, medial to the quadrilateral plate, and in the space of Retzius) either via a Pfannenstiel incision or a longitudinal (laparotomy), midline incision (Artwork by Bernie Kida)

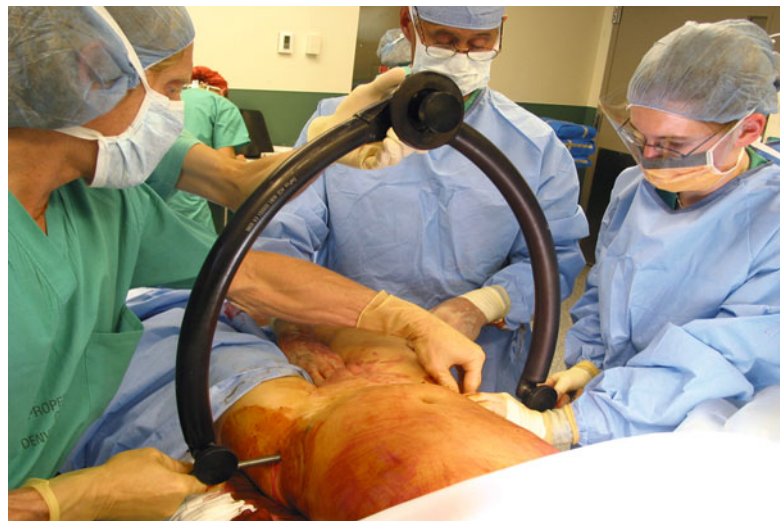

Fig. 7.7 Placement of a C-clamp for pelvic ring injuries, particularly those that involve the posterior ring. The C-clamp is ideally placed using fluoroscopy

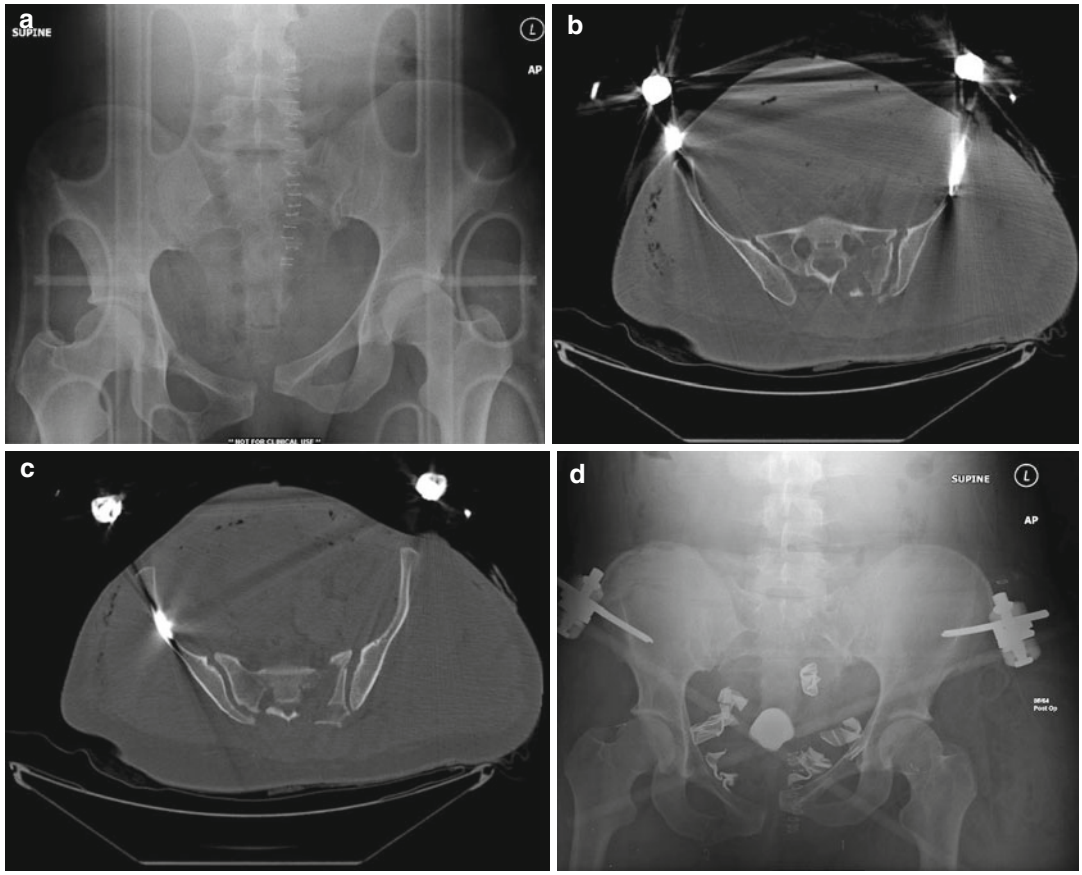

Fig. 7.8 (**a**) An anterior/posterior (AP) pelvis X-ray that demonstrate a combined mechanism pelvic ring injury. (**b**, **c**) A computed tomography (CT) scan demonstrates external fixation pins within the tables of the ilium in addition a left sacral fracture and left sacroiliac joint injury. (**d**) AP pelvis X-ray after pelvic external fixation and packing

with early angiography without packing to a protocol with mechanical stabilization, packing and angiography if needed. The packing group required less blood products in the first 24 h and had a reduced need for pelvic embolization [64]. There were no acute deaths from blood loss in the pelvic packing group; in those cases that required angiography, the angiographic intervention happened in a more timely fashion compared to the group that did not undergo pelvic packing. Figure 7.8 demonstrates a patient with a combined mechanism (CM) injury initially stabilized with pelvic packing and external fixation. Definitive fixation is shown in Fig. 7.9.

Intraoperative angioembolization (IAE) has not been extensively studied, but potentially offers

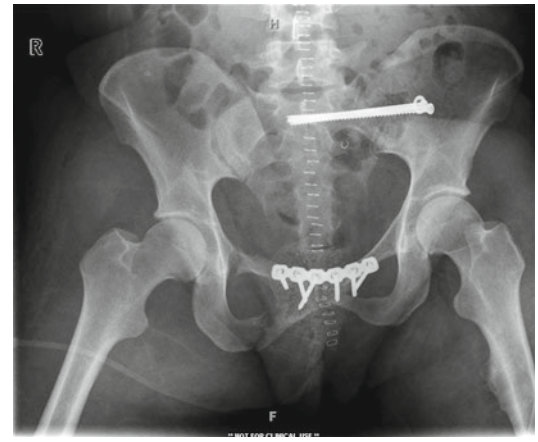

Fig. 7.9 An AP pelvis X-ray after anterior ring fixation and placement of two left iliosacral screws to stabilize the posterior ring

a more efficient means of controlling physiologic instability from pelvic hemorrhage. A recent case series by Cherry et al. demonstrated that IAE is beneficial in patients with a base deficit >13, those who did not require >6 units of PRBC prior to IAE, and those patients who did not sustain a vertically unstable pelvis fracture. The study was limited secondary to low patient numbers and limited resolution of the IAE images compared to the angiography suite [65]. More randomized controlled trials should be performed to assess the efficacy of IAE on patient survival. IAE, if timely and effective, would prove itself as an ideal complement to pelvic packing. For instance, a patient with a pelvic fracture who is still unstable despite pelvic packing could undergo simultaneous IAE in the operating room instead of having to be transported to the interventional angiography suite. Furthermore, other damage-control procedures could be performed in the OR with no delay.

After initial damage-control management of pelvic fractures, definitive treatment of unstable pelvis fractures should be performed when the patient is hemodynamically stable. Definitive treatment is based on the stability of the posterior ring. Type A fractures, for example, rarely need treatment. For type B and C fractures, the posterior ring is involved to some degree. Although external fixation has been used in the past to definitively treat type B and C fractures, studies have demonstrated the superior functional outcome of internal fixation over external fixation for pelvic fractures [66, 67]. The timing of definitive treatment has not been elucidated. Vallier et al. demonstrated that early fixation (<24 h) of pelvic ring injuries and/or acetabular fractures in multiply injured patients reduces morbidity (acute respiratory distress syndrome) and length of intensive care unit stay [68]. However, numerous authors have demonstrated low mortality and morbidity when fixation is delayed several days until bleeding has slowed and the patient is physiologically stable. Further studies need to be performed to determine the physiologic or hemodynamic parameters that represent "stability" in these patients (i.e., base deficit, lactate levels). Figure 7.10 summarizes the authors' management of hemodynamic instability in patients with pelvic injuries.

If the SI joint is subluxed or dislocated, the authors recommend attempted closed reduction and percutaneous iliosacral screw placement using fluoroscopic guidance. However, if an adequate reduction cannot be achieved, open reduction via the anterior or posterior approach to the SI joint should be performed. If there is a concomitant symphyseal injury, anterior plating is recommended to allow for mobilization. If the posterior ring injury is a sacral fracture component of an LC III injury, this can also be fixed with an iliosacral screw and anterior symphyseal plating, particularly if there is distraction at the sacral fracture. For injuries with bilateral sacral fractures, recommended treatment methods are bilateral iliosacral screws or posterior tension band plating. For the latter treatment, particular attention should be paid to the soft tissue over the buttock, especially in recumbent patients, since this area is prone to breakdown. In many of these injuries, after the anterior ring is addressed, the posterior ring will reduce and facilitate fixation. The reader is referred to evidence-based biomechanical fixation of the pelvic ring for more details [69].

Although open pelvic fracture comprise less than 5 % of all pelvic fractures, the mortality rate has been shown to be 20–50 % [70–72]. An open pelvic fracture is one in which there is either exposed fracture or direct communication between the vagina, rectal, and perineum and the fracture. These injuries require early diagnosis and aggressive management since infection and resulting sepsis can be disastrous. Open wounds over the fracture site and perineal lacerations are obvious clues to an open injury, but small rectal or vaginal tears are more insidious. Air in retroperitoneal soft tissue is often a clue of an occult communication between the fracture and the vagina or rectum. In patients with rectal or perineal wounds, a diverting colostomy or ileostomy with a washout must be the first consideration to prevent contamination. If the patient has a vaginal tear with a pelvic fracture, irrigation with primary repair should be first consideration. Closed, degloving injuries of the pelvis are called Morel Lavallée lesions. These injuries are insidious and can lead to sepsis and death; they should be carefully monitored and debrided at the first concern

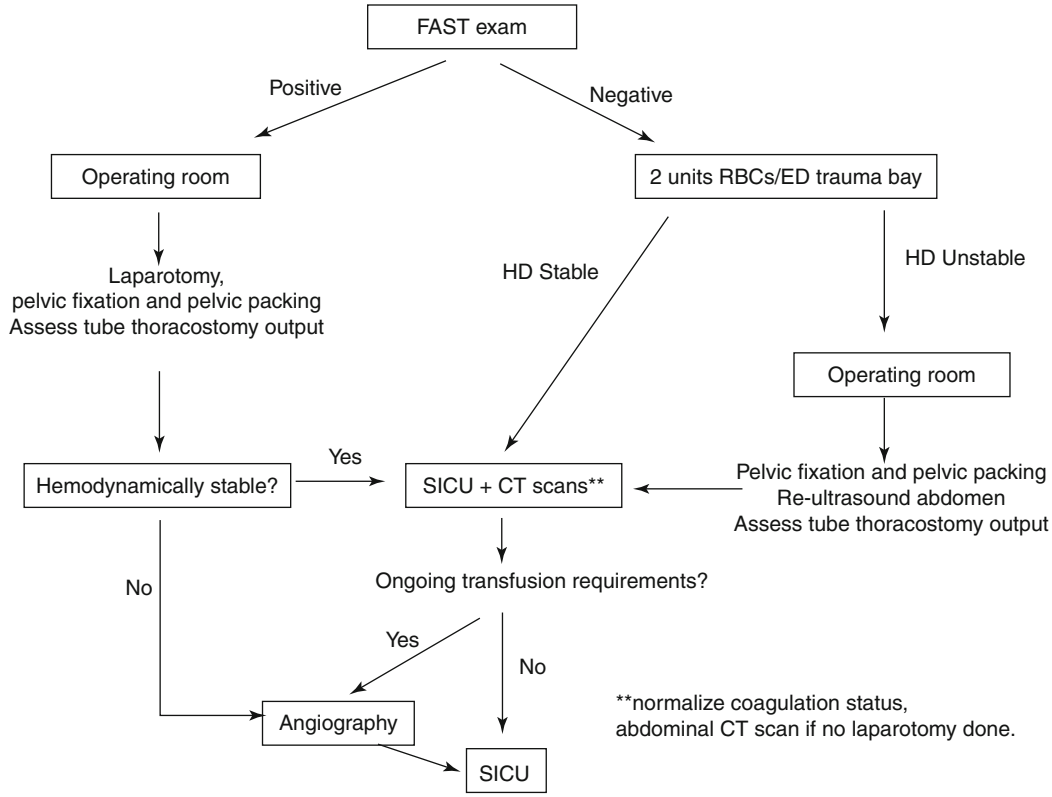

Fig. 7.10 Flow diagram protocol for patients who have sustained a pelvic ring injury with accompanying hemodynamic/physiologic instability

of infection. Tseng and Tornetta [73] demonstrated that small incisions could be used to introduce brushes to effectively debride these wounds. The smaller incisions make wound healing less complicated and reduce the need for skin grafting and tissue transfer.

Lastly, pelvic fractures resulting from civilian gunshot wounds rarely cause pelvic instability, but coordination with the general surgery team is vital since many of these injuries may involve visceral organs. Intra-articular bullet fragments (i.e., hip joint or sacroiliac joint) should be removed.

Future Directions

As technology evolves in the identification of physiologic and hemostatic deficiencies in the trauma patient, the treatments will likely entail more efficient, targeted therapies. Randomized controlled trials on the efficacy of DCR as well as targeted coagulation factor replacement need to be performed to confirm efficacy and justify cost. Furthermore, newer assessment tools such as point-of-care rapid thromboelastography may further demonstrate temporal patterns to factor deficiencies with potential for treatments on the field, in addition to the emergency room. Other treatments, such as tranexamic acid, an antifibrinolytic, have been shown to decrease bleeding when administered early [74]. Recombinant factor VIIa (rFVIIa) has been shown to decrease need for blood products [75]. More randomized clinical trials in trauma cohorts need to be performed to evaluate the efficacy and timing of these new hemostatic agents. These newer agents may decrease transfusion requirements and subsequent mortality since blood products have been shown to be an independent risk factor for post-injury multiple organ failure [76]. Finally, mechanical means of hemorrhagic tamponade such as pelvic packing will benefit from more

multicenter randomized controlled trials as well as surgeon education.

However, there is currently abundant data to support implementation of standardized protocols for pelvic injuries at all modern trauma centers. Unfortunately, in the United States of America, national and state verification and designation groups do not mandate such protocols for any trauma centers. The variability from center to center is significant and usually unknown to the general public despite appellations such as "level 1" or "level 2." The greatest impact on mortality can be made by incorporating already established "best" practices. In addition, all trauma centers that currently treat pelvic trauma should not only establish and follow protocols but also review their results. Further improvements in patient care and technology are showing promise, but an immediate impact can be felt by following best practices today.

Conclusions

Unstable pelvic fractures are potentially fatal due to hemorrhage and other associated injuries. Classification schemes are based on the integrity of the posterior arch of the pelvis, as this is the key to rotational and vertical stability. APC II/III, LC III, VS, and CM pelvic injuries are the most unstable fracture patterns. These injuries are usually associated with high-energy trauma but can be caused by low-energy trauma in elderly, osteoporotic individuals. The initial management of pelvic fractures includes the ATLS protocol. *Age >60*, *shock*, and *need for blood products* are easily identifiable and have been shown to be early predictors of mortality in unstable pelvis fractures. Since the majority of pelvic bleeding originates from cancellous bone and the overlying venous plexus, the authors recommend external fixation and pelvic packing as a rapid and efficient means to aid in hemorrhage control and initial stability. Angiography and embolization are reserved for those patients with continued hemodynamic and physiologic instability despite packing and external fixation. Mechanically stable pelvis fractures can be associated with hemodynamic instability, especially in patients with poor bone stock, although studies on this are lacking. In the final analysis, pelvic fracture patients continue to die at high rates despite massive transfusion protocols, damage-control resuscitation, improved critical care, and new technologies for mechanical and hemostatic stabilization. Rapid decision-making and early shock interventions appear to be the keys to decreasing mortality. Protocol-based approaches clarify and streamline decision-making across disciplines; however, controversy exists as to the precise order of interventions. Newer diagnostic technologies such as rapid thromboelastography continue to evolve as well as studies evaluating the efficacy of targeted coagulation factor replacement and antifibrinolytic therapy. No doubt exists, however, that pelvic fracture protocols need to be regularly and objectively evaluated for efficiency and efficacy. Further investigations are needed with large cohorts in order to improve patient outcome. The lack of prospective randomized data establishing ideal management of acute pelvic fractures should not preclude implementation of protocols at modern trauma centers. Thoughtful consideration of the patient's needs leads to the conclusion that standardization of care of these complex injuries reduces errors and improves patient outcomes.

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Spine Fractures

8

Todd F. VanderHeiden

Introduction

Spinal injury must be suspected in any polytrauma patient. These patients require comprehensive care and coordinated input from multiple medical and surgical providers. Surgeons must maintain a high level of respect when considering the delicate nature of the physiological status of these complex patients. That respect must be maintained when considering the need for other treating specialties to impart urgent and emergent treatment as well as utilize diagnostic modalities. At all times during initial assessment, a spinal injury must be assumed present until proven otherwise. As such, spinal precautions must be maintained to protect injured spinal segments as well as protect against potential neurological deterioration. All involved parties must appreciate that multiply injured patients can suffer from acute respiratory compromise and hemodynamic collapse. Alone, or in combination, these problems can cause hypotension and/or hypoxemia that can further exacerbate spinal cord injury. Adding to the stressfulness of treating these patients is that patients are usually unable to participate in the informed consent process and families may not be available in the acute setting. This increases the burden upon the treating trauma surgeon to quickly and accurately diagnose spinal injuries and swiftly and appropriately provide treatment. Once treatment is completed, patients and families can expect a prolonged recovery including a lengthy hospitalization, protracted rehabilitation period, and an extended convalescence. Even though this can be predicted, the significant impact of spinal injury, especially spinal cord injury, upon the patient, the family, and the medical community cannot be overstated. Case 8.1 demonstrates the complex nature of treating multiply injured patients with concomitant spinal injury.

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Case 8.1

A 35-year-old male was involved in a high-speed, single-car, motor vehicle collision. The car hit a tree at highway speeds. "Texting-while-driving" was suspected as a possible cause since all toxicology studies were negative. The patient was emergently transported to the level 1, regional trauma center. He was maintained on a backboard and a cervical, field collar was utilized to immobilize the cervical spine. Advanced Trauma Life Support (ATLS) protocol was followed. Bilateral chest tubes were placed for hemopneumothoraces. Secondary survey then ensued. This showed an obvious open femur fracture. Step-off was also detected at the thoracolumbar junction upon palpatory examination resulting in a high suspicion for severe spinal injury. Neurological examination was difficult due to obtundation, but the patient lacked both rectal tone and bulbocavernosus reflex. Damage-control-orthopedics proceeded with washout, external fixation, and provisional reduction of the femur fracture. The patient was then ushered to the surgical intensive care unit for further resuscitation. Once adequately resuscitated, full radiographical analysis of the patient's spine was possible. This analysis showed a T9–10 fracture-dislocation and spinal cord injury (Figs. 8.1 and 8.2) as well as a C1–2 vertical distraction injury (Fig. 8.3). Within 24 h of the injury, the patient returned to surgery for open C1–2 reduction, fixation, and fusion with concomitant open

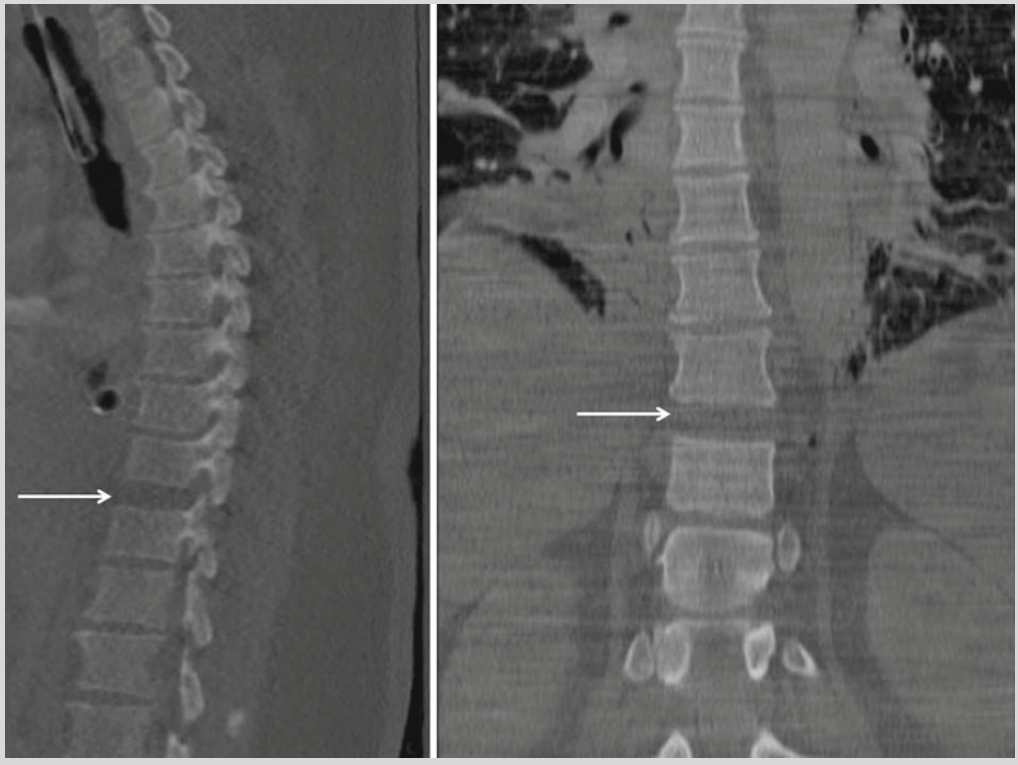


Fig. 8.1 Sagittal and coronal computed tomography (CT) reformations of the thoracic spine. The *arrows* point to the severely injured T9–10 segment. Vertical distraction injury is present resulting in severe spinal instability

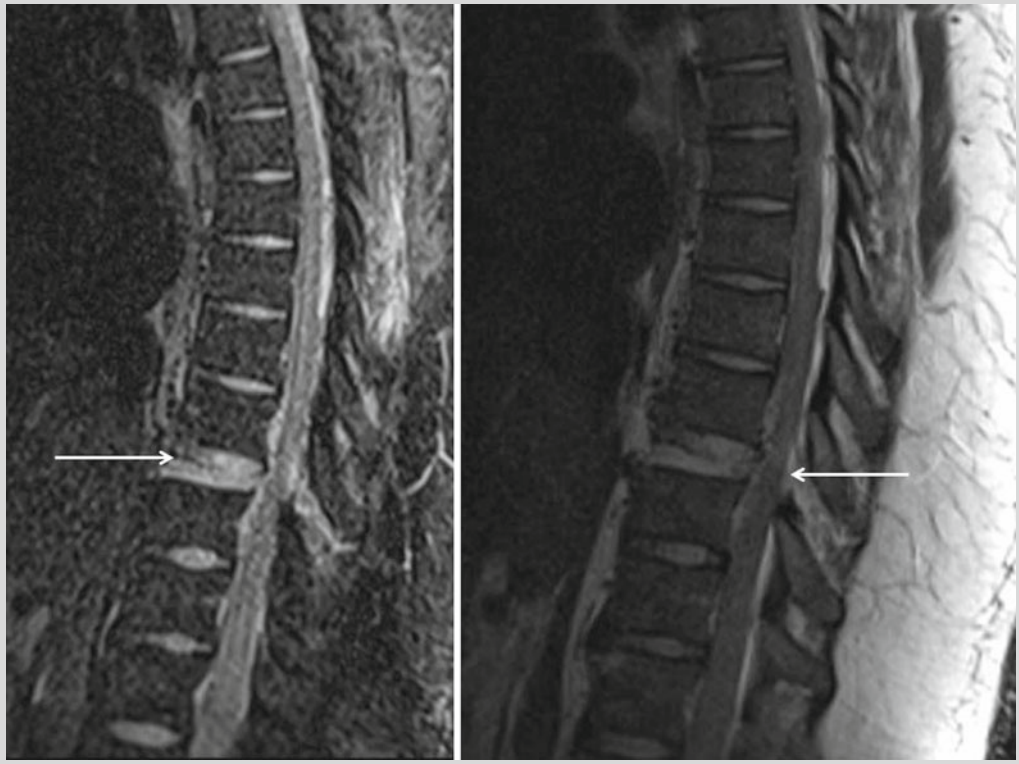


Fig. 8.2 STIR sequence (*left*) and T2-weighted (*right*) magnetic resonance imaging (MRI) pictures demonstrating severe damage to the T9–10 segment with complete compromise of the intervertebral disk (*left arrow*), the longitudinal ligaments, and the posterior ligamentous complex (PLC) (*right arrow*). Also note the spinal cord injury edema – this resulted in a complete neurological injury

T9–10 reduction, fixation, decompression, and fusion (Figs. 8.4 and 8.5). Despite an extensive hospitalization complicated by polymicrobial pneumonia and colon infection, the patient made a substantial recovery. It was determined that his spinal cord injury was complete (American Spinal Injury Association, Type "A" or ASIA-A) at the level of the T9–10 injury. However, he remarkably maintained complete function of his upper extremities following stabilization of his high-cervical injury. This occurred despite near-catastrophic occipitocervical dissociation. This case demonstrates all of the important concepts in treating polytrauma patients with concomitant spinal injuries including proper field care, coordination of care with other specialties upon arrival at the treating institution, accurate and full recognition of all injured bodily systems, adequate resuscitation, appropriate timing of surgery, proper execution of spinal treatment interventions, aggressive aftercare strategies, and thorough rehabilitation protocols. When adherence to these important guidelines is accomplished, treating teams can be successful in restoring maximal function to these severely injured and complex patients.

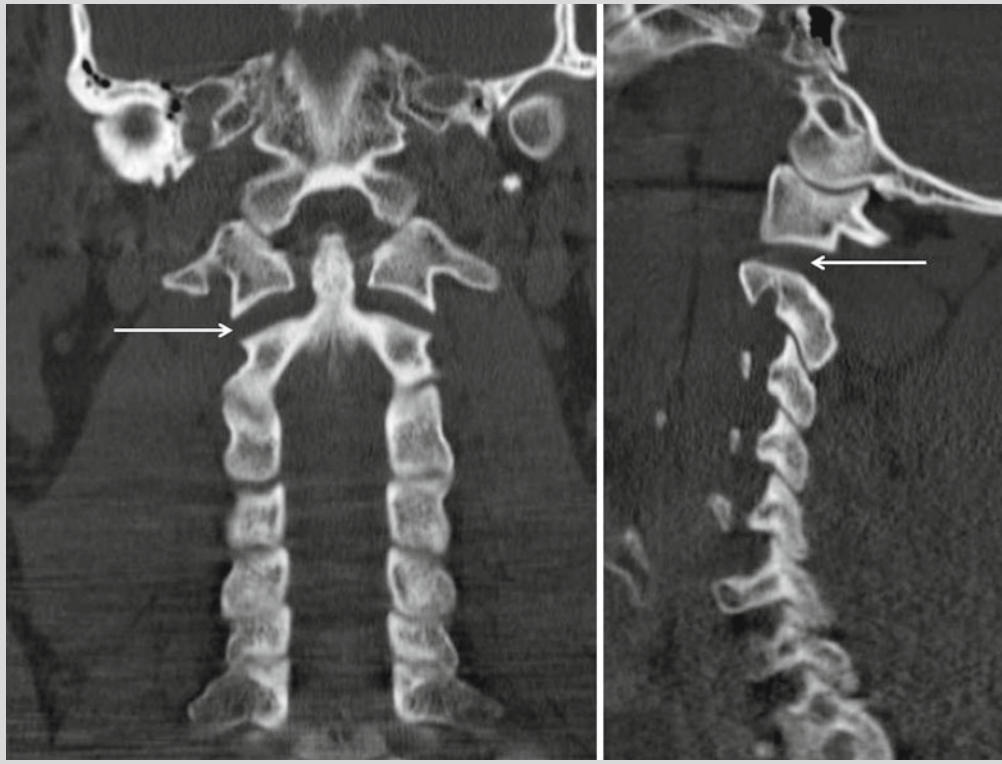


Fig. 8.3 Vertical distraction injury at the occipitocervical junction. Coronal and left parasagittal computed tomography (CT) images demonstrate a vertical distraction injury. This represents a type 2B occipitocervical dissociation in which the connection between the atlantoaxial articulation is lost. The skull and atlas ring have migrated in a vertical fashion away from the axis bone. The *arrows* point to severe atlantoaxial facet joint diastasis. This represents a highly unstable craniocervical relationship

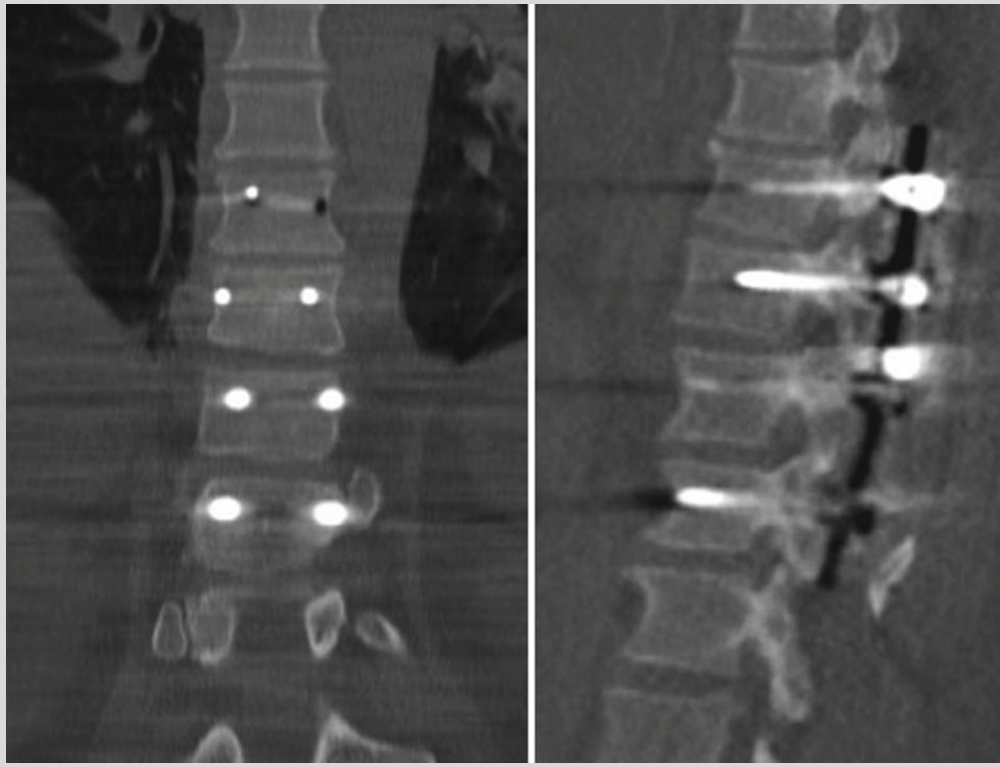


Fig. 8.4 Postoperative computed tomography (CT) images demonstrate T8–11 open reduction and internal fixation with instrumented posterior spinal fusion of the T9–10 fracture-dislocation

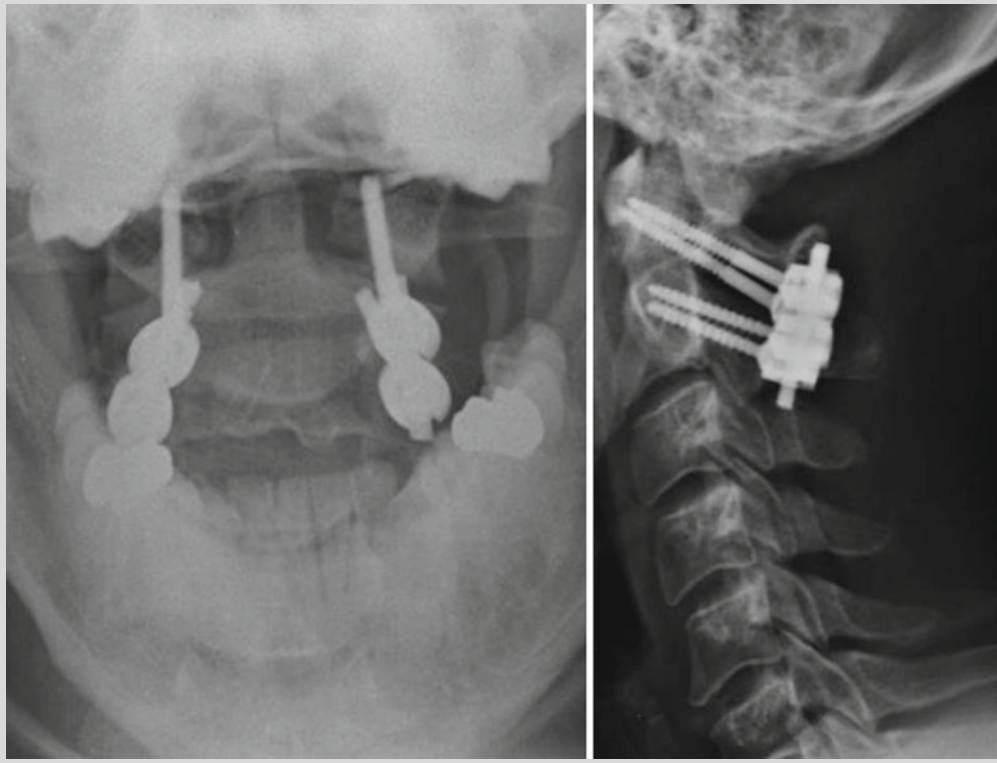


Fig. 8.5 Anteroposterior and lateral radiographs of the atlantoaxial open reduction and internal fixation with instrumented posterior C1–2 Harms-type spinal fusion. From both a cervical and thoracic standpoint, the patient garnered immediate stability which enabled instant mobilization without bracing. This facilitated easier therapy and nursing care in the surgical intensive care unit

Epidemiology

Spinal injury is common in polytrauma patients. Associated spinal cord injury (SCI) is arguably the most devastating survivable injury a patient can endure. SCI was formerly considered a death sentence, but modern medical care has enabled much improved quality of life and life span [1]. However, life expectancy is still foreshortened compared to the general population [2–4]. Incidence of SCI is approximately 429 cases per million person-years in the United States of America [5]. On a worldwide scale, the number is more difficult to calculate, but the incidence lies between 10 and 80 cases per million population [6]. Prevalence is increasing given the longer life expectancy of SCI survivors. As with most traumatic injuries in the United States of America, young white males are most affected. These patients are also more commonly single, uneducated laborers. Mechanism of injury includes motor vehicle accidents, falls from significant height, and gunshot wounds as the most common forms. Injuries usually occur in summer months and on weekends and are associated with other bodily injury at least half of the time. Injuries that accompany SCI most often include facial fractures, head injury, trunk injury, and long bone fractures [7]. Neurological injury level is most often cervical, followed by thoracic, and then lumbar. Patients affected by SCI suffer from pneumonia, thromboembolic events, and pressure sores and typically succumb to afflictions of the respiratory tract. Cost of SCI is a huge burden

to patients, families, communities, and the total health-care system. Polytrauma patients that suffer severe spinal injuries with associated neurological compromise should be treated at institutions well versed in trauma and acute SCI care. Many tertiary care centers go to great lengths to develop comprehensive SCI protocols. These guidelines often include every detail regarding the care of these complex patients. Examples include directions about maintaining goals for mean arterial pressure [8] and specifics about skin care and bowel programs. Every detail of care is contained in these protocols in order to guide the caretakers. These protocols involve the interactions of innumerable health-care providers and are coordinated from the time these complex patients enter the trauma bay and continue throughout their hospital stays. These interdisciplinary efforts progress until patients are fully stabilized and ready for transport to the secondary rehabilitation center. These secondary centers are often specialized institutions that deal directly with the prolonged recovery and rehabilitation associated with spinal injury and neurological deficits that polytrauma patients can sustain.

Field Care

Emergency medical personnel must assume spinal injury is present when stabilizing and transporting polytrauma patients. Injured individuals are secured to spinal boards and further immobilized in cervical collars often with the addition of sandbags and sturdy tape. Log-rolling is essential when movement of injured patients is necessary. Field personnel prioritize treatment in the sequence of life, limb, and function. Injured athletes and motorcycle riders should have helmets and shoulder pads left intact until safe removal in the emergency department of the treating hospital. In-line immobilization of the head-neck unit (while aligned with the trunk) should occur while attempting to limit flexion and extension. Understanding that the head circumference of a child in proportion to the body size is larger than an adult is important during immobilization and transport. The torso of a child should be elevated with padding or a special child spine board should be utilized with a cutout for the occiput to protect against undue cervical flexion. Emergency personnel can also be extremely useful in communicating presence of neurological deficits to the treating specialists upon arrival at the trauma center. Furthermore, the emergency transport team can relay important details regarding the accident scene to the treating doctors during the hand-off in the trauma bay. This can provide useful information to care providers regarding injury mechanism and pattern-of-injury which may be significantly important during evaluation of complex polytrauma patients. Upon arrival to the emergency department, rapid assessment and evaluation needs to begin immediately in order to allow early removal of the spinal board and enable proper examination of the entire spinal column. It should also be noted that the "one-size-fits-most" cervical immobilizers applied in the field by emergency personnel are often not adequate for proper immobilization of the cervical spine in these highly injured patients. It should be a goal of the treating caretakers to rapidly assess if a cervical injury exists. If cervical injury is absent and the treating team can determine that the spine is "cleared," then these field collars should be removed as soon as possible. If the treating team determines that a cervical injury is present, then a more properly fitted rigid cervical immobilizer should be applied and the spine surgeon should be contacted immediately for further evaluation.

Initial Assessment

Initial assessment of the polytrauma patient must start with acquiring and assimilating all available data related to the patient's injury mechanism and circumstances. Special attention should be paid to the pattern-of-injury. A classic "injury pattern" is the presence of visceral injuries that may accompany a flexion-distraction "Chance" fracture in the setting of a motor vehicle crash where a patient has employed only a lap belt (Figs. 8.6, 8.7, and 8.8). Severe injuries can also occur to the large vessels when flexion-distraction

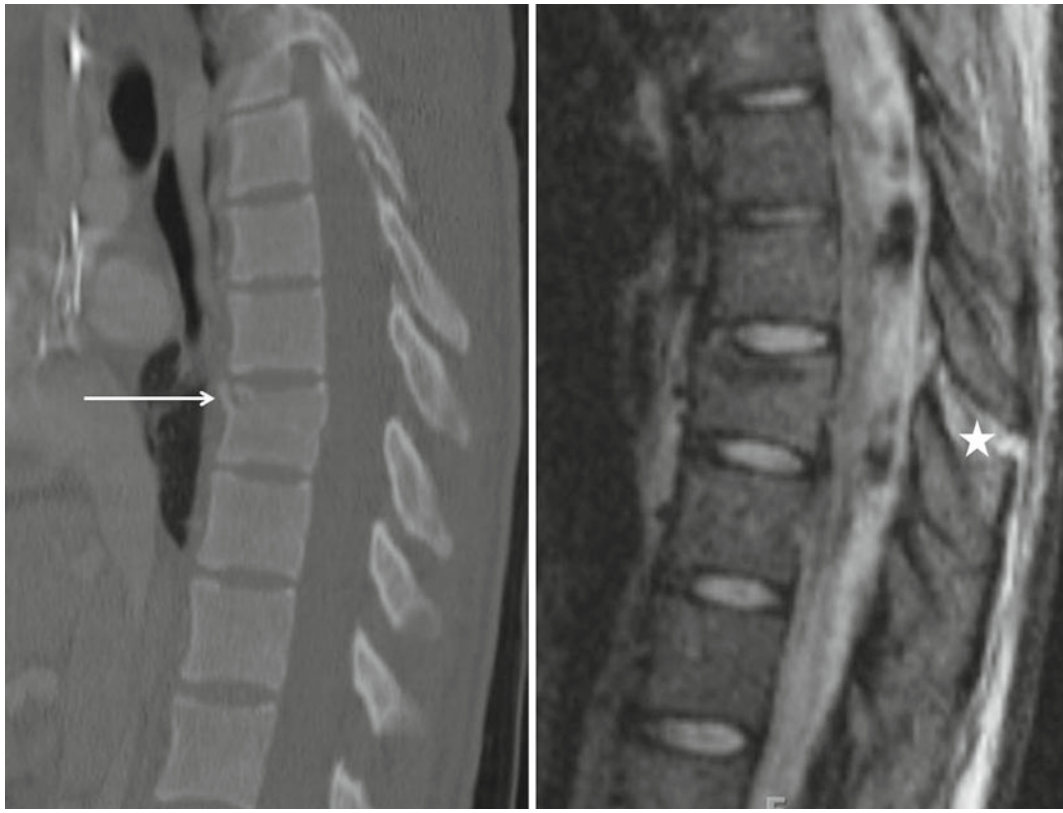


Fig. 8.6 Midsagittal computed tomography (CT) image and STIR MRI image depict a flexion-distraction injury to the thoracic spine. This poor young woman sustained severe abdominal injuries including splenic and liver rupture, pancreatic injury, and inferior vena cava laceration. These injuries often accompany lap belt-type spinal injuries ("Chance fractures") that are the result of flexion-distraction mechanisms. This patient underwent emergent exploratory laparotomy with inferior vena cava repair, splenectomy, and liver packing. Once properly resuscitated and physiologically stabilized, the patient returned to the operating room 24 h later for an abdominal washout followed by closure. She was then rolled prone in the same setting and underwent operative spinal stabilization. The *left arrow* represents a small avulsion teardrop-type fracture that should alert the spinal surgeon to the possibility of a flexion-distraction fracture. The *star* is positioned about the posterior ligamentous complex rupture

moments are applied to the thoracolumbar spine [9]. Another example of a spinal "injury pattern" that must not be overlooked includes the presence of head and facial lacerations, abrasions, and fractures that may be coupled with an extension-compression cervical fracture mechanism. Extension-compression cervical injuries can also be associated with central cord syndrome especially when involving a spondylitic, stenotic spinal column (Figs. 8.9 and 8.10) [10]. This incomplete spinal cord injury syndrome typically manifests with greater deficits in the upper extremities compared to the lower extremities. Falls from significant height may show concomitant pelvis, acetabular, and lower extremity fractures coupled with lumbar and thoracolumbar axial-loading injuries [11]. Severe chest injuries may be linked to thoracic fractures and fracture-dislocations [12, 13]. All of these examples point to the importance of considering and understanding the pattern-of-injury that accompanies certain traumatic accident mechanisms. These patterns can often be deduced from an analysis of the patient's complete list of injuries as well as discussing with emergency personnel the particular details of the injury scene along

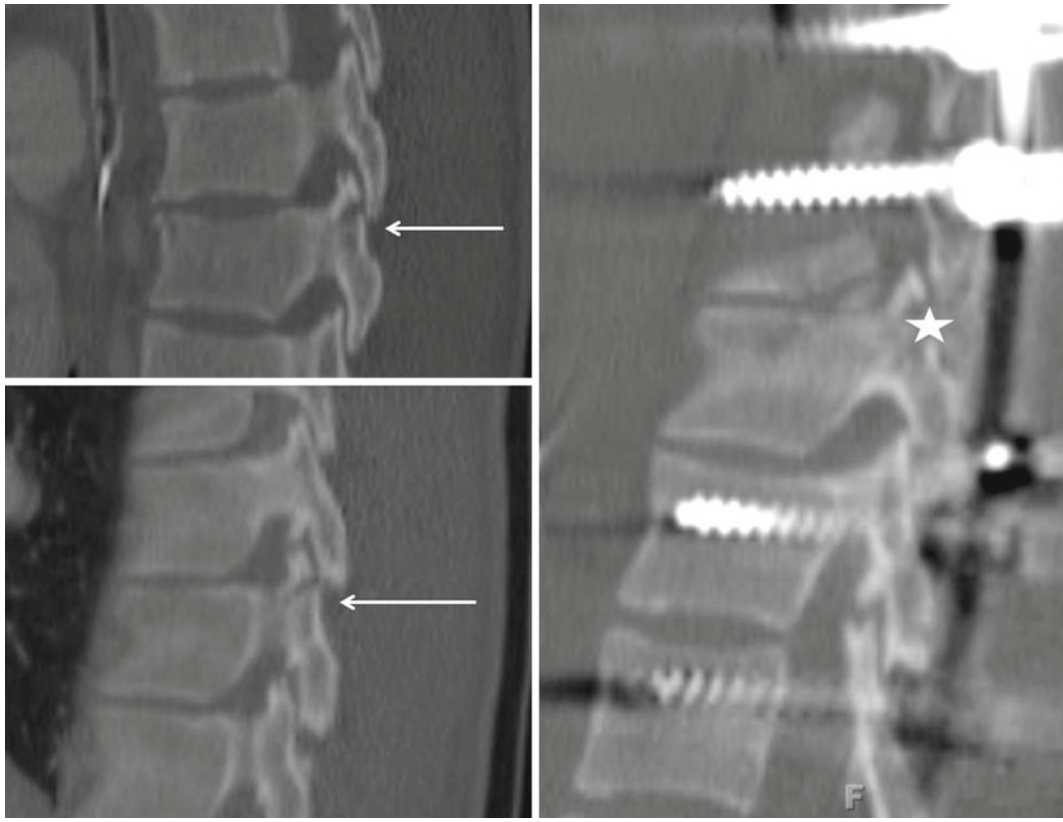


Fig. 8.7 Left and right parasagittal computed tomography (CT) images show bilateral pars/facet fracture-subluxations (*arrow*) which add to three-column spinal instability. The postoperative parasagittal image shows a nicely reduced and stabilized facet joint (*star*)

with eyewitness accounts. Prior to a thorough analysis of the injury pattern, treating teams should always adhere to ATLS protocol. Spinal evaluation can start concurrently with the primary survey and resuscitative measures through gross inspection. Secondary survey quickly follows with identification of neurological deficits, exposure and visualization of the entire spinal column, recognition of severely unstable injuries, as well as determining the presence of spinal and/or neurogenic shock. A detailed neurological examination is essential as the next step in evaluation. The ASIA classification can serve as a guide to proper neurological assessment (Fig. 8.11). Care providers need to understand the extreme importance of a proper and thorough rectal examination (Table 8.1). This examination can serve as a "window to the spinal cord" in obtunded patients. Spinal specialists should keep in mind the possibility of a "double-crush" phenomenon that may alter the rectal examination findings where two separate neurological insults exist in the same patient. This can lead to a drastically altered neurological exam that requires more diagnostic data usually in the form of MRI scan. Additionally, spinal specialists should be aware of conus medullaris level injuries that can lead to a mixture of upper and lower motor neuron signs. Injuries at this level can produce confusion in the early stages of spinal assessment. They can also terminally destroy the bulbocavernosus reflex arc. Following thorough examination, and once adequate physiological stability is provided, polytrauma patients can then receive appropriate radiological evaluation. Obtunded patients require total spinal imaging with CT scan. Injured spinal segments often require further analysis with MRI scan to

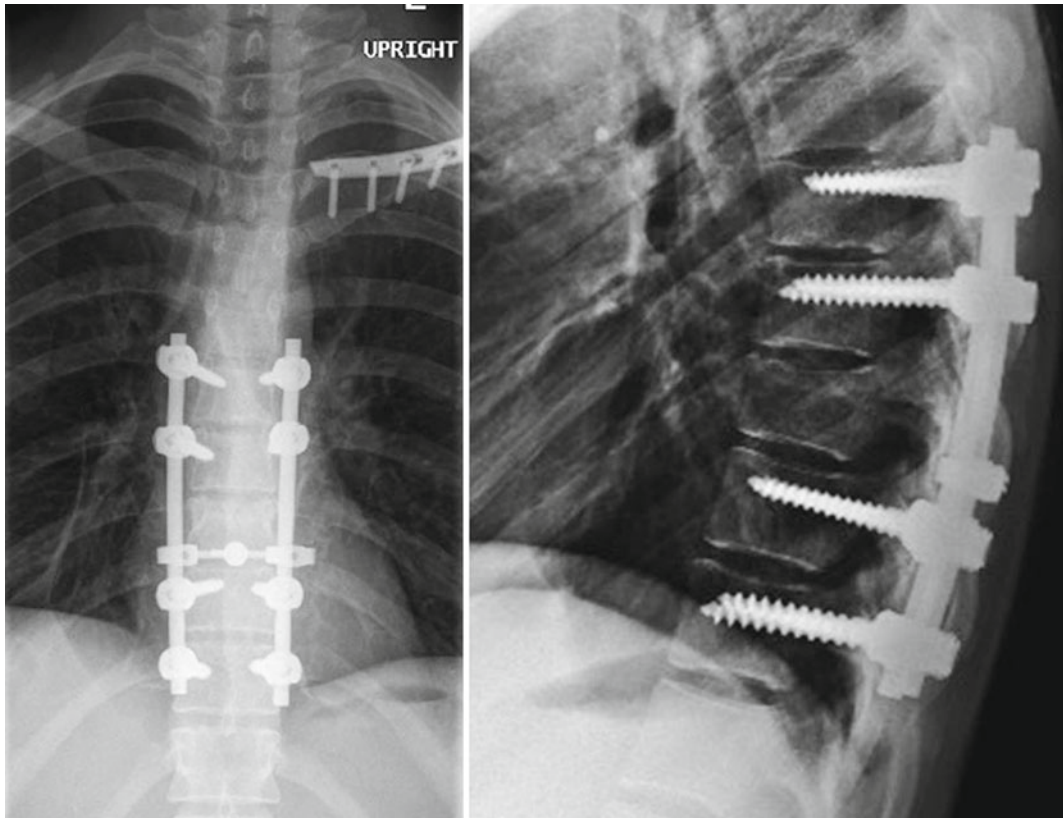


Fig. 8.8 Postoperative anteroposterior and lateral X-ray images of the thoracic spine showing T7–11 open reduction and internal fixation with instrumented posterior spinal fusion for the T9 "Chance fracture." Notice the image is taken with the patient upright. The immediate spinal stability provided by the procedure enables early mobilization and proper nursing care. It also obviates the need for bracing, which could be quite detrimental to a patient with multiple abdominal injuries. Also, notice that the clavicle has been repaired. This reduction and fixation event was completed by the orthopedic traumatologist 2 days after spinal fixation. This demonstrates the multidisciplinary approach to polytrauma care that these complex patients demand

evaluate the soft tissues and neural structures. With this information, spinal injuries can then be classified adequately, and appropriate treatment is then chosen by the spinal specialist.

Initial Management

Polytrauma patients with concomitant spinal injuries should be kept on log-roll precautions until definitive management strategies can be employed. Log-roll precautions include rigid cervical collar immobilization. The entire care team should understand the concepts and goals of log-roll precautions including frequent turning to avoid decubitus ulcer formation. Alternatively, a rotating frame bed may be utilized. The spine specialist should act quickly to determine an effective treatment plan that will enable mobilization of the patient and easier nursing care. More recent protocols recommend spinal stabilization within 24 h of injury to facilitate early mobilization and intensive care delivery [14]. This early interventional approach is a key component in reducing the incidence of complications in these multiply injured patients including reduced pulmonary, thrombotic, and integumentary adverse events. When early surgery cannot be employed due to a patient's compromised physiological status,

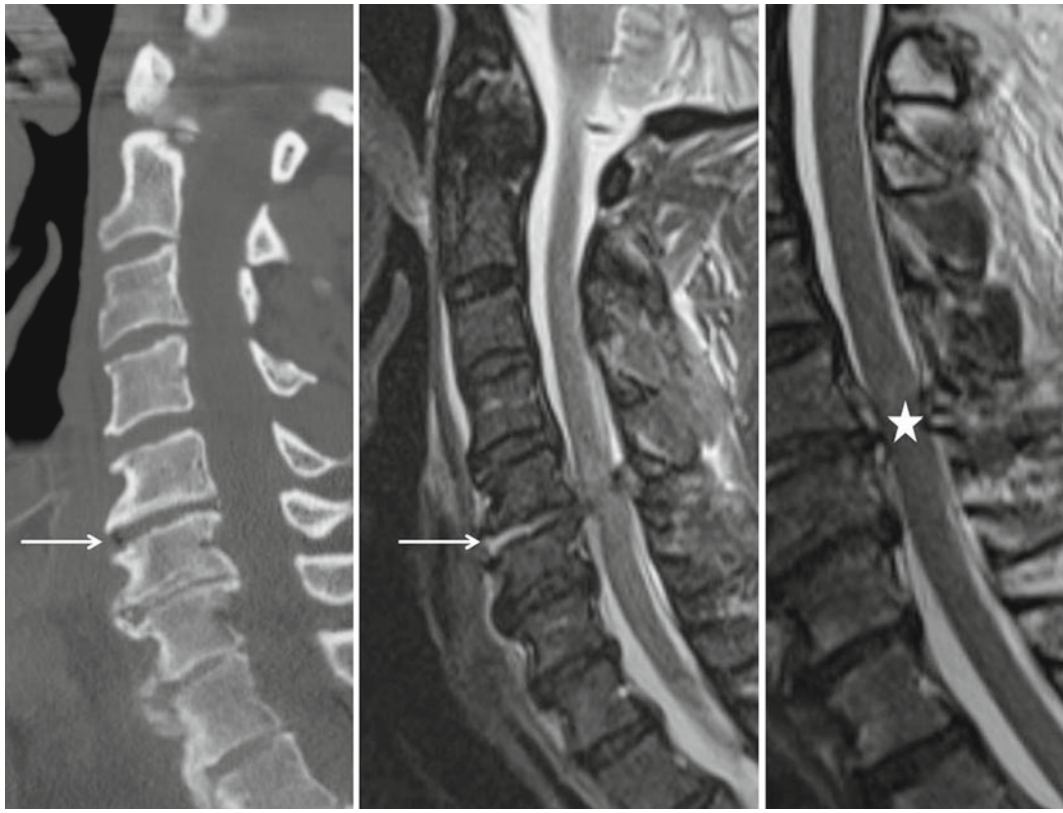


Fig. 8.9 Midsagittal computed tomography (CT) image (*left image*) of the cervical spine demonstrating maintained alignment and significant spondylosis. The only hint of an injury is the air diskogram at C5–6 (*arrow*). However, considering the "pattern-of-injury" in this patient, one develops much more concern. A fall from height with significant force impacting the facial region (multiple facial abrasions, lacerations, and a nasal bone fracture) coupled with a neurological exam showing mainly distal upper extremity deficits (consistent with central cord syndrome) led to an emergent MRI scan. Midsagittal STIR sequence and T2-weighted MRI cervical spine images (*middle* and *right image*) demonstrate C5–6 disk injury (*arrow*) along with stenosis, yellow-ligament buckling, and spinal cord injury edema (*star*)

then alternative methods can be used. These methods include application of Gardner-Wells tongs traction, halo-ring traction, or halo-fixator application to reduce subluxations/dislocations in a closed manner. Halo-vest fixator application can also temporarily reduce and stabilize unstable occipitocervical and cervical injuries until internal fixation strategies can be employed. It can also be effective to utilize fluoroscopic analysis of injuries in the intensive care unit setting to ensure that alignment has been restored with these closed methods. This can eliminate the need to transport patients to the radiology suite and can also improve image quality compared to portable X-rays.

When the treating spinal specialist cannot employ closed means of stabilization such as halo-vest fixator placement or Gardner-Wells tongs traction and manipulation, it is important to relay the appropriate spinal precaution data to the primary caretakers. For instance, if a patient's physiological status precludes definitive stabilizing surgery, but an unstable thoracic or lumbar spinal fracture exists, the care team should have specific instructions regarding the care, mobilization, and treatment of that patient from the spine perspective. Considerations include "head-of-bed" instructions/restrictions, frequency of neurological assessments, location and type of temporary brace fitting, delivery of medications,

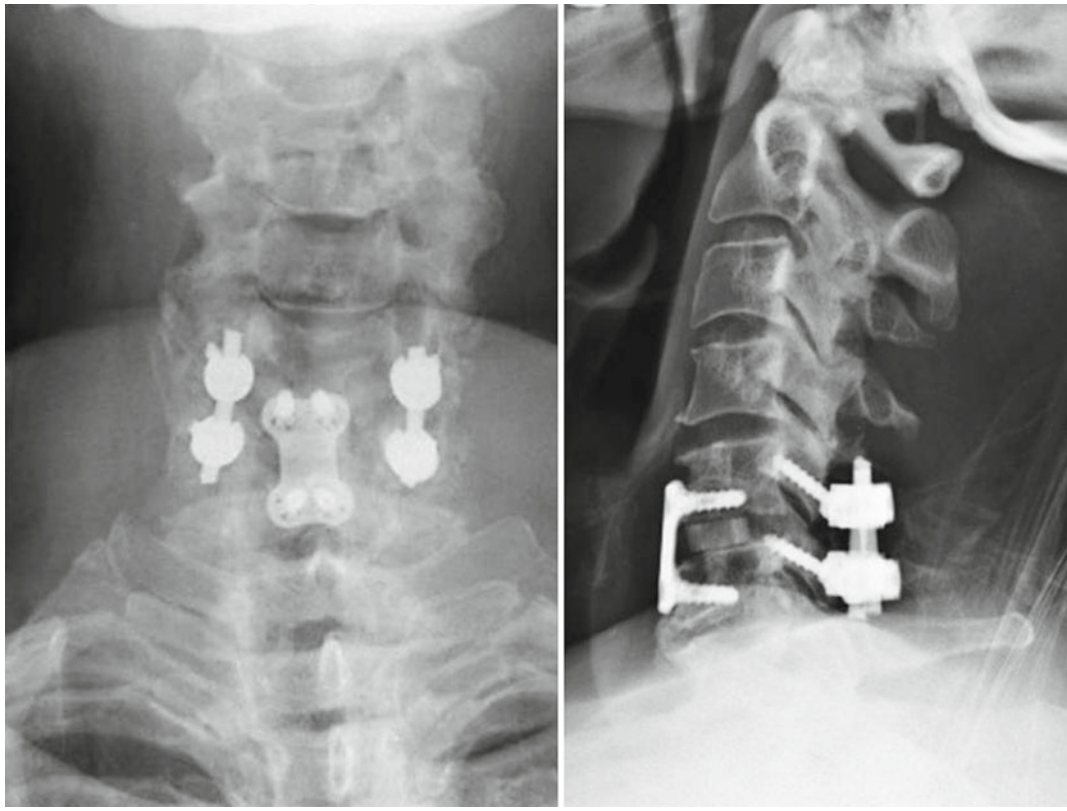
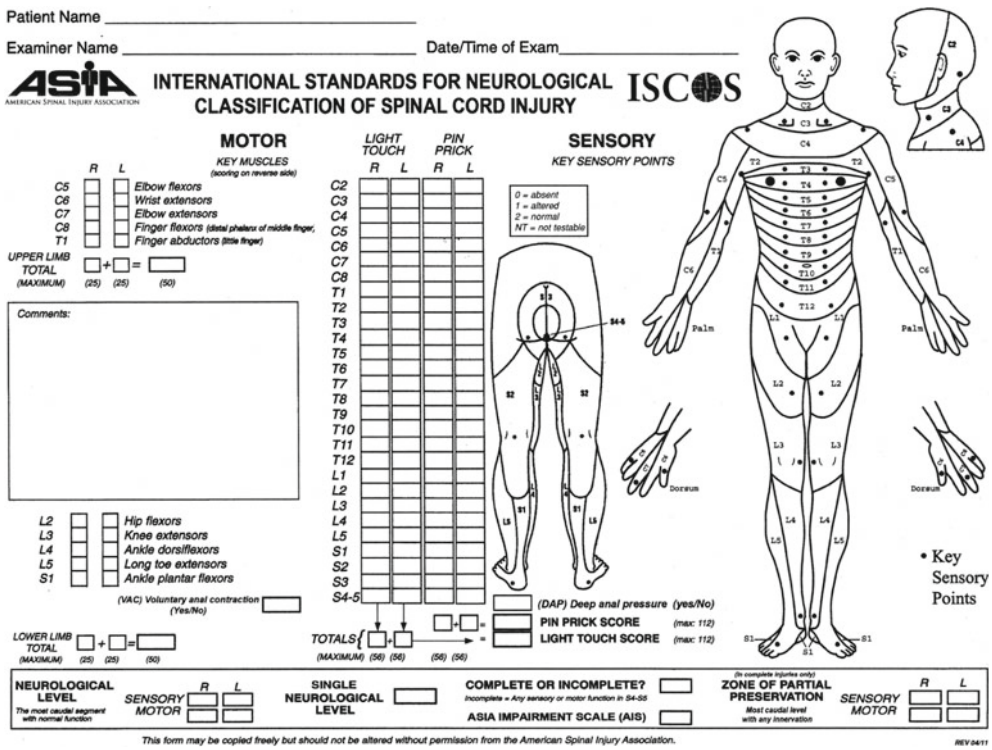


Fig. 8.10 Postoperative anteroposterior and lateral X-rays depicting C5–6 anterior cervical discectomy and instrumented fusion with insertion of machined, lordotic, allograft, interbody spacer coupled with instrumented posterior C5–6 fusion and decompression which includes C5 and C6 partial laminectomies and excision of the buckled ligamentum flavum with thorough decompression of the spinal cord. The X-rays are taken with the patient standing. This patient made a substantial postoperative neurological recovery

involvement of physical and occupational therapy (if indicated), and an explicit description of the definitive treatment plan. Once the entire care team understands the spinal treatment plan, patients can receive quicker and more appropriate interventions while awaiting the definitive spinal treatment solution.

Early care of polytrauma patients with spinal injuries also includes recognizing spinal shock and neurogenic shock and understanding the difference. Spinal shock is a transient syndrome of sensorimotor dysfunction. It is characterized by flaccid areflexic paralysis and anesthesia below the level of a spinal cord injury. The syndrome typically lasts between 24 and 72 h and has ended when reflexic activity resumes below the injury. The classical notion is that return of the bulbocavernosus reflex heralds the end of spinal shock. However, this topic is currently under debate by many physicians that routinely treat spinal cord injury [15]. The important message is that during the spinal shock phase which follows an SCI, the treating team cannot appropriately predict spinal injury level, spinal injury severity, or spinal recovery prognosis. Additionally, one should consider injuries directly to the conus medullaris where the bulbocavernosus reflex arc may be terminally destroyed. In contradistinction to spinal shock is the concept of neurogenic shock. This syndrome results from impaired sympathetic outflow tracts as a result of SCI and is accompanied by hypotension and bradycardia. It is diagnosed only after ruling out hemodynamic shock in the polytrauma patient. It is

a



b

Muscle Function Grading

0 = total paralysis

1 = palpable or visible contraction

2 = active movement, full range of motion (ROM) with gravity eliminated

3 = active movement, full ROM against gravity

4 = active movement, full ROM against gravity and moderate resistance in a muscle specific position.

5 = (normal) active movement, full ROM against gravity and full resistance in a muscle specific position expected from an otherwise unimpaired peson.

5* = (normal) active movement, full ROM against gravity and sufficient resistance to be considered normal if identified inhibiting factors (i.e. pain, disuse) were not present.

NT = not testable (i.e. due to immobilization, severe pain such that the patient cannot be graded, amputation of limb, or contracture of >50% of the range of motion).

ASIA Impairment (AIS) Scale

☐ **A = Complete.** No sensory or motor function is preserved in the sacral segments S4-S5.

☐ **B = Sensory Incomplete.** Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5 (light touch, pin prick at S4-S5: or deep anal pressure (DAP)), AND no motor function is preserved more than three levels below the motor level on either side of the body.

☐ **C = Motor Incomplete.** Motor function is preserved below the neurological level**, and more than half of key muscle functions below the single neurological level of injury (NLI) have a muscle grade less than 3 (Grades 0-2).

☐ **D = Motor Incomplete.** Motor function is preserved below the neurological level**, and at least half (half or more) of key muscle functions below the NLI have a muscle grade ≥ 3.

☐ **E = Normal.** If sensation and motor function as tested with the ISNCSCI are graded as normal in all segments, and the patient had prior deficits, then the AIS grade is E. Someone without an initial SCI does not receive an AIS grade.

**For an individual to receive a grade of C or D, i.e. motor incomplete status, they must have either (1) voluntary anal sphincter contraction or (2) sacral sensory sparing with sparing of motor function more than three levels below the motor level for that side of the body. The Standards at this time allows even non-key muscle function more than 3 levels below the motor level to be used in determining motor incomplete status (AIS B versus C).

NOTE: When assessing the extent of motor sparing below the level for distinguishing between AIS B and C, the ***motor level*** on each side is used; whereas to differentiate between AIS C and D (based on proportion of key muscle functions with strength grade 3 or greater) the ***single neurological level*** is used.

Steps in Classification

The following order is recommended in determining the classification of individuals with SCI.

1. Determine sensory levels for right and left sides.
2. Determine motor levels for right and left sides.
   Note: in regions where there is no myotome to test, the motor level is presumed to be the same as the sensory level, if testable motor function above that level is also normal.
3. Determine the single neurological level.
   This is the lowest segment where motor and sensory function is normal on both sides, and is the most cephalad of the sensory and motor levels determined in steps 1 and 2.
4. Determine whether the injury is Complete or Incomplete. (i.e. absence or presence of sacral sparing)
   If voluntary anal contraction = **No** *AND all S4-5 sensory scores =* **0** *AND deep anal pressure =* **No,** *then injury is COMPLETE. Otherwise, injury is incomplete.*
5. Determine ASIA Impairment Scale (AIS) Grade:
   Is injury Complete? If **YES,** AIS=A and can record ZPP (lowest dermatome or myotome on each side with some preservation)
   NO ↓
   Is injury motor Incomplete? If **NO,** AIS=B (Yes=voluntary anal contraction OR motor function more than three levels below the motor level on a given side, if the patient has sensory incomplete classification)
   YES ↓
   Are at least half of the key muscles below the single neurological level graded 3 or better?
   NO ↓ AIS=C
   YES ↓ AIS=D
   If sensation and motor function is normal in all segments, AIS=E
   Note: AIS E is used in follow-up testing when an individual with a documented SCI has recovered normal function. If at initial testing no deficits are found, the individual is neurologically intact; the ASIA Impairment Scale does not apply.

Fig. 8.11 American Spinal Injury Association (ASIA) standard neurological evaluation worksheet (**a**). ASIA Grading/Scale/Classification Tool (**b**). (Used with permission of the American Spinal Injury Association)

Table 8.1 A thorough and reliable rectal examination

1. Inspection
Visualize the anus
Presence of feces can signify incontinence
2. Sensory evaluation
Check perianal light touch
Check perianal pin prick
3. Reflex assessment
Check anal wink reflex
Check bulbocavernosus reflex (requires insertion of Foley catheter in women)
4. Motor assessment*
Check resting tone
Check voluntary tone if possible

The sequence and elements of a proper and thorough rectal examination are shown. This is an important test that should be performed on each and every polytrauma patient as part of the secondary survey and neurological examination

Performing the test in this order (1 → 4) can minimize patient discomfort

*Check digit for blood – significant pelvic trauma may exist

typically associated with more cephalad levels of spinal injury. Fluid resuscitation should proceed carefully while monitoring the markers of resuscitation including base deficit and lactate levels. Treatment of neurogenic shock should focus on increasing peripheral vascular resistance. Additionally, treatment should focus on definitive management of the spinal column injury with restoration of normal anatomical alignment and stabilization along with decompression of impinged neural elements as soon as patients can medically tolerate such procedures.

Not only do patients with neurogenic shock require careful blood pressure monitoring and pharmaceutical interventions to adjust for this potentially labile problem, but so too do patients with SCI even in the absence of neurogenic shock. There is increasing data to suggest that maintaining mean arterial blood pressure above 85–90 may be beneficial to SCI patients with regard to diminishing secondary injury [16]. The primary spinal cord injury occurs as a result of the physically deformed or compressed neural tissue usually as a result of fracture fragments, misalignment, ongoing compression from disk material, or a combination of factors. These offenders should be dealt with as soon as patient's resuscitative status allows. Secondary injury to the spinal cord comes from diminished blood flow to the neural structures which can theoretically be benefited by maintaining adequate systemic blood pressure. Secondary injury can also be induced by vascular changes, endothelial damage, vascular clotting, hemorrhage, inflammation, production of free radicals, programmed cell death, electrolyte dysfunction, and release of neuroexcitatory transmitters that prove to be cytotoxic. To date, there is no single pharmaceutical medication that can heal the spinal cord. However, there are many potential interventions that can attack the problem at the level of the secondary injury. The future hopefully holds a strategy to successfully treat these spinal cord injuries. Restoring neural tissue and function is certainly one of medicine's holy grails. One strategy that has been employed for many years for injured neural tissue is the administration of corticosteroids. Although it is still a topic of much debate, the delivery of high-dose steroid protocols has largely been abandoned at institutions that routinely treat spinal cord injured patients. The potential recovery of neural function is outweighed by the adverse effects on infection and pulmonary complications [17].

Many challenges exist with delivery of medications to labile polytrauma patients. This is certainly the case when concomitant spinal column and spinal cord injuries are present. However, even when those problems do not exist, patients can experience issues with the most simple treatment modalities. Polytrauma patients invariably arrive at the treating center in a rigid cervical field collar. It is important that protocols exist on the institutional level to rapidly assess the need for such devices as well as determine their adequacy. Providers must also appreciate that maintenance of a cervical immobilizer is not a benign measure. Respiratory compromise and skin ulcerations are just two of the problems that can accompany these items. This presents the concept of "cervical spinal clearance" (Fig. 8.12). Clearance protocols for the cervical spine are now essentially ubiquitous. Nonetheless, the treating team, and most importantly, the treating

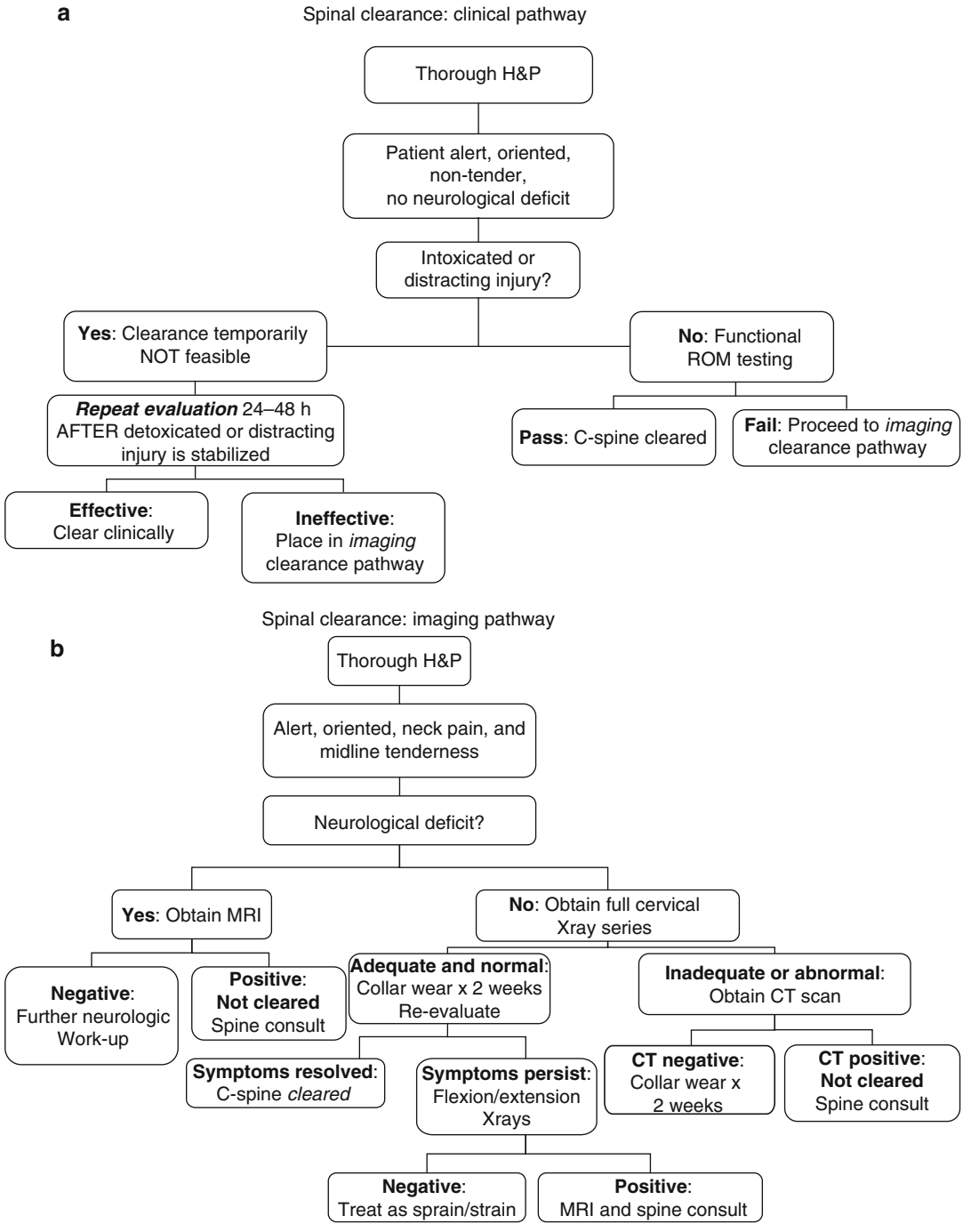


Fig. 8.12 (**a**) Spine clearance: Clinical pathway. An example of a "spinal clearance pathway" in an awake, alert and oriented, non-painful, non-tender, neurologically intact patient. In this setting, the physician can usually rely on clinical findings. (**b**) Spinal clearance: Imaging pathway. An example of a "spinal clearance pathway" in an awake, alert, and oriented patient that has spinal pain and tenderness on examination. In this setting, the physician requires imaging data in addition to clinical information to support decision making. (**c**) Spinal clearance: Obtunded pathway. An example of a "spinal clearance pathway" in an obtunded patient. In this setting, the physician typically relies upon the imaging data to inform clinical decision making

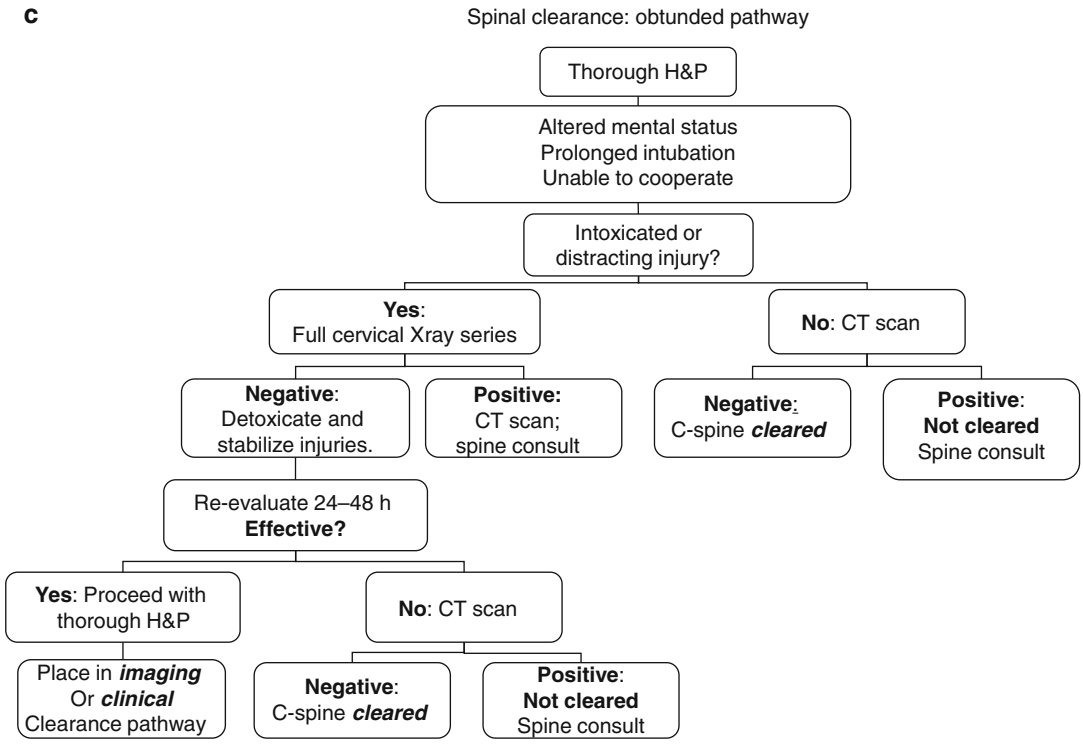


Fig. 8.12 (continued)

spinal specialist, needs to understand these concepts and be able to fully employ their directives. Spinal clearance can also be extrapolated from the neck to include the thoracic and lumbar spinal regions. "Clearance" implies that a thorough investigation of the spine has occurred and that no injury has been identified requiring treatment. Once that has been determined, all immobilization may be removed and log-roll precautions repealed. "Spinal clearance" is a challenging endeavor and may also be a time and labor-intensive process. It involves thorough history taking, complete physical examination, and thorough analysis of the radiological data. It may prove useful to divide the patients into four separate groups: asymptomatic, temporarily nonassessable, symptomatic, and obtunded. Once this is accomplished, the provider can easily run through the appropriate algorithm to determine the best course of treatment. Symptomatic patients require further radiographic assessment to determine diagnosis and treatment approaches. Asymptomatic, examinable patients without distracting injuries or intoxicated status can be clinically assessed and potentially cleared. Temporarily nonassessable patients require temporary rigid cervical immobilization until clearance of intoxicating substances, return of normal mentation, and/or stabilization of distracting injuries before they can be reliably assessed and potentially cleared. Obtunded patients require imaging to rule out spinal injuries. Polytrauma patients typically fit into the obtunded or temporarily nonassessable categories. Prolonged obtundation can present the treating spinal specialist with a dilemma regarding spinal clearance. Obviously, if an injury is identified, then a definitive treatment plan is determined and executed. If no injury is identified on CT scan, then the spinal specialist

must decide whether to remove the rigid cervical collar. Some physicians believe that an MRI scan is mandatory in this situation. Others feel that monitored flexion and extension radiographs (or fluoroscopic analysis) are warranted. However, the incidence of occult unstable spinal injuries occurring in the presence of a normal CT scan is extremely low. Therefore, many institutions remove the cervical collar in obtunded patients that have no identified spinal injury on CT scan [18].

Definitive Management

Once a spinal injury is identified in a polytrauma patient, a spinal specialist should be summoned for further characterization and treatment. Guiding the spine surgeon are fracture classification and injury schemes that enable thorough description, concise understanding, and precise treatment delivery recommendations [19]. The AO classification system, when coupled with the neurological status of the patient, can prove extremely useful for guiding the spine surgeon (Fig. 8.13). In addition, the Thoracolumbar Injury Classification and Severity Score (TLICSS) can help the surgeon determine if spinal injuries enter the operative range (Table 8.2) [20]. The most important components of assessing spinal injury include the mechanism and morphology of the injury, the neurological status of the patient, and the posterior ligamentous complex (PLC). The PLC is comprised of the supraspinous ligament, the interspinous ligament, the facet joint capsules, the intertransverse ligaments, and the ligamentum flavum. These components should be thoroughly analyzed. In addition to proper analysis and scoring, the physiological status of the patient, along with the presence of other injuries should be thoroughly considered when determining a definitive treatment plan. From a cervical spine standpoint, the Subaxial Spine Injury Classification and Severity Score (SLICSS) also places a very high priority on the diskoligamentous complex [21]. Once the treating spinal specialist determines the treatment to be initiated, swift execution of the plan is mandatory.

The goals of treatment for spinal column injury include protecting uninjured neural tissue, maximizing the recovery of injured neural tissue, and optimizing the conditions for the recovery of the musculoskeletal portions of the spine. Optimizing conditions for the spinal column include reducing deformity and providing solid stabilization. The ultimate goal is to restore the maximum amount of function to the injured person. This end goal is best achieved with an early start to the process. The timing of surgery, although left to some debate, is most appropriately determined by the physiological status of the patient in conjunction with consideration of the severity of the spinal injury. The overwhelming trend in spinal treatment is to provide early surgical reduction, stabilization, and decompression. The purpose of this type of intervention relies upon the premise that early mobilization avoids the complications associated with prolonged recumbency including respiratory deterioration, disuse atrophy, thromboembolic disease, integument breakdown, and infections. Surgical delay has been associated with increased pulmonary complications, urinary tract infections, and prolonged hospital stays, along with more wound complications and hospital-acquired infections. As such, many institutions have advocated for spinal reduction, stabilization, and decompression within a reasonably expedient time frame. Other institutions have more aggressive protocols mandating treatment within 24 h of the injury. These institutions argue that earlier mobilization, increased effectiveness of ICU care, and a "commonsense" approach to decompression (the soonest possible relief of ongoing neural impingement) mandate the quickest care possible [22].

After selecting the treatment modality, the spinal surgeon also chooses the approach, the method of fixation, the construct length and spinal levels to incorporate, as well as whether to fuse the spinal segments. Other considerations include combined anterior and posterior

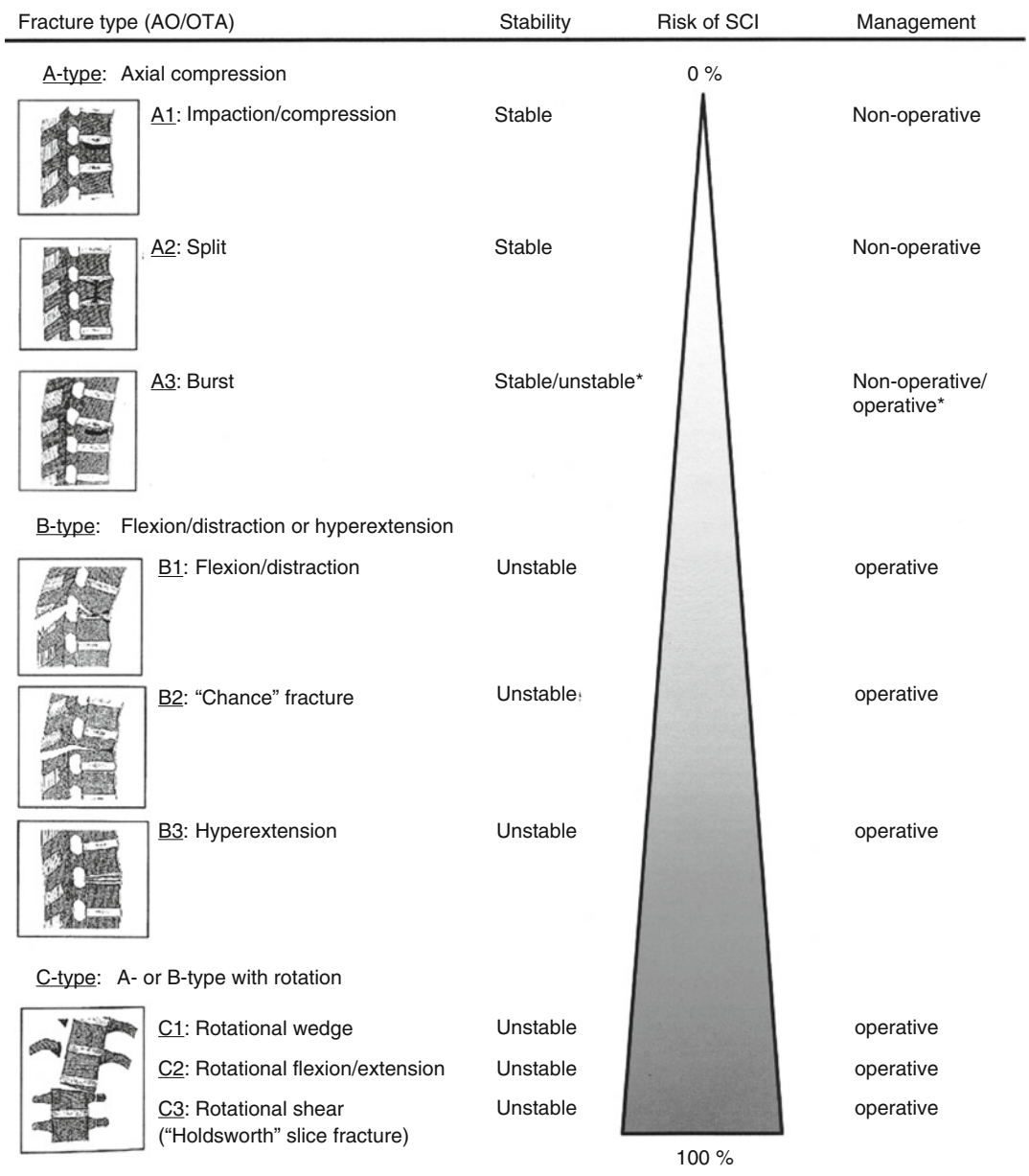


Fig. 8.13 Modified AO classification of spine fractures including fracture type, assessment of stability, neurological status, and treatment recommendation. A-type injuries result from mainly axial forces applied to the spinal column and produce anterior and middle column injuries ("A" = axial). B-type injuries involve bending forces that can couple both compression and tension depending on the location of the center of rotation ("B" = bending). The posterior ligamentous complex (PLC) is typically ruptured in these injuries. C-type injuries involve multidirectional forces and produce highly unstable injuries involving 360 ° of the spinal column ("C" = circle) including rupture of the PLC. When spine specialists couple these fracture mechanisms, the morphology of the injury, along with the neurological status of the patient, it can become fairly straightforward to determine stability and incorporate a surgical treatment strategy (Reproduced with permission from: Stahel et al. [27])

Table 8.2 The Thoracolumbar Injury Classification and Severity Score (TLICCS)

	Score
Fracture morphology	
Compression injury	1
Burst fracture	+1 = 2
Translational/rotational injury	3
Distraction injury	4
Neurological injury	
Intact	0
Nerve-root injury	2
Complete injury	2
Incomplete injury	3
Cauda equina injury	3
PLC	
Intact	0
Injury suspected	2
Injured	3
Summation	*Total score*
Nonoperative	<4
Grey zone	=4
Operative	>4

This scoring system has gained wide acceptance among the spine surgery trauma community and can prove very helpful in deciding to treat spinal injuries with surgical intervention. As shown, the important components (which receive point scores) are the mechanism and morphology of the injury, the neurological status of the patient, and the supporting posterior ligamentous complex (PLC). The sum of the point values in each category produce a total score that can help guide treatment

procedures, types of bone grafting and bone graft extenders/substitutes, decompression strategies, and whether to use postoperative external immobilizers to enhance immobility. Each one of these topics can easily encompass its own lengthy manuscript. As such, it makes more sense to reiterate the spinal surgical principles. Surgeons should rely on their expertise to deliver rapid reduction of spinal deformity, immediate stabilization that can withstand the rigors of postoperative mobilization and rehabilitation, as well as thorough decompression of impinged neurological structures in the safest manner possible. Surgeons should have a stocked rmamentarium of approaches, tools, instrumentation options, and backup plans to enable the smoothest delivery of invasive spinal trauma care to the polytrauma victim. Further priorities include the minimization of complications and maximizing functional outcomes through facilitating early mobilization and rehabilitation. Spine surgeons should also partner closely with their orthopedic traumatology colleagues to coordinate the care of polytrauma patients that most likely have concomitant appendicular skeletal trauma. This includes close discussion regarding the timing of multiple surgeries and the implementation of damage-control-orthopedics and spine-damage-control practices. One interesting area of overlap for orthopedic surgeons is lumbosacropelvic trauma. Case 8.2 demonstrates a pertinent example. Traumatic injuries to this area can often benefit from a team approach striving to provide lumbopelvic fixation and/or triangular osteosynthesis. This method is employed in a variety of kyphotic sacral fractures, vertically unstable sacral fractures, and lumbopelvic dissociation [23]. The spine surgeon and the orthopedic traumatologist can couple their efforts to provide stable fixation to the lumbosacral junction through utilizing a combination of pedicle screws, iliac bolts, and iliosacral screws. This enables patients to immediately bear full weight. In fact, patients can mobilize with severe lumbosacropelvic injuries that were previously treated with long periods of protected weight bearing [24]. Spine surgeons well versed in orthopedic traumatology applications can also offer full treatment of the entire spectrum of lumbosacropelvic trauma. This capability can only come about through rich communication and focused teaching efforts provided by orthopedic traumatology colleagues.

Case 8.2

An unfortunate 17-year-old female sustained a four-story fall through the roof of a factory. She injured her lumbosacral junction. This resulted in traumatic spondyloptosis and lumbosacral dissociation (Figs. 8.14 and 8.15). She had multiple injuries consisting of a humeral fracture, a hemopneumothorax, an open ankle fracture-dislocation, along with splenic and colonic contusions. She also sustained a cauda equina injury with loss of bladder control, vaginal numbness, and plantar flexion weakness. She was taken urgently to the operating room where she received tube thoracostomy, washout and splint stabilization of the ankle fracture, splint stabilization of the humeral fracture, and temporary reduction and traction stabilization of the lumbosacral dislocation utilizing bilateral distal femoral skeletal traction pins. She returned to the operating room 24 h later, following adequate resuscitation, for definitive spinal treatment. She received open reduction of the lumbosacral dislocation with stabilization and fusion utilizing lumbopelvic fixation methods (Fig. 8.16). She also received cauda equina decompression. At that time, inspection of the nerve roots revealed severe injury to bilateral S1. Her dural sac was also suture repaired. No further

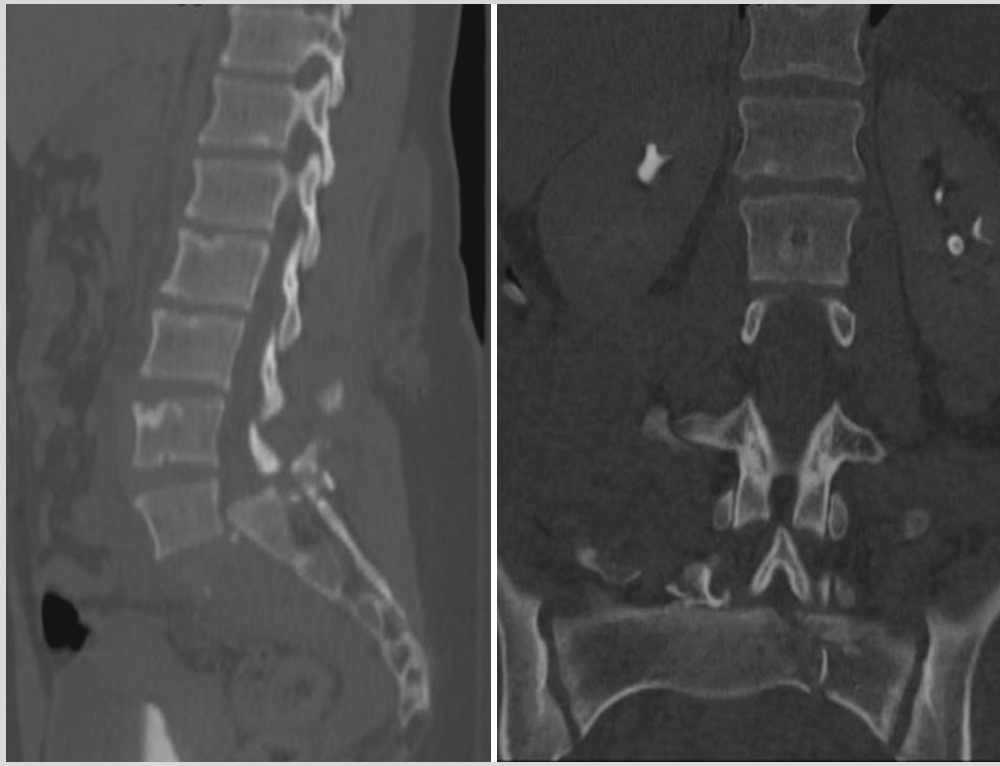


Fig. 8.14 Midsagittal and coronal computed tomography (CT) images of the lumbosacral spine demonstrating severe traumatic disruption of the lumbosacral junction. This traumatic L5–S1 Meyerding grade 5 spondylolisthesis contributes to a highly unstable lumbosacral dissociation

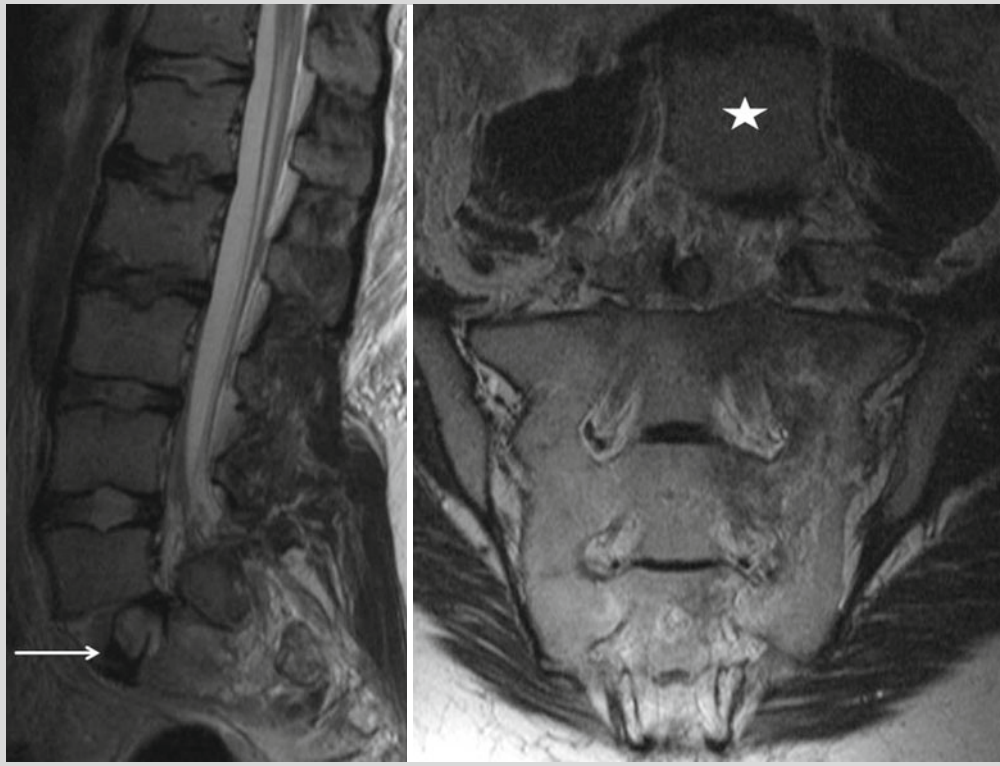


Fig. 8.15 T2-weighted midsagittal and coronal MRI images of traumatic lumbosacral dissociation. A severe injury to the cauda equina and the sacral nerve roots occurred which is typically associated with a traumatic spondyloptosis. Also, note the completely enucleated L5–S1 intervertebral disk (*arrow*). One can see a coronal view of the sacrum and an axial view of the L5 corpus (*star*) on the same image (*right side*)

sacral root inspection was performed. Later she received definitive fixation of her ankle and humeral fractures. She was discharged from the hospital approximately 2 weeks after her injury. Her sacral roots made some recovery, and she no longer required self-catheterization. Her vaginal sensation is also improved. She is now ambulating without gait aid. This case demonstrates the need for a multidisciplinary approach to polytrauma victims that also sustain spinal injuries. The patient's initial course involved the general surgery team, the orthopedic surgery and the spine team, along with the intensive care unit team. Working together enabled this young lady to make a marked improvement after a potentially devastating injury. This case also demonstrates the importance of knowing the initial management strategies for severe spinal injuries and further highlights the need to smoothly transition to definitive treatment methods. Early reduction, stabilization, and decompression enabled this patient to mobilize immediately following surgery and to avoid potentially severe complications associated with delayed treatment.

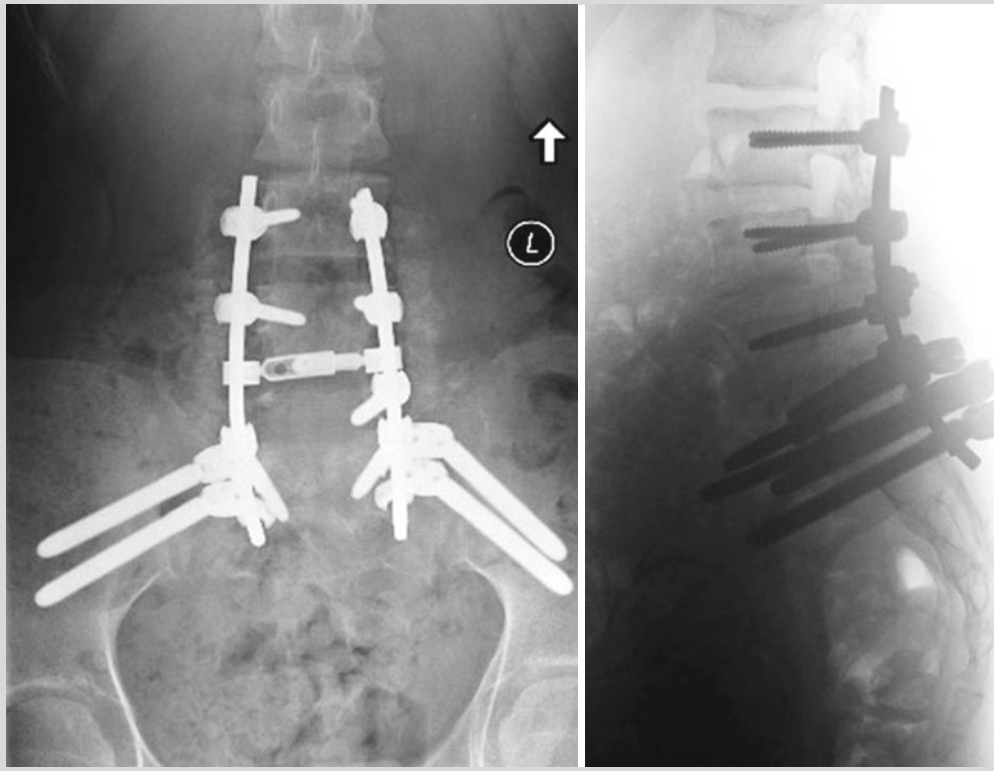


Fig. 8.16 Postoperative anteroposterior and lateral X-rays following open reduction and internal fixation with instrumented L3 iliac posterior lumbosacropelvic fixation and fusion. The patient also received a thorough decompression of involved neurological structures and repair of traumatically lacerated dural sac

Aftercare

Following polytrauma, patients require significant effort from all members of the treatment team to enable early mobilization and care of multiple injuries. When spinal injuries accompany the situation, this can complicate the delivery of other needed interventions. One goal of early and definitive internal fixation and stabilization of spinal injuries is to eliminate the need for external bracing which may interfere with nursing and other care modalities. External orthoses can disrupt respiratory care, exacerbate abdominal conditions, and contribute to integumentary breakdown and ulceration. Especially in polytrauma patients, eliminating the need for bracing can translate into an easier and smoother transition to the state of active rehabilitation. After surgery, considerations include treatment of traumatic spinal fluid leaks, presence and management of wound drains, proper incisional care, implementation of adaptive equipment for neurological injury, and placement in appropriately equipped care facilities. The list of caretakers and providers is quite extensive. This speaks to the complexity of providing care for these victims. Institutions frequently employ health-care providers that

can enable the highest level of care for these patients from every possible angle. These include nursing staff, physical therapists, occupational therapists, speech therapists, orthotists, prosthetists, respiratory therapists, wound care specialists, medical social workers, spiritual care providers, and physicians from all specialties. It is important to quickly involve all appropriate caretakers to enable delivery of needed interventions in a timely fashion. With this complex interplay at work, it is advisable to protocolize treatment delivery through the establishment of institutional guidelines that are easily followed and strictly enforced. This will ensure the best care at all time points throughout the treatment course of polytrauma patients with severe spinal injuries. After the initial recovery phase, patients may need further evaluation to assess for successful spinal fusion. Or, alternatively, they may require planned return to the operative suite to remove implanted spinal fixators.

Future Directions

As the treatment of spinal injuries in polytrauma patients evolves, it is becoming clearer that early intervention is both warranted and desirable. The goals of treatment remain well identified, and the delivery methods continue to improve. Options for fixation and stabilization continue to expand, while possibilities for less morbid and minimally invasive exposures are quickly developing. It is incumbent upon the treating spinal surgeon to remain educated with regard to the newest treatment strategies that are proven to be beneficial to patient care. One concept in orthopedic surgery that has gained widespread acceptance and implementation is that of "damage-control-orthopedics" [25]. This concept enables fast and effective stabilization with minimal invasion. Patients are temporarily stabilized with external measures that reduce tissue damage and secondary injury with the plan to return for definitive internal fixation once resuscitated and physiologically stable. This concept can be extrapolated to spinal injuries in polytrauma patients through the idea of "spine-damage-control" [26]. Case 8.3 demonstrates a pertinent example of spine-damage-control and is contrasted to an early-total-care model in Case 8.4. The spine-damage-control treatment concept relies upon the rationale that patients receiving the earliest possible stabilization of spinal injuries benefit from improved ability for intensive care and avoidance of complications associated with immobilization, log-roll precautions, and recumbency. The delivery of spine-damage-control involves the reduction and internal stabilization of spinal injuries through a posterior approach within 12–24 h of the injury to enable improved ICU care. Spine-damage-control can even occur in the same setting as external fixation of other fractures. Once resuscitated, the patient returns for definitive anterior stabilization, decompression, and fusion. Spine-damage-control mainly applies to thoracolumbar trauma associated with neurological insult. Spine-damage-control is best delivered by experienced surgeons that can provide a rapid exposure, place pedicle screws through a pure anatomical approach while avoiding wasted time for fluoroscopic analysis, and ensure a rapid and thorough decompression without injury to the meninges or neural elements with minimal blood loss. The next frontier for spine-damage-control will likely involve the implementation of percutaneous techniques for pedicle screw placement, rod passage, and spinal stabilization. This technique would not preclude the implementation of open techniques once patients are appropriately physiologically stabilized. Furthermore, spine-damage-control could successfully be coupled with staged anterior stabilization, decompression, and fusion in a delayed fashion.

Case 8.3

A 33-year-old male crashed his motorcycle at highway speeds and sustained multiple and severe injuries. The patient initially received an emergent exploratory laparotomy with splenectomy, bowel repair, liver packing, and bladder repair. This was done concomitantly with closed reduction and external fixation of femur and tibia fractures along with splint immobilization of upper extremity injuries. The patient was then resuscitated in the surgical intensive care unit. Reasonable physiological stability was obtained which enabled the patient to undergo thorough spinal imaging. Another survey also demonstrated lower extremity neurological deficits. It was determined that the patient had a thoracolumbar fracture-dislocation with associated spinal cord injury (Fig. 8.17). Within 24 h, the patient returned to the operating suite for spine-damage-control. This involved open reduction of the spinal deformity, reconstitution of the normal spinal alignment, stable internal fixation, instrumented posterior spinal fusion T10–L2, with decompression of the damaged neural elements (Fig. 8.18). Following the posterior spine-damage-control procedure, the patient recovered in the surgical intensive care unit for many days. During that interval, the patient received many procedures to internally stabilize and fixate long bone fractures and upper extremity injuries. The intra-abdominal injuries were also monitored and definitively treated. On hospital day 14, the patient returned to surgery for the final

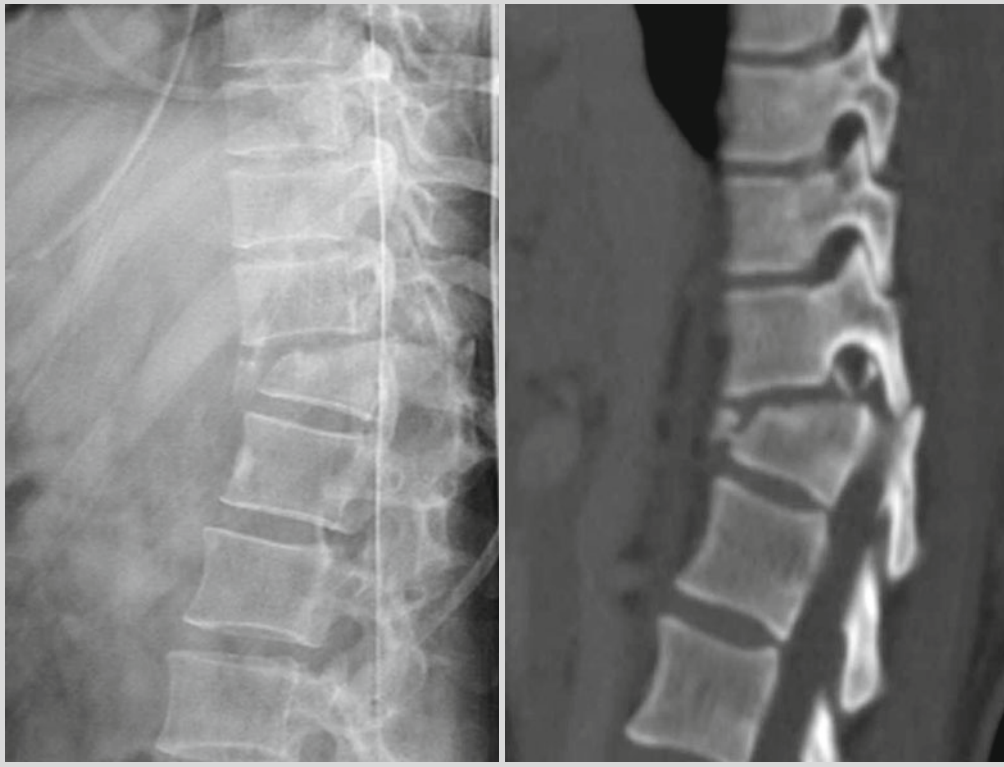


Fig. 8.17 Portable-lateral radiograph and parasagittal computed tomography (CT) images demonstrating thoracolumbar fracture-dislocation with bilateral facet fracture-dislocations and translational deformity

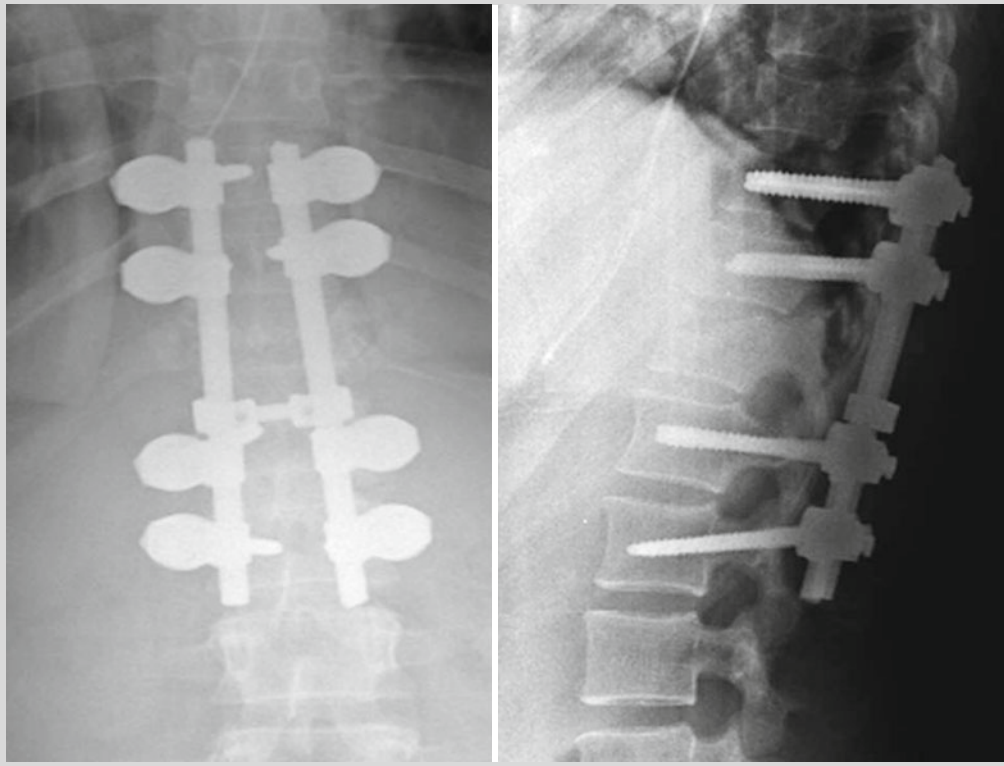


Fig. 8.18 Anteroposterior and lateral radiographs in the postoperative setting following "spine-damage-control" procedure demonstrating reduction of spinal deformity, stable fixation, and instrumented T10–L2 posterior spinal fusion with decompression of the injured levels

phase of his spinal treatment. This included an anterior approach with corpectomy, cage reconstruction, and instrumented anterior spinal fusion with screw/plate construct from T11 to L1 (Fig. 8.19). The concept of spine-damage-control allows patients immediate stability of devastating spinal injuries to enable proper ICU care without the significant morbidity of an early-total-spinal-care model. The victim is allowed to properly recover before returning to surgery for the definitive anterior procedure. Proponents of spine-damage-control point to several reasons for adding an additional anterior procedure: increased anterior column support, more thorough decompression of the spinal canal, and an increased chance of spinal fusion through the addition of anterior column and middle column bone grafting. Opponents to spine-damage-control feel that spinal injuries such as these can be adequately treated through an all-posterior approach (see Case 8.4). Nonetheless, there are situations where many surgeons are in agreement that patients need both anterior and posterior procedures for their spinal injuries. In those cases, it makes sense to provide provisional stability and decompression from a posterior approach and delay the more morbid anterior surgery until the polytrauma victim has been thoroughly stabilized.

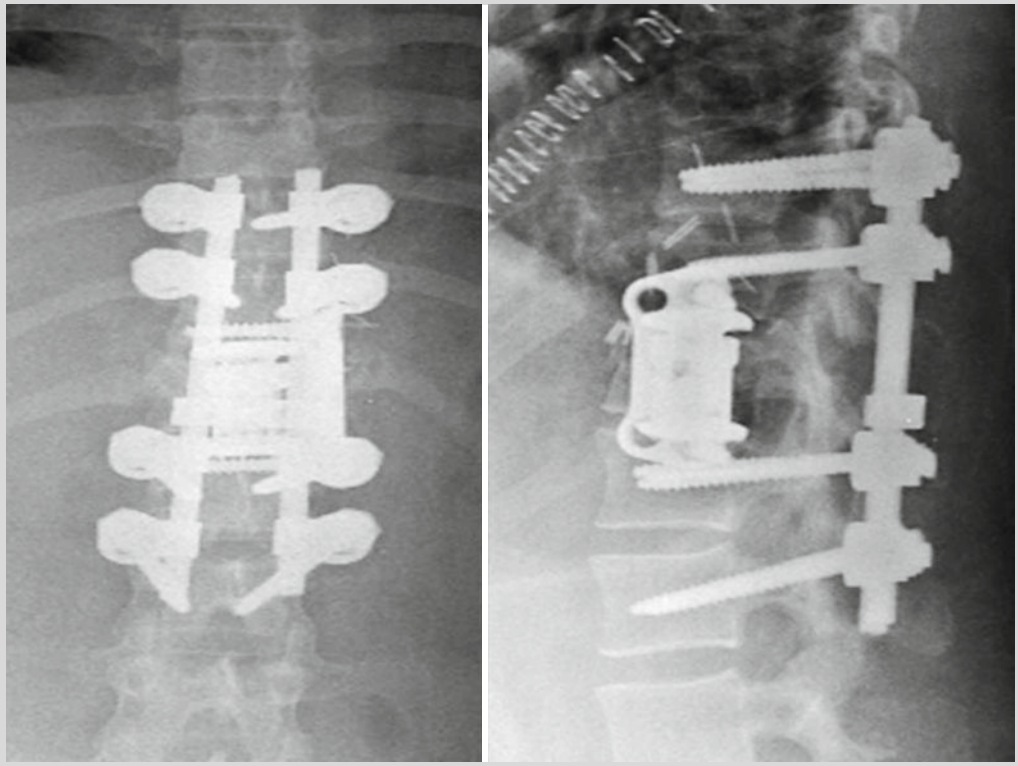


Fig. 8.19 Anteroposterior and lateral radiographs after definitive treatment of the spinal injury to include corpectomy of T12, cage reconstruction, and anterior spinal fusion with instrumentation T11–L1. This portion of the procedure enabled more thorough decompression of the spinal canal along with rigid stability over the injured segment with fixation and anterior column support

Case 8.4

A 31-year-old male was struck by a train. He sustained multiple severe injuries. A closed head injury was present. The left upper extremity was traumatically amputated at the proximal humeral shaft level. A nonoperative left ankle fracture was sustained. The patient was also noted to have dorsiflexion, plantar flexion, and toe flexion-extension deficits along with altered rectal examination. Despite those observations, the patient required emergent surgery for completion guillotine amputation of the left upper extremity and control of severe brachial arterial bleeding. His ankle was concomitantly splint immobilized. He was then transferred to the surgical intensive care unit for further resuscitation. Once adequately stabilized he received total spinal imaging. A CT scan showed a severe L4 burst fracture with spinal canal obliteration and traumatic relative kyphosis (Fig. 8.20). An MRI was then emergently obtained. This showed severe cauda equina compression that was consistent with the patient's acute cauda equina syndrome (Fig. 8.21). His neurological picture showed 0/5 strength in bilateral dorsiflexion, plantar flexion, and great toe flexion-extension. He also had diminished rectal tone and altered perianal sensation. Within 24 h of the injury, this patient was

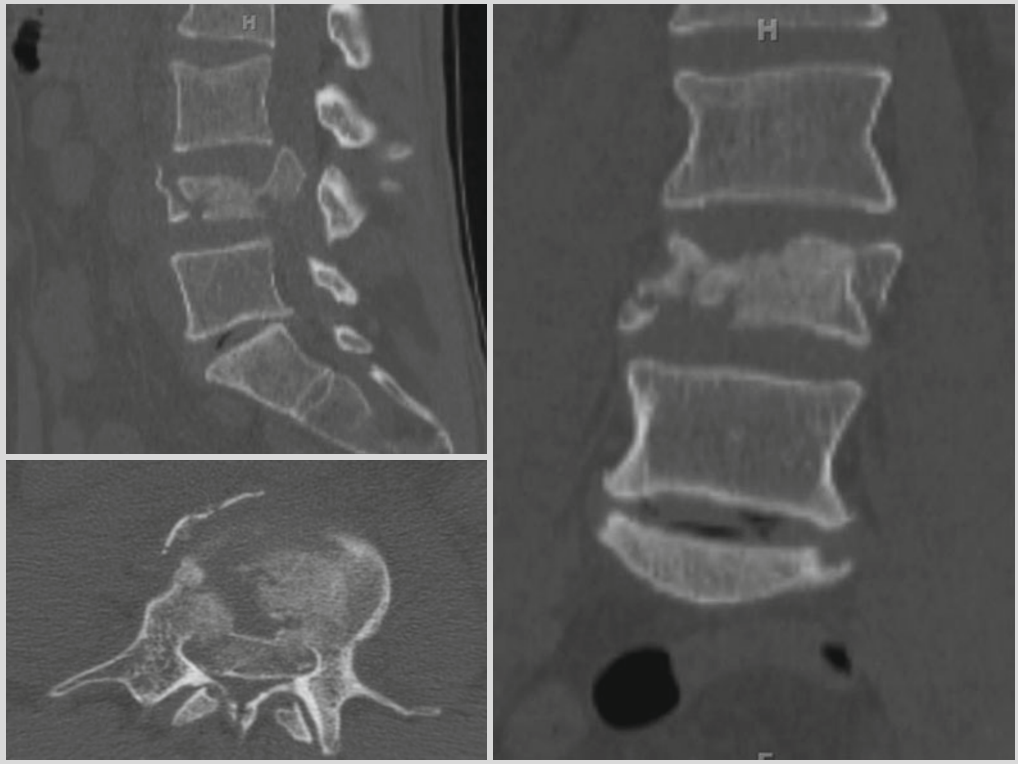


Fig. 8.20 Midsagittal, coronal, and axial computed tomography (CT) images demonstrating a severe L4 burst fracture with spinal canal compromise

taken by the spine surgeon for open reduction and internal fixation and stabilization of the L4 burst fracture utilizing an L3–5 instrumented spinal fusion construct (Fig. 8.22). Application of this construct also enabled the surgeon to fully restore the normal sagittal contour of the lumbar spine while simultaneously enabling an indirect fracture reduction and spinal canal decompression through ligamentotaxis. After those maneuvers were completed, the treating spinal surgeon proceeded to fully decompress the cauda equina over the injured segment through laminectomy and partial facetectomies. Further analysis intraoperatively showed that the burst fragment position could still be improved (Fig. 8.23). Through bilateral posterolateral approaches, a formal and direct burst fragment reduction was performed with footed tamps (Fig. 8.24). A postoperatively obtained CT scan confirmed thorough canal decompression and excellent position of the spine and the implants (Fig. 8.25). Postsurgical neurological examinations revealed marked improvement in neurological function with nearly full strength in the previously flaccid areas. After the spinal surgery, the patient returned to the operating room several times for definitive treatment of the traumatically amputated left upper extremity. In this case, the treating spinal surgeon chose to avoid a return trip to the operating room for anterior corpectomy and cage reconstruction with anterior instrumented spinal fusion. This surgeon cited solid fixation, stout stabilization from a posterior-only approach, and thorough decompression utilizing the posterior and posterolateral mediums as reasons for avoiding further

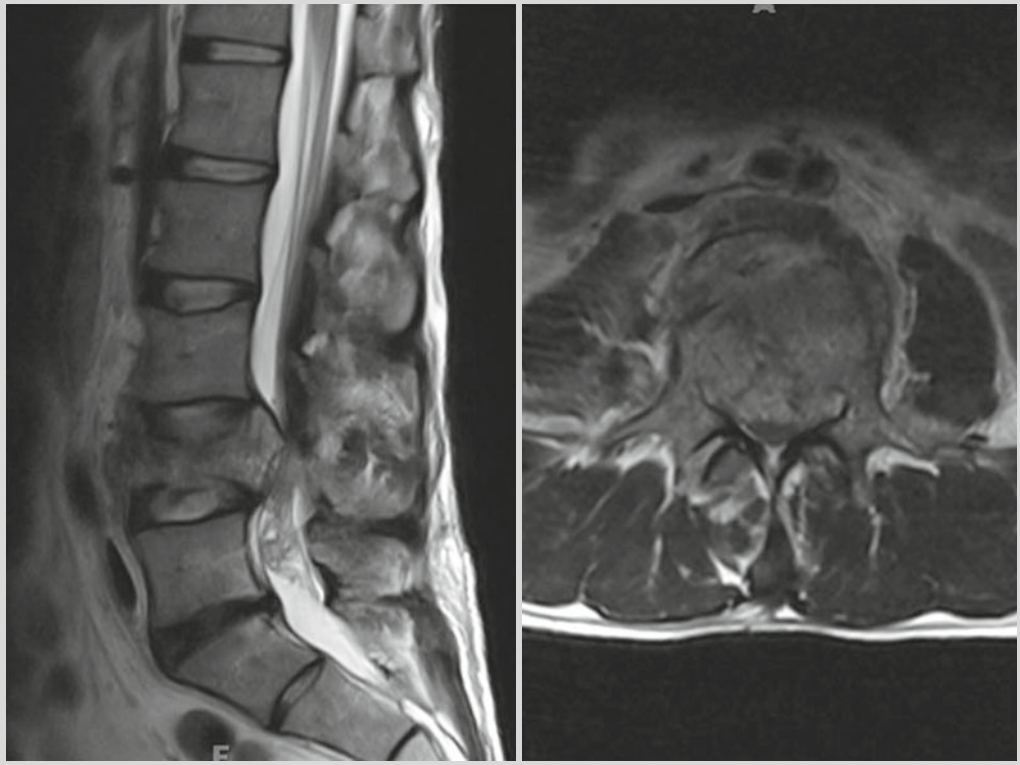


Fig. 8.21 T2-weighted MRI midsagittal and L4 axial images showing burst fracture fragment retropulsion and complete obliteration of the spinal canal. Notice the redundancy of the nerve rootlets indicating severe stenosis

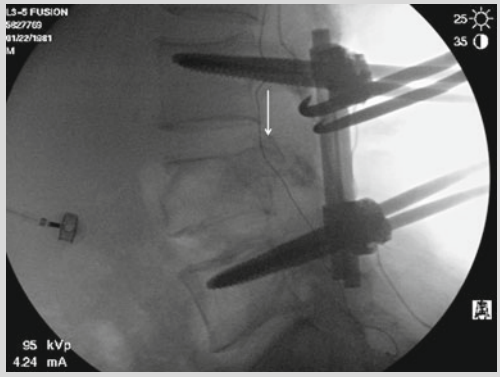


Fig. 8.22 Intraoperative fluoroscopic image demonstrating application of an L3–5 spinal fixator. After maneuvers to enable reconstitution of L4 vertebral body height, realignment of the normal lordotic posture, and partial reduction of the burst fragment through distraction and ligamentotaxis, a residual burst fragment remained (*arrow*)

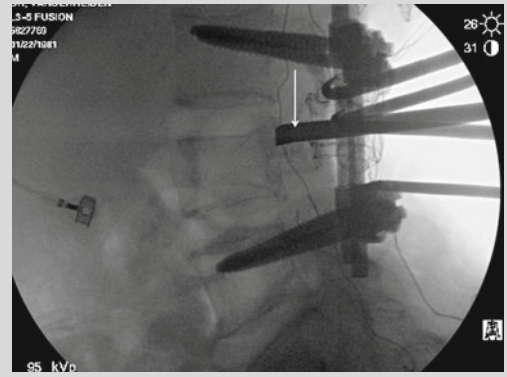


Fig. 8.23 A posterolateral approach to the burst fragment allows footed tamp application (*arrow*) to the burst fracture fragment and further reduction of the spinal canal stenosis. This was done sequentially in a bilateral fashion with gentle retraction of the cauda equina

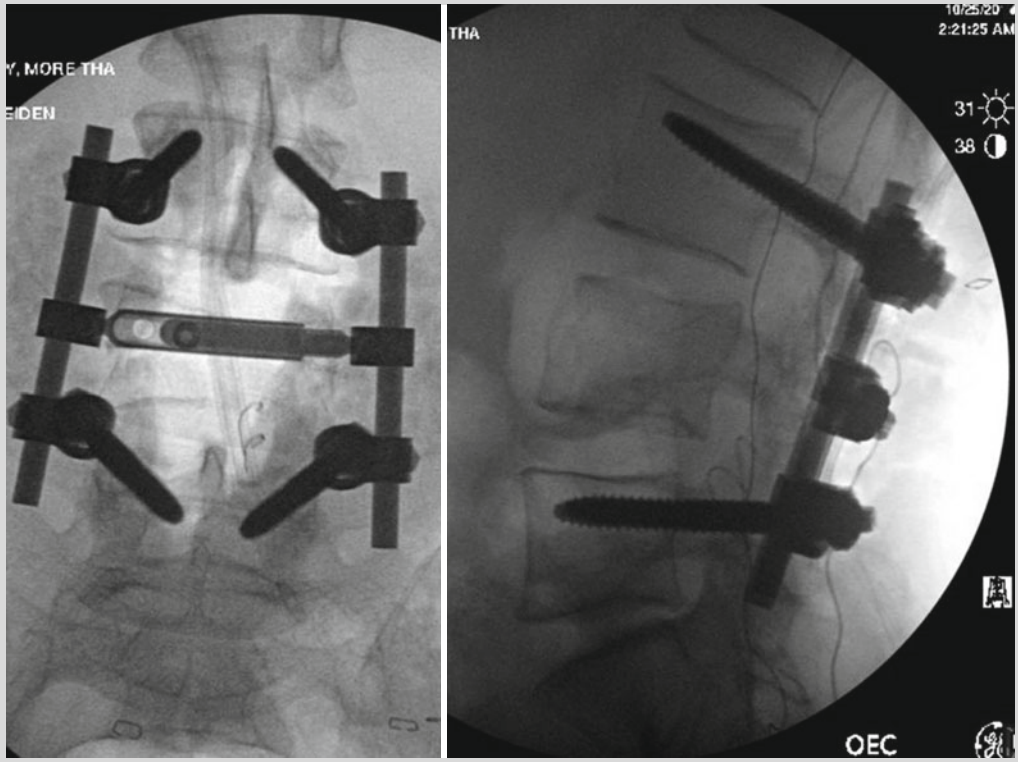


Fig. 8.24 Final intraoperative anteroposterior and lateral fluoroscopic images demonstrating reconstituted spinal alignment and near-normal configuration of the L4 corpus along with stable internal fixation and instrumented posterior spinal fusion L3–5. Note the completely reduced burst fragment

anterior surgery. The surgeon also considered the reconstituted configuration of the vertebral body and the reasonable provision of anterior column support through the well-aligned fracture fragments of the L4 vertebral bone. This surgeon essentially provided the patient with the definitive surgical solution in the setting of spine-damage-control concepts and timing. This patient will avoid the morbidity associated with the anterior procedure. However, that treatment could be easily recommended in a delayed fashion in order to complete a 360° approach to this significant spinal problem. As it occurred, the patient ambulated from the hospital in good condition 2 weeks after the spine surgery without the assistance of a gait aid and no need for catheterization or focused bowel program. With immediate spinal stability provided by the early surgery, the patient will be able to focus more closely on upper extremity rehabilitation after the loss of his arm.

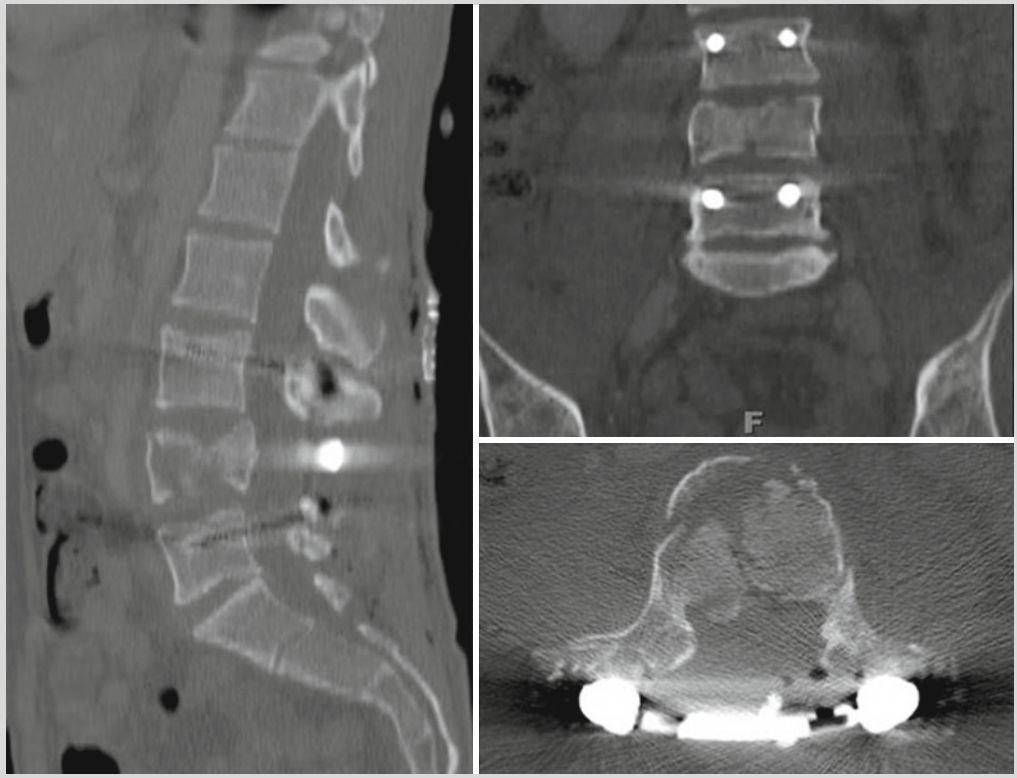


Fig. 8.25 Postoperative computed tomography (CT) scan images depicting midsagittal, coronal, and L4 pedicle-level axial pictures after treatment of L4 burst fracture associated with cauda equina syndrome

Conclusions

Polytrauma victims very frequently sustain spinal injuries that require surgical treatment to ensure early stability, early mobility, and early care of other injured bodily systems. The orthopedic traumatologist should understand the need to quickly and accurately diagnose spinal injuries so that the treating spine surgeon can become involved very early in the treatment process. The entire treatment team should also understand the goals of spinal surgery and the need to get them accomplished in an expedient fashion. Early intervention from a spinal standpoint is becoming a care standard which enables superior functional outcomes in the later stages of recovery for these severely injured patients. Furthermore, many of the feared complications associated with polytrauma and prolonged immobilization can be confidently avoided. The guiding principle to deliver these care strategies is a unified team approach between all involved members of the trauma team. The orthopedic traumatologist and the spine surgeon can prove to be invaluable team captains that lead the charge towards successful treatment of multiply injured victims.

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Open Fractures and Limb Salvage

9

Cyril Mauffrey, Kyros Ipaktchi, and James C. Beazley

Introduction

The management of severe open lower limb fractures will remain one of the greatest orthopedic challenges of our time. The energy transfer during the accident often causes injuries to other body systems such as the chest, abdomen, and/or brain with an inflammatory response that contributes to increasing patients' morbidity and mortality. A multidisciplinary approach is paramount and the concept of managing the patient as a whole following strict evidence-based algorithm (ATLS) crucial. The general management of multiply injured patient is beyond the scope of this chapter, and our focus will lie on the severe open lower limb injury in a stabilized patient. The dilemma between salvage and amputation of the injured limb remains a controversy. Recently, the Lower Extremity Assessment Project study looked at both options, but no significant difference in return to work, functional outcomes, or even cost of treatment (including the prosthesis) was identified. A few decades ago, clinical scores flourished in an attempt to help in the decision process of amputation versus salvage but none were very reliable. However, there is a consensus that the key to limb viability seems to be the severity of the soft tissue injury [1]. Factors such as associated injuries, patient age, and comorbidities (such as diabetes) also should be considered. Our chapter will focus on the management of open fractures and limb salvage options. The first section will be dedicated to the acute management with an emphasis on antibiotic prophylaxis, timing and technique of the initial debridement, current evidence behind local antibiotics treatment, vacuum-assisted therapy, and options for immediate fracture stabilization. Our second section is dedicated to soft tissue management and reconstruction. Finally, we address an area where good evidence is lacking and present an algorithm highlighting our preference for the management of segmental bone loss.

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Acute Management of Open Fractures

Patient Assessment

Which Centers Should Manage Severe Lower Limb Fractures?

The multifaceted aspects of the management of complex lower limb fractures necessitate that

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such fractures be managed by centers with orthopedic and plastic surgeons both regularly exposed to lower limb trauma. Hospitals that are not experienced in dealing with such injuries should have specific arrangements with their local trauma centers for urgent referral and management of these patients, and even the initial debridement and stabilization of the injury should ideally take place in the trauma center. When determining if a patient should be referred to a trauma center, clinical and radiological features pertinent to the injured limb should guide the decision process, as they may imply a large energy transfer to both bones and the soft tissues surrounding them. For the tibia, these include [2]:

- A tibial fracture with an open wound that is not primarily closeable
- Presence of a limb threatening vascular injury
- Obvious muscle necrosis at the time of the initial debridement
- Bone loss either at the time of the injury or following the initial debridement

Centers managing severe lower limb injuries should have dedicated OR time during which microvascular and orthopedic surgeons can collaborate and establish a treatment plan for bony stabilization and soft tissue management. Presence and early involvement of the infectious disease team is a plus [2].

Management in the Emergency Department

When seeing the patient in the emergency department, physicians and other healthcare providers should follow the advanced trauma life support (ATLS) guidelines established by the American College of Surgeons and its Committee on Trauma. The airways, cervical spine, breathing, and circulation are assessed and the patient stabilized. The detailed management of unstable patients extends beyond the scope of this chapter. When assessing the lower extremity injury, the clinician should have in mind the potential for a vascular injury and the initial examination should be used to exclude such an injury. Vascular injuries have been classified based on clinical features at presentation and may include hard and soft signs [3]. The former include:

- Bruit over an injured artery
- Pulsatile bleeding
- Signs of distal ischemia such as pale lower extremity
- Visible expanding hematoma

Soft signs include the following:

- Large hemorrhage found on history taking
- A decreased pulse compared to the contralateral side
- Any neurologic abnormality

These clinical findings should always be correlated with measurements of ankle-brachial index [3]. Physical examination of the injured patient should guide the treatment algorithm and the necessity to perform more imaging with arteriography or a Doppler. If a vascular injury is identified, the microvascular team should be involved to participate in the decision process of acute amputation versus surgical repair. If the limb is not under immediate threat, then radiographs and/or photographs of the injury should be taken and the wounds dressed appropriately using a sterile soaked gauze; the patient's tetanus immunization status should be checked and, if necessary, a tetanus vaccine given. Intravenous antibiotics should be given as early as possible (see section on antibiotics prophylaxis).

Antibiotic Prophylaxis

Along with the timing of the first debridement, antibiotic prophylaxis is an aspect of management where the gold standard is still unclear. Controversy surrounds both the type of antibiotics to be given to prevent contamination of the tissues and/or bone and the ideal duration of prophylaxis.

Antibiotic Efficacy

The use of antibiotic therapy to prevent infection in open fracture is now well established. A thorough debridement is essential, but without intravenous antibiotic prophylaxis, the infection rate can be as high as 25 % [4]. Trials comparing antibiotics to placebo are published and have shown that a first-generation cephalosporin was more effective than both, a placebo, or a combination of

penicillin with streptomycin [5, 6]. The Cochrane group performed a meta-analysis looking at this specific question identifying a significant reduction in infection rate when antibiotics were used (5.5 %) versus no antibiotics (13.4 %) [7].

Organisms Involved

The value of cultures in the emergency department or at the time of debridement has been questioned and has a low predictive value in case of infection. Lee reviewed the cultures of 245 open fractures. Only 8 % of bacteria identified at the pre-debridement stage later caused infection, while 7 % of open fractures with negative cultures during the pre-debridement phase eventually developed infection [8]. Similar conclusions were reached by Valenziano et al. showing a 6 % wound infection rate with nearly three quarters of them having negative bacterial growth at the pre-debridement stages [9].

These microbiological studies allowed us to understand the nature of contaminant at the time of an open fracture and later during the debridement. Robinson et al. identified nearly 100 types of organisms in as many cases of open fractures. In decreasing order of prevalence, aerobic Gram-negative rods grew in 40 %, followed by *Staphylococcus epidermidis* (34 %) and *Staphylococcus aureus* (26 %) during the pre-debridement stages. Cultures taken a day later in the post-debridement stages grew either no organisms (59.5 %) or saprophytic organisms that were nonpathogenic [10]. Patients that grew the same organism in the pre- and post-debridement stages ended up developing an infection. An interesting finding from a double-blind randomized trial is that when Gram-negative coverage only is given to patients with an open fracture, a prevalence of infection with Gram-positive bacteria will be seen should patients develop an infection, while patients that are given prophylaxis against Gram-positive bacteria will tend to grow Gram-negative bacteria should they develop an infection [11]. The growing concerns of infection with MRSA are well founded. Indeed, *Staphylococcus aureus* stands out as the number one cause of surgical site infection [5, 6, 11], methicillin-resistant *Staphylococcus aureus* (MRSA) accounting for nearly a third of the total staphylococcal infections [11]. Given the overzealous use of antibiotic in the general population over the last decades, we are now faced with a new concern, one that was probably not highlighted in clinical trials now more than a decade old: the evolving epidemiology and susceptibility of the colonizing organisms in general and for open fractures in particular.

Antibiotic Selection

Based on the data summarized above, coverage for *Staphylococcus aureus* is crucial. On the other hand, according to the study by Carsenti-Etesse et al., the risk of developing an infection from Gram-negative organisms is elevated if the antibiotic is solely aimed at preventing Gram-positive infection [11]. The last decade has also witnessed an increase in Gram-negative infections, attributed to extensive use of cephalosporin antibiotics and to the increase in hospital-acquired infections. This statement was verified by Patzakis et al. who compared the use of ciprofloxacin alone to a combination of cefamandole and gentamicin. The difference in infection rates between the two groups in type I or II open fractures was not significant. However, a significantly lower infection rate was identified in the group treated with Gram-negative prophylaxis (7.7 % versus 31 %) for type III fractures [12]. The recently published East guidelines also emphasize the importance of Gram-negative coverage for type III fractures [13].

Gustilo and Anderson classified open factures after the first wound debridement. There is a tendency to underestimate the severity and type of an open fracture until the time of operative debridement. In addition, antibiotics should be given in the emergency department as soon as the patient arrives and ideally within 3 h of the injury. Hence, the assumption that the patient who arrives with a wound measuring less than 1 cm in the anteromedial aspect of his tibia should be treated (in terms of antibiotic prophylaxis) as a type I fracture can have serious consequences. The authors believe that the antibiotic prophylaxis protocol should not be based on the initial size of the open wound as it presents to the emergency department, but rather

the protocol should be chronologically based, following a clear algorithm with a prophylaxis that is tailored to the surgical management of the open wound, the fracture, associated bone, and vascular/muscle injury. Cefazolin, a first-generation cephalosporin, covers "sensitive" Staphylococcus, Streptococcus, and some of the "friendly"—i.e., more common—Gram-negatives (such as *E. coli*). It does not cover anaerobes, MRSA, Pseudomonas, or Acinetobacter. Vancomycin is a glycopeptide that covers Gram-positives (including MRSA) and some anaerobes (like Clostridium) but has no Gram-negative coverage. A lincosamide, clindamycin covers Staphylococcus (including some MRSA) and Streptococcus and most anaerobes, though it also has no Gram-negative coverage. Gentamicin, an aminoglycoside that covers Gram-negatives, including most Pseudomonas and Acinetobacter has no Gram-positive coverage (by itself) or anaerobic coverage. The authors propose systematic coverage against Gram-positive bacteria [13] using early IV cefazolin, supplemented with IV vancomycin if there is a previous history of MRSA infection. (If there is gross contamination by farmyard waste or sewage, cefazolin is replaced by clindamycin.) One dose of gentamicin is given at the time of the initial debridement to cover for possible Gram-negatives if the incision and debridement reveal bone loss, skin flaps that will not close or muscle necrosis. Fluoroquinolones offer no advantages compared with a combination of a cephalosporin and gentamicin; furthermore, fluoroquinolones may actually have a detrimental effect of fracture healing and increase infection rate in type III fractures [13].

Duration of Antibiotic Therapy

Most clinicians would agree that patients should be started on IV antibiotics within 3 h of the injury, and this has indeed been shown to reduce the rate of infection from 7.4 to 4.7 % when compared to prophylaxis started more than 3 h after the injury [12]. One question still debated, however, is the total duration of antibiotic prophylaxis for open fractures. Dellinger et al. performed a randomized trial in an attempt to answer this question; this trial, albeit more than 20 years old, showed that a 24-h course of antibiotics (cefonicid sodium) was as good as a prolonged course (5 days of cefonicid or 5 days of cefamandole) in preventing infection in open fractures. The infection rates were, respectively, 12, 11.8, and 13.1 % [14].

Proposed Guidelines for Antibiotics

Antibiotics should ideally be administered within 3 h of the injury. The antibiotic of choice is cefazolin (1 g IV q8h for patients <80 kg or 2 g IV q8h for patients ≥80 kg), with this regime continued for 24 h after the first debridement (excision). If the patient has a history of MRSA, 1 g of vancomycin IV should be added. For grossly contaminated wounds or farmyard injuries, the antibiotic of choice is clindamycin 600 mg IV q8h for a duration of 24 h after the initial debridement. Intraoperative assessment of the injury during the first debridement will determine the following: If the wound edges are not closeable or if there is muscle necrosis or bone loss, a single dose of gentamicin 5 mg/kg IV should be added at the time of the initial debridement. Furthermore, one should consider application of local antibiotics (antibiotic bead pouch) or a negative pressure dressing.

At the time of definitive skeletal stabilization and skin closure, patients should be given one dose of cefazolin 1 g IV < 80 kg or 2 g IV ≥ 80 kg (plus vancomycin 1 g IV if a previous history of MRSA exists). Patients with anaphylaxis to penicillin should receive clindamycin (600 mg IV pre-op/qds) in place of a cephalosporin (Fig. 9.1).

Initial Debridement

Timing

In 2009, the guidelines on the management of open tibial fractures from the British Orthopaedic Association (BOA) and the British Association of Plastic, Reconstructive and Aesthetic Surgeons (BAPRAS) were modified to change the recommended timing of debridement from within 6 h of injury to within 24 h of injury [15]. In fact, the historical cutoff of 6 h was based on scarce evidence.

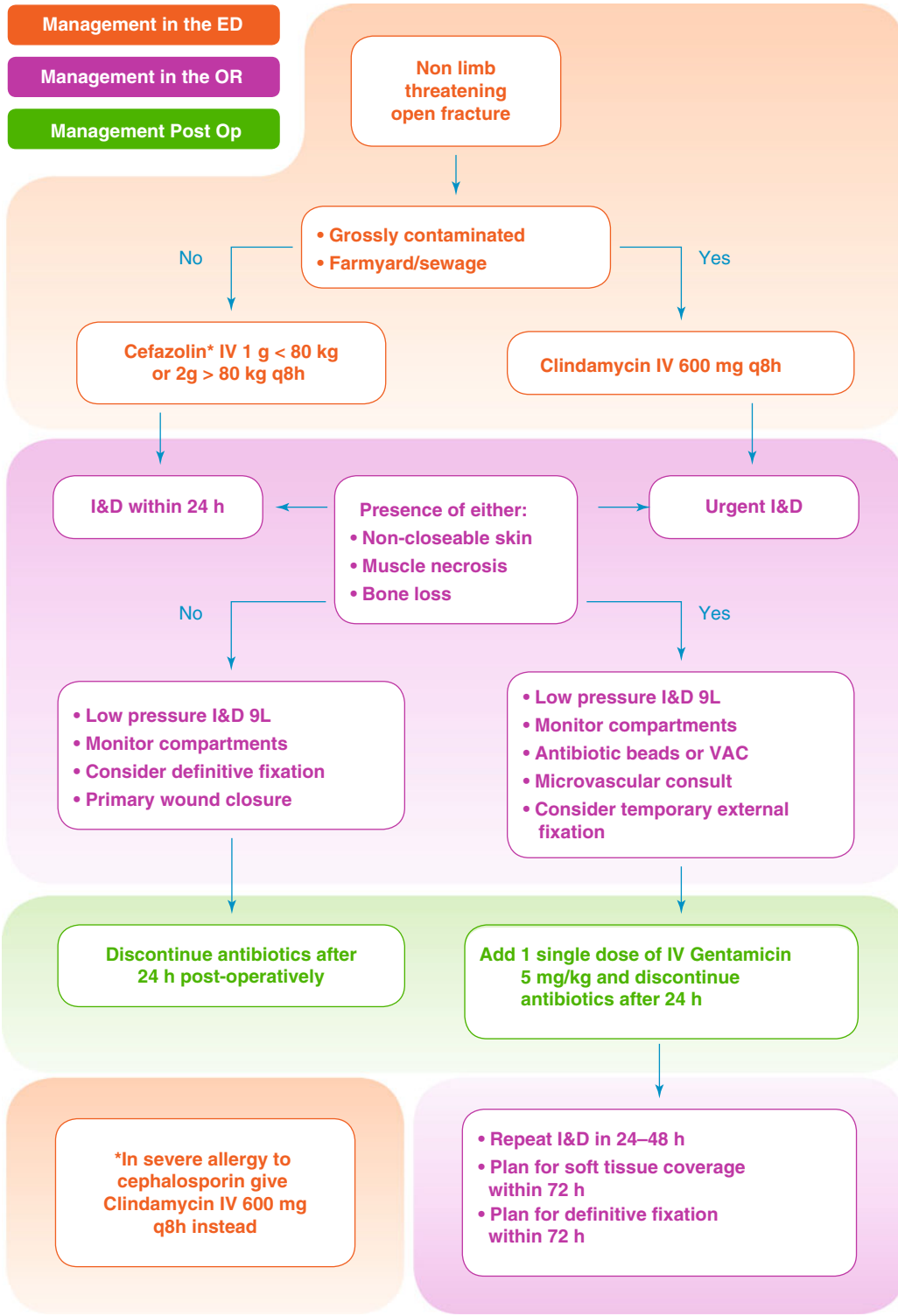


Fig. 9.1 Algorithm for the acute management of open fractures (Used with permission from Slack Orthopedics: Mauffrey et al. [116])

More recently, new clinical evidence has arisen supporting the change in practice from emergent debridement to debridement within 24 h. Patzakis and Wilkins looked at the relation between infection rate and timing of debridement. Seven percent was the rate of infection in both groups treated within 12 h of injury or more than 12 h after their open fracture [16]. Other authors confirmed the lack of correlation between urgent debridement and reduced infection rates both in humans [17–19] and in animal models [20].

It appears that the most important aspect of management of open fractures in reducing the risk of infection is by the early initiation of prophylactic intravenous antibiotics. The timing of debridement does not appear to relate to the development of deep infection if performed within 24 h.

Surgical Technique

In 2010, the British Orthopaedic Association (BOA) and the British Association of Plastic, Reconstructive and Aesthetic Surgeons (BAPRAS) working party on the management of open tibial fractures [15] agreed on a protocol that is now followed nationwide across the United Kingdom. This includes antibiotic prophylaxis but also a well-defined step-by-step approach for initial wound debridement that includes:

- Removal of all dead tissue.
- Systematic debridement in the following sequence:
  - Application of a pre-prep with a soapy solution.
  - Preparation of the limb with a chlorhexidine alcohol solution, avoiding direct contact of the chlorhexidine with the open wound.
  - Wound extensions are done ideally following potential fasciotomy incisions.
  - Systematic assessment of the tissues, from superficial to deep and from the periphery to the center of the wound.
- In order to ensure bone viability during the debridement process, deflation of the tourniquet should be performed to assess bleeding of the bony segment. At this stage, nonviable fragments or loose fragments of bone with no attachment to soft tissue should be discarded.
- Once the debridement has been performed, thorough irrigation can be achieved.

It is only following this radical and systematic approach that the injury can be classified and a multidisciplinary treatment plan be elaborated by the orthopedic and microvascular surgery teams.

Acute Wound Management

Delayed vs. Primary Wound Closure

When closing the tissue defect created by an open fracture, the surgeon much choose whether to close it soon after the injury or wait and close it later as a means to control potential infection. Primary closure, defined as approximation of wound edges immediately following debridement or cleaning within 6 h of injury, has the benefit of rapid wound healing but presents the threat of increased wound infection. Delayed closure, defined as approximation of wound edges more than 48 h after debridement or cleaning, is often used for wounds judged to be "dirty" or contaminated—i.e., with a risk of infection perceived to be higher due to the environment and circumstances surrounding the injury. However, no good evidence exists to guide this decision; as of July 2011, a review of the Cochrane Database by Eliya and Banda indicated there were no randomized controlled trials comparing primary versus delayed wound closure [21].

Negative Pressure Wound Therapy

Negative pressure wound therapy, in which vacuum suction is applied across an airtight topical dressing, has been used in the treatment of chronic and surgical wounds. The negative pressure is thought to aid the drainage of excess fluid, reduce infection rates, and increase localized blood flow. It is also known as topical negative pressure (TNP) therapy, vacuum-assisted closure (VAC), and sealed surface wound suction (Fig. 9.2). A systematic review in 2008 by Ubbink et al. found only a small number of flawed trials and thus little evidence to support the use of negative pressure wound therapy in the treatment of wounds [22].

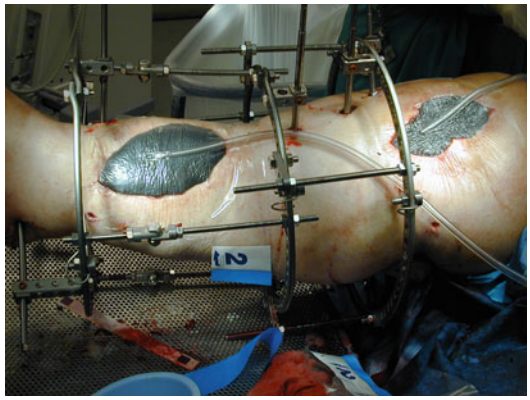


Fig. 9.2 Application of vacuum-assisted closure (VAC) dressing

In Situ Antibiotics

The use of antibiotic beads is advocated by Ostermann and Seligson in a paper reviewing 1,085 consecutive cases of severe open fractures [23]. The first group was managed solely with systemic antibiotics and debridement at the time of presentation, while the second group was supplemented with local aminoglycoside-polymethylmethacrylate (PMMA) beads. The difference between the overall infection rates in both groups was shown to be statistically significant ($p<0.001$), with a 12 % infection rate in patients managed with IV antibiotics and debridement only versus a 3.7 % infection rate in the group managed with additional placement of antibiotic beads. The authors also analyzed fracture subtypes and concluded that while both acute infection and osteomyelitis were reduced in the second group for all fractures types overall, the reduction reached the level of statistical significance only for type IIIB and IIIC fractures for acute infection and type II and IIIB fractures for chronic osteomyelitis (Fig. 9.3).

Other papers have shown similar results. Henry et al. treated 404 open fractures, with similar distributions of fracture types. Around two thirds of patients were treated with IV antibiotics and tobramycin antibiotic bead chains, while the remaining third was treated with IV antibiotics only. The group supplemented with antibiotic beads had a statistically significant lower rate of infection (4.2 %) than the group that did not receive beads (21.4 %) [24, 25].

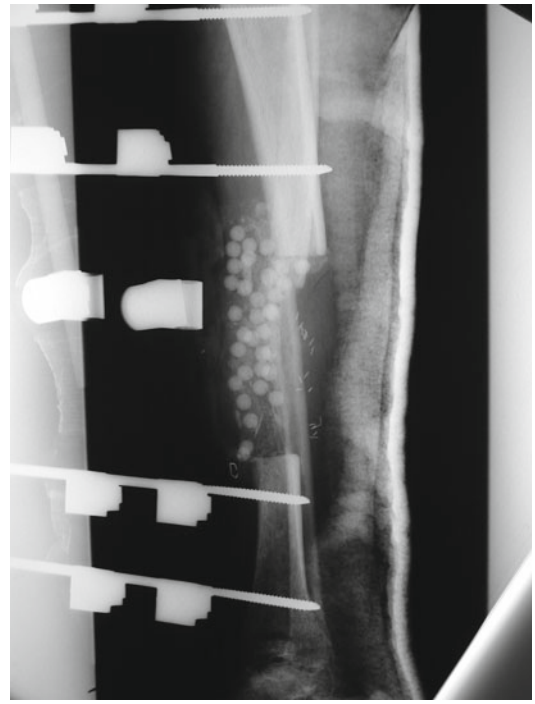


Fig. 9.3 Lateral radiograph of a tibia with segmental bone defect and antibiotic beads placement

Fracture Stabilization in the Acute Setting

The surgeon has several options to stabilize an open fracture, including techniques commonly employed during an immediate fracture presentation such as splinting, casting, and traction, and other operative interventions like external fixation, plating, and intramedullary nailing. The choice of methods is dependent upon a number of factors, including the bones involved, the location, and qualities of the fracture, and several studies have attempted to address this question specifically, as Okike and Bhattacharyya summarize in their 2006 article [26].

Fractures of the Femur

Beginning with a report by Winquist et al. in 1984, early intramedullary nailing has been shown to be effective in the treatment of femoral shaft fractures [27]. Okike and Bhattacharyya identified several articles studying early intramedullary nailing with reaming for open femoral

shaft fractures. A prospective, randomized, controlled trial by Bone et al. in 1989 compared early (within 24 h) and late (after 48 h) stabilization of 178 femoral fractures, which demonstrated no differences for patients with isolated injuries but decreased complications for poly-trauma patients. Specifically, these patients, when undergoing stabilization within 24 h, were shown to have a decreased rate of pulmonary complications, shorter hospital stays, and less time in the ICU [28]. In 1988, Lhowe and Hansen concluded that immediate intramedullary nailing could be accomplished safely with an acceptable rate of complications after their series of 67 patients with open femoral shaft fractures. Only 2 of 67 patients suffered a wound infection, and all fractures healed within 4 months of injury [29]. Brumback et al. published a series of 89 open femoral fractures stabilized by intramedullary nailing with reaming and reported no infections among 62 type I, II, and IIIA fractures. Of the 27 type IIIB fractures, only 3 became infected, and the rate of infection did not differ for patients treated early (within 24 h) or late (after 48 h) [30]. More recently, 2009 study by Taitsman et al. identified open fractures as a risk factor for nonunion for fractures treated by intramedullary nailing [31], but the preponderance of evidence still supports early reamed intramedullary nailing as the preferred method for treatment of open femoral shaft fractures.

Fractures of the Tibia

The current literature favors intramedullary nailing for fixation of open tibia fractures. In their 2006 paper, Okike and Bhattacharyya chronicle the evolution of opinion regarding treatment for open tibia fractures. They list two articles written in the 1980s that addressed external fixation in tibia fractures: Edwards et al. concluded that external fixation was successful for treatment of severe open tibia fractures [32], and Bach and Hansen [33] reported fewer complications with external fixation than with internal plate fixation. The 1990s saw a shift away from external fixation toward intramedullary nailing, and Okike and Bhattacharyya point out several studies making this argument. Henley et al. found fewer incidences of malalignment (8 % vs. 31 %, $p<0.001$), fewer subsequent procedures (mean 1.7 vs. 2.7, $p=0.001$), and lower rate of infection (13 % vs. 21 %, not significant) comparing 104 patients treated with unreamed intramedullary nailing to 70 patients treated with external fixation [34]. Schandelmaier et al. reported a retrospective study of 114 patients with fresh tibial shaft fractures with severe soft tissue injury; 48 were treated with unreamed tibia nails and 66 were treated with external fixators. The unreamed tibia nail group underwent fewer subsequent procedures and achieved better functional outcomes, though the authors articulate that this result may not hold true for patients with less severe injuries [35]. Similarly, in 1994 Tornetta et al. compared intramedullary nailing with external fixation in a randomized, controlled trial of 29 patients with severe (grade IIIB) open tibia fractures. Though they detected no significant differences in healing or range of motion, they considered intramedullary nailing superior due to patient preference and easier fracture management [36]. More recently, the usefulness of external fixation for "damage control orthopedics" has been described by Lebel et al. following their experiences in the aftermath of the 2010 Haiti earthquake [37]. Furthermore, a review by Bhandari et al. has shown that intramedullary nailing after external fixation achieves good union and infection rates and may be superior to casting for definitive treatment, as casting has similar rates of infection but significantly higher rates of nonunion. Removal of the external fixator within 28 days may reduce the risk of infection by 83 % [38].

There has been some controversy as to whether intramedullary nails should be inserted reamed or unreamed, and several smaller trials were inconclusive [39–41]. A meta-analysis by Bhandari et al. in 2001 presented strong evidence in favor of intramedullary nailing over external fixation but failed to find any definitive evidence for reamed versus unreamed nailing, suggesting a much larger study was necessary to analyze the question [42].

Accordingly, the SPRINT trial, a multicenter, blinded randomized trial completed in 2007 and reported in 2008, studies reamed versus unreamed

nails in both closed and open tibia fractures on a much larger scale. One of the largest orthopedic studies ever conducted, over 1,226 patients with 1,248 fractures of the tibial shaft were included. Of these, 400 patients with 406 open fractures (108 [26.6 %] Gustilo type I, 161 [39.7 %] Gustilo type II, 107 [26.4 %] Gustilo type IIIA, 30 [7.4 %] Gustilo type IIIB) were treated with reamed (210 total) and unreamed (196 total) intramedullary nails. As its primary outcome, the study evaluated reoperation and/or autodynamization within 1 year. A total of 106 patients experienced a primary event (reoperation)—60 from the reamed group and 46 from the unreamed group. There was no statistically significant difference between these two groups ($p = 0.16$), and the study concludes that the optimal nailing technique for open fractures remains uncertain [43].

Soft Tissue Reconstruction

Extremity soft tissue defects continue to be a challenging problem for orthopedic trauma surgeons and complicate the management and salvage of severe extremity injuries. In the last decades, an increase of civilian high-energy trauma and significant battlefield trauma together with improved trauma survival has increased the incidence of complex extremity wounds, necessitating soft tissue reconstruction. Open fractures are associated with higher rates of infection, malunion, nonunion, as well as limb loss [44]. The individuality of each injury and patient characteristics call for custom solutions. Limb salvage has to be seen in context with the injury profile of the patient and expected functional demands. Traumatologists need to closely coordinate damage control surgery, definite fixation, and soft tissue reconstruction in order to achieve optimal outcome. This calls for a close interdisciplinary cooperation between orthopedic trauma specialist and reconstructive plastic surgeons or special, orthoplastic trained surgeons. This section will give an overview of current algorithms and options for soft tissue reconstruction in the lower extremity. The continuous development and refinement in flap reconstructions and increased utilization of adjuvant techniques such as vacuum-assisted closure have changed the reconstructive practice and will be highlighted.

General Principles

Epidemiology

The majority of extremity soft tissue defects is seen in the lower extremity and overwhelmingly related to trauma. Most commonly these injuries occur after motor vehicle accidents followed by penetrating injuries, blast trauma, and burn injuries. Soft tissue loss due to management of necrotizing infections and tumor resections often necessitate soft tissue reconstruction. The focus of this review will be on traumatic injuries.

Indication for Soft Tissue Reconstruction

The indication and decision for soft tissue reconstruction arise after formal debridement of the traumatic wound at which point a decision regarding extent, depth, and involvement of vital structures can be made. Many mangling limb injuries in a multi-injured patient preclude salvage based on ATLS principles of life before limb. The indication for reconstruction can only be seen in a stabilized patient.

Most commonly, open fractures and associated soft tissue injuries are classified according to the Gustilo and Anderson classification (Table 9.1) [45].

Table 9.1 Gustilo and Anderson Classification for open fractures

Type	Definition
I	Open fracture, clean wound, wound <1 cm in length
II	Open fracture, wound >1 cm in length, limited soft tissue damage
III	Higher-energy open fractures with extensive soft tissue injury
IIIA	Type II, adequate periosteal coverage of the bone, primary closure possible
IIIB	Type II, extensive soft tissue loss necessitating soft tissue management including flaps
IIIC	Type III, arterial injury requiring emergent repair, irrespective of soft tissue injury severity

The indication for soft tissue reconstruction in the lower extremity is usually seen in IIIB type injuries with extensive soft tissue defects, which cannot be closed without soft tissue management or flaps and in the majority of IIIC injuries, which are soft tissue injuries with an arterial injury necessitating vascular repair for perfusion of the foot. In addition to this classification concept, the type of defect, location, and exposed structures dictate the scale and complexity of reconstruction. Generally speaking: Exposed neurovascular bundles, tendons, bone, and joints mandate in most instances flap coverage. This said, full thickness skin loss in functionally important regions such as the popliteal and antecubital fossa and the wrist/hand benefit from early, higher complexity reconstructions, such as a flap surgery instead of skin grafting, to provide for full thickness coverage of vital structures and allow better functional rehabilitation, prevention of contractures, and disability.

Indication for Primary Amputation

The question of amputation versus salvage has been extensively addressed by the LEAP study consortium, which showed similar functional outcomes over time in regard to patients with mangled lower extremity injuries treated with either amputation or salvage [46]. The group found significantly higher lifetime costs associated with amputations. There was no significant difference in the two groups with regard to return to work or patient satisfaction scores. The research highlighted the significant impact soft tissue injuries had on the decision for or against salvage. The evidence gained from this study showed a poor predictive value and clinical utility of existing extremity injury scores in the decision of limb salvage versus amputation. It was found that mangled extremities with initially insensate feet regained a protective level of sensation and were of benefit to patients. Thus the denervated foot cannot be seen as an indication for amputation [47].

Clear indications to amputate are currently seen:

- Whenever there is an imminent risk of death. For the lower extremity injured patient, uncontrollable hemorrhage is the foremost indication to amputate. This is seen in mangled and blast-type injuries inflicted by IED and mines.
- In limb ischemia times greater than 6 h, by which time myolysis and tissue necrosis has set in. Attempts of salvage could expose the patient to ischemia reperfusion injury [48].
- Segmental limb loss in the lower extremity of greater than one-third of length.
- Incomplete amputations with a mangled distal part.

Timing

Soft tissue reconstruction has to follow resuscitation and stabilization of the patient. In the case of a monotrauma to a limb, early or immediate reconstruction is feasible and had been initially propagated by Godina, who described favorable results with one stage—primary free flap reconstructions for complex injuries [49]. This has to be differentiated from emergent flap reconstruction in IIIC injuries where a combined vascular and flap reconstruction is performed to revascularize a limb. Some authors have termed all flap surgeries performed within 24 h after trauma as "emergency" flaps; this practice dilutes the true meaning of the term—emergency flap [50]. In a new classification, Ninkovic proposes calling flaps performed within 12–24 h after initial debridement "primary flaps." Flaps done between 2 and 7 days after debridement are termed "delayed primary flaps," and all other flaps performed after 7 days are "secondary flaps" [51].

The proponents of primary reconstruction demonstrated improved outcomes and less flap failures as opposed to the outcomes after secondary reconstruction. Inflammatory changes to the wound bed leads to more friable vessels and induration of periadventitial fat. This together with bacterial wound colonization or frank infection can all contribute to the adverse outcomes seen in late reconstructions. Most authors favor a delayed primary reconstruction within the first 72 h to 5 days after trauma [52]. With this approach, one can address and condition wound contamination, establish definite fixation, and optimize availability of best-qualified surgical resources.

History and Classification of Flaps

The term "flap" derived from the Dutch word "flappe"—designating a loose piece of tissue hanging on to only one side. Flaps have been reportedly used well before Christ for nasal reconstructions in India [53]. Major advances in the field have been associated with our increased anatomic knowledge of vascular supply to bone, musculature, and skin. One can generally categorize flaps based on blood supply, the tissue entity of which the flap is composed of, such as musculocutaneous or fasciocutaneous flaps, and finally the shape or location of the donor tissue—as, for example, in local V-Y advancement flap of the fingertip.

Early flaps were elevated on a random pattern of dermal blood supply and were limited in their arc of rotation and tissue advancement. The development of tubed and pedicled flaps and knowledge of revascularization of transferred tissue allowed for sequential, distal originating tissue transfers—such as groin to forearm to face transfers for reconstruction of facial defects as practiced during World War I. Detailed anatomic studies and definition of axial blood supply allowed the harvest of longer skin flaps based on a dominant vascular pedicle (e.g., groin flap). Studies by Nahai and Mathes differentiated muscle perfusion into five subtypes. This knowledge allowed for the controlled transfer of muscles based on defined pedicles. These findings significantly enhanced the development of free-tissue transfer based on reliably encountered vascular pattern pedicles [54].

While in the past the primary aim of flap coverage was defect closure and prevention of infection, in today's practice free flap reconstruction has advanced to the point of not just covering wounds but individually addressing functional deficiencies of composite defects by restoring bony support [55] and musculotendinous function [56], including innervation and tissue coverage as in composite or chimeric flaps, which include several tissue types pedicled on a common blood vessel [57]. Increasingly restoration of function and form—meaning the aesthetic result—is seen as the ultimate goal in reconstructive surgery. Patients pleased with the aesthetic result were found to have much higher level of functionality and improved quality of life [58]. Newer developments are seen in "cut as you go" refined distal perforator flaps, prefabricated and pre-expanded flaps, as well as supermicrosurgery flaps in the below than 1 μm level [59, 60].

Fig. 9.4 The reconstructive ladder describing reconstructive options for soft tissue defect management. Climbing up the rungs, both technical complexity and risk of morbidity increases

Reconstructive Ladder

Wound closure can be achieved by various techniques ranging from wet to dry dressing changes stimulating granulation and secondary healing as the most basic form of achieving healing all the way to complex free flaps. Most authors define a ladder of reconstruction, which increases in complexity and skills needed as you go up the rungs and potentially also leading to an increase in patient morbidity [61] (Fig. 9.4). In this concept the lowest rung of the ladder—healing by secondary intention—starts out with what is a non-surgical wound closure and climbs up through primary, delayed primary closure to skin grafts in the middle rungs of the ladder. At this level a secondary defect is created due to tissue harvest. The benefit of achieving wound closure at the primary site must outweigh the risk associated

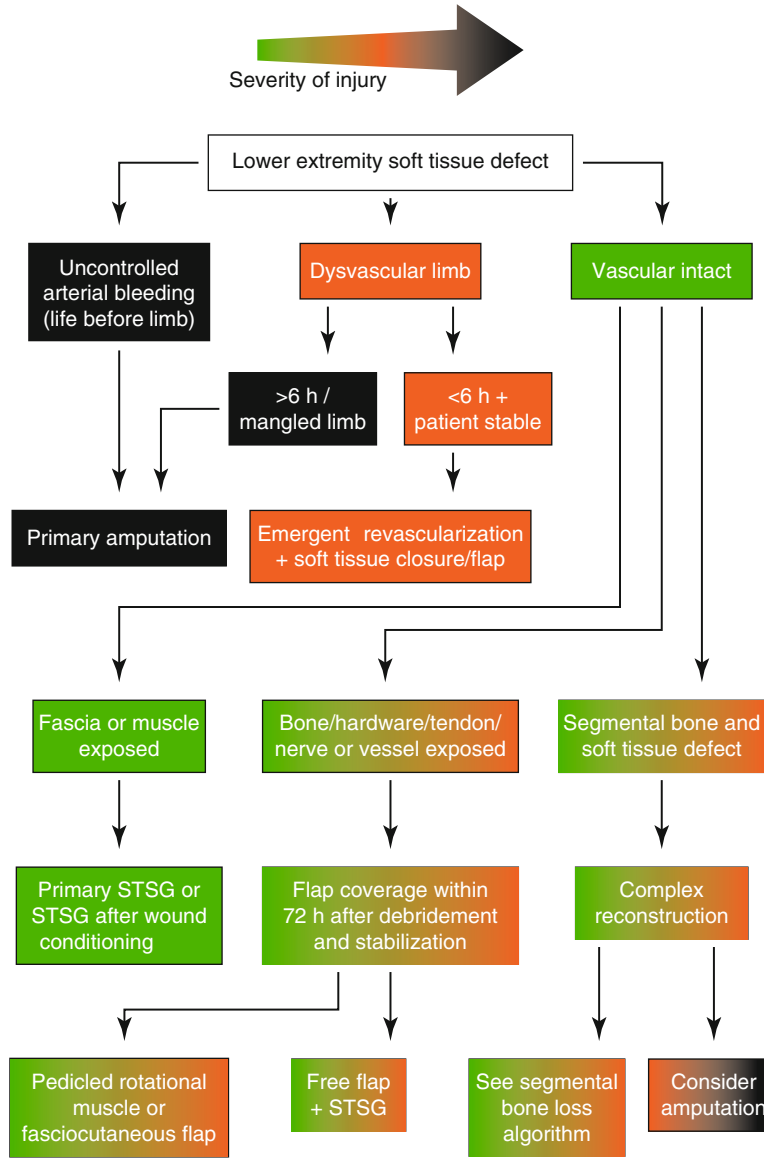


Fig. 9.5 Reconstructive algorithm for severe open lower extremity injury

with harvest wound problems. This said, the decision for each reconstructive option is based on defect requirements, functional demands, and donor site morbidity. The more complex flap will not necessarily yield the better result. It is the practice of most surgeons to utilize the most reliable, technically less demanding procedure as preferred choice. However, when encountering defects in functionally high demand areas, such as the dorsum of the hand, jumping up the ladder to more complex reconstructions, such as pedicled flaps or free flaps, can be necessary. When deciding on which type of flap to choose, the size of the recipient site, depth and bulk needed for reconstruction as well as anticipated pedicle length, and need for composite tissue reconstruction and options for innervation all have to be considered. The reconstructive team must either be able to offer all surgical options or refer the patient to an appropriate center. One should not choose a solution based on what is feasible for the team but what is optimal for the patient.

Figure 9.5 shows a reconstructive algorithm.

Monitoring

Whenever tissue is transferred—pedicled or free—there is the risk for graft or flap loss. Successful reconstructions rely on a competent team which includes the primary surgical team as well as nursing and midlevel support staff which are often the first to detect in the postoperative course alterations to the flap condition [62]. Parallel to the reconstructive ladder, the complexity and demand in monitoring increases. Most flap failures are seen in the first 24–48 h after surgery, and during this time, patients need to be closely monitored in an intensive or intermediate care level unit which allows for hourly checks of color, turgor, temperature, and recapillarization of the flap as well as Doppler evaluation of pulse signals. A recent study showed a salvage rate of 62 % in failing flaps with higher salvage rates seen in outflow (venous) occlusions [63]. Although newer techniques such as implantable laser flow Doppler probes suggest optimization of technical flap monitoring—still clinical exam and expertise appears to the most important factor in flap monitoring [64, 65].

Complications

Soft tissue reconstruction of injured extremities must be considered a major surgery. One has to differentiate intraoperative complications from early postoperative and late complications. Intraoperative cardiopulmonary problems due to volume and blood loss must be prevented, and volume and blood component substitution must be administered judiciously. This is especially true in free flaps where adequate tissue perfusion must be maintained and vasopressors avoided. Given the extensive surgery times and often multiple operative sites, hypothermia and associated coagulopathy are feared. Technical problems must be avoided through training and planning. With regard to surgical problems and complications, surgical strategy and plan must include evaluation of donor tissue options as well available recipient vessels. The anastomosis of a free flap must be positioned well outside the zone of injury; this in turn might entail longer pedicles or the need to plan for interpositional vein grafts. One must have a backup solution available should the planned flap prove unsuited or not feasible (e.g., anomalous perforators). Proper microsurgical training is essential as well as establishing a well-trained team. There are ample possibilities for intraoperative complications, even a tight dressing compressing the flap pedicle of what was until then smooth procedure can end up in flap loss. Continuous alertness and extensive experience is mandatory. In the early postoperative course, flap loss is the most feared complication and leaves the patient with two defects and the surgeon with an even greater reconstructive problem. Undersizing the flap and underestimating the injury zone with later demarcation of dead tissue around a vital flap are feared complications. The late amputation of a "salvaged" limb due to dysfunctionality or a multitude of eventually unsuccessful surgeries to address follow-up problems such as infections and nonunions must be seen as a disastrous late complication.

Alternate Techniques

With the wide usage of wound vacuum-assisted techniques, there are many instances where deficient soft tissue coverage can be successfully managed without surgical reconstruction. This is especially true for defects with full thickness skin loss, exposing muscle but not structurally important tissues as defined above. Wound vacuum-assisted therapies can be also utilized in between debridements as a bridge to early primary flap closure.

Soft tissue defects in the extremities can be successfully closed by dynamic closure "Jacobs Ladder" type using various elastic sterile material [66]. This technique together with full thickness skin incisions as relaxation incisions—often called "pie crusting"—can obviate the need for skin grafting and/or flap coverage. In these techniques proper wound border mobilization can be helpful to achieve closure. Given the appropriate indication, these techniques can result in an aesthetic and functional superior result and higher patient satisfaction.

Lower Extremity Reconstructive Options

Thigh and Knee

Although flap reconstructions at the thigh level are uncommon given the copious amounts of well-perfused muscular tissue surrounding the

femur, one can commonly see traction injuries, resulting in epifascial degloving of thigh and peripelvic tissue. These Morel-Lavallee lesions can be extensive and are for the most part located around the lateral side of the thigh and may extend high up the pelvis. The management relies on drainage and dead space management. Demarcating degloved skin needs to be excised at which point split thickness skin grafts in combination with a suction drainage or a wound vacuum-assisted device are used [67]. On rare occasions, critical soft tissue defects in the medial thigh have to be addressed such as in exposed vascular reconstructions where either local rotational flaps or free flaps may need to be employed for coverage.

Due to its exposed location and the lack of muscular coverage in the anterior aspect, traumatic soft tissue problems around the knee are common. Based on the defect size, rotational medial or lateral gastrocnemius muscle flaps are the workhorses of this body region. These flaps are pedicled on the medial and lateral sural artery respectively and can cover various soft tissue defects such as exposed patellar bone or hardware. The medial gastrocnemius has a longer muscle belly and can be easier harvested and rotated. The use of the lateral gastrocnemius is somewhat more complicated due to the peroneal nerve coursing the zone of dissection and the fibular head, which can—if the muscle is not completely transposed—shorten the rotational arc. Epimysial incisions are commonly used to increase flap length. Unilateral or bilateral gastrocnemius flaps can cover the entire anterior knee region when completely detached from their condylar insertions and skeletonized on the vascular pedicle [68]. Split thickness skin is used to epithelialize muscle flaps. The use of the tibialis anterior muscle as a proximally based rotational flap is described but seldom used due to its important role in foot dorsiflexion. As a modification the elevation of a slip of tibialis anterior for smaller defects is possible [69]. In cases of significant sized defects around the knee area, which are not amenable to rotational flap coverage—free flap reconstruction is indicated.

Leg: Proximal Third

The proximal third of the leg often requires soft tissue coverage. Defects are either due to direct trauma or seen subsequent to procedures necessitating extensile approaches to the tibial plateau. Hardware exposure is commonplace. Here similar to the already described coverage of the knee area, local rotational gastrocnemius flaps are most commonly used. The flaps can be elevated to include a strip of triceps surae tendon to allow secure flap fixation in sublay technique on the far end of the wound border. Use of suction drains in the harvest area is common practice for most surgeons. In addition to the abovementioned epimysial incisions and flap mobilization, knee flexion can increase flap length. In planning the procedure one has to position the patient such as to allow extensile dissection into the popliteal fossa.

Leg: Middle Third

The middle third of the leg is the most common area necessitating soft tissue reconstruction. The flap choice depends on defect size and injury severity. If local tissues are intact, rotational flaps can be utilized. One has to be mindful that already injured calf flexors can lose residual vitality after elevation and rotation, in which case it is more prudent to plan for a free flap. The preoperative use of angiography can be helpful to determine available recipient vessels, especially in high-energy trauma. However, one can opt for an intraoperative exploration during initial or second look debridement and perform a surgical Allen's test—which means to clamp the recipient vessel and ascertain foot perfusion.

In going through the reconstructive ladder, simple dermal defects such as seen after full thickness necrosis of skin blisters or abrasions or fasciotomy wounds are usually successfully managed with wound debridement and split thickness skin grafting. Once a IIIA level is encountered, closure can be facilitated by delayed primary closure in a second look procedure once the swelling subsides or use of adjuvant techniques such as pie crusting the skin or dynamic skin closure. Soft tissue defects as seen in IIIB defects usually calls for elevation of a pedicled flap. This can be a muscle flap—such as the

gastrocnemius and soleus flap or a fasciocutaneous flap as the sural artery neurovascular flap. The majority of low energy IIIB defects in the middle third of the leg are easily manageable by coverage with a proximal based soleus or hemisoleus flap. Some authors have described a recent increase in usage of rotational flaps and reduction in free flap reconstructions [70]. Extensile coverage can be achieved by using combined gastrocnemius-soleus flaps and skin grafting on top of the muscle flaps [71]. Once one chooses to elevate a flap, one has to be certain that the remaining soft tissues are viable and not undergoing necrosis. Oversizing the flaps to allow for secondary coverage of late demarcating injury zones and inverting the flap ends into the defect zone can be salvage options to prevent the need for additional flap coverage. If the injury severity precludes rotational flaps, one has to resort to various free flaps for coverage. Also immediate flow through flaps as emergency flaps may be indicated to restore pedal blood flow and cover the defects. A rarely used but robust reconstruction is a cross-leg flap which can salvage limbs in patients medically unsuited for extensive free flap surgery or void of recipient vessels [72]. This technique is valuable for attempts of limb salvage in underserved areas of the world. A full thickness flap is raised on the healthy contralateral leg and rotated—"cross leg" into the defect. The flap needs to be secured in place for 2–3 weeks at which point revascularization from the injured limb can occur and the flap can disconnected from the origin.

Structural vascularized bone graft such as a contralateral free fibula is an option for treating segmental defects seen after trauma or sequestrectomy [73, 74]. Vascularized free fibula flaps have a great versatility, can be harvested with attached skin islands and soleus muscle as well as the peroneal vessels as a flow through option for vascular reconstruction, and offer an option to treat segmental bone loss.

Leg: Distal Third

Whereas the proximal and middle one-third of the leg are the domains of rotational flap coverage, in the distal one-third of the leg the indication for free flaps was traditionally seen due to scarcity of tissue for transposition. This algorithm has changed with the development of axial fasciocutaneous and individual "cut as you go" perforator flaps, which allow for regional flaps to be utilized for coverage of distal leg and as well as hind- to midfoot defects [75, 76]. Tissue thickness mismatch can be a greater problem in the distal one-third of the leg where circumference, shoe fit, and esthetics are special importance. This must be a consideration when choosing a free flap. Thinner more pliable flaps such as a free radial forearm flap or free fascia flaps can be advantageous for restoring contour. One of the most commonly used flaps in the lower one-third of the leg is the sural artery flap, a fasciocutaneous flap pedicled on the sural artery. This flap can be elevated with relative ease and does not require microsurgical skills. It is usually pedicled distally and hinges on a peroneal perforator about 5 cm above the tip of the fibula [77].

Foot and Ankle

Crush and avulsion injuries are commonly seen in the foot. One has to differentiate between weight bearing and non-weight bearing areas in the foot and be cognitive about the need to restore or maintain sensation to the plantar aspect of the foot.

If the injury involves the forefoot, salvage options have to be critically evaluated. Often an amputation at a functional proximal level is indicated. If the crush injury involves only part of the toes, a local rotational flap involving a full thickness debulked flap from the remaining toes—such as greater toe filet flap—will provide adequate sensate coverage to maintain length and leverage for push-off during ambulation.

Complex midfoot trauma, as seen in penetrating gunshot injuries, can leave a full thickness defect with exposed tarsal bones. Here salvage is possible with thin fasciocutaneous flaps such as a free radial forearm flap on the dorsum of the foot, which does not add excessive bulk which could preclude use of regular shoes. The local rotational flap of the midfoot involving the non-weight bearing instep based on the medial plantar artery is described but not often used. Similarly

local extensor digitorum brevis muscle flaps are an option; however, dissection can be very tedious and in the situation of a complex trauma not possible.

Often—especially seen in motorcyclists—hindfoot injuries involving the heel pad are encountered. The heel pad is a unique tissue entity with its dense septated structure and difficult to replace. Heel avulsions if incomplete and still maintaining partial blood supply can be salvaged by careful adaptation and weight shielding or may end up undergoing partial tissue loss. In complete heel avulsions reconstruction usually requires free flap coverage. Given the large surface area of the calcaneal tuber and the required thinness of the flap, the rectus abdominis muscle is often used and can be folded around the heal and covered with split skin coverage [78]. This reconstruction usually results in some form of disability and for the most part in absent sensation, leaving the patient with gait problems and an ongoing risk for pressure ulcers. Custom orthopedic footwear is necessary.

In partial heel defects or perimalleolar defects with exposed hardware of full thickness defects such as seen in ankle dislocations, a local, retrograde pedicled neurovascular—sural artery flap is often used, allowing a 180° arc of rotation and can reach any area around the ankle and the tuber of the calcaneus. Local muscle flaps exist in the form of the peroneus brevis flap, which offers a simple low morbidity reconstructive option for isolated defects on the lateral ankle. For more extensive hindfoot and ankle defects, one has to climb the reconstructive ladder and use various free flap options. Again one needs to strive for thin pliable flaps in these non-weight bearing areas and use free fasciocutaneous or free fascia flaps with skin graft coverage.

Flaps to Salvage Functional Amputation Levels

When faced with an amputation, the primary indication for flap reconstruction is the salvage of a more functional amputation level. Generally speaking, transtibial and transfemoral levels are the most beneficial amputation levels. Most common indications are attempts to salvage a below knee amputation stump. Critical factors are the condition of the bony skeleton of the resulting stump. Comminuted proximal one-third tibia shaft fractures and involvement of the knee must be seen as relative contraindications for salvage. Reconstruction can be performed via either a free flap—such as a latissimus dorsi or rectus abdominis muscle flap—or utilization of an intact foot in the form of a foot filet flap [79, 80]. When planning the reconstruction, flap size needed to cover a three-dimensional stump and mechanical stability of the flap are important factors. Consideration must be given into availability of recipient vessel for anastomosis of a free flap. Often the tibialis anterior or the medial geniculate arteries are used for inflow. Venous outflow reconstruction can be more challenging and one has to critically reevaluate flap salvage once interpositional grafts are necessary for vascular anastomosis.

Management of Segmental Bone Defects and Options for Bony Reconstruction

Historically many fractures with significant bone loss were treated by primary amputation. Modern techniques of fracture stabilization and soft tissue reconstruction have enabled many more severely injured limbs with bone defects to be salvaged. The reconstruction of segmental bone defects (BD) represents a substantial clinical challenge to the orthopedic trauma surgeon.

Defects can be characterized by their length and or the percentage of circumferential involvement [81]. A “critical-sized defect” is a defect that will not heal spontaneously despite surgical stabilization and will require further intervention to achieve union. The threshold size of a critical defect varies according to both anatomical location and degree of soft tissue injury, but as a general rule, defects that involve and length superior than 2 cm and over half of the circumference are considered critical-sized defects [81].

The choice of fixation method for fractures with a critical BD is of particular importance. Not only must the method of fixation achieve the

normal prerequisites of skeletal stabilization and restoration of length and alignment and allow for preservation of function, but the fixation method must also allow for further management of BD. The choice of fixation is therefore influenced by the anticipated method of managing BD. Established methods of managing BDs include acute limb shortening, distraction osteogenesis techniques, nonvascularized bone graft, and free vascularized bone transfer. The relative rarity of these injuries means there is a paucity of high-level evidence to guide management. This section reviews the different treatment options available for the management of bone defects secondary to trauma and gives the authors preferred treatment algorithm.

Acute Limb Shortening

Acute limb shortening can be performed at the time of initial stabilization to close a segmental BD. It is the simplest of all treatment options and can be performed in nonspecialist centers with most methods of fixation. It has the shortest treatment time and results in the least complications [82]. It improves stability, relaxes tension on soft tissues, and helps facilitate primary closure of open injures [83]. Shortening is better tolerated in the humerus than in the lower limb as inequality of upper limb length is less important to functionality than inequalities of lower limb length [84]. Case series of tibias and femurs electively shortened to restore limb length equality with up to 5 and 10 cm of shortening, respectively, suggest both muscle power and limb function return to near pre-shorted levels within 2 years [85, 86]. Lower limb length discrepancy from acute shortening can however lead to both functional and cosmetic concerns. Discrepancies of greater than 3 cm leads to postural imbalance and an uneven gait [87] and may result in contracture of the Achilles tendon, lower back pain, scoliosis, pelvic tilt, and hip pain [88, 89]. Isolated acute shortening of up to 3 cm in the lower limb is generally accepted [90, 91].

For BDs greater than 3 cm in size, acute shortening does remain an option; however, further procedures are generally required to address the resulting limb length discrepancy. One option is to acutely shorten the injured limb and electively shorten the contralateral limb at a later date. Case series reporting good outcomes in adults with up to 10 cm of elective shortening in the femur and 5 cm in the tibia have been reported [92, 93]. Little data exists on the functional outcome of acutely shortened limbs in the trauma setting, but generally, this treatment option is reserved for cases with less than 6 cm of shortening [90]. Another option is to acutely shorten the injured limb then lengthen the limb using distraction osteogenesis techniques.

Distraction Osteogenesis

Distraction osteogenesis techniques offer the potential to either acutely shorten a fracture then restore length or maintain length and transport a segment of bone into the defect (segmental bone transport).

Ilizarov developed his external fixator design in the 1950s and by chance discovered distraction osteogenesis in a patient who had mistakenly distracted his frame instead of compressing it [94]. He subsequently defined the optimum conditions for distraction osteogenesis over a 10-year period with a series of canine experiments [95].

In segmental bone transport, the limb is stabilized with a circular external fixator and a bone transport segment is produced by corticotomy of the metaphysis. After a 5-day latent period, this segment is transported at 1 mm/day in four increments until the diaphyseal segmental bone defect is eliminated. The new defect is filled with new bone by the process of distraction osteogenesis (Fig. 9.6). The docking site heals in compression by fracture callus. Generally 2–3 days of consolidation are required for each day of distraction. In most cases, both ends of the bones should be freshened and autologous bone graft used to increase healing potential [83]. This technique can be used to fill a defect of any size [83].

An alternative technique involves acutely shortening the limb with subsequent lengthening of the limb at a distal corticotomy site. This technique

has the theoretical advantage of faster healing of the traumatic fracture as it does not require waiting until docking is achieved to begin callous healing. Furthermore, in cases where a wound is present over the segmental bone loss, shortening can facilitate wound closure. One has to be aware of the potential risk of redundant tissue if the shortening is excessive [83]. A French working group recommended against using this technique for defects greater than 6 cm in size [90].

Although both above techniques were developed using a circular frame, unilateral rails and more recently intramedullary devices have been used for distraction osteogenesis techniques.

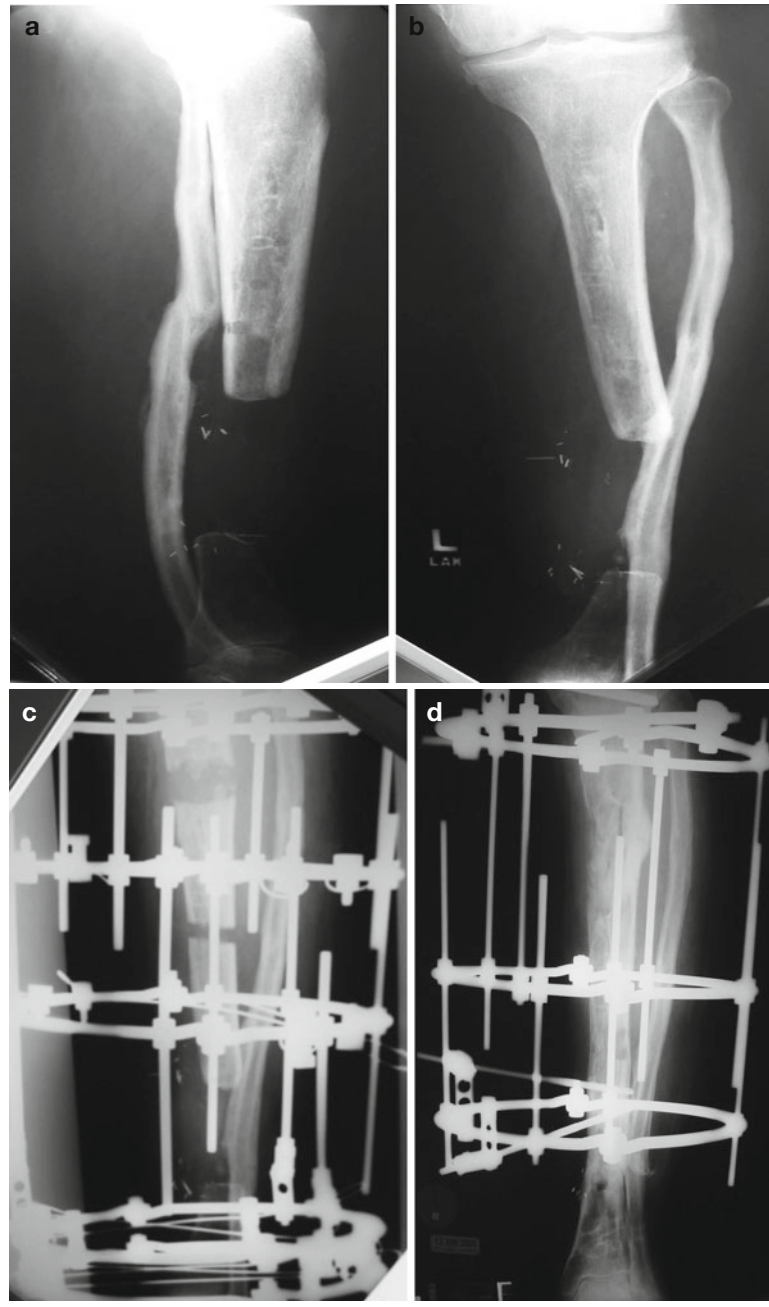
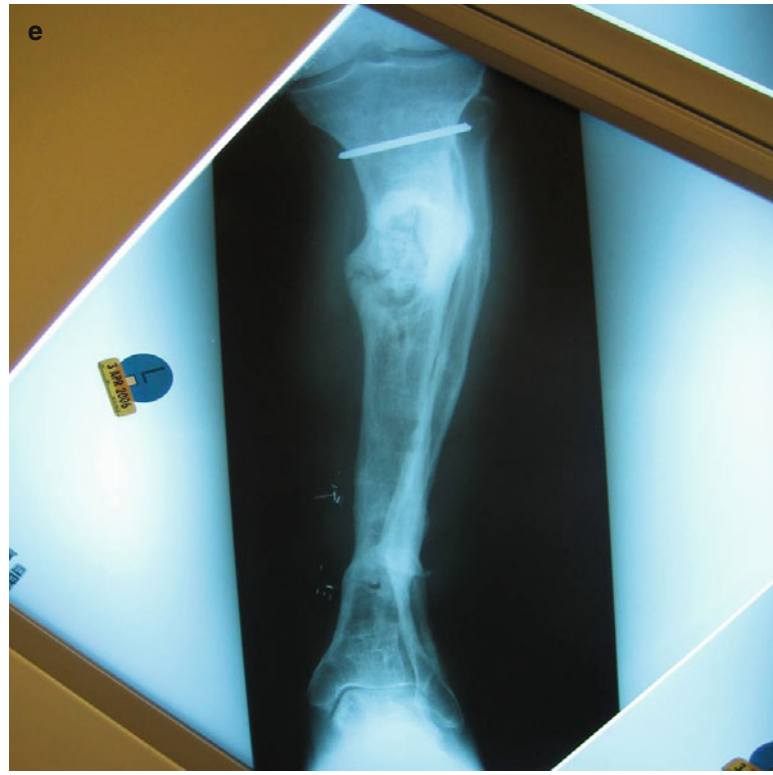


Fig. 9.6 (**a**–**e**) Segmental bone transport. Bone transport segment created by proximal corticotomy and distracted at 1 mm/day to fill distal defect. Bone formed in new proximal defect by distraction osteogenesis. (**a**, **b**) Lateral and AP radiographs of a tibia and fibula following resection of segmental bone loss. (**c**, **d**) Distraction osteogenesis using a ring fixator

Fig. 9.6 (continued)



Both the above techniques require prolonged periods of treatment and are associated with high complications rates. A recent case series of acute shortening followed by lengthening reported a mean time in external fixation of 7.1 months and an average of 2.1 complications per patient of which 38.5 % were considered major complications [96]. Despite these problems, some form of distraction osteogenesis is probably the most commonly used method of managing intermediate and large BDs [83].

Autologous Nonvascularized Bone Graft and Bone Graft Substitutes

Autologous nonvascularized cancellous bone grafting remains a common method of managing critical-sized posttraumatic BDs. Skeletal stabilization is performed with external fixation, intramedullary rods, or plates and may be done at normal length or with some shortening. Bone grafting is generally delayed for 6 weeks. Delaying grafting 6 weeks after free-tissue transfer allows complete epithelialization of the flap and therefore decreases bacterial contamination. When tissue transfer is not required, delaying grafting for 6 weeks allows wound healing and revascularization of marginally viable tissues [83]. Incorporation of bone graft is improved by grafting onto a host bed with stable vascularity. To improve the local blood supply to graft, all avascular scar tissue should be meticulously debrided and the medullary canal recanalized, thus reestablishing the medullary blood supply.

Several grafting location are available for tibial defects. Harmon described a posterolateral approach with subsequent placement of the bone graft on the interosseous membrane to obtain a long fibular synostosis spanning the tibial defect [97]. With the advent of free flap coverage, the choice of surgical approach is largely determined by the location of flap pedicle. For example, if the flap is anastomosed to the posterior tibial

artery, an anterolateral approach can be utilized. Furthermore, the graft can also be placed directly in the tibial defect, known as central bone grafting. Central bone grafting has been shown to be as effective as posterolateral grafting [98].

The first documented case of bone grating was in 1668 by a Dutch surgeon who described filling a bony defect in a soldier's cranium with a piece of canine skull [99]. Several different contemporary sites exist for the harvest of cancellous autologous bone graft including the anterior iliac crest, posterior iliac crest, distal femur, proximal tibia, and distal tibia. Equivalent levels of osteoinductive growth factors have been shown among the different graft sites [100], but the volumes available have been shown to be slightly different. In fact in decreasing order of volume, the sites are: posterior iliac crest > anterior iliac crest > distal femur > proximal tibia > distal tibia [81]. Harvest from the anterior superior iliac spine remains the most popular option [81].

Significant limitations of anterior iliac crest harvest however exist. A recent meta-analysis reported a complication rate of 19.6 %, with chronic donor site pain occurring in 7.75 % of the 6,449 patients undergoing anterior iliac crest harvest [101]. Furthermore, due to the limited volume of graft obtainable from the iliac crest, the size of BD treatable with iliac crest graft is restricted to defects 5–7 cm in size [91]. As a consequence of these limitations alternatives to conventional cancellous harvest have been developed, both in terms of new graft harvest sites and alternative graft material.

The intramedullary canal of long bones represents another donor site for autologous bone graft and is a rich source of osteoprogenitor cells and growth factors [102]. The reamer aspirator irrigator (RIA) system has been used to harvest bone graft from the intramedullary canal of the femur and tibia. This technique has been shown to provide the highest volume of autologous bone available of all other anatomical sites (25–90 cm^3) [101], and there is some in vitro work demonstrating RIA bone grafts have elevated levels of osteoinductive growth factor and osteoprogenitor/endothelial progenitor cell types relative to iliac crest graft, suggesting it may represent a biologically superior graft source [81]. Furthermore, the overall complication rate associated with RIA harvest in a recent meta-analysis was 6 % in 233 patients [101] (it is however worth noting that 1.6 % of theses were fractures).

As a consequence of the limitations of autologous cancellous bone grafting, a significant quantity of research has been directed at finding an alternative. The optimal bone substitute should be osteoconductive, osteoinductive, and osteogenic, without risk of transferring infection, readily available, manageable, biocompatible, and bioresorbable [102]. In order for a fracture to heal, prerequisite such as presence of osteogenic cells, growth factors, osteoconductive matrix, and a stable mechanical environment are essential [102]. Autologous graft can satisfy the first three prerequisites (and potentially the fourth if a strut graft is utilized). The various commercially available bone graft substitutes satisfy the diamond concept prerequisites to varying degrees.

Bone graft substitutes can be broadly classified as allograft, biological bone substitutes such as demineralized bone matrix (DBM), or synthetic substitutes such as calcium phosphate, calcium sulfate, or bioactive glass. All are osteoconductive but contain variable quantities of growth factors depending on preparation. Growth factors such as BMP-2 can be used in conjunction with graft substitutes. Although there is limited Level I data available on the performance of bone graft substitutes compared with autologous cancellous graft, the limited available data is encouraging. In a study by Jones et al. patients were randomized to receive either autologous iliac crest bone graft or recombinant human BMP-2 combined with cancellous allograft at a tibial bone defect site. The average defect size was 4 cm. There were no significant differences in complication rates or functional outcomes between the two groups. The authors concluded that the use of allograft/recombinant human BMP-2 was equal in efficacy to the use of iliac crest graft for the treatment of segmental bone defects in the tibial diaphysis [103]. Bone graft substitutes do not have to be used in isolation. There are several case series reporting good results of segmental defects treated with

DBM and bone marrow aspirate concentrate [104] and cancellous autologous graft, concentrated bone marrow aspirate and scaffold [105]. Masquelet has recommended bone graft substitutes can be used to bulk up autologous grafts so long as the ratio of graft to substitute does not exceed a 1:3 ratio [106].

Masquelet Technique

Limits exist to the size of defect treatable with conventional nonvascularized autologous techniques. Generally cancellous autologous graft is not advocated when the defect is over 5 cm in size [107]. When diaphyseal defects larger than 6 cm in size are reconstructed with autologous bone graft, healing is limited by graft resorption, even in a well-vascularized muscular envelope [108, 109]. The use of periosteal flaps to provide a well-vascularized envelope enabling large nonvascularized grafting showed promising results in animal models [110] but was limited in practice by the size of flap available in humans. In 1986 Masquelet developed the induced membrane technique to reconstruct large defects with nonvascularized autologous bone graft. In the early 1980s Masquelet observed that the foreign body-induced membrane created by the use of a polymethylmethacrylate (PMMA) cement spacer provided a well-vascularized graft bed onto which large defects could be grafted without graft resorption. Later experimental studies confirmed that the richly vascularized membrane secretes growth factors including VEGF, TGF Beta 1, and BMP-2 and stimulates bone marrow cell proliferation and differentiation to osteoblastic lineage [110], thus reducing graft resorption.

The Masquelet technique is a two-stage technique. The first stage comprises of radical debridement, soft tissue repair, and the insertion of a polymethylmethacrylate (PMMA) cement spacer into the bone defect. Originally the technique was described in conjunction with external fixation, but both IM nails and plates have subsequently been used with the technique [107]. The second stage is performed 6–8 weeks later. The spacer is removed with the PMMA induced membrane being left in place. The cavity is then filled up with morcellized cancellous bone autologous graft harvested from the iliac crests and the membrane is closed around the graft, creating a biological containment system. Although Masquelet recommended cancellous iliac crest graft, promising results have since been obtained with RIA graft [111, 112]. For lower limb reconstruction, full weight bearing is usually initiated at 5–6 months with the protection of the external fixator with removal of the fixator at the 8-month stage.

Between 1986 and 1999, Masquelet reported on a series of 35 reconstructions of long bone segmental defects ranging from 5 to 24 cm. Thirty-one of thirty-five patients (89 %) healed their bone defect. Full weight bearing without protection was acquired in the mean time of 8.5 months (range: 6–17 months). Four patients sustained a late fracture through the grafted defect after it was considered healed. All were treated successfully with cast immobilization [107]. In a larger multicenter retrospective French series, 84 posttraumatic diaphyseal long bone segmental defects were reconstructed using the induced membrane technique. Union was obtained in 90 % of cases with a mean time to union of 14.4 months. Interestingly mean of 6.1 interventions was necessary to obtain union [113].

Vascularized Bone Transfer

Free vascularized fibular grafts are autologous grafts with structural strength and can be used to bridge defects of up to 20 cm in size [90]. The bone is vascularized and therefore remains viable. The contralateral fibula is isolated with its nutrient artery and veins and transferred to the BD, fixed in situ and vessels anastomosed. Five centimeters of distal fibula must be left at the donor site to avoid ankle problems, and 7 cm of proximal fibula usually is left to avoid knee and peroneal nerve problems. The time to union varies but most authors quote 3–6 months [82] with a union rate up to 90 % [114].

The principal disadvantages of free fibular grafting include the risk of failure of the vascular anastomosis, nonunion of graft, and fracture of

graft. A recent comparison of free fibular grafting with bone transport in the femur by Song et al. indicated that superior results were obtained with the latter method [115].

Conclusions and Authors' Preferences

Segmental bone defects in trauma patients continue to represent a considerable clinical challenge to the orthopedic trauma surgeon. Although many different treatment options have been described in the literature, high-level evidence to help guide the clinician is lacking. Outcomes of the same technique vary from center to center and techniques are often combined, clouding the treatment effect of individual interventions. At our center we have adopted the following algorithm for management of segmental bone defects (Fig. 9.7).

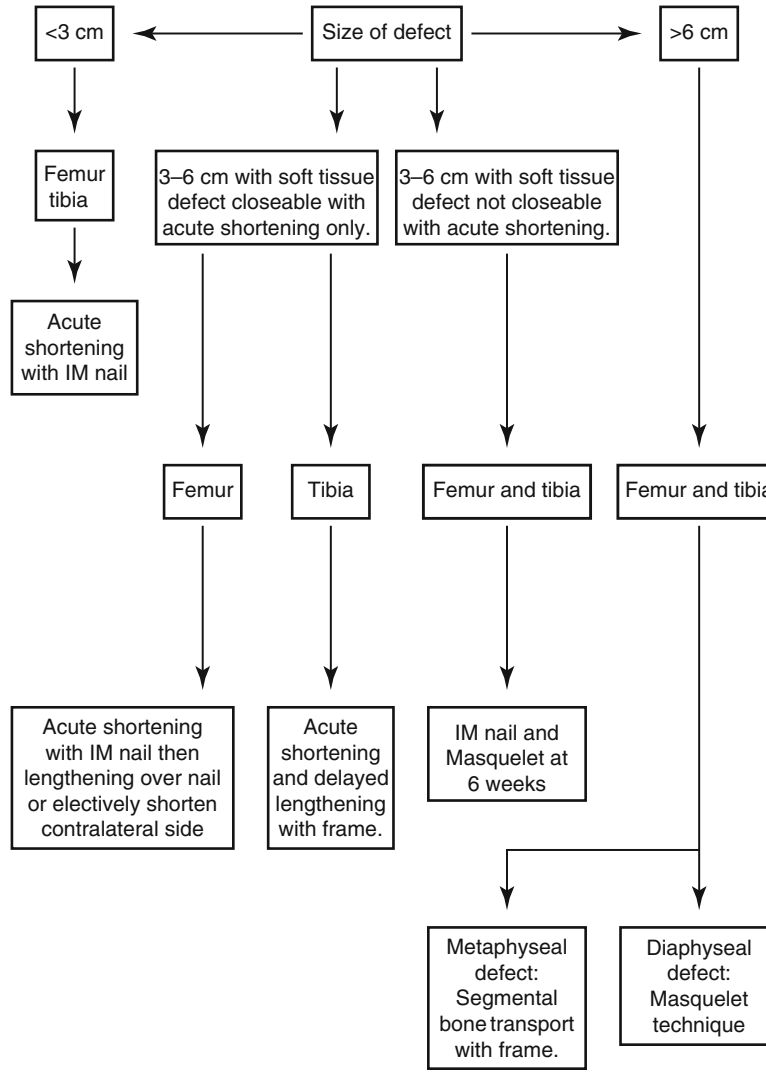


Fig. 9.7 Algorithm for the management of segmental bone loss (authors preference)

Conclusion

Severe open lower limb fractures remain an injury that should be managed by a team of experts in the field of orthopedic traumatology. Early involvement of microvascular surgeons is paramount for optimum planning of long-term care and reconstruction options. It is rarely wrong to stage the treatment with early application of external fixator with an aim to cover the skin defect and definitively fix the fracture within 72 h. Early antibiotic prophylaxis alongside thorough debridement of the wound is critical and if delayed or not performed in a systematic approach will increase the infection rate. The initial debridement and the presence of a muscle necrosis, bone loss, or a wound that will not close are the basis upon which the new OTA classification is based upon. Presence of either one of the above signifies a higher-energy transfer and risk of infection from devitalized tissue making prophylactic Gram-negative coverage advisable in these cases. The use of local application of antibiotics in the form of antibiotic beads is currently being evaluated through prospective trials. In the meantime, the retrospective evidence available seems to show significant reduction of infection rates for type II and III fractures. Soft tissue reconstruction for limb salvage still remains an art and requires great technical expertise. Patients' comorbidities and compliance with treatment will have a significant impact on the feasibility and the outcome of limb salvage. The reconstructive ladder describes reconstructive options for soft tissue defect management. Climbing up the rungs increases both technical complexity and risk of morbidity. Simpler options should be attempted first. When segments of long bones are missing following a traumatic event a number of options are available from shortening to bone transport, vascularized grafts, and induction membrane technique. The rarity of injuries causing segmental bone loss combined with the large number of options (including means of fixations and types of grafts) available to treat them means that performing a randomized control trial is challenging. There is no supremacy of one method over the other, but our institutional algorithm helps guide the treatment based upon the site of bone loss and the size of the defect.

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Compartment Syndrome

10

David A. Forsh and Philip R. Wolinsky

Introduction

Compartment syndromes occur within closed osseofascial compartments of the extremities. The pathophysiology has been well delineated, and the treatment, early and complete fasciotomy of the involved compartments, is clear. Although the clinical symptoms of a compartment syndrome are a mantra that has been drilled into our heads as residents, their clinical utility is actually unclear. The clinical symptoms and physical exam consistent with a compartment syndrome have a high negative predictive value, but their positive predictive value, specificity, and sensitivity are less than optimal. They can be difficult to evaluate even in an awake, alert patient and cannot be used for patients with an altered sensorium or for pediatric patients. Compartment pressures can be directly measured and used as a diagnostic adjunct when the clinical symptoms are unclear or cannot be obtained, but pressures do not directly measure tissue ischemia. Numerous absolute or relative pressure values have been proposed to use as a threshold for decompression. Other modalities are being investigated that might be used in the future for the detection and diagnosis of compartment syndrome, but none are currently ready for routine use.

Patients who have had decompressive fasciotomies have long-term sequelae, but it is unclear if those sequelae are the result of treatment or the injury. What is clear is that the outcome after a missed compartment syndrome is devastating to the patient as well as the treatment team, and the legal system may become involved after a missed compartment syndrome. The mainstay of treatment is to be alert to the possibility of compartment syndrome in the appropriate clinical situation and using the clinical signs and symptoms when obtainable, with or without pressure measurements, to determine whether a compartment syndrome is present. If it is an emergent, decompression is needed.

History

It has been over a century since Volkmann first described a condition characterized by paralysis and muscular contractures of a limb that he attributed to over-compression by tight circumferential bandages around injured extremities [1]. Since his landmark description, many studies have developed our understanding of what we now know as compartment syndrome.

Volkmann hypothesized that this condition, later called "Volkmann's ischemic contracture" was caused by disruption of blood flow to the muscles. Less than a decade later, it was noted

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that ischemic contractures could occur in the absence of external compression [2]. During World War I wounded soldiers were found to develop contractures thought to be secondary to arterial injuries. In 1928 Jones stated that these contractures "may arise from pressure within the limb or without … it is generally a combination of both" [3]. Since vasospasm of damaged arteries was thought to contribute to the vascular occlusion, Leriche and Griffiths proposed the use of reflex arc sympathectomy or sympathetic blockade to prevent ischemic contractures [4, 5].

Modern studies have revealed that this condition has many etiologies and elucidated the pathophysiology that leads to the compromised blood supply to the tissues. Murphy suggested that increased pressure within the deep fascia obstructed venous circulation and led to muscle damage [6] and was one of the first to advocate splitting of the deep fascia (fasciotomy) to relieve this pressure.

Over the course of the mid-twentieth century, cases of ischemic necrosis in the absence of injury or trauma were noted. Though unpublished, Vogt is credited with first describing exercised-induced ischemic muscle necrosis in marching cadets [7]. Hughes suggested that this necrosis of the anterior compartment of the leg was attributed to spasm of the anterior tibial artery [8]. Decompressive fasciotomies were effective in relieving pain symptoms in professional football players [9].

In 1958, Ellis suggested that unrecognized vascular damage or spasm could cause complications even for minor fractures [10]. MacGowan realized that a limb could still develop a Volkmann's contracture when palpable pulses were present and noted the difficulty in identifying the early clinical signs and symptoms of ischemia [11]. Seddon reported on 15 cases of ischemic necrosis in the leg and advocated the recognition of early signs of ischemia as the essential step to prevent late complications. He was a proponent of early fasciotomy to decrease the ischemic injury to muscle and described evacuating submuscular hematomas [12].

Continued research on Volkmann's initial ischemic contracture, which Matsen later referred to as compartment syndrome (CS) [13], led to the measurement of intracompartmental pressures (CPs) and defining normal and abnormal pressures. The first recorded measurement of interstitial fluid pressure using needle cannulation was reported in 1884 [14]. Prior to the turn of the twentieth century, the role of hydrostatic and oncotic forces in influencing movement of fluid across capillary membranes was defined (Starling forces) [15]. It wasn't until years later that these concepts were applied to the pathophysiology of extremity injuries.

Epidemiology

The incidence and risks for developing a compartment syndrome were described using data from the United Kingdom. McQueen et al. [16] reviewed 164 patients with CS over an 8-year period. Sixty-nine percent of patients with compartment syndrome had a fracture. The two most common fractures were diaphyseal tibia (36 %) and distal radius fractures (9.8 %). Twenty-three percent of cases only had a tissue injury. Most cases occurred in young men (149 pts, average age of 32 years) versus 15 cases that occurred in women. The incidence of compartment syndrome was three times lower in patients over 35 years of age. The annual incidence was 7.3/100,000 for men and 0.7/100,000 for women. They concluded that patients most at risk of developing acute compartment syndrome are young men with diaphyseal tibial fractures, high-energy forearm and/or distal radius fractures, or fractures of the tibial metaphysis. Patients with bleeding diatheses with soft tissue injuries also have a significantly higher risk.

There are a variety of injuries and conditions that may cause compartment syndrome (Table 10.1): fractures, soft tissue trauma or crushing injuries, tight casts or wraps, and ballistic injuries are some of the more commonly cited etiologies [16–26]. While most cases occur in the lower extremity, a compartment syndrome can occur anywhere a closed osseofascial space exists. The gluteal region, thigh, calf, foot, deltoid, upper arm, forearm, and hand all have compartmentalized musculature

Table 10.1 Common causes of compartment syndrome

Fracture
Anticoagulation/bleeding diatheses
Soft tissue injury
IV fluid extravasation
Dialysis/nephrotic syndrome
Burns
Snake bites
Revascularization
Exercise induced
Tight casts/dressings[a]
Crush injury[a]
Pneumatic antishock garments[a]

[a]Source of external compression

encompassed within fascia making them susceptible to the development of a compartment syndrome.

The use of traction and the position of a limb affect tissue pressures. Shakespeare et al. demonstrated that leg compartment pressures were proportional to the amount of calcaneal traction that was applied. There was a rise in pressure within the deep posterior compartment of >5 % for every 1 kg added [27]. The position that the ankle is immobilized affects intracompartmental pressures. Plantar flexion elevates pressure in the anterior compartment, while dorsiflexion raises pressures in the posterior compartments; the elevations in the deep and anterior compartment pressures can fluctuate upwards of three- to sevenfold. Positioning the ankle between 0 and 37° of flexion is most protective against elevated pressures in both the anterior and deep posterior compartments [28].

Studies have evaluated the changes in compartment pressures that occur during intramedullary nailing. Moed et al. found that intramedullary nailing of closed tibial fractures in a canine model increased pressures particularly in the anterior compartment [29]. The pressure increases normalized over time and were sustained in only 2 of 10 canines. Tornetta et al. found that unreamed tibial nailing caused transient intraoperative pressure elevations up to 58 mmHg, and these elevations returned to normal by the end of the procedure [30]. McQueen and Court-Brown using continuous intraoperative monitoring had similar findings [18]. Based on these studies, it seems that intramedullary nailing causes a transient increase in compartment pressures that returns to normal over time.

Polytrauma patients are at high risk for a delay in the diagnosis of a compartment syndrome compared to patients with isolated injuries [21, 31–34]. Anesthetized or intubated patients are unable to participate in a clinical exam. Other criterion (compartment measurements, pressure differentials, etc.) should be used in order to make the diagnosis in these patients.

Pathophysiology of a Compartment Syndrome

Understanding of the events that lead to the development of a compartment syndrome is essential for diagnosis and treatment. The initial inciting event leads to an increase in intracompartmental pressure. If high enough, this will decrease tissue perfusion secondary to a decreased arterial-venous gradient. Homeostasis between venous pressure, arteriolar flow, and tissue pressure is vital. Ischemic histologic changes in muscle can be seen in as little as 2 h, and changes that have clinical implications occur within approximately 3–4 h [35].

The normal relationships that exist between arterial pressure, venous pressure, and interstitial tissue pressure create an arteriovenous gradient (AV gradient) that provides adequate tissue perfusion. Since veins are collapsible, the pressure inside venules must be the same pressure as the pressure in the interstitial space. Once interstitial pressure increases, so does the pressure in the venous system. This decreases the AV gradient and tissue perfusion—this is the mechanism by which a compartment syndrome develops. Once there is an imbalance, a vicious cycle ensues where ischemia leads to further edema within the compartment that further compromises blood flow. Following the onset of ischemia, irreversible changes in nerve tissue and skeletal muscle can be seen in as little as 8 h [36] since tissue metabolic requirements are unable to be met [21–26, 31–34].

Some authors hypothesize that the difference between compartment pressure and blood pressure is vital for tissue perfusion [21, 37–41]. Hargens et al. proposed a microvascular occlusion theory where capillary occlusion is the primary factor that reduces tissue blood flow [42]. Using a canine model, they found that compartment pressures averaging 25 mmHg were sufficient to reduce tissue perfusion enough to cause capillary membrane damage resulting in an increase in permeability leading to leakage of plasma proteins which causes increased edema and decreased lymphatic drainage.

Muscle tissue can tolerate ischemia for up to 4 h before irreversible changes occur. Changes in nerves remain reversible for up to 8 h, fat up to 13 h, skin up to 24 h, and bone for 4 days. Type I and type II muscle fibers demonstrate differences in their susceptibility to ischemia. Most muscles contain a combination of red and white fibers, which are named based in the amount of myoglobin they contain. Type I, or red slow twitch fibers, rely predominantly on oxidative metabolism of triglycerides as their energy source and are particularly vulnerable to ischemia. These fibers can be found in the anterior compartment of the leg. Conversely, type II white fast twitch fibers predominantly use anaerobic metabolism of glycogen as their energy source and are more resistant to ischemia. These fibers are found in the muscles of the posterior compartment of the calf [38].

Clinical Diagnosis

Numerous papers and textbooks describe the clinical signs and symptoms of compartment syndrome. The key is a high degree of suspicion that a CS may be present and then to have the clinical acumen to diagnose it. The history, mechanism of injury, and radiographs can help identify patients at risk. The classic signs of a CS are referred to as the 6 Ps: pain out of proportion to injury, pain with passive stretch of the muscles in the involved compartments, paresthesias, pallor, paralysis, and pulselessness [26].

Pain is described as one of the earliest signs of an impending compartment syndrome, particularly pain out of proportion to the injury, or pain that was well controlled then suddenly increases. Judging pain levels can be difficult since:

- Perception of pain levels varies widely from patient to patient.
- Patients may have pain due to other injuries (distracting injury).
- It may be impossible to determine if a patient's pain is due to a developing compartment syndrome or to their initial/associated injury.
- Patients with a neurologic injury may not be able to complain of pain.
- Patients who have had regional or local blocks will not be able to complain of pain.
- Patients with an altered sensorium due to intoxication or head injury may not be able to communicate adequately.
- Patients who are intubated and sedated will not be able to complain of pain.
- Younger pediatric patients may be unable to effectively communicate as well.

Therefore, assessing pain may be possible in an alert patient with an unaltered sensorium, but that is not always the clinical situation for a trauma patient, and other diagnostic modalities should then be considered [17, 18, 21].

Paresthesias are another early sign of CS and occur secondary to nerve ischemia. Cessation of conduction occurs after approximately 75 min of complete ischemia [43].

A "swollen or full" feeling compartment may be an early physical exam finding suggestive of compartment syndrome and may be the only detectable sign in an obtunded or unconscious patient. Although the presence of fullness should trigger the thought that a CS may be present, the degree of "fullness" is not a reliable indicator as to whether a CS is present or not. The correlation between the subjective finding of compartment "firmness" and the pressure consistent with compartment syndrome is quite poor. Shuler and Dietz reviewed physicians' (junior/senior residents and attending surgeons) ability to manually detect elevations in intracompartmental pressures of the leg and showed overall sensitivity of 24 % with a specificity of only 55 %. The positive and negative predictive values were 19 and 63 %, respectively [44]. It seems that the presence of "firmness" should arouse suspicion for a CS,

but the degree of firmness cannot and should not used to determine whether a CS is present or not.

Pallor and paralysis are late signs of a CS and indicate the presence of significant tissue damage with poor prognoses. Paralysis is a sign of irreversible nerve damage with a low probability of functional recovery. Bradley et al. reported functional recovery in 13 % of patients presenting with paralysis [45]. Pulselessness is not typically seen with a CS and usually indicates that an arterial injury is present. In order to not have a pulse, compartment pressures would need to rise close to the systolic blood pressure.

Although compartment syndromes have been recognized and treated for many years, the methods that are available for diagnosing them still have issues. It is said that in an awake, alert patient without the presence of distracting injuries, the diagnosis of a compartment syndrome is a clinical one. Although the 6 Ps have been described as the cardinal symptoms of a compartment syndrome, paralysis is a late finding, pulselessness requires very high pressure on the order of systolic blood pressure that is rarely seen, and pallor would reflect ischemia of the limb as a whole rather than individual compartments. Studies evaluating pain out of proportion to injury, "fullness" of a compartment, and paresthesias emphasize the high negative predictive value of these findings, meaning that their absence is a relatively good way to exclude a compartment syndrome. However, the positive predictive values of these signs are poor, meaning that when they are present there may or may not be a compartment syndrome. In addition, the literature is confounded by the absence of a defined, universally applied, diagnosis of what a compartment syndrome or what a missed compartment syndrome is.

Compartment Pressure Measurements

The diagnosis of compartment syndrome may be made based on the physical examination in patients who are lucid, without gross neurological compromise, who can effectively communicate, and have clear finding on a physical exam. It is more difficult to make the diagnosis in patients in whom the clinical exam is unclear or when a patient is obtunded or intubated. In these scenarios the measurement of intracompartmental pressures (CPs) may be helpful. However, CPs do not directly measure tissue ischemia, they measure tissue pressures which are used as an indirect marker of tissue ischemia.

Pressure Thresholds

There is a range of absolute pressures cited in the literature for the diagnosis of a compartment syndrome. These pressures range from 30 to 45 mmHg [46–49]. Mubarak defined 30 mmHg as the critical pressure for decompression since this is the pressure at which the perfusion of muscles and nerves is decreased. He also found that pain and paresthesias were noted by his osteotomy patients once their compartment pressures rose over 30 mmHg [50]. Matsen considered absolute pressures over 45 mmHg as an indication for a fasciotomy. He also felt that an isolated elevated pressure value was of limited utility for decision making due to individual variations in tolerance to elevated compartment pressures [49].

Unfortunately, there is no accepted absolute value at which a fasciotomy should be performed. The numbers vary due to factors including (1) individual differences in the sensitivity of tissues to ischemia, (2) the AV gradient is dependent on a patient's blood pressure and, (3) damaged muscle is more sensitive to ischemia compared to non-damaged muscle. Because of the lack of consensus on an absolute critical pressure, relative or differential pressures are often used to aid in the diagnosis of CS.

Whitesides was the first to recommend the use of the difference between diastolic blood pressure and compartment pressure (Δ[DELTA]*P*). He felt that the clinical symptoms of a CS were variable and therefore not the most reliable indicator for the presence or absence of a compartment syndrome. His experimental work revealed inadequate tissue perfusion once the tissue pressure is within 10–30 mmHg of diastolic blood pressure. He recommended a fasciotomy when

the Δ*P* reached these levels [46]. When absolute tissue pressures were in the 20–30 mmHg range, he recommended close monitoring of the patient with repeat measurements every 1–2 h. Normotensive patients with diastolic pressures of 70 mmHg and suspected compartment syndrome should be decompressed once the absolute tissue pressures reach 40–45 mmHg.

The most common pressure measurement used clinically is the difference between diastolic blood pressure and compartment pressure. Court-Brown and McQueen, based on a study where they continuously monitored compartment pressures in 116 patients with diaphyseal tibia fractures, recommended the threshold for fasciotomy when the difference between diastolic blood pressure and the compartment pressure was a Δ[DELTA]*P* of 30 mmHg or less (Δ[DELTA]*P*) [18]. Using these criteria only 3 of the 116 patients developed an acute compartment syndrome, and there were no symptoms of any missed cases seen during the follow-up period. At follow-up of 6 months, none of the patients with a Δ[DELTA]*P* >30 mmHg had any sequelae from a missed compartment syndrome.

Using Δ[DELTA]*P* requires a reliable and reproducible diastolic blood pressure. Patients under anesthesia have a decreased diastolic blood pressure (DBP) that returns to the preanesthetic level postoperatively. Therefore the preoperative and not the intraoperative DBP should be used to calculate Δ*P* intraoperatively to make the decision whether or not to perform fasciotomy. Tornetta et al. found an average drop in diastolic pressure of 18 mmHg +/− 13 during surgery when they reviewed 242 anesthetized patients undergoing tibial intramedullary nailing [51].

At present there is no universally accepted absolute value used to define compartment syndrome, and many clinicians use relative pressures. Controversy remains about whether the existing thresholds are reliable. Prayson et al. measured compartment pressures in patients with isolated lower extremity fractures who had no clinical signs of a compartment syndrome and used the contralateral non-injured limb as a control. The average compartment measurements in the injured leg were 35.5 mmHg versus 16.6 mmHg in the uninjured leg, and 58 % of the patients had a Δ[DELTA]*P* of 20. Yet, despite not having a fasciotomy, no patient had sequelae of an unrecognized compartment syndrome at 1 year of follow-up [52]. The authors suggest that the current pressure criteria used to define a compartment syndrome should be interpreted with caution and highlighted the "normal" elevations in compartment pressures that occur in the presence of a fracture.

Continuous Pressure Monitoring

While there is debate regarding critical pressures, there is a consensus that the early diagnosis of compartment syndrome is of paramount importance to prevent late sequelae. The use of continuous pressure monitoring has been investigated as a method that could be used to detect early pressure rises and therefore earlier detection and treatment of compartment syndromes. Court-Brown [18] used continuous pressure monitoring and did not miss any compartment syndromes, which suggests that continuous pressure monitoring is helpful. Harris et al. randomized 200 extra-articular tibia fractures in alert patients into a continuously monitored group (100) and an unmonitored group (100). All patients were also monitored with repeated physical examinations. Five patients in the unmonitored group developed compartment syndrome, while none in the monitored group did. At 6 month of follow-up, there were no significant differences in complication rates or late sequelae between the two groups. The authors concluded that elevated postoperative compartment pressures did not correlate with the development of a compartment syndrome, and the clinical exam by itself is sufficient to detect a CS. They also concluded that continuous pressure monitoring of tibia fractures is not indicated in awake, alert patients who can be adequately observed [53].

Methods to Measure Compartment Pressures

The most common devices used to measure pressures use either a side-ported needle or a slit catheter. A slit catheter is used for continuous

pressure monitoring, while a side-ported needle is used for a static measurement. No difference has been shown between the two devices [54]. Pressure measurements using a standard 18G needle (which does not have a side port) result in higher pressures than either a slit catheter or a side-ported needle and should not be used.

There is a pressure gradient within compartments. The highest pressure is located at the fracture site and just adjacent to it. The pressure decreases the further you measure from the fracture site. Therefore, to increase the accuracy of pressure measurements, it is recommended to check pressures at multiple sites, including within 5 cm proximal and distal to the fracture [55].

Newer Noninvasive Diagnostic Modalities

Near-Infrared Spectroscopy

Due to the issues with the clinical diagnostic criteria used to diagnose CS as well as with compartment pressures, alternative diagnostic modalities are being investigated. Tissue ischemia correlates with the degree of muscle oxygenation. Shuler et al. directly measured tissue oxygenation using near-infrared spectroscopy. Near-infrared spectroscopy samples deep tissue below the skin to determine the concentration of oxygenated and deoxygenated hemoglobin. Fourteen patients with a diagnosis of compartment syndrome secondary to trauma were evaluated. Spectroscopy was used to record values in the affected extremity, and readings from the contralateral uninjured limb were used as a control. Thirty-eight compartments had pressure evidence of ischemia with a Δ[DELTA]*P* <10 mmHg. Near-infrared spectroscopy values in the affected anterior, lateral, deep, and superficial posterior compartments of the injured extremity were decreased by an average of 10, 10, 9, and 16 % compared with the corresponding contralateral compartments of the uninjured leg. The authors concluded that normalized near-infrared spectroscopy values decreased significantly with decreased limb perfusion pressures [56]. Near-infrared spectroscopy has potential for use in the early diagnosis of acute compartment syndrome; however, clinical trials are lacking. Technical improvements are needed since the low depth of tissue penetration is a limiting factor in its utility. More investigation is needed prior to the widespread clinical application of this device.

Metabolic Biomarkers

Metabolic biomarkers associated with muscle ischemia may be another noninvasive means to diagnose early compartment syndrome. Creatine kinase (CK), myoglobin (Mb), and fatty acid-binding protein (FABP) are markers present in skeletal muscle and may be elevated after muscle injury and necrosis. Lampert et al. found that CK values >2,000 units/L following surgery may be a warning sign for impending compartment syndrome in the anesthetized patient [57].

The Mb/FABP ratio has also been shown to be useful in identifying skeletal muscle injury. In myocardial tissue the normal ratio is approximately 5, while in skeletal muscle it is about four times higher [58]. Frequent measurements of these values following injury or fracture could theoretically detect the early stages of an impending compartment syndrome; however, these markers lack sensitivity and specificity and are not clinically useful.

Inadequate tissue perfusion leads to an anaerobic metabolism and a low pH; elevated lactate levels within an affected compartment that occur as a result may be an indicator of an early compartment syndrome. Ischemic modified albumin (IMA) has recently been identified as a marker of myocardial ischemia, is transiently decreased when skeletal muscle is ischemic, and returns to normal quickly when tissue perfusion is restored [59]. While these serologic markers may be a clue to early diagnosis, they lack reliable sensitivity and specificity, since they are usually elevated in inflammatory conditions and after trauma.

Imaging Studies

Advanced imaging studies have limited utility in the diagnosis of CS. MRIs show compartment edema; however, it is unable to differentiate

ischemic muscle from generalized soft tissue inflammation secondary to trauma. Radionuclide scintigraphy has been used to evaluate myocardial perfusion. This technique was used to evaluate limb perfusion in chronic exertional compartment syndrome. Edwards et al. showed good positive and negative predictive values with scintigraphy using 99-technetium-methoxyisobutylisonitrile (99Tc-MIBI) [60]. While low cost and minimal invasiveness are strengths of this technique, its use is limited because of the lack of specificity, time needed to perform the scan, and the difficulty repeating it [61].

Standard ultrasound has been shown to be ineffective, but a relatively newer technique, pulsed phase-locked loop (PPLL), has shown promise. Initially designed to monitor intracranial pressure, PPLL transmits ultrasound waves through the soft tissues and records the reflected waves through a transducer, which detects fascial micromotion corresponding to arterial pulsations. Decreased motion is indicative of increased compartment pressure; however, there are normal variations in fascial movements, and this limitation needs further investigation. Perfusion can be evaluated by transilluminating tissue and analyzing the light backscattered by moving red blood cells (laser Doppler flowmetry—LDF). LDF has promise, but it has only been evaluated in one study of chronic exertional compartment syndrome [62].

Direct Nerve Stimulation

Sheridan et al. used direct nerve stimulation to differentiate between neuropraxia secondary to acute compartment syndrome versus a more proximal nerve injury [63]. The absence of muscular contraction with stimulation suggests pathology from elevated pressures, while if the muscle contracts there may be a more proximal injury to the nerve itself. This method is not particularly useful in monitoring of at-risk patients for compartment syndrome because of the difficulty directly stimulating a nerve. Alterations in vibratory sensation have been shown to correlate with increased intracompartmental pressures [64].

Treatment

Once the diagnosis has been made, timely decompression is necessary. Prior to going to the operating room, anything causing external compression should be removed. Intracompartmental pressures are lowered after bivalving and spreading circumferential casts and dressings which allows room for increased swelling [65, 66]. To maximize tissue perfusion, the extremity should be placed at heart level. Placing it above the level of the heart reduces arterial inflow pressure and placing it below heart level increases venous pressures [67].

Once a compartment syndrome has been diagnosed, complete decompression via a fasciotomy is mandatory. Long skin incisions are used since incompletely releasing the skin results in persistently elevated pressures. Gaspard et al. described cases where the skin continued to cause compression after limited skin incisions, but they did not measure compartment pressures [68]. Cohen et al. made long fascial incisions using short 8 cm skin incisions in posttraumatic CS, and in over 30 % of cases, the pressures remained over 30 mmHg. When the skin incisions were extended to an average of 16 cm, pressures were significantly lowered to an average of 13 mmHg [69]. The use of long skin incisions to ensure complete decompression during a fasciotomy is mandatory.

Techniques

Calf Fasciotomy

The four compartments in the calf (anterior, lateral, superficial, and deep posterior) can be released using one lateral incision or a lateral incision combined with a medial incision. Both techniques are effective. When using the dual incision technique, the anterior and lateral compartments are decompressed through a long lateral incision placed just anterior to the fibula and extending from 5 cm distal to the fibular neck to 5 cm above the tip of the lateral malleolus (Fig. 10.1). Anterior and posterior skin flaps are then raised. The intermuscular septum is identified and the lateral and anterior compartments are

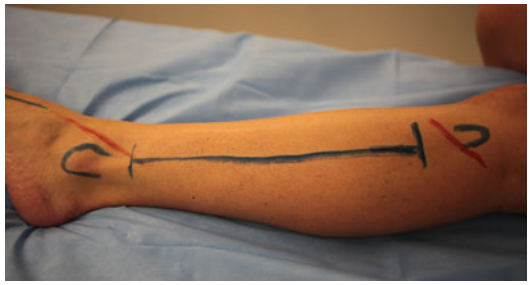

Fig. 10.1 Calf fasciotomy: the lateral incision used for a calf fasciotomy is drawn in *blue*. The proximal and distal ends of the fibula are outlined in blue proximal and distal to the incision. The proximal *red line* shows the course of the common peroneal nerve, and the distal red line shows the course of the superficial peroneal nerve in the distal calf

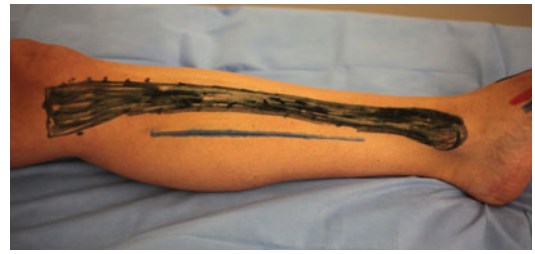

Fig. 10.2 Calf fasciotomy: the incision used for a medial calf fasciotomy is outlined in blue and is located 1–2 cm posterior to the subcutaneous border tibia (outlined in *black*). The superficial and deep posterior compartments are released using this incision when performing a two-incision technique

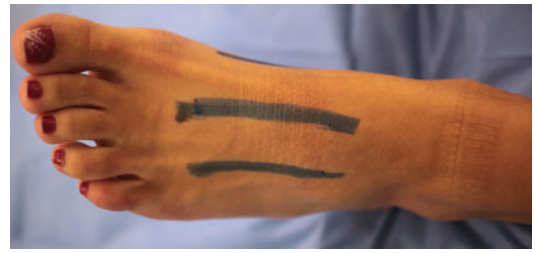

Fig. 10.3 Foot fasciotomy: the two dorsal incisions used for a foot fasciotomy are outlined in *blue*. They are located over the dorsal border of the second and fourth metatarsals. To ensure that the foot is completely decompressed, the deep dissection is carried down between the metatarsals

released by incising the fascia. The superficial branch of the peroneal nerve needs to be identified and protected where it pierces the fascia over the lateral compartment in the distal one-third of the leg. The medial incision is made 2 cm posterior to the medial tibial border (Fig. 10.2). The fascia of the superficial posterior compartment is visualized by retracting the skin posteriorly. The deep posterior compartment is released by releasing the soleal muscular leash from the medial face of the tibia. Since the musculature of the superficial posterior compartment is located proximally and the musculature of the deep posterior compartment is located distally, the medial incision needs to be long to perform an adequate decompression of both muscles.

When a single lateral incision technique is used, the same long lateral skin incision as the two-incision technique is used. The skin is elevated off the fascia anteriorly and posteriorly, and the septum between the anterior and lateral compartments is identified, and the fascia is released. The contents of the lateral compartment are then elevated from the posterior intermuscular septum, and the superficial posterior compartment is released by incising this fascia. To decompress the deep posterior compartment, the intermuscular septum is followed down to the fibula and subperiosteal dissection of the fascia off the lateral and posterior parts of the fibula decompresses the deep posterior compartment. It is important to maintain subperiosteal dissection around the back of the fibula to avoid the peroneal vessels that are nearby and can be easily damaged.

Foot Fasciotomies

Foot fasciotomies are performed using two longitudinal dorsal incisions placed just medial to the second metatarsal and lateral to the fourth metatarsal (Fig. 10.3). It is important to dissect between the metatarsals to completely release the fascia of the intrinsic muscles of the foot and achieve adequate decompression. Manoli and Weber described 9 ft compartments (medial, lateral, superficial central and deep, four interosseous, and calcaneal) and advocated making an accessory medial incision placed just plantar to the first metatarsal to release the medial and central compartments (Fig. 10.4) [70]. Others prefer using the extensile medial approach of Henry

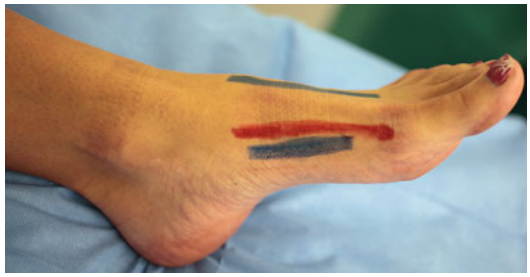

Fig. 10.4 Foot fasciotomy: the medial foot incision used to release the medial, central, and lateral compartments is outlined in *blue* and is located just plantar to the subcutaneous border of the first metatarsal which is outlined in *red*

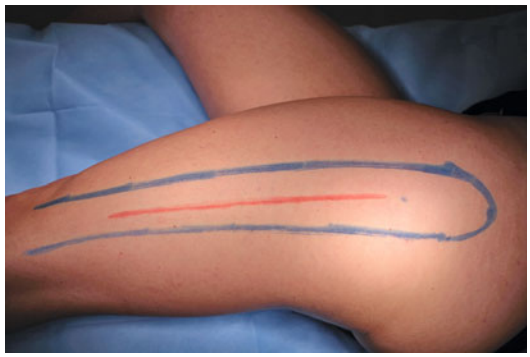

Fig. 10.5 Thigh fasciotomy: the lateral thigh incision used to decompress the anterior and posterior compartments of the thigh is outlined in *red*. The incision is centered over the shaft of the femur, which is outlined in *blue* and is in line with the longitudinal axis of the femur. The hip is to the right and the knee is to the left

to release all compartments [71]. Care must be taken to preserve the medial neurovascular bundle with this approach.

Thigh Fasciotomy

There are three compartments in the thigh: medial, anterior, and posterior. A laterally based incision is used to decompress the anterior and posterior compartments (Figs. 10.5 and 10.6). A second medial incision may be needed to release the adductor compartment if it is not decompressed after decompression of the anterior and posterior compartments (Figs. 10.7 and 10.8). Typically the pressures in the medial compartment are decompressed via the lateral incision; however, the pressures in the medial compartment should be measured after decompression of the lateral and posterior compartments and if still elevated a medial incision must be made to decompress the medial compartment.

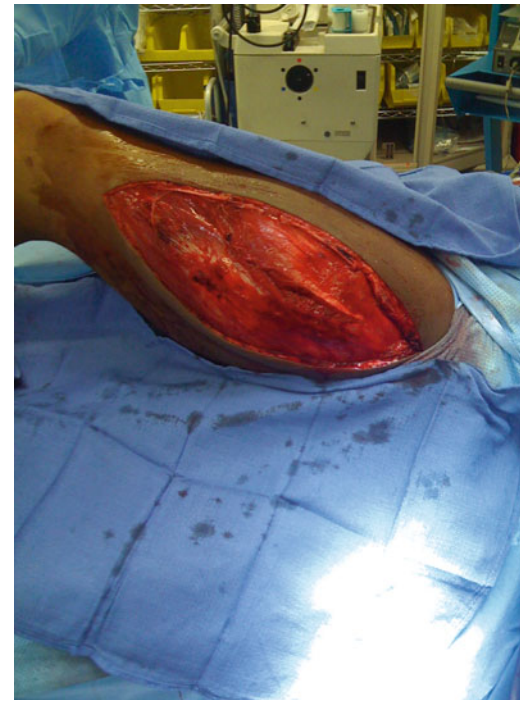

Fig. 10.6 Thigh fasciotomy: clinical picture of a thigh after a thigh fasciotomy using a lateral incision. Note the swelling and bulging of the muscles

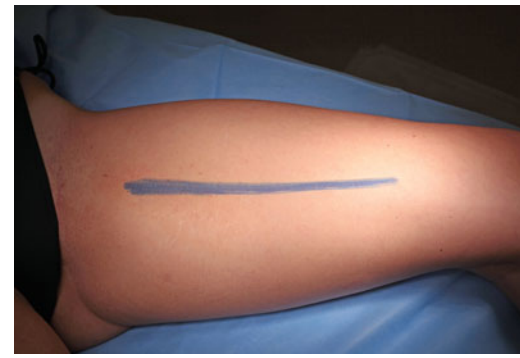

Fig. 10.7 Thigh fasciotomy: the medial thigh incision used for a fasciotomy of the adductor muscles is outlined in *blue*. This incision is based over the center of the hip adductor muscles and is used when the medial thigh compartment is not decompressed after a lateral thigh fasciotomy. The hip is to the left and the knee is to the right

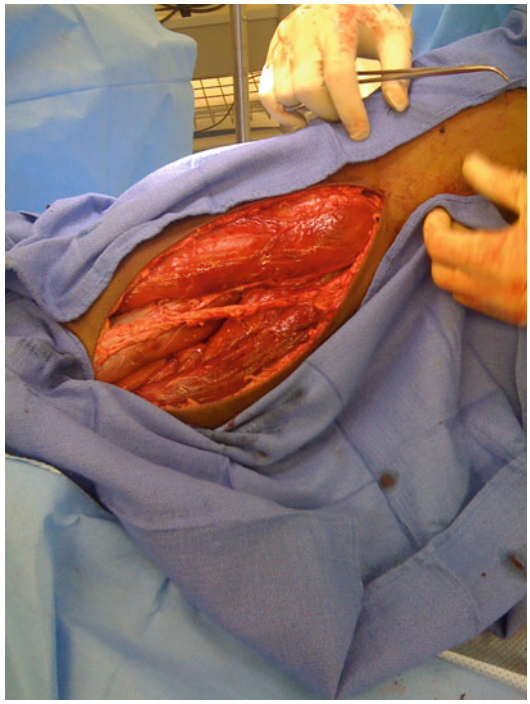

Fig. 10.8 Thigh fasciotomy: an intraoperative photo following a decompressive fasciotomy of the medial compartment of the thigh using a medial incision. Note the course of the saphenous vein traversing the field

Gluteal Fasciotomy

The gluteal region contains three distinct compartments: the tensor, gluteus medius-minimus, and gluteus maximus compartments. While rare, when a compartment syndrome develops in any of the gluteal compartments, a Kocher-Langenbeck incision is used for decompression. This approach also allows exploration of the sciatic nerve (Fig. 10.9).

Forearm Fasciotomy

Longitudinal volar and dorsal incisions over the forearm are used to decompress the forearm. Usually the dorsal compartment is decompressed after a volar fasciotomy; however, a dorsal incision extending from 2 cm distal to the lateral epicondyle towards the midline of the wrist is used if dorsal pressures remain elevated (Fig. 10.10). The mobile wad compartment containing the brachioradialis, extensor carpi radialis longus, and extensor carpi radialis brevis must be released separately.

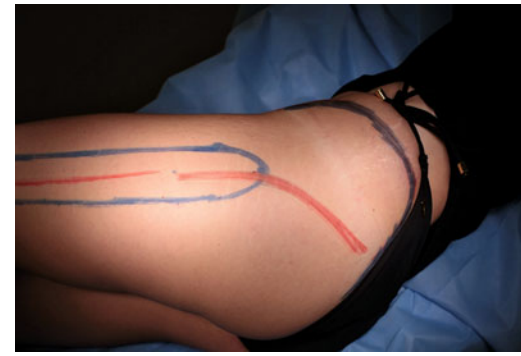

Fig. 10.9 Gluteal fasciotomy: a gluteal fasciotomy is performed using the Kocher-Langenbeck approach. The incision that starts just lateral to posterior superior iliac spine and curves over the posterior third of the greater trochanter and down the axis of femur is outlined in *red* and *blue*. This incision can be combined with the lateral incision for a thigh fasciotomy if needed. The model is positioned on her side: the hip is to the right, the knee to the left, up is anterior, and down is posterior

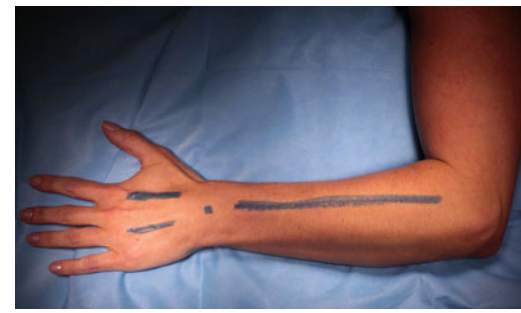

Fig. 10.10 Dorsal forearm fasciotomy: a dorsal fasciotomy of the forearm (*blue marks*) is performed using a straight incision on the dorsum of the forearm that begins 2 cm distal to the lateral epicondyle and extends towards Lister's tubercle (*blue dot*) and can be used to decompress the extensor compartment of the forearm. The deep dissection is in the interval between the extensor digitorum communis and the extensor carpi radialis brevis interval. The mobile wad can be released using this incision

The volar incision is curvilinear and extends from the proximal ulnar aspect of the forearm to gently curve radially and finally return to ulnar side. It then extends into the mid-palm just ulnar to thenar crease to decompress the carpal tunnel, which is a mandatory part of a forearm fasciotomy, as is releasing the lacertus at the elbow (Fig. 10.11). This incision allows for adequate

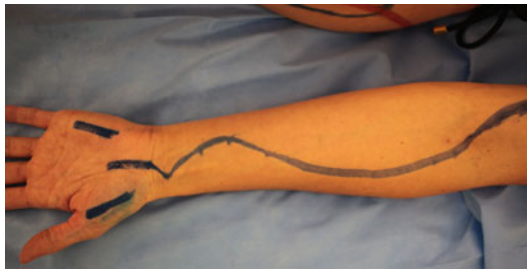

Fig. 10.11 Volar forearm fasciotomy: the volar compartment of the forearm is released using a curvilinear volar incision. The incision that is selected must cross the elbow flexion crease proximally and extend either anteromedial or anterolateral to the biceps tendon in order to decompress the lacertus. Distally the incision must cross the wrist flexion crease in order to release the carpal tunnel. Several different incisions are possible to use to decompress the forearm. The *blue marks* on hand are for release of the thenar and hypothenar eminences

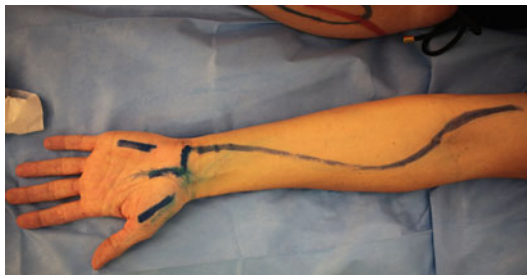

Fig. 10.12 Volar forearm fasciotomy: alternatively the volar compartment may be decompressed using a slightly different incision. Note the same gentle radial curvilinear course proximally with a more ulnar deviation distally. The wrist crease is traversed obliquely and extended into a straight incision over the carpal canal. The lacertus is released similarly as described in the previous figure. The *blue marks* on hand are for release of the thenar and hypothenar eminences

coverage of the median nerve. The carpal tunnel incision is closed, and the rest of the incisions are left open. Different curvilinear incisions may also be used to decompress the volar forearm and allow release of the lacertus proximally as well as the carpal canal distally (Figs. 10.12 and 10.13). There may be two separate volar compartments, superficial and deep, and the surgeon needs to make sure that the deep volar structures have been adequately decompressed.

Hand Fasciotomy

The compartments of the hand are released using two dorsal incisions placed over the second and

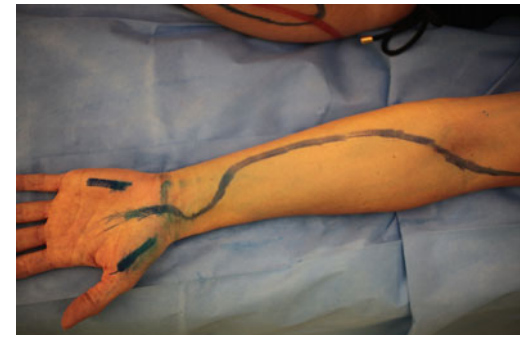

Fig. 10.13 Volar forearm fasciotomy: the volar forearm may also be decompressed via an incision with a long gentle ulnar-sided curvilinear incision which courses radially over the flexor carpi radialis. Again, the wrist crease is crossed obliquely into a carpal tunnel release. The *blue marks* on hand are for release of the thenar and hypothenar eminences

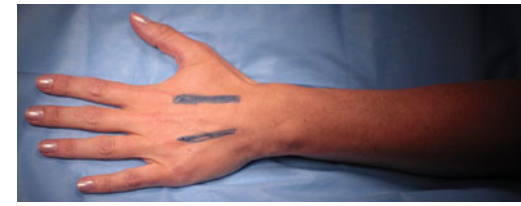

Fig. 10.14 Fasciotomy of the hand: the two dorsal incisions located over the dorsal subcutaneous borders of the second and fourth metacarpal used to release the dorsal and volar interosseous compartments are shown in *blue*

fourth metacarpals (Fig. 10.14). The overlying fascia between the metacarpals is incised to decompress, and each muscle should be individually identified to ensure adequate release. If necessary, the thenar and hypothenar eminences are decompressed via two additional incisions placed over the glabrous borders of each (Fig. 10.15).

Upper Arm and Deltoid Fasciotomies

Compartment syndrome of the arm is rare; the anterior and posterior compartments are separated by the humerus and the medial and lateral intermuscular septae. They can be released using a single incision placed either medially or laterally (Figs. 10.16 and 10.17). Occasionally a single posterior incision is used to release the triceps compartment (Fig. 10.18). The deltoid can be subdivided into anterior, middle, and posterior compartments and can be released through a standard deltopectoral incision (Fig. 10.19).

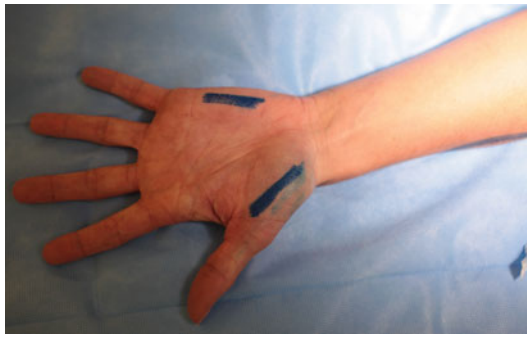

Fig. 10.15 Fasciotomy of the hand: the incisions located at the radial and ulnar-sided glabrous borders used to release the thenar and hypothenar compartments are outlined in *blue*

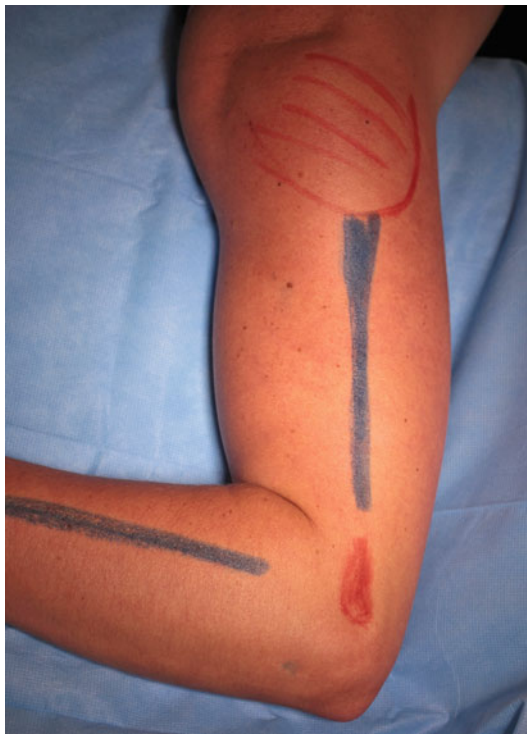

Fig. 10.16 Arm fasciotomy: the anterior and posterior compartments of the upper arm can be released using a lateral incision made in line with the long axis of the humerus. The incision is shown in *blue*, the deltoid muscle is to the right and is outlined in *red*, and the lateral condyle is to the left and outlined in *red*. This incision can be combined with the dorsal incision used for decompression of the forearm that is also show in *blue*

Alternatively, the deltoid compartment may be released through a straight lateral incision over the middle head of the muscle (Fig. 10.20). If a single medial or lateral incision is used to fasciotomize the upper arm, proximal extension should be considered to release the deltoid.

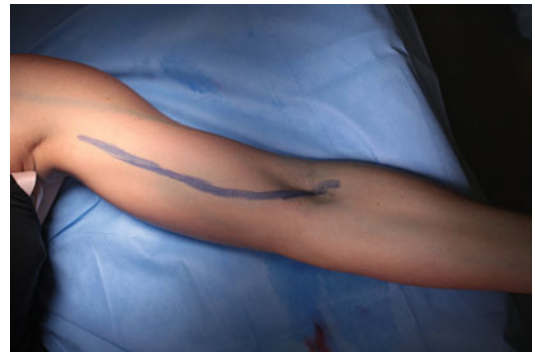

Fig. 10.17 Arm fasciotomy: the anterior and posterior compartments of the arm can also be released using an incision placed anteromedial to the biceps muscle. The incision is shown in *blue*. Exploration of the vascular structures can also be performed using this approach, and it can be combined with the volar incision to decompress the forearm when needed

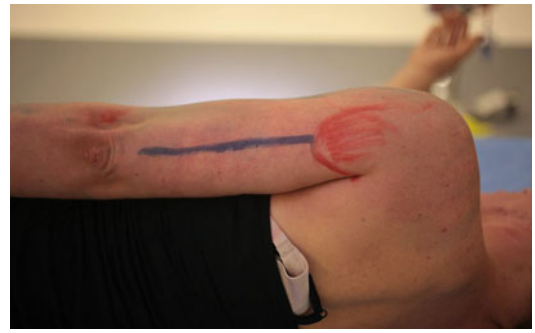

Fig. 10.18 Arm fasciotomy, posterior compartment: a straight posterior incision that can be used to release the posterior compartment of the arm is outlined in *blue*. The more proximal deltoid muscle is outlined in *red*

Post Fasciotomy Care

Fasciotomy wounds should never be closed primarily because of the risk of re-elevating compartment pressures [72]. Following decompression, a sterile dressing is applied, and the wound can be explored 48 h later to assess muscle viability. Delayed primary closure may be performed if the swelling and soft tissues are amenable; otherwise split-thickness skin grafts

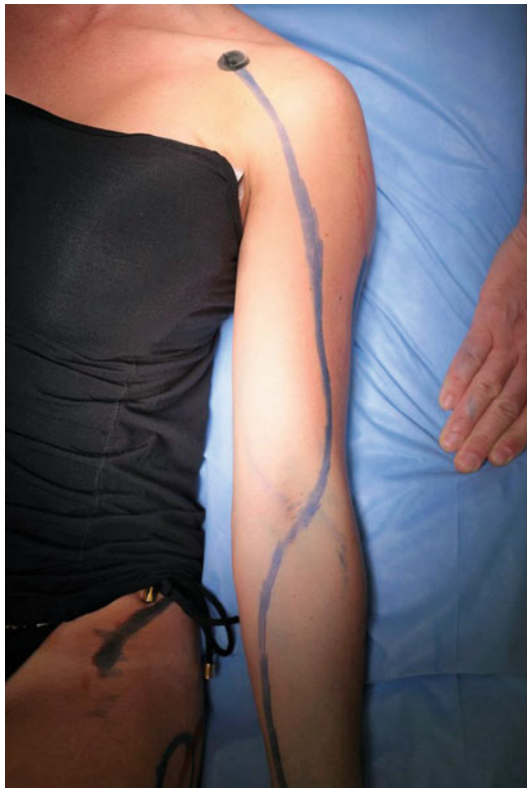

Fig. 10.19 Deltoid fasciotomy: a deltopectoral approach can be used to release the deltoid compartment and is outlined in *blue*. The coracoid, which is located at the most proximal end of the incision, is outlined in *black*. This incision can be connected with an anterolateral incision to decompress the arm, as well as a volar incision to decompress the forearm, when needed

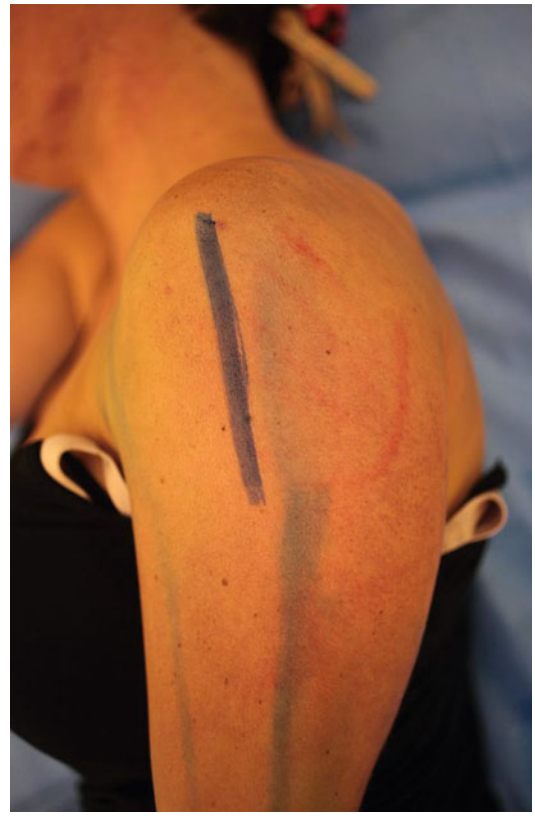

Fig. 10.20 Deltoid fasciotomy: alternatively, the deltoid compartments can be released using a straight incision located over the middle head of the deltoid that is outlined in *blue*

are used to cover the wound(s). All necrotic muscle should be debrided prior to definitive closure. Negative pressure dressings are often used to cover fasciotomy sites and serve several functions. This is a sealed dressing that is placed in the operating room and may prevent secondary contamination on the floor/ICU. By stimulating the formation of granulation tissue, it enhances the take of split-thickness skin grafts, and by potentially decreasing muscle, edema may allow some wounds to be closed and/or decrease the size of the skin graft that is needed [73]. "Roman sandal" sutures may be used by stapling elastic vessel loops to the skin edges. The purpose of this technique is to prevent the skin wound edges from retracting further; caution must be exercised as over tightening has been associated with edge necrosis as well as elevation of compartment pressures [74].

Complications

Timely recognition and emergent treatment are the most important factors to prevent complications and adverse sequelae secondary to CS. Delays of more than 6 h have been shown to increase the incidence of muscle contractures, weakness, sensory loss, infection, and nonunion of fractures [18, 29, 39–42, 75]. In severe cases amputation may be necessary because of infection or lack of function secondary to delayed treatment [76].

Timely recognition that a compartment syndrome has developed followed by an emergent

decompressive fasciotomy is the paradigm for managing a compartment syndrome. A delay in diagnosis and fasciotomy increases morbidity and can fatal consequences due to hyperkalemia or rhabdomyolysis. It is very difficult to determine how late is too late to perform fasciotomy after a compartment syndrome has been diagnosed. If the patient has no demonstrable ability to contract the involved muscles or if the delay has been more than 8 h (which is almost impossible to determine), then decompression should not be performed and the limb splinted in a functional position, allowing the muscles to scar and fibrose. Reis et al. noted increased rates of sepsis and associated complications with fasciotomy following long delays [77].

Ernst felt that delayed decompression exposes already necrotic and nonviable tissue to bacterial contamination and increases the risk of infection [78]. Finklestein reported on 5 patients who underwent delayed fasciotomies after more than 35 h: 1 patient died of multiorgan failure and septicemia. The remaining 4 patients required lower limb amputation because of infection and septicemia [76]. Because a delayed fasciotomy converts a closed injury into an open one and does not reverse the nerve and muscle damage that has already occurred, many authors recommend foregoing decompression when a compartment syndrome has been present for more than >8 h. It can be very difficult however to pinpoint the exact time that a compartment syndrome developed. Supportive care should be given to prevent renal dysfunction secondary to myoglobinuria [76]. By comparing 22 patients treated with fasciotomy <12 h to 22 patients decompressed >12 h, Sheridan and Matsen noted that 68 % of patients in the early decompression group had normal function compared to 8 % in the delayed group [79].

Outcomes After Fasciotomy

There is limited data on the sequelae of fasciotomy wounds. Fitzgerald et al. described the long-term sequelae of upper and lower extremity fasciotomy wounds in 60 patients followed for an average of 59 months. 95 % of the patients had persistent altered sensation and/or paresthesias that were restricted to the margins of the fasciotomy wound in 77 % of the cases. Patients who were skin grafted had more marked symptoms when compared to those who had a delayed primary closure. Over half the cohort had continued pain, 7 % had tethered tendons, and 13 % suffered from recurrent ulcerations within the wound closure area. Twelve percent of the patients changed their occupation as a result of problems with their fasciotomy incisions [80]. These findings parallel those reported by Rorabeck: 22 % of patients in his cohort had "unacceptable" outcomes with persistent neurological sequelae requiring secondary reconstructive surgery or amputation (6 %) [81].

One issue with the limited information that is available on the outcome after fasciotomy is that aside from local wound problems it is unclear if these adverse outcomes are related to this injury itself or to the treatment of the injury. Not performing a fasciotomy in a timely fashion may result in loss of function of a limb—that is clear.

Legal Implications of Compartment Syndrome

Diagnosing an acute compartment syndrome can be difficult. A delay in the diagnosis and/or treatment or missing the diagnosis can result in contractures, limb paralysis, amputation, infection, and death. This morbidity and mortality has medicolegal implications. Orthopaedic surgery ranks in the top five specialties for number of claims filed and amount of financial payments [82].

The economic burden resulting from missed compartment syndromes is significant with an average indemnity payment of over $224,000 in one series and $426,000 in another [83, 84]. Data from the Canadian Orthopaedic Association showed that 35 out of 64 (54 %) litigated cases were settled or judged in favor of the plaintiff [85]. Bhattacharyya and Vrahas noted that an early fasciotomy (within 8 h) improved

patient outcomes and decreased indemnity risk [84]. Common clinical issues related to delayed diagnosis leading to increased indemnity payments include poor physician-patient communication, delayed decompression or surgical fracture stabilization, inadequate documentation or physical examination, and increased number of cardinal signs (5 Ps) [83–85].

In a medicolegal era of increased malpractice premiums and litigations, orthopaedic surgeons must remain diligent in the assessment and timely treatment of compartment syndrome. According to the American Academy of Orthopaedic Surgeons, 80 % of practicing orthopaedic surgeons have had at least one malpractice claim [86]. Appropriate examination, documentation, and treatment, as well as surgeon-patient communication, are of prime importance in decreasing the risk of malpractice claims and indemnity payments.

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Closed Fractures and Dislocations

11

Colin D. Booth, Bruce H. Ziran, and Luis Lozada Jr.

Introduction

Trauma patients present with a combination of soft-tissue and boney injuries. The bone injuries have a wide range of presentation from closed to open, simple to comminuted. Despite the specific presentation, the common goal for fractures and dislocations is reduction and stabilization. Reduction and stabilization need to be achieved relatively quickly, especially in the polytrauma patient, to help decreases the release of proinflammatory cytokines, relieve stress on surrounding neurovascular structures as well as soft tissue, and aide in slowing hemorrhage from fractured bone. Until final fixation can be achieved, invasive and noninvasive techniques are employed to stabilize the fractures in a manner that the patient's hemodynamic status is not compromised further. One particular treatment approach that has been developed is called Damage Control Orthopaedics (DCO), which takes into account the patient's status and the need for stabilization.

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Integrating into Trauma Care Protocols and Damage Control Orthopaedics

Providing proper orthopaedic care to trauma patients must be done in a manner that is in line with current trauma protocols so that no further damage is done to the patient. When it comes to trauma patients, stable or unstable, constant reassessment is needed especially those with multiple fractures. Such patients need constant monitoring of lung function, temperature, fluid requirement, and absence of coagulopathy [1]. Doing so allows an adaptive treatment plan and timing of definitive fixation to exist that can adjust to the patient's current condition. If operating room time is needed to achieve initial or definitive fixation, it is a general rule that operating room time should be 2 h or less to avoid worsening the patient's physiological status [1]. Additionally, if the patient is stable, definitive treatment of fractures should be achieved within the first 24 h of injury. While these rules are commonplace today, it was not always as such and the need for Damage Control Orthopaedics developed.

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The timing of treating orthopaedic injuries has shifted over the years. Until recently, appropriate management of polytrauma patients was fixation of all fractures as soon as possible despite physiological distress [2]. The rationale behind immediate fixation stemmed from the thought that stabilization of long bones and subsequent soft tissue would decrease the inflammatory load and help the patient to be positioned upright for adequate pulmonary toilet. Under this reasoning, all fractures, regardless of patient status, were fixed within 24 h [2]. While the modern approach to fixation continues to evolve, it is important to understand how fixation philosophy changed over the last 50 years. During the 1960s patients with long-bone fractures and numerous traumatic injuries were developing fat embolism syndrome (FES) and other pulmonary issues [1]. So, fixation was delayed for up to 10–14 days using splints, casts, or traction, until symptoms of fat embolism syndrome resolved and pulmonary, cardiovascular, neurological, and coagulation profiles stabilized.

Such delays in fixation did not come without problems. The main issues encountered were pneumonia, decubitus ulcers, vascular abnormalities, psychological disturbances, and GI stasis [1]. A study in the mid-1980s showed that delayed fixation was associated with a longer intensive care unit stay and more incidences of fever and leukocytosis [3]. Furthermore, deferred stabilization and fixation prevented the start of physical therapy, which translated into major joint stiffness [1]. As general trauma knowledge and resuscitation improved, and R Adams Cowley demonstrated the effects of ongoing shock, patients were surviving and more amenable to orthopaedic fixation strategies. As a result, more patients were receiving better fluid resuscitation and early fixation. While an improvement over traction, a new issue arose; it was the increased incidence of acute respiratory distress syndrome (ARDS). The paucity of fat embolism syndrome in the current era may be due to the effects of resuscitation, wherein FES is a pulmonary manifestation of an under-resuscitated state without orthopaedic fixation, and ARDS is the pulmonary manifestation of an over-resuscitated state with acute orthopaedic fixation.

One of the first studies evaluating orthopaedic fixation timing was that of Johnson et al. They found that acute fixation benefited trauma patients and that the more severely injured patients saw the greatest benefit. While a retrospective study with several flaws, it heralded the era of Early Total Care (ETC). Another study by Bone et al. analyzed the timing between fixation of long-bone fractures and the development of ARDS [4]. They determined that patients with traction and late femoral fixation had the highest incidence of ARDS. These studies encouraged early fixation, which subsequently lowered average time in traction from 9 to 2 days [1]. Despite these studies' findings, problems with secondary and remote organ injury still arose. It was probably that the idea of early fixation was interpreted too literally, and aggressive protocols of fixation, without consideration to other physiological variables, led to increased number of complications. The rise in complications led surgeons to further investigate the timing of fracture fixation and in the end culminated in the idea of Damage Control Orthopaedics (see Fig. 11.1).

Damage control was a term originally coined by the United States navy to describe tactics needed to keep compromised vessels afloat. Originally adopted by Rontondo et al., "damage control surgery" was a method of treatment that used rapid but non-definitive control of hemorrhage to avoid the lethal triad of acidosis, hypothermia, and coagulopathy in patients exsanguinating from penetrating abdominal wounds [5]. This idea was later adapted to fit orthopaedic protocols of fixation. Damage Control Orthopaedics is an adaptive treatment strategy defined as the provisional stabilization of musculoskeletal injuries in order to allow the patient's overall physiology to improve to tolerate longer more taxing procedures required for definitive fixation [2]. Under this definition, the primary tools for early stabilization were splinting, traction, and external fixation. With use of DCO, orthopaedic intervention in the acute injury phase is limited to temporary stabilization of structures that could contribute to physiological compromise. Too early an intervention could potentially add or trigger greater physiological injury, called the "2nd hit."

The "2nd" hit concept describes the additive orthopaedic impact after the "1st hit" which is the initial trauma. The 1st hit can activate inflammatory

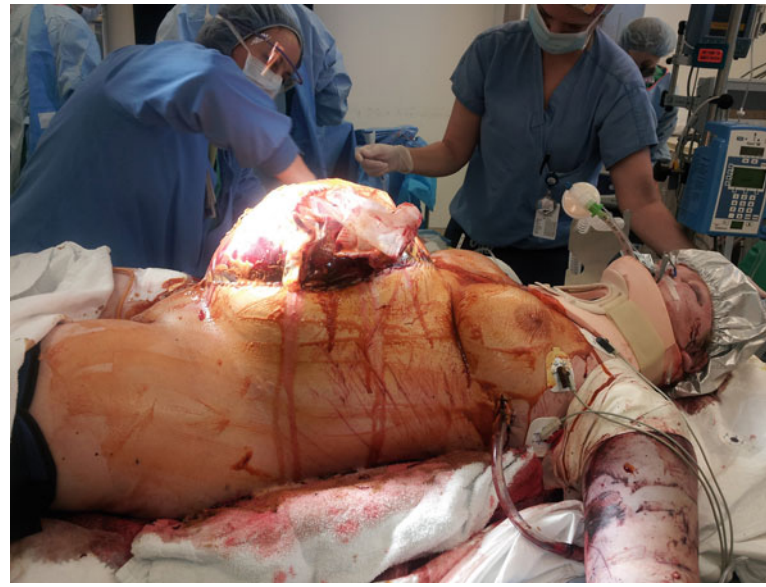

Fig. 11.1 Trauma patient. The figure depicts a trauma patient with multisystem pathology. Such patients can have numerous orthopaedic injuries and management of these injuries needs to be in accordance with damage control orthopaedic protocols

mediators in some patients (interleukins) resulting in a hyperreactive physiology. The subsequent "2nd hit" can trigger an even greater response that can result in further end-organ injury and patient demise. Too early an orthopaedic intervention can be the cause of the 2nd hit via substantial blood loss and/or further soft-tissue damage. The end result may be hypoperfusion, hypoxia/ischemia, reperfusion, and tissue damage causing local necrosis, inflammation, and acidosis [2]. This inflammatory response to trauma, whether it be the "first" or "second" hit, that causes release of proinflammatory cytokines, proteins, and hormones was probably the cause of the major complications seen with aggressive, early fixation like ARDS and multiorgan failure [2]. DCO arose with a purpose of avoiding worsening of physiological parameters by delaying definitive fixation until the patient's physiology permitted.

The decision to employ DCO can be decided upon using genetic and numerous biochemical markers, but these analyses are not available in a timely manner at most facilities so physiological signs can be used to guide decision-making [2]. Some particular physiological markers were defined by Pape et al. to be serum lactate greater than 2.5 (mmol/L), base excess of more than 8 (mmol/L), a pH of less than 7.24, and temperature less than 35 °C, surgical time more than 90 min, any coagulopathy, and transfusion of more than 10 units of packed red blood cells [6]. Some fractures should still be approached with caution even if all physiological parameters indicate lower risks for second hit phenomenon. Recognizing that the above parameters are proxies for complex interactions, there are undoubtedly physiological or genetic predispositions that remain unrecognized that could still result in complications in the absence of overtly abnormal markers. From an orthopaedic standpoint, femur fractures in polytrauma patients, pelvic ring injuries with substantial hemorrhage, and geriatric polytrauma patients are those at risk [2]. While definitive treatment will be covered in different chapters, the decision to undergo definitive surgery can be done when the criteria in Table 11.1 or when fluid balance is negative [2]. For this chapter, however, we will focus on noninvasive and invasive methods to initially stabilize fractures in trauma patients.

Assessment

Initial presentation of the trauma patient needs to be handled in a systematic way to ensure proper assessment and workup usually according to BLS/ACLS/ATLS protocols [7]. Regardless of the protocol chosen, the ABCs (airway,

Table 11.1 Parameters to consider when deciding to implement damage control orthopaedic protocol

Polytrauma with Injury Severity Score of >20 points with additional thoracic trauma (Abbreviated Injury Scale score of >2 points)
Polytrauma with abdominal and pelvic injuries and hemorrhagic shock (systolic blood pressure <90 mmHg)
Injury Severity Score of ≥40 points without additional thoracic injury
Initial pulmonary artery pressure of >24 mmHg
Increased pulmonary artery pressure of >6 mmHg during intramedullary nailing
Difficult resuscitation
Platelet count <90,000/μ[micro]L (<90 × 10^9/L)
Hypothermia (e.g., temperature of <35 °C)
Transfusion of >10 units of blood
Bilateral lung contusion on initial chest radiograph
Multiple long-bone fractures and truncal injury
Prolonged duration of anticipated surgery (>90 min)

Table outlines the criteria as defined by Schmidt et al. for the appropriate time to employ damage control orthopaedic protocols [1]

breathing, and circulation) need to be assessed first to ensure all vital systems are competent. Next, the rest of the patient is assessed with higher priority ailments receiving the most attention. The general rule in trauma is life over limb, but at times extremity injuries can be life threatening.

From the orthopaedic standpoint, a proper examination of the extremities includes inspecting for open wounds or soft-tissue defects and palpation for point tenderness or crepitus over all boney structures. Next, one should range all major joints paying particular attention to decreased or painful range of motion, indication of possible dislocation, with additional investigation for laxity or instability hinting at ligamentous injury. Pulses and neurological status need to be assessed to determine if any vascular or nerve damage is present in the limb. If such a deficit is found, need for further investigation is indicated and urgent. Such exams like ankle-brachial index or computed tomography angiography are used rule out vascular injury/compromise. Presence of neurological deficits, while usually not critical, can hint at a larger problem such as central neurological compromise or severe peripheral nerve damage that could affect future functioning of the limb. In patients with altered consciousness, only signs of grimacing or withdrawal may be the only indicators of injury. In obtunded or intubated patients, the clinical exam requires more attention as it is rather common to miss orthopaedic injuries in these patients.

Each limb needs to be assessed for compartment syndrome due to the devastating effects of a missed diagnosis that can ultimately end in loss of function or even amputation of a limb. Compartment syndrome is also a leading cause of litigation in orthopaedics. It is usually not what is done, but rather, what is not done that becomes the focus of litigation in such cases. Providers need to recognize that the presence of an open fracture does not exclude a compartment syndrome [8] since the small opening of the skin and fascia do not usually result in an adequate decompression, and there are other compartments remote from the open injury that can be involved. All details of compartment syndrome are discussed in more detail in Chap. 10.

Radiography

Once the full assessment is completed and fluid resuscitation according to ATLS has begun [8], x-rays of areas that are suspicious for fracture need to be evaluated radiographically. The basic tenets of appropriate radiographic evaluation are to obtain images of joints proximal and distal to observed fractures. This rule is important because forces in any given bone are by definition transmitted to the joint "above and below" and can result in remote injury. For example, axial loads from the foot can result in injuries to the hip or knee. The physics of load transfer helps explain a special situation that exists in the forearm and lower leg. The presence of the interosseous membrane in the forearm and lower leg, where it contributes to a structure called the syndesmosis, acts as a structure distributing forces around the region causing associated fractures and dislocations [9, 10] (see Fig. 11.2). This explains why severe external rotation about the ankle can cause

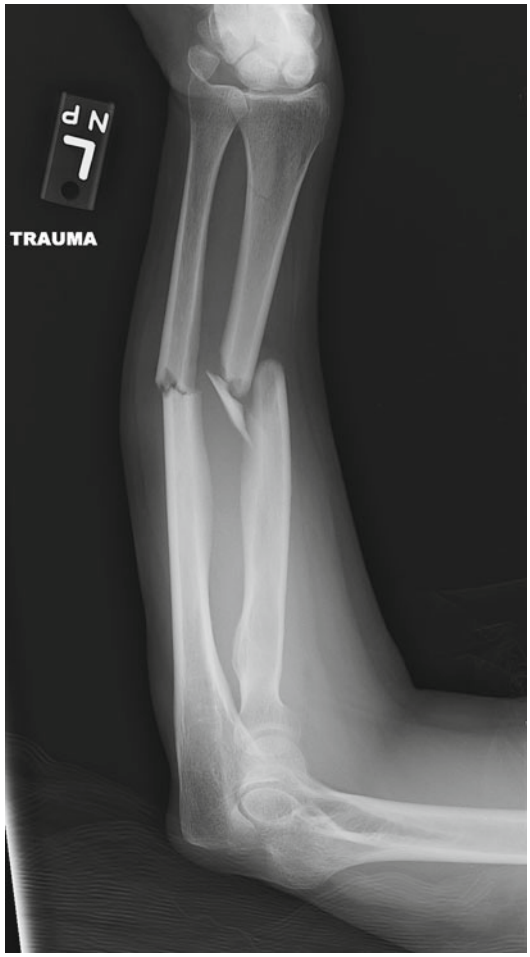

Fig. 11.2 Both-bone forearm fracture. An AP x-ray showing a both-bone forearm fracture with comminution of the radial shaft fracture

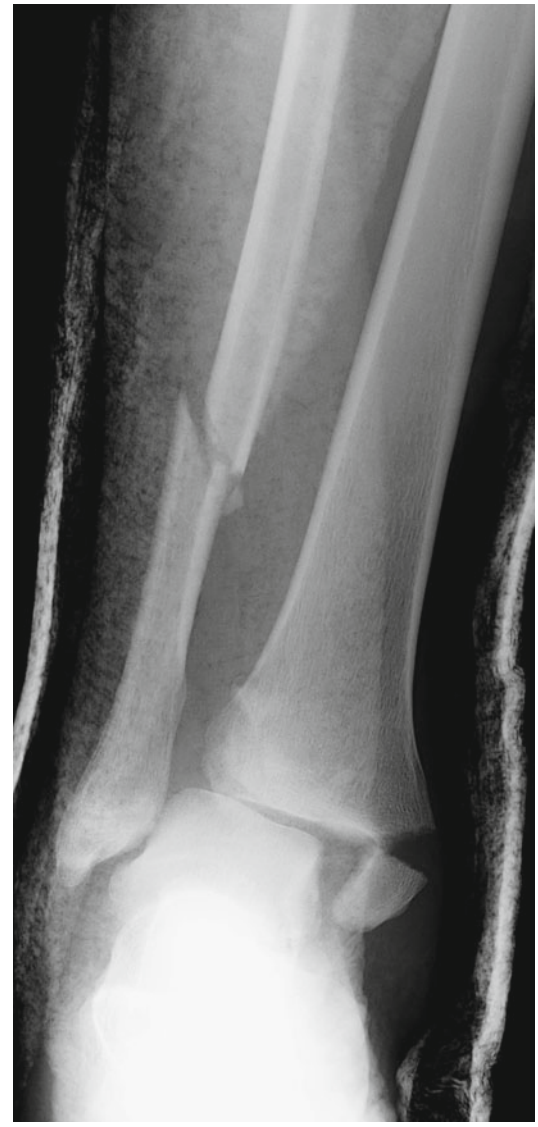

Fig. 11.3 Ankle fracture with associated syndesmosis injury. The figure shows a distal fibular fracture along with a medial malleolar fracture. A syndesmosis injury can also be noted secondary to the increased space between the fibula and tibia where overlap between the bones is usually expected

fractures in the proximal lower leg about the knee or why a fracture of the radial or ulnar shafts can cause a secondary dislocation or fracture of the other bone (see Fig. 11.3).

If pelvic fractures are suspected from the initial screening, AP pelvis film, Judet films, and inlet and outlet views should be taken. An AP film is a good screening tool that can direct the need for different sets of films (see Fig. 11.4). Judet films allow for the evaluation of posterior and anterior columns as well as the posterior and anterior acetabular walls. Inlet views of the pelvis allow for proper evaluation of any anterior/posterior displacement of the sacroiliac joint sacrum or iliac wing as well as determining rotation deformities of the ilium and sacral impaction injuries. Outlet views allow for evaluation of vertical displacement of the hemipelvis (see Fig. 11.5). However, with the implementation of new technology, CT scanning is generally the most informative and thus definitive radiographic technique. Newer CT radiographs may supplant

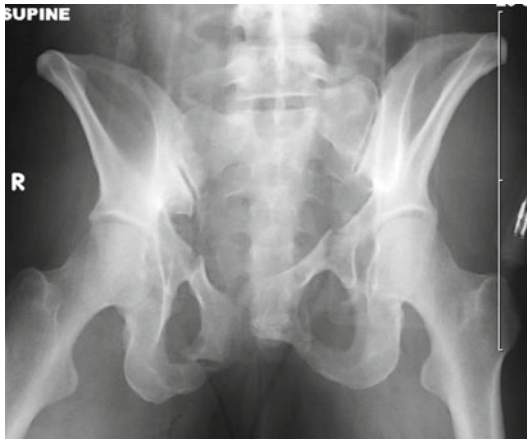

Fig. 11.4 Bilateral inferior/superior rami fractures. An AP film of the pelvis depicting a bilateral superior and inferior pubic rami fractures in a trauma patient (Photo courtesy of Evan Siegall, MD)

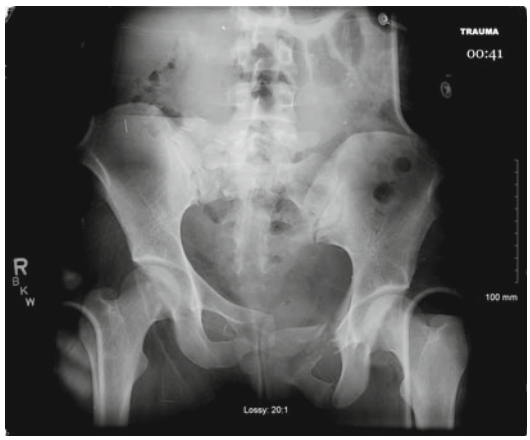

Fig. 11.5 Vertical sheer pelvis. This is an AP x-ray of the pelvis showing displacement of the left hemipelvis secondary to severe trauma. Such injuries are called vertical sheer injuries and are indicative of high-energy trauma

the use of common x-rays, especially when done in a suboptimal manner.

Noninvasive Techniques of Treatment

Closed fractures and dislocations can be dealt with numerous ways in trauma patients; the least traumatic techniques are noninvasive ones, which are reduction, traction, and splinting. When diagnosed

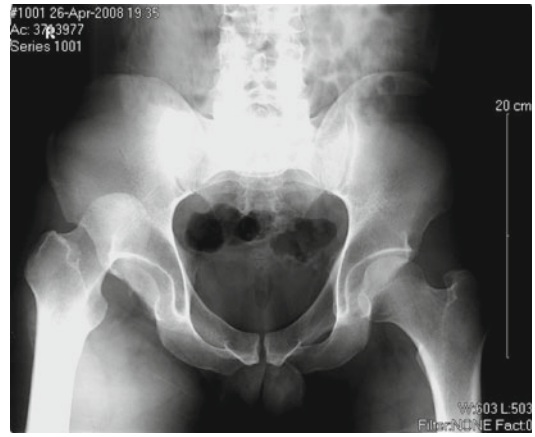

Fig. 11.6 Dislocated hip. The AP pelvic film shows a traumatic, posterior dislocation of the left femoral head. A left posterior acetabular wall fracture should be suspected and needs to be investigated with a pelvic CT scan or Judet views of the pelvis. Pubic diastasis should also be noted

clinically and confirmed radiographically, all dislocations need to be reduced as soon as possible. The need for immediate joint reduction is owed to the fact that joint dislocations can stretch/compromise nearby neurovascular structures. Neurological or vascular deficits may result as a result of prolonged dislocation that either compresses or stretches such structures. Specific examples of this can be seen in patients with dislocated hips. These patients are at an increased risk of osteonecrosis of the femoral head secondary to the hip capsule being stretched or torn which contains the vessels that supply the femoral head. Sciatic nerve injury can also occur the longer the femoral head stays dislocated and compressing the sciatic nerve (see Fig. 11.6). The effects of nerve damage secondary to dislocation are also seen in the shoulder where injury to the axillary nerve or brachial plexus can occur (see Fig. 11.7).

There are many techniques to reduce joints but all require some form of mild sedation to allow the patient and muscle spasms to relax for an adequate reduction. Popular choices for sedation are etomidate, propofol, ketamine, and versed to name a few. The choice of sedation will depend on specific protocols at each individual's hospital. Once reduced, neurovascular status distal to the joint, especially pulses, needs to be

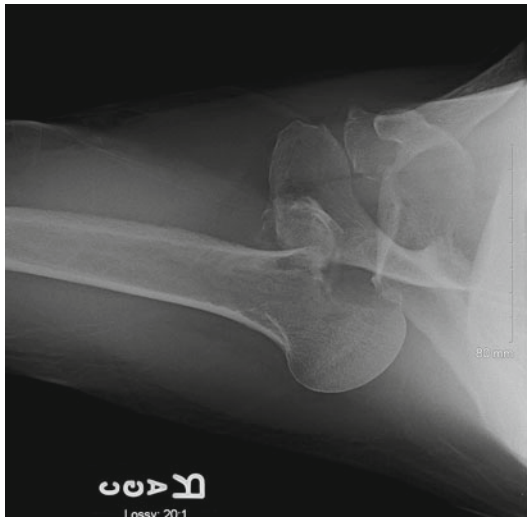

Fig. 11.7 Dislocated shoulder. This patient has an anterior dislocation of the right shoulder with associated proximal humerus fractures. Axillary views are needed to confirm what type of dislocation is present

Fig. 11.8 Elbow external fixator. This patient's right elbow fracture is being managed with an external fixator. The fixators can be hinged or fixed. Hinged fixators allow the patient to range the elbow and decrease chance of stiffness after the fractures have healed

reassessed, and the joint needs to be ranged to determine the arc of motion where stability exists. If the joint remains unstable after ranging, it should be immobilized for a short period of time and then reassessed for range of motion. If stable, passive range of motion and strengthening should be started. However, if the joint still remains unstable, further intervention for short-term stability in preparation for long-term reconstruction is needed.

Closed fractures need to be assessed and stabilized in a timely manner according to the patient's physiological tolerance. One of the main methods for noninvasive stabilization is splinting. The benefits of splinting are no need for OR time, and it is an effective choice for upper extremity or lower-energy fractures [11]. Disadvantages, however, are that soft tissues are not easily evaluated while the splint is in place and re-splinting to evaluate the soft tissues reintroduces fracture instability. Also, splinting inhibits the patient from mobilizing uninjured joints, which can increase long-term stiffness and pain.

Proper splinting technique involves immobilization of the fracture as well as the joint above and below to remove any possible lever arm that would increase motion around the fracture site. Sir John Charnley was one of the first orthopaedist to describe the physics of proper reduction and splinting techniques [12]. Charnley realized that the displacement of fracture pieces has much more to do with the surrounding soft tissues and the soft-tissue attachments that still exist on those fracture pieces. The understanding of these attachments allows one to reduce the fracture as close to anatomically possible as allowed by the fracture pattern.

Upper extremity fractures are usually best initially stabilized with splinting. Wrist and forearm fractures are placed in a sugar tong splint, which is a splint that covers the volar and dorsal aspect of the forearm wrapping around the elbow and extending to the metacarpophalangeal (MCP) joint of the hand. Elbow and distal humerus fractures are splinted with the elbow in flexion, as needed for soft-tissue condition, with the splint spanning from mid-arm to mid-forearm on the dorsal aspect of the arm. Sidebars should be added to the medial and lateral aspect of the splint around the elbow to provide more stability. If not possible, an alternative can be an external fixator (with or without a hinge) (see Fig. 11.8).

Mid-humerus fractures should be splinted in a coaptation splint that spans from the axilla around the elbow and to the neck. Proximal humerus and shoulder fractures can be placed in a shoulder immobilizer. Clavicle fractures are not technically splinted but best treated with a shoulder sling or clavicle brace, mostly for comfort. Metacarpal fractures should be splinted in the intrinsic plus position which involves a splint on the dorsal aspect of the hand extending from midforearm to the distal phalanx with 90° flexion at the MCP joint and about 30–45° of extension at the wrist. Finally, finger fractures can be either buddy taped or placed in a "frog" splint.

Lower extremity fractures can also be splinted but this usually does not apply to hip or femur fractures. Ankle and distal tibia fractures are commonly splinted with a posterior slab extending from the toes on the plantar aspect of the foot to proximal tibia on the posterior aspect of the leg. A side aspect, which wraps around the plantar aspect of the foot and stabilizes the medial and lateral aspect of the lower leg, should always be added to the splint. This type of splint is commonly referred to as a "Cadillac" or "posterior sugar tong" splint. For foot fractures a simple hard sole shoe will be sufficient. Tibia plateau fractures, proximal tibia fractures, and distal femur fractures can be splinted in a knee immobilizer as long as the immobilizer has sufficient enough size to decrease motion around the fracture site. A long posterior splint running the length of the plantar aspect of the foot to the upper posterior thigh can also be used but is usually opted against due to its bulky nature and inability to provide sufficient medial/lateral stability. Calcaneus fractures do not receive special splints, but rather very padded dressings called a Bulky Jones. Here, multiple layers of splint padding are wrapped around the foot and heel to create a large, bulky pad to protect the calcaneus. Unless surgical fixation is indicated, this is usually all a calcaneus fracture needs. Finally, when mechanical thromboembolic prophylaxis is required with lower extremity splinting, a foot pump or calf pumps placed inside the plaster splint can be used.

Besides deciding what type of splint is needed for a given fracture, the splint needs to be applied with the proper technique. Charnley viewed fractures in three different categories: fractures without stability against shortening, fractures with complete stability against shortening, and fractures with potential stability against shortening. The first category comprises fractures of a spiral or oblique nature and highly comminuted fractures. These type fractures require traction, which will be discussed below, to prevent shortening. The second category refers to transverse fractures. Once the bone is manipulated to a satisfactory degree of end-on-end contact, a splint is just needed to preserve shape until the bone is healed. It should be noted, however, transverse fractures of the femur are not suitable to this closed stabilization secondary to continual shrinkage of the thigh muscles that will eventually displace the end-on-end reduction.

The last category, blunt oblique fractures whose fracture line is less than 45° from the transverse line, falls into this category and is where proper splinting technique becomes important. To counteract shortening/deforming forces with a plaster splint, Charnley determined that a "three-point" splint was necessary to maintain the reduction. Here, the physician applies pressure to certain areas of the limb around the fracture to essentially overcorrect the deformity. The rationale behind the technique uses the remaining soft-tissue attachments to the proximal and distal fracture segments and places these connections under tension with the overcorrection of the fracture to maintain the limbs' length and reduction.

Charnley was also a big proponent of traction for lower extremity fractures. Traction, as viewed by Charnley, was a type of splinting that counteracted fracture dislocation from continual shortening forces of the existing muscle attachments. The use of traction produces a relative fixation, like splinting, by conferring tension to the surrounding soft tissues. Charnley likens such an action as to observing a chain in tension where the chain acts like a solid unit in tension. Traction is very useful for lower extremity fractures, especially those of the femur and providing stability to hip dislocations with associated unstable posterior acetabular wall fractures. Not only does the traction aid in relative fixation, it also aides in pain relief and tamponading of the bleeding bone. Traction can be

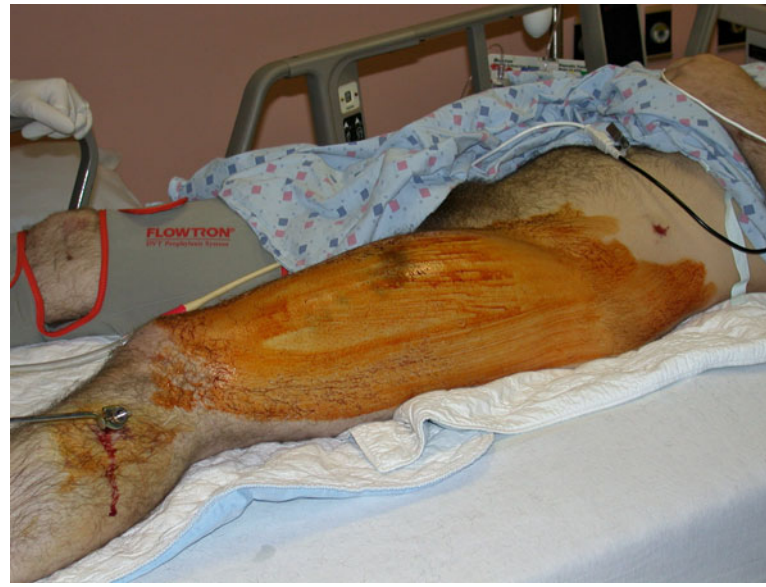

Fig. 11.9 Skeletal traction. This patient has a tibial skeletal traction pin in his left tibia. Weights are attached to the pin to pull traction on the fracture to help stabilize the fracture. Traction pins can also be placed in the distal femur if the tibia is not suitable for traction

done using either a Buck's boot or skeletal traction, which involves drilling a pin through either the distal femur or proximal tibia and attaching the traction apparatus to the pin. Either way, proper traction only requires about 10–15 lb of weight to achieve the desired results (see Fig. 11.9).

While pelvic fractures are discussed in more detail in Chap. 7, we will briefly touch on closed reduction and treatment of pelvic fractures here as well. Pelvic fractures are treated noninvasively with neither splinting nor traction. The most effective method to quickly reduce fractures, close down widening of the pelvic girdle, and tamponade any bleeding that may be present secondary to the fractures is the application of a pelvic binder or sheets wrapped around the patient with the middle of apparatus at the level of the greater trochanters of the femur (see Fig. 11.10). Research has shown that a tension of 180 N is most effective and if done properly can decrease transfusion requirements, length of stay, and mortality [13].

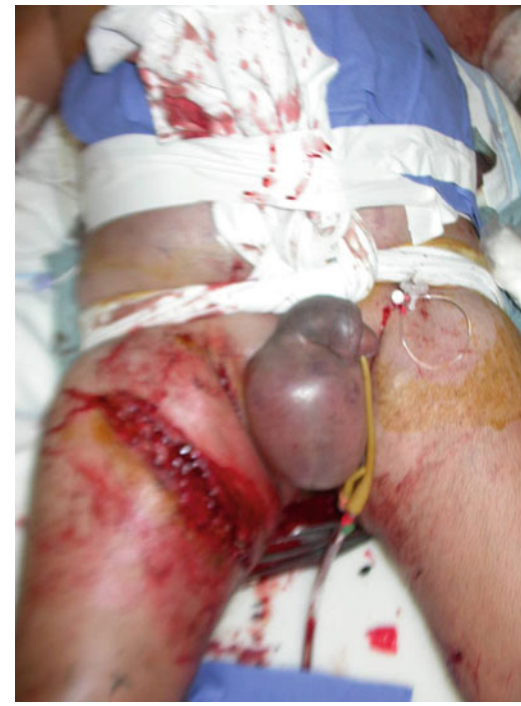

Fig. 11.10 Pelvic binder. The trauma patient above has a "makeshift" pelvic binder composed of tape. Sheets and commercially made binders are more commonly used. Whatever material is used, the binder should be centered over the greater trochanters of the femur and tightened to help close down the pelvic girdle. The applied pressure of the binder increases internal pelvic pressure which can decrease or stop bleeding into the pelvis secondary to fractures

Invasive Techniques

If a fracture cannot be properly or effectively stabilized with splinting or traction, more invasive techniques should be employed. While splinting

is usually enough in the hand or foot, the presence of unstable joint dislocations, significant, unstable fractures, or rotational deformities of the phalanx may require more stable fixation in the form of percutaneous pinning.

The external fixator is quite useful for numerous reasons when it comes to treating trauma patients with fractures. First, applying the external fixator allows the orthopaedic surgeons to limit operating room time to less than 2 h with minimal blood loss, and this is quite important in keeping with the principles of Damage Control Orthopaedics described above [1]. An external fixator acts like a splint that can hold traction allowing for near anatomical reduction without having to use open reduction techniques and subject the patient to long orthopaedic procedures. It is indicated for use in unstable fractures with associated vascular injury, multiple injury patient, segmental bone loss with positionally dependent perfusion, unstable fractures with soft tissue requiring frequent evaluation, closed unstable extremity fracture, and complex periarticular fractures (see Fig. 11.11) [11]. One of the only disadvantages of external fixation is the risk of pin tract infections. However, if proper pin care is administered, this complication can be easily avoided. The key to pin care is the stability of the soft tissues around the pin. Areas like the anterior tibia rarely have problems due to the lack of soft tissues around the pin. In fleshy part, soft tissues move about the pin resulting in irritation, inflammation, and subsequently an exudate. The compromised pin tract is easily colonized and subsequently infected. Proper pin care involves stabilizing the soft tissues around the pin to avoid motion or pistoning that causes the ensuing inflammation. Gauze dressings are wrapped around the pins to create slight pressure onto the underlying skin, such that the skin motion is minimized during motion of the limb. No special cleaning or chemical agents are required and several studies have demonstrated that covering and stabilizing the pins are better than any cleaning regimen.

The main utility of external fixators is in pelvic and femur fractures (see Fig. 11.12). The benefits of external fixating the pelvis include reducing pelvic volume and minimizing the risk of disrupting the initial pelvic clot [13]. The initial hemorrhage into the pelvis is counterintuitively protective. The blood contains clotting factors, and the initial stages of shock serve to reduce the extravasation pressures. If this initial hemorrhage can be stabilized around the injured vasculature, pelvis bleeding can be stopped or slowed. With ongoing shock requiring aggressive resuscitation, the intravascular volume is replaced with clotting factor, depleted fluid, and the pressure head is increased to normotensive levels. If the initial clot in the pelvis is dislodged, the ensuing hemorrhage is with a higher pressure head and with less coagulant factors in the fluid. Theoretically, this will contribute to ongoing extravasation. Thus, the benefit of early external fixation is the potential protection of that initial pelvic clot.

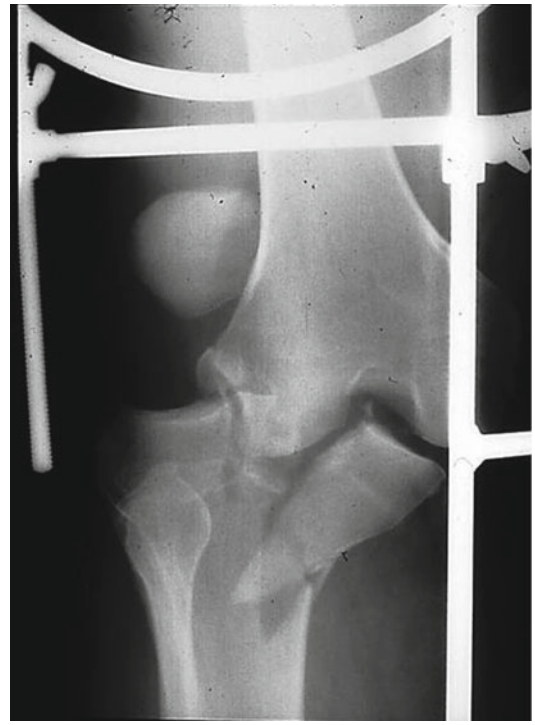

Fig. 11.11 Medial, inter-articular tibial plateau fracture. The x-ray shows a displaced, medial tibial plateau fracture caused by severe axial loading of the leg. The patella and femur appear to be dislocated as well. It should be noted that medial tibial plateau fractures are associated with knee dislocations, compartment syndrome, and vascular injuries. These patients should be examined for such injuries and monitored closely

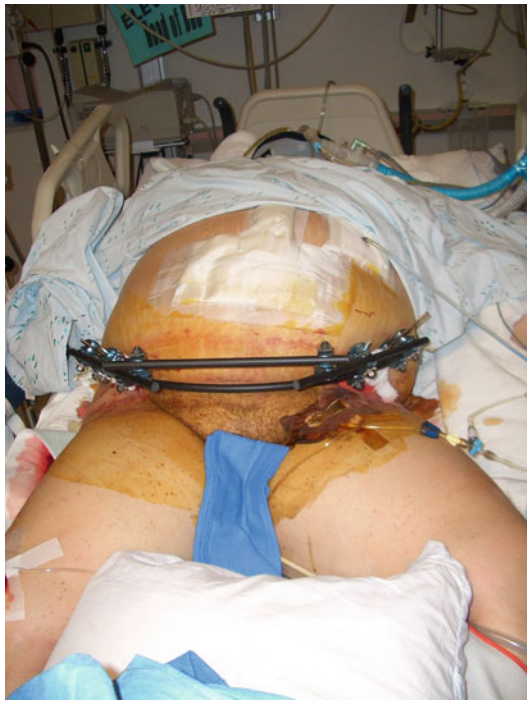

Fig. 11.12 Pelvic external fixator. The patient shown above has a pelvic external fixator. In very unstable patients, a pelvic external fixator is a quick and relatively harmless option for stabilizing severe pelvic fractures

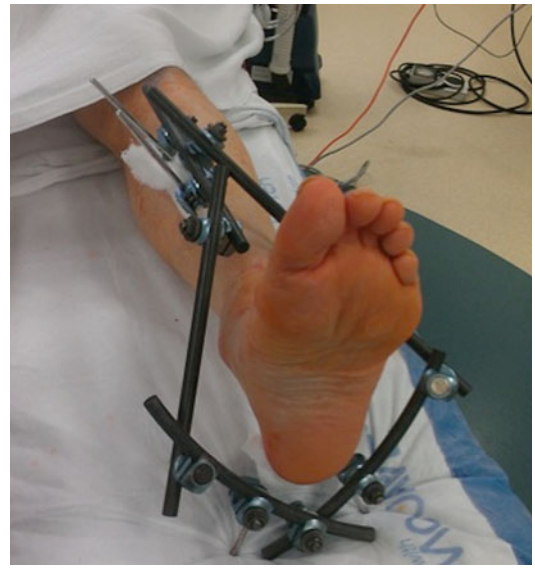

Fig. 11.13 Ankle external fixator. The figure depicts an external fixator used to stabilize an ankle fracture. Technique uses two posterior calcaneus pins and two anterior tibial pins to help elevate the foot off the bed (Technique courtesy of Bruce Ziran, MD. Picture courtesy of Evan Siegall, MD)

For the femur, reducing blood loss, tissue damage, and pain are the main reasons for acute external fixation. Cumbersome skeletal traction during trips to scanners or the operating room is also avoided with use of external fixation, which functions like "portable traction." If ipsilateral lower extremity injury is present, the fixator can be extended all the way to the foot and span ankle and knee injuries. In the more distal parts of the extremity, including the upper extremity, external fixation serves more as damage control for the extremity. It helps stabilize the soft-tissue envelope, provides visualization and access to the soft tissues, and provides comfort.

Applying external fixation is a fairly standard procedure regardless of the area of injury or manufacturer of the product. Proper fixation requires placement of Schanz screws proximal and distal to fracture in stable bone. For pelvic fractures, screw placement is usually not proximal and distal but rather to the left and the right of the fracture and most commonly seen in cases of pubic diastasis. Schanz screws come in a variety of sizes and diameters for the type of bone being used in fixation; smaller diameter screws are used in upper extremity and foot fractures, while larger diameter screws are for pelvic, femur, tibia, and ankle fractures. In general, joint spanning external fixators are avoided if possible so the patient can mobilize all uninjured joints. However, if the fracture needing stabilization involves a joint, such as an elbow, tibial plateau, or ankle fracture, spanning external fixators are needed (see Fig. 11.13).

Screw placement is an important step in using the external fixation. Choosing where to place the screws for the fixator requires detailed knowledge of the neurovascular structures around the area desired for screw placement. In general, there is an arc of varying degrees in which screw placement has a decreased risk of damaging neurovascular structures. For example, in the humerus, the axillary nerve is of main concern and the knowledge that its path from proximal to distal changes from posterior to lateral,

respectively. Another example is the femur where the main concern is the femoral nerve and artery. This neurovascular structure lies medially to the femur, so as long as screw placement does not violate the medial compartment, nerve and artery damage is less likely. In addition to avoiding neurovascular damage, screw placement should not violate a joint capsule which is especially important in placing screws for tibial external fixation. Finally, placing the pins as close to the fracture, proximally and distally, as permitted by increases the stability of the construct. Stability can also be increased by simply placing the external fixator bars close to the skin or by adding more bars to the construct.

Very little dissection is needed once screw placement is determined since screws are placed percutaneously with only small stab incisions needed to gain access to the bone. Fluoroscopy is used to check placement and depth of the screws to ensure proper and stable placement in the bone. After placement, bars connecting the Schanz screws are attached loosely to the screws and the fracture is manipulated under fluoroscopy to try to achieve anatomic alignment. When a satisfactory reduction is achieved, the clamps holding the bars to the Schanz screws are tightened, and the reduction is now held until the clamps are loosened at some point in the future. The limb can then be manipulated without fear of displacing the fracture. For exact techniques about applying external fixators, please refer to the manufacturer's technique guide.

Summary

Closed fractures and dislocations are common in the trauma patient and need to be dealt with in a timely manner. These problems can lead to increased morbidity and even contribute to mortality if not dealt with properly. With thorough examination, assessment, and radiographs, the treatment of closed fractures and dislocations can be planned within trauma protocols. It is important that fractures are stabilized according to the patient's physiological status. Damage control protocols are very effective at dealing with a patient's current status. These protocols are also very useful to help guide the type of orthopaedic fixation with the key being to reduce the patient's inflammatory reaction to fracture. Stabilization can be achieved with simple means such as splinting and/or traction according to principles originally defined by Charnley. This choice of stabilization is quite common and effective for upper extremity fractures and low-energy fractures.

More invasive measures, most specifically external fixation or percutaneous pinning, are sometimes indicated. Percutaneous pinning is useful in the hand or foot for unstable joint dislocations, small, unstable fractures, or fractures that cause significant rotation deformities of the digit. External fixation is a great choice if soft-tissue injury is a major concern or the fracture pattern is highly unstable and/or comminuted. The other added advantage of external fixation or percutaneous pinning is that they can be used as definitive treatment. This is quite common for percutaneous pinning but less common for external fixation where it is only used definitively if the patient is too unstable to tolerate long procedures required for internal fixation or the fracture is significantly comminuted. Whatever method is chosen for treatment of closed fractures and dislocations, it is important to not compromise the patient's physiological status in effort to achieve orthopaedic fixation.

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Part III

Damage Control Strategies in the Presence of Critical Associated Injuries

Shock and Coagulopathy

12

Jeffrey N. Harr, Fredric M. Pieracci, and Ernest E. Moore

Background

The understanding of shock has followed a remarkable course due to early misconceptions of the circulatory system. In the second century A.D., Galen first proposed that arteries carry blood, instead of air, which passes from the right heart to the left through small pores in the interventricular septum [1]. It was nearly 1,500 years later when Vesalius challenged Galen's conclusions and demonstrated that blood did not flow from the right to left ventricle directly [2]. William Harvey ultimately described the circulatory system in detail proposing it was a closed system in which the heart served as the pump for the blood, and in 1740, Stephen Hales was the first to observe the physiological relationship between venous return, stroke volume, and cardiac output [3, 4].

It was not until 1743 that the term "choc" was introduced by the French surgeon Henri François Le Dran, but this term described the causative traumatic event rather than the resulting physiological consequences [5]. In fact, Le Dran proposed blood-letting, a common therapy of the time, as the remedy for shock. As the recognition of shock progressed, so did the definition. John Collins Warren, the founder of the *New England Journal of Medicine and Surgery*, defined shock as "a momentary pause in the act of death" characterized by "imperceptible" or "weak, thread-like" peripheral pulses, and in 1872, Samuel V. Gross contributed a physiological component to the definition as "the manifestation of the rude unhinging of the machinery of life" [6].

Claude Bernard was the first to propose that an organism maintains constancy of the "milieu interne" despite external forces acting to disrupt this equilibrium [7]. However, it was not until the early twentieth century when Walter B. Cannon introduced the term "homeostasis," suggesting that the body attempts to maintain constancy in the internal environment, and failure to do so resulted in shock [8]. From his battlefield observations during World War I, he proposed shock resulted from both a disturbance of the nervous system and a "toxic factor" producing vasodilation and hypotension. Soon after, Alfred Blalock, from his series of sophisticated animal studies, ultimately put the pieces together, demonstrating that shock from hemorrhage was the result of volume loss with decreased cardiac output. After further investigations, he subsequently proposed four categories of shock: cardiogenic, hypovolemic, neurogenic, and vasogenic [9].

Our understanding of the pathophysiology and management of shock continues to evolve, as we now realize that the effects of shock are also linked to systemic inflammatory responses and

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alterations in coagulation. Extensive research in resuscitation and the use of rapid point-of-care assays continue to elucidate the complex pathophysiological responses to shock, especially in the trauma setting.

Shock

Shock results from the body's inability to provide adequate substrate to cells for the maintenance of aerobic metabolism, energy production, and homeostasis. There are multiple etiologies of shock (Table 12.1), but all lead to a final common pathway—impaired oxygen delivery, transport, or utilization. Early intervention is necessary in the management of shock, as prolonged periods of ischemia may lead to irreversible shock despite appropriate treatments. Three phases of shock (compensated, decompensated, and irreversible) have been described based on Wiggers' animal model of graded-hemorrhagic shock [10]. Both compensated and decompensated shock may be treated, but as the term implies, irreversible shock surpasses the threshold for intervention, and death shortly ensues in spite of all therapeutic measures.

Pathophysiology of Shock

The pathophysiology of shock is complex and is ultimately driven by tissue hypoperfusion leading to altered cellular metabolism and activation of the immune system. Prolonged ischemia has many detrimental effects at the cellular level (Table 12.2) but eventually results in a shift to anaerobic metabolism and a failure to synthesize adenosine triphosphate (ATP) for maintaining homeostasis [11]. Although the results of ischemia are detrimental, the restoration of blood flow and reoxygenation intensifies the inflammatory response and exacerbates tissue injury [12]. This phenomenon is commonly known as ischemia/reperfusion injury. Ischemia initially results in impaired endothelial cell barrier function and increased vascular permeability, but it is the reperfusion which leads to the activation of cell death pathways, autophagy-associated cell death, and necrosis [13, 14]. These apoptotic pathways are highly regulated by ischemia-induced transcription of genes, such as NF-κ[kappa]B and HIF [15].

Ischemia/reperfusion injury leading to apoptosis and necrosis is highly immunostimulatory and leads to inflammatory cell infiltration and cytokine production. Release of intracellular products from injured cells such as high-mobility group box 1, heat shock proteins, mitochondrial peptides, heparin sulfate, and RNA has paracrine and endocrine-like effects on distant tissues stimulating inflammatory responses [16]. These molecules that are released have been termed damage-associated molecular patterns (DAMPs), and their effects are physiologically known as danger signaling. DAMPs are recognized by cell

Table 12.1 Categories of shock

Category	Mechanism	Treatment
Hemorrhagic	Loss of circulating blood volume	Intravascular volume replacement Restore O_2 carrying capacity
Traumatic	Loss of circulating blood volume with tissue injury and release of DAMPs	Control of bleeding and intravascular volume replacement
Cardiogenic	Cardiac failure	Improve cardiac perfusion, may require vasopressors/inotropes or devices (IABP/LVAD)
Neurogenic	Brain or spinal injury with disruption of the sympathetic regulation of the cardiovascular system	Intravascular volume replacement and vasopressors
Obstructive	Impaired right ventricular diastolic filling or obstruction of right ventricular output	Early diagnosis and resolution of decreased venous return/ventricular filling
Septic	Decrease in systemic vascular resistance with release of PAMPs	Intravascular volume replacement and vasopressors

Table 12.2 Cellular effects of ischemia

Cellular acidosis
Altered intracellular ion distribution
Altered membrane potential
Cellular swelling
Cytoskeletal disorganization
Increased hypoxanthine
Decreased oxidative phosphorylation and ATP synthesis
Decreased phosphocreatine
Decreased glutathione
Increased nucleotide phosphohydrolysis and adenosine signaling
Increased leukocyte adhesion molecule expression

surface pattern recognition receptors, such as Toll-like receptors (TLRs), which through intracellular signaling amplify the immune response. Remarkably, similar effects are generated from molecules associated with pathogens, such as bacterial lipopolysaccharide, termed pathogen-associated molecular patterns (PAMPs).

Tissue-resident macrophages or mast cells are the first local cellular responders to ischemia releasing eicosanoids, histamines, and cytokines (IL-1, IL-6, MIP, TNF-α[alpha]) and chemokines (IL-8), which further amplify the immune response (Fig. 12.1) [17]. Macrophage activation subsequently leads to neutrophil priming, chemotaxis, leukocyte-endothelial cell adhesion, and transmigration into the ischemic/injured tissue resulting in further release of TNF-α[alpha] and other cytokines [18]. However, tissue-resident macrophages may take several days to achieve their full inflammatory response, and the complement cascade may play a larger role in the initial inflammatory process [19].

The complement cascade is activated via several pathways following severe injury, hemorrhagic shock, or infection and produces anaphylatoxins (C3a and C5a), which further recruit and activate macrophages but are also potent attractants and activators of neutrophils. In both injured patients and patients in hemorrhagic shock, the degree of complement activation is proportional to the magnitude of injury or depth of shock [20–22]. Generally, the complement system is known to be activated by either the classical, alternative, or lectin pathways [23]. Activation of the classical pathway requires the formation of antigen-antibody complexes, which binds C1 and initiates the cascade. Following ischemia/reperfusion, cells can express neoepitopes (β[beta]2-glycoprotein, β[beta]-actin, annexin IV, and non-muscle myosin heavy chain type II) or form microparticles, which are bound by natural antibodies and activate complement [24]. The lectin pathway is activated by mannose residues on bacterial surfaces, which bind the mannose-binding lectin (MBL). This complex subsequently activates the MBL-associated serine proteases (MASP-1 and MASP-2), which act similar to the C1 complex. The alternative pathway may be activated by cell surface factors on yeast or bacteria or may be activated spontaneously. Simply, these three pathways converge to a common pathway with the formation of C3 and C5 convertases, which cleave C3 to form C3a and C3b and C5 to form C5a and C5b (Fig. 12.2) [25]. The binding of C3a and C5a to their corresponding receptors (C3aR, C5aR, and C5L2) on both myeloid and nonmyeloid cells incites proinflammatory signaling which acts as a chemoattractant for neutrophils, activates an oxidative burst and lysosomal enzyme release, stimulates mast cell and basophil degranulation, induces expression of adhesion molecules on endothelial cells, promotes smooth muscle cell contraction, and enhances the acute phase response of the liver [26–28]. C3b acts as an opsonin for both prokaryotic and eukaryotic cells, and C5b initiates the assembly of the membrane attack complex (MAC), composed of C5b–C9, which promotes cellular lysis.

However, two additional complement activation pathways have been recently described—the properdin and thrombin pathways (Fig. 12.2). The properdin pathway is activated after recognition of DAMPs on foreign and apoptotic cells and allows for the assembly of C3 convertase on the cell surface [29]. DAMPs expressed following ischemia or tissue disruption implicate the properdin pathway as an important link between innate immunity and inflammation following shock. The fifth complement activation pathway is through the clotting factor thrombin. Thrombin may directly act as a C5 convertase linking both the complement and coagulation systems [30, 31].

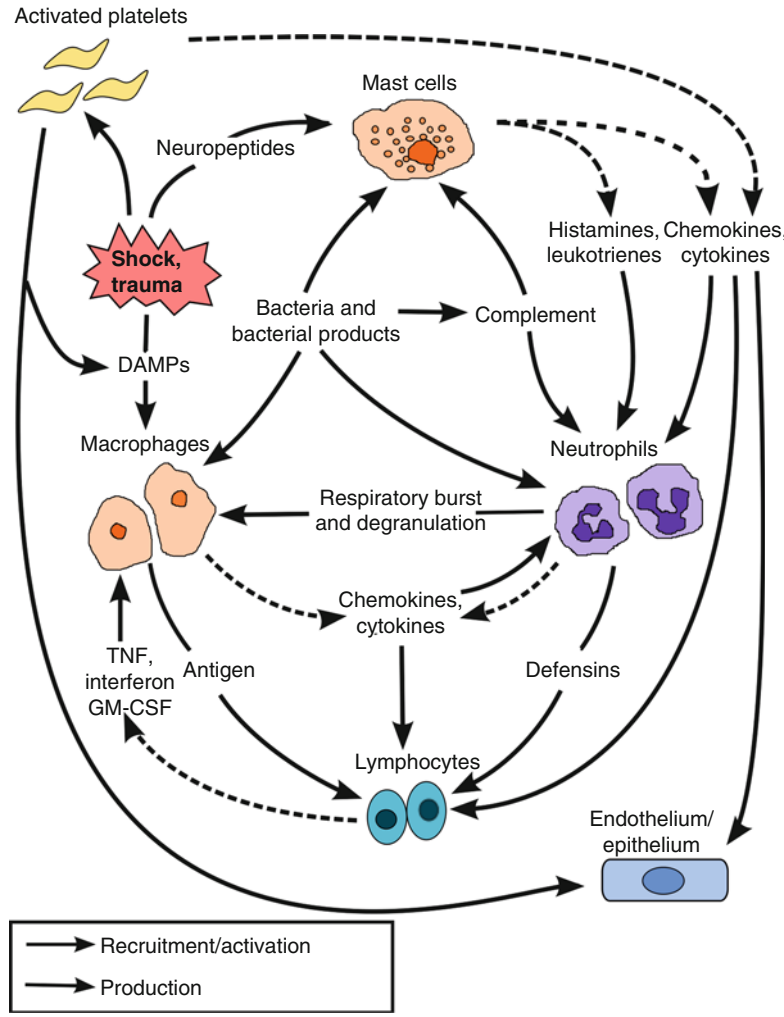


Fig. 12.1 Flow of signaling following trauma and shock resulting in the recruitment and activation of the innate immune system and the amplification of the inflammatory process. *DAMPs* danger-associated molecular patterns, *TNF* tumor necrosis factor, *GM-CSF* granulocyte-macrophage colony-stimulating factor

Neutrophils are the predominant effector cells of the innate immune system, and the ultimate outcome of both the macrophage and complement response to shock is neutrophil recruitment to the ischemic/injured tissue. Moreover, recent data suggest that danger signals associated with ischemic or necrotic cells (nucleotide oligomerization domain receptors—NLRP3 inflammasome activation, intravascular gradient of chemokine C-X-C ligand 2, and signaling through N-formyl peptide receptor-1) guide neutrophils through healthy tissue to sites of sterile inflammation [32]. Neutrophils are highly regulated, effective phagocytes whose main function is to seek out and destroy pathogenic microorganisms. However, these same cellular and biochemical events in response to infection paradoxically contribute to severe inflammation and tissue injury in the setting of ischemia/reperfusion and tissue disruption.

Neutrophils contain four types of intracellular granules: azurophilic (primary), specific (secondary), gelatinase (tertiary) granules, and secretory vesicles that contribute to tissue injury [33]. Granule-associated proteins delivered to the neutrophil surface following activation facilitate cell adhesion and chemotaxis, and others aid in microbial killing or the pathologic response to shock. Neutrophil elastase is one such factor found in the azurophilic granules and is a serine protease which functions as part of the phagolysosome. However, once

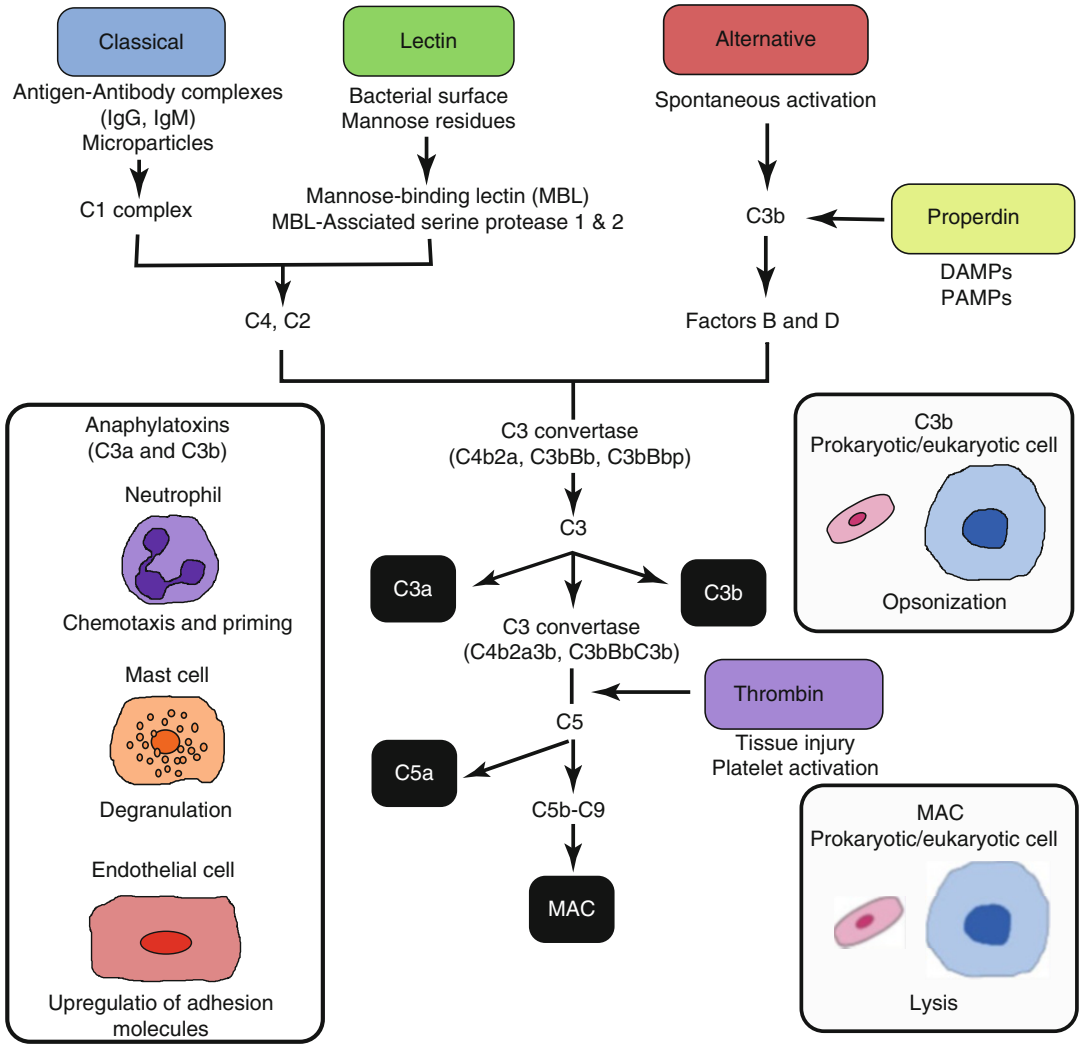


Fig. 12.2 Overview of complement activation pathways and biologic effects mediated by complement pathway products. *MAC* membrane attack complex

released extracellularly following pronounced inflammation, elastase stimulates the release of growth factors and proinflammatory cytokines, formation of reactive oxygen species, and cellular apoptosis [33–35]. Matrix metalloproteinases (MMPs) are zinc-dependent endopeptidases found in both specific and gelatinase granules and influence tissue remodeling, cell migration, and angiogenesis. The release of MMP-2 (gelatinase A) and MMP-9 (gelatinase B) from gelatinase granules contributes to tissue injury [36, 37]. However, MMP-8, which is released from the specific granules, may have anti-inflammatory properties through cleavage of neutrophil-derived cytokines and upregulation of anti-inflammatory cytokines [38]. α[alpha]-Defensins are released from the azurophilic granules and are cationic peptides that can associate with bacterial cell walls, form pores, and disrupt the cell wall integrity. α[alpha]-Defensins also trigger macrophage release of TNF-α[alpha] and interferon-γ[gamma] further amplifying the immune response [39]. Myeloperoxidase, a major constituent of primary granules, and NADPH oxidase are responsible for neutrophil generation and release of

reactive oxygen species [40]. Reactive oxygen species disrupt intercellular tight junctions, increase endothelial permeability, and accelerate apoptosis. [41, 42]

There is ample evidence demonstrating the strong coupling of the effects of shock and inflammation. After an initial insult, neutrophils become activated and localize to the injured/ischemic tissue resulting in more localized inflammation. This response is often tolerated if the injury is limited. However, a second insult (i.e., infection, ischemia/reperfusion, or tissue injury) following trauma and shock may activate systemic primed neutrophils and incite an unbridled inflammatory response resulting in multiple organ failure and mortality [43]. Therefore, it is important to understand the pathophysiology of shock and the maladaptive immune response following severe injury.

Physiological Response to Shock

The pathophysiology of shock is complex, but the physiological response to shock is ultimately driven by tissue hypoperfusion. While this physiological response may differ depending on the etiology of shock, the initial priority is to preserve cerebral and coronary perfusion. The cardiovascular response to differing etiologies of shock is an illustration of this point, manifesting as variations in the systemic vascular resistance, central venous pressure, and cardiac index (Table 12.3).

The body has multiple, complex systems in place that regulate these responses and are sensitive to small decreases in blood pressure and pH and rapidly respond to correct these changes. Stretch receptors in the heart and baroreceptors located in the carotid sinuses and aortic arch sense decreases in blood pressure and respond by a reflexive activation of the sympathetic nervous system. The end result of sympathetic activation is an increase in peripheral vascular resistance and cardiac output through increased vasoconstriction, heart rate, and ventricular contractility [43]. Activation of the sympathetic nervous system further induces the release of catecholamines from the adrenals. The carotid and aortic bodies also detect changes in partial pressures of oxygen and carbon dioxide as well as arterial pH. Central chemoreceptors are also present in the medulla oblongata, which primarily detect changes in pH within the cerebrospinal fluid but have also been shown to respond to hypercapnia and hypoxia. Activation of these receptors results in an increase in respiratory rate as well as peripheral vascular resistance with a corresponding decrease in heart rate, which is known as the Cushing reflex [44]. The renin-angiotensin-aldosterone system also responds to decreases in blood pressure. The kidney's macula densa senses a decrease in blood pressure and signals the juxtaglomerular cells to secrete renin. Plasma renin is then responsible for the conversion of angiotensinogen to angiotensin I in the liver. Subsequently, angiotensin I is primarily converted to angiotensin II in the lungs. Angiotensin II is a potent vasoactive peptide, which increases blood pressure through vasoconstriction and also stimulates the adrenal cortex to secrete aldosterone. The role of aldosterone is to promote sodium reabsorption in the distal tubules and collecting ducts of the nephron, thus expanding intravascular volume and subsequently increasing blood pressure [44].

Other hormones besides aldosterone are also involved in physiological responses to shock. Hypoperfusion activates the hypothalamic-pituitary-adrenal axis stimulating the hypothalamus to release corticotropin-releasing hormone and, subsequently, release of adrenocorticotropic hormone (ACTH) from the anterior pituitary. ACTH stimulates the adrenal cortex to release cortisol, which promotes a catabolic state as well as kidney reabsorption of sodium. The posterior pituitary releases antidiuretic hormone (ADH) in response to both hypovolemia and increased

Table 12.3 Hemodynamic responses to shock

	Cardiac index	SVR	Venous capacitance	CVP/ PCWP	SVO_2
Hemorrhagic	↓	↑	↓	↓	↓
Traumatic	↓	↑	↓	↓	↓
Cardiogenic	↓	↑	↑	↑	↓
Neurogenic	↓	↓	Variable	Variable	↑
Obstructive	↓	↑	↑	↑	↓
Septic	↑	↓	Variable	Variable	↑

plasma osmolality, which increases water permeability in the distal tubule and collecting duct of the kidney, resulting in an increase in intravascular volume. ADH, which was first described as arginine vasopressin, acts a peripheral vasoconstrictor, shunting blood from splanchnic organs to the cerebral and coronary circulation [44].

Categories of Shock

Classically, four categories of shock were described by Blalock based on hemodynamic profiles, which are still useful today: hypovolemic, cardiogenic, distributive, and neurogenic shock [45]. However, clinicians who manage trauma patients prefer to add two additional categories due to their unique pathophysiology: traumatic and obstructive shock (Table 12.1). Treatment of all forms of shock is empiric until the etiology can be determined. Initially, a secure airway, to ensure adequate oxygenation (pO_2) and ventilation (pCO_2), and adequate intravenous access must be established, with the initiation of volume restoration. Subsequently, a systematic physical exam and appropriate diagnostic studies are performed to identify the cause(s) of shock.

Hemorrhagic Shock

In trauma patients, loss of circulating volume from hemorrhage is the most common etiology of shock. Therefore, hypotension in the trauma patient should be presumed to be from hemorrhage until proven otherwise. The physiological response and clinical presentation of hemorrhagic shock have been characterized according to the volume of blood loss (Table 12.4). Blood loss of less than 15 % of the total circulating blood volume results in very few clinical symptoms and may be overlooked. Mild symptoms of tachycardia and anxiety are usually evident in patients with up to 30 % blood loss. However, trauma patients usually do not become hypotensive, confused, or develop significant tachycardia until a blood loss of greater than 30 %. If trauma patients lose greater than 40 % of their circulating blood volume, they are usually obtunded on presentation with severe hypotension and tachycardia and are at significant risk for death. Young patients, with greater physiological reserve and stronger compensatory mechanisms, may tolerate larger volumes of blood loss while exhibiting fewer clinical signs. On the other hand, elderly patients, due to their preexisting diseases or medications to manage comorbidities, may be at a greater risk for bleeding (warfarin, platelet inhibitors, and direct thrombin inhibitors) and inability to compensate for hypovolemia (antihypertensive agents and β[beta]-blockers).

It is important to identify the source of bleeding in patients with hemorrhagic shock following trauma, since controlling the hemorrhage is imperative to resuscitate the patient. The potential sites capable of large volume blood loss include external, intrathoracic, intra-abdominal, retroperitoneal spaces, as well as long bone and pelvic fractures. These can often be rapidly diagnosed based on a focused physical exam besides ultrasound (FAST exam) or plain radiographs. Treatment consists of controlling the blood loss and commencement of intravenous volume resuscitation.

Resuscitation strategies may vary by institution and region but continue to evolve as the pathophysiology of hemorrhagic shock, and how

Table 12.4 Classification of hemorrhagic shock

	Class I	Class II	Class III	Class IV
Blood loss (mL)	Up to 750	750–1,500	1,500–2,000	>2,000
Blood loss (% BV)	Up to 15 %	15–30 %	30–40 %	>40 %
Heart rate (beats/min)	<100	>100	>120	>140
Blood pressure	Normal	Orthostatic	Decreased	Severely decreased
Pulse pressure	Normal	Narrowed	Narrowed	Very narrow or unobtainable
Hourly urine output	>0.5 mL/kg	>0.5 mL/kg	<0.5 mL/kg	Minimal
Mental status	Normal	Anxious	Confused	Obtunded

it relates to inflammation and coagulation, is better understood. There remains much controversy behind resuscitation strategies, and two of these proposed strategies include hypotensive resuscitation and the minimization of dilutional coagulopathy. However, a balance must exist to ensure adequate perfusion in order to minimize the post-resuscitation inflammatory response and further coagulopathy. A recent management strategy in the severely injured trauma patient is a combination of these strategies known as damage control resuscitation [46]. The principal components are (1) to employ "hypotensive resuscitation" to a systolic blood pressure of 80–90 mmHg in order to minimize ongoing hemorrhage and clot disruption, (2) to minimize dilutional coagulopathy by limiting crystalloid transfusion, and (3) to administer preemptive blood components to replace whole-blood loss. While this strategy has been reported to improve survival, the benefits of the individual components remain to be established [47]. However, hypotensive resuscitation should not be employed in traumatic brain injury patients, in which systolic blood pressures should be maintained greater than 110 mmHg or greater than 120 in settings without invasive intracranial monitoring capabilities, in order to sustain cerebral perfusion pressures and minimize secondary insults [48]. Closed head injuries are at increased risk for disturbances in autoregulation and elevated intracranial pressure, thus lowering cerebral perfusion. If the cerebral perfusion pressure falls below 50 mmHg, patients are at high risk for ischemic brain injuries, and therefore cerebral perfusion pressure should be maintained between 50 and 70 mmHg [49]. Attempts to maintain cerebral perfusion pressure greater than 70 mmHg have been associated with an increased risk of ARDS without any improvement in neurological outcomes [50].

The optimal initial resuscitation fluid given to patients in hemorrhagic shock continues to be debated. Crystalloids are the standard of care in the United States. Colloid fluids, such as albumin, dextran, gelatin, or hydroxyethyl starch, are hypothesized to be optimal fluids since they are retained in the intravascular plasma volume. However, to date, no randomized controlled trials have shown a survival benefit of colloid solutions compared to crystalloid solutions. The administration of human albumin has been extensively studied for over two decades without any improved mortality and is significantly more expensive than crystalloids [51, 52]. The starch-containing colloids are currently used in Europe but have been associated with nephrotoxicity and increased bleeding, and no randomized trials have demonstrated convincing improved outcomes [53, 54]. The same holds true for hypertonic saline. Although hypertonic saline (HTS) is a plasma volume expander and has immunomodulatory effects, a recent randomized trial failed to show improved survival. It is postulated that HTS may have delayed blood product transfusion or increased coagulopathy, leading to increased mortality [55]. Ultimately, intravascular volume should be restored with blood products as early as possible in patients with persistent hemorrhagic shock [56].

The endpoints of resuscitation remain unclear and extend beyond the normalization of blood pressure, heart rate, and urine output. Even when these parameters normalize, up to 85 % of severely injured patients still have evidence of tissue hypoperfusion reflecting compensated shock [57, 58]. Therefore, other parameters have been proposed including supranormal hemodynamic parameters (cardiac index, O_2 delivery, and O_2 consumption), mixed venous oxygen saturation (SvO_2), arterial base deficit and lactate, gastric tonometry, and near-infrared spectroscopy (NIRS). Although achieving supranormal hemodynamic parameters (cardiac index >4.5 L/min/m, O_2 delivery >600 mL/min/m^2, and O_2 consumption >170 mL/min/m^2) were proposed to improve survival and decrease of MOF, no adequately powered randomized prospective trials demonstrated improved outcomes [59–62]. Many studies showed no difference in outcomes and suggested that the ability of patients to achieve these parameters was predictive of survival, rather than the supranormal endpoints themselves [63–65]. In fact, recent data indicate supranormal resuscitation results in excessive crystalloid infusion, abdominal compartment syndrome, MOF, and mortality [66, 67]. Although SvO_2 should reflect

the adequacy of O_2 delivery to tissues, resuscitating critically ill patients to normal SvO_2 (>70 %) did not improve survival or MOF [68]. Both arterial base deficit and lactate levels reflect the degree of tissue ischemia and are proportional to the depth and severity of shock. Although several studies have shown that initial values and time to normalization are predictive of transfusions, MOF, and mortality, it has not been proven that normalizing base deficit or lactate levels as an endpoint for resuscitation improves survival [60, 69–72]. Gastric tonometry allows for the detection of subclinical ischemia through the measurement of gastric pCO_2 and calculated intramucosal pH (pHi). The difference between intragastric pCO_2/pHi and arterial pCO_2 correlates with the degree of gastric ischemia and is predictive of MOF and mortality in trauma patients [73–75]. However, measurement requires withholding gastric feeding and suppressing gastric acid secretion, and normalization of pHi or pCO_2 gap as endpoints for resuscitation has not been shown to improve outcomes. NIRS can simultaneously measure tissue pO_2, pCO_2, and pH, and preclinical studies have suggested that measurement of tissue pO_2, pCO_2, and pH of solid organs and skeletal muscle may be a better predictor of outcomes and provide better endpoints for resuscitation [76–78]. NIRS-derived tissue oxygenation saturation has also been found to predict the need for blood transfusion in trauma patients who initially appeared hemodynamically stable [79]. Although preliminary, and no studies showing improved outcomes with NIRS resuscitation endpoints have been performed, this technology provides clinicians a tool to quickly assess tissue oxygenation in a noninvasive manner and shows promise to guide resuscitation as well as triage patients in the field.

Traumatic Shock

Traumatic shock is a variant of hemorrhagic shock, combining the effects of tissue injury and long bone fractures with substantial blood loss. Traumatic shock initiates a massive proinflammatory response that increases the systemic inflammatory response syndrome (SIRS) and risk for acute respiratory distress syndrome (ARDS) and multiple organ failure (MOF), which rarely occur following simple hemorrhagic shock [80]. In severely injured trauma patients, the inflammatory response occurs within 30 min following injury, and this early increase in cytokines is associated with worse outcomes [81, 82]. This magnified inflammatory response may be due to the combination of both ischemia and direct cellular injury leading to a greater release of endogenous molecules, called DAMPs or alarmins, which interact with cells of the innate immune system and initiate an inflammatory response (Fig. 12.3) [16, 83, 84]. Mechanical cell rupture from trauma, or ischemia-induced apoptosis, leads to the passive release of intracellular contents into the extracellular environment, which contain DAMPs [85]. High-mobility group box (HMGB) 1, heat shock proteins (HSPs), and mitochondrial DNA have been reported to be elevated following trauma, and plasma HMGB1 levels correlate well with injury severity score and base deficit [86–88]. S100 proteins are calcium-binding proteins which are found primarily in oligodendrocytes, astrocytes, and Schwann cells and act as ligands for the transmembrane receptor for advanced glycation endproducts (RAGE), which have direct effects on the innate immune system and inflammation [89]. Elevated plasma levels of S100B have been identified in traumatic brain injury patients and correlated well with poor outcomes [90, 91]. Secretory RAGE (sRAGE) is cleaved from RAGE and is elevated in the plasma within 30 min following injury. High levels have been associated with coagulopathy and complement activation in trauma patients [92, 93]. Furthermore, cell surface saccharides, heparan sulfate and hyaluronic acid, act as ligands for TLR4 and have been linked to SIRS [94]. The net result of these events is a "genomic storm," resulting in a dramatic increase in both proinflammatory and anti-inflammatory signaling [95]. Treatment of traumatic shock focuses on minimizing the immune dysregulation via prompt control of hemorrhage, adequate volume resuscitation, restoring coagulation capacity, debridement of nonviable tissue, and stabilization of fractures.

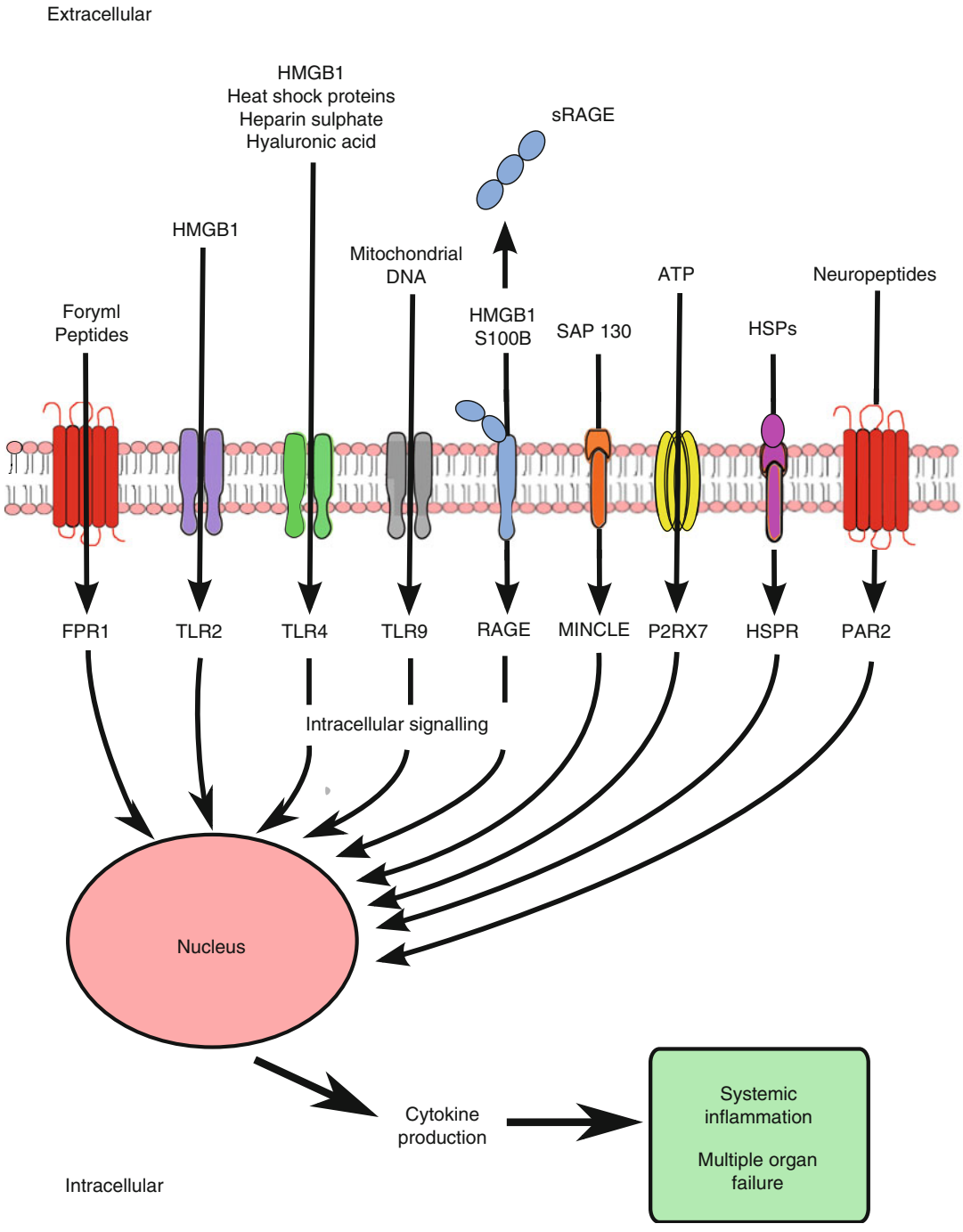


Fig. 12.3 DAMPs or alarmins resulting from trauma and shock and their receptors for proinflammatory immune activation. *HMGB* high-mobility group box protein, *(s)RAGE* (secretory) receptor for advanced glycation endproducts, *ATP* adenosine 5′-triphosphate, *HSP(R)* heat shock protein (receptor), *FPR* formyl peptide receptor, *TLR* Toll-like receptor, *Mincle* macrophage-inducible C-type lectin, *P2RX7* purinergic receptor, *PAR* proteinase-activated receptor, *NF* nuclear factor

Distributive Shock

Distributive shock is characterized by an overall decrease in systemic vascular resistance from the failure of vascular smooth muscle contraction, which results in hypotension and poor tissue perfusion. There are multiple etiologies for distributive shock (Table 12.5), but all result in endothelial and vascular dysfunction from exogenous and endogenous inflammatory mediators or as a response from prolonged and severe hypoperfusion [96]. Ultimately, all forms of untreated and prolonged shock result in distributive shock.

The most common etiology for distributive shock is severe sepsis, which is increasing in incidence and kills approximately a third of those diagnosed in the United States [97, 98]. However, distributive shock can occur with advanced traumatic shock [96]. Most etiologies have a common pathway involving the production of nitric oxide (NO). NO is synthesized by nitric oxide synthase, which converts L-arginine to NO and L-citrulline. NO may then act locally or diffuse into nearby cells where it activates cytosolic guanylate cyclase forming cyclic GMP (cGMP). In the vascular smooth muscle, increases in cGMP result in smooth muscle relaxation and vasodilation. There are currently three recognized isoforms of NOS in eukaryotes: endothelial NOS (eNOS) and neuronal NOS (nNOS), which are calcium dependent and constitutively expressed, and the inducible isoform (iNOS), which is calcium independent, and its expression is induced by cytokines and inflammatory mediators [99]. Normally, NO production through eNOS regulates microvascular homeostasis and regulation of blood flow. However, in sepsis and inflammation induced by protracted shock, inflammatory stimuli (LPS, IL-1β[beta], macrophage migration inhibitory factor [MIF], and IL-6) induce iNOS expression and the creation of large amounts of NO (Fig. 12.4) [99]. This results in systemic vasodilation, hypotension, early neutrophil activation, decreased immune cell function (monocyte/macrophages, dendritic cells, T-lymphocytes, and late neutrophil function), and ultimately organ dysfunction.

Table 12.5 Causes of distributive shock

Sepsis
Noninfectious
Pancreatitis
Burns
Adrenal insufficiency
Anaphylaxis
Prolonged and severe hypotension
Hypovolemic shock
Cardiogenic shock
Obstructive shock
Metabolic
Lactic acidosis
Carbon monoxide poisoning

Early goal-directed resuscitation for patients in septic shock has been shown to improve survival [100]. Within the first 6 h after the diagnosis of septic shock, the goal of resuscitation is to achieve a central venous pressure of 8–12 mmHg, mean arterial pressure greater than or equal to 65 mmHg, urine output greater than or equal to 0.5 ml/kg/h, and a central venous or mixed venous oxygen saturation greater than or equal to 70 % or 65 %, respectively [101]. Vasopressors may be needed to maintain the mean arterial pressure greater than or equal to 65 mmHg, and norepinephrine is the initial vasopressor of choice [101]. Vasopressin may be subsequently added to norepinephrine, and dobutamine may be used in patients with myocardial dysfunction for inotropic support. Empiric antibiotics must be started as soon as the diagnosis is suspected and chosen based on the most likely pathogens. The use of steroids remains controversial but should be considered in patients when hypotension responds poorly to adequate fluid resuscitation and vasopressors [101].

Neurogenic Shock

Acute spinal cord injury (SCI) above the level of T6 has been associated with a higher incidence of neurogenic shock [102, 103]. In developed countries, SCI has an incidence of about 50 cases/million population per year, but less than 20 % results in neurogenic shock [104, 105].

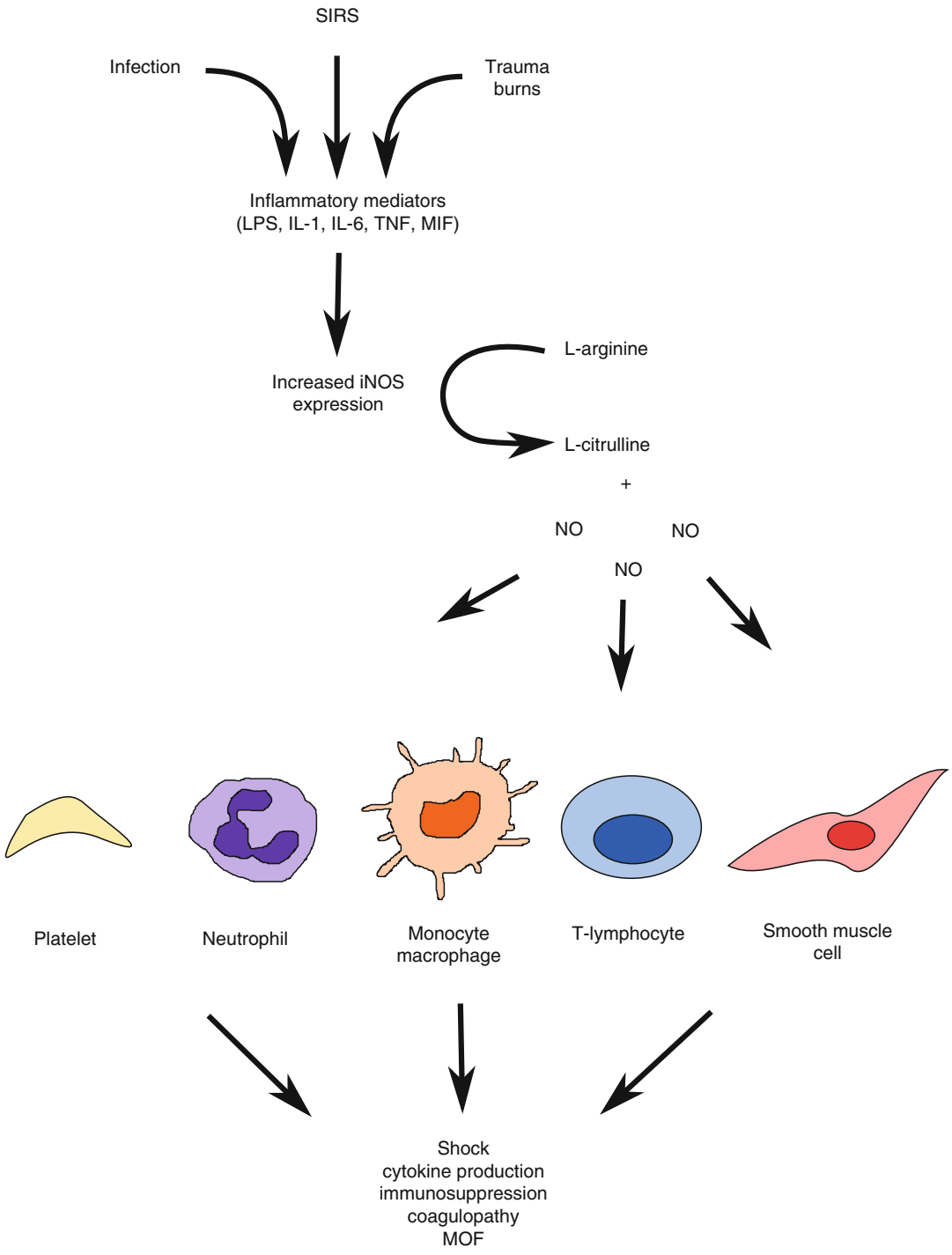


Fig. 12.4 Role of NO in the pathophysiology of shock following trauma, systemic inflammatory response syndrome (*SIRS*), and infection

Neurogenic shock occurs secondary to spinal cord injuries usually from vertebral body fractures of the cervical or high thoracic spine, which disrupt the sympathetic regulation of the cardiovascular system. It may also result from compression of the spinal cord from an epidural

hematoma. Although, often considered a form of distributive shock, the pathogenesis of neurogenic shock is unique in that shock results from the inhibition of multiple systems to regulate blood pressure. This occurs from disruption of the descending medullary basal sympathetic tone to the sympathetic preganglionic vasomotor neurons in the thoracolumbar cord, which affects four organs: (1) the heart, resulting in bradycardia and decreased inotropic function; (2) the peripheral vascular system, increasing systemic vasodilation; (3) the adrenal medulla, decreasing circulating epinephrine and norepinephrine; and (4) the kidney, decreasing stimulation of the juxtaglomerular cells which activate the renin-angiotensin-aldosterone axis [106]. It is important to note that neurogenic shock is a different entity than spinal shock. Although spinal shock often accompanies neurogenic shock, spinal shock does not refer to circulatory collapse and is characterized by a marked reduction or abolition of somatic and/or reflex functions of the spinal cord caudal to the injury site (loss of sensation accompanied by motor paralysis and areflexia/hyporeflexia). On the other hand, neurogenic shock is characterized by hypotension following injury to the central nervous system [102].

Diagnosing neurogenic shock can be difficult in patients with multisystem trauma, and hemorrhagic shock must first be ruled out. Neurogenic shock should be suspected in patients with hypotension (SBP <90 mmHg), relative bradycardia, warm extremities, motor and/or sensory deficits suggestive of a spinal cord injury, and radiographic evidence of vertebral column fractures. Spinal cord injuries can later be defined by an MRI once the patient is hemodynamically stable. Initial treatment consists of securing an airway. If the patient requires endotracheal intubation, it should be performed by experienced personnel, with standard precautions, including in-line immobilization. Subsequent interventions should be focused on expanding intravascular volume to maintain a systolic blood pressure greater than 90 mmHg or a cerebral perfusion pressure greater than 50 [107–109]. It is imperative to correct the hypotension rapidly since decreased spinal cord perfusion is detrimental to the initial SCI and perpetuates a secondary injury [110]. If hypotension persists after adequate volume has been given, vasopressors and inotropic agents should be used. In the case of decreased peripheral vascular resistance and adequate cardiac output and heart rate, norepinephrine is preferred. In the unusual situation where cardiac output and heart rate are the dominant factors, dopamine may be optimal [110].

Cardiogenic Shock

Clinically, cardiogenic shock is defined by tissue hypoxia secondary to myocardial dysfunction in the presence of adequate intravascular volume. It is characterized by a systolic blood pressure of less than 90 mmHg for at least 1 h that is (1) not responsive to fluid administration alone and (2) associated with a cardiac index of less than 2.2 L/min/m^2 in the setting of a pulmonary capillary wedge pressure (PCWP) greater than 18 mmHg [111]. The leading cause of cardiogenic shock is left ventricular failure secondary to acute myocardial infarction [112]. However, there are multiple potential etiologies of cardiogenic shock following trauma (acute myocardial infarction, cardiomyopathy, valvular heart disease, arrhythmias, myocardial injury), but all ultimately result in cardiac ischemia, leading to cardiac dysfunction and, thereby, promoting further ischemia resulting in a vicious cycle. Consequently, mortality from cardiogenic shock can be as high as 80 % [113]. Once a critical mass of ischemic/necrotic left ventricular myocardium reaches approximately 40 %, there is a life-threatening decrease in pumping capability, stroke volume, and cardiac output [111].

In the trauma patient, cardiogenic shock needs to be considered even in the setting of hemorrhagic shock, and a rapid physical assessment should be performed in the emergency department. Patients in cardiogenic shock will usually present with hypotension and jugular venous distension. The presence of a cardiac murmur, ascites, or peripheral edema suggests a preexisting cardiac problem. The diagnosis can be further pursued with an ECG, laboratory values (elevated troponin and BNP), chest radiograph, and echocardiography. The initial step in management should be to ensure an adequate airway. If the likely etiology is an acute myocardial infarction, prompt revascularization is the only intervention

that has consistently been shown to reduce mortality in patients with cardiogenic shock [112]. Fibrinolytic therapy should be avoided in the trauma patient and is not as effective once cardiogenic shock has developed [114]. If hypotension is unresponsive to fluid challenges, vasopressors should be used. Norepinephrine and dopamine are considered first-line drugs of choice in this situation. If patients continue to be nonresponsive to pharmacologic therapy, devices, such as intra-aortic balloon pumps or left ventricular assist devices, may be required to improve cardiac perfusion and allow time for the myocardium to recover.

Extracardiac Obstructive Shock

Extracardiac obstructive shock results from an acute obstruction to circulatory flow by either impaired diastolic filling of the right ventricle (tension pneumothorax or cardiac tamponade) or obstruction of right ventricular output (massive bronchovenous air embolism/pulmonary embolism or pulmonary hypertension). There are several etiologies of obstructive shock, which involve IVC obstruction/compression or increases in intrathoracic/intracardiac pressures, but tension pneumothorax and cardiac tamponade are the most common following trauma.

The rapid diagnosis of a tension pneumothorax is imperative and should be made on clinical examination. Clinical findings include hypotension, respiratory distress, diminished breath sounds over one hemithorax, jugular venous distention, and tracheal deviation. A pneumothorax develops secondary to a breach in the visceral, parietal, or mediastinal pleura, and in a tension pneumothorax, the pleural defect functions as a one-way valve with air entering the pleural cavity on inspiration but is unable to exit on expiration [115]. This leads to an increase in intrathoracic pressure and a decrease in venous return to the heart, effectively reducing cardiac output and resulting in shock. Empiric treatment is pleural decompression with a tube thoracostomy; however, if a chest tube is not readily accessible, as in the prehospital setting, decompression with a large-caliber needle may be performed as a temporizing measure. Following pleural decompression, there should be a rapid resolution of the hypotension and improvement in respiratory parameters.

Cardiac tamponade results from the accumulation of fluid within the pericardial sac. In trauma, cardiac tamponade is seen in both penetrating and blunt trauma scenarios in which the myocardium or vessels are injured and blood acutely fills the pericardial space, increasing the intracardiac pressures. This decreases right atrial filling and, ultimately, compresses the right ventricle, resulting in a significant reduction in cardiac output. Clinical findings may include those of Beck's triad (hypotension, muffled heart tones, and jugular venous distention), but this may be absent in acute tamponade. The diagnosis can be confirmed by a rapid bedside ultrasound, demonstrating pericardial fluid, right atrial collapse, and poor distensibility of the right ventricle. Treatment consists of an emergent pericardiocentesis with placement of a catheter to confirm the diagnosis and to relieve the tamponade. If the patient is in circulatory arrest, an emergent left thoracotomy for pericardial decompression should be performed. Patients may benefit from a fluid bolus if hypovolemic, but in cases of normovolemia or hypervolemia, fluid administration may be deleterious resulting in pulmonary eduma [116]. Isoproterenol may also be beneficial until pericardial decompression can be performed. Isoproterenol increases heart rate and cardiac output and decreases right atrial pressure and systemic vascular resistance [117].

Bronchovenous air embolism is another cause of right ventricular outflow obstruction and can occur following blunt or penetrating thoracic trauma with damage to the great veins or air entry through disrupted smaller venous vessels. The incidence following thoracic trauma has been estimated at 4–14 %, with mortality as high as 80 % in blunt trauma patients [118, 119]. Bronchovenous air embolism may be diagnosed by CT or echocardiography. The initial management of venous air embolism is to immediately give the patient 100 % supplemental oxygen and to place the patient in Trendelenburg with left lateral decubitus positioning. This may prevent air from traveling through the right side of the heart

into the pulmonary arteries. A central venous catheter may then be placed into the right atrium and aspiration performed to remove the air from the venous circulation. Hyperbaric oxygen therapy may also be considered as a possible therapy in symptomatic patients.

Coagulopathy

Trauma continues to be the leading cause of morbidity and mortality worldwide with road traffic injuries, self-inflicted injuries, and interpersonal violence accounting for the three leading causes of death in persons aged 15 through 44 years [120]. Uncontrolled hemorrhage is the second leading cause of death in trauma patients and accounts for nearly 40 % of early mortality, which is preceded only by central nervous system injury [121]. Moreover, approximately one-third of trauma patients have a coagulopathy on arrival to the emergency department, further contributing to hemorrhage, and these patients are four times more likely to die than those without a coagulopathy [122–124].

Coagulopathy, or post-injury hemorrhage that persists despite control of surgical bleeding, was originally described over 60 years ago and has been referred to by many names: medical bleeding, diffuse bleeding diathesis, posttransfusion bleeding disorder, medical oozing, and disseminated intravascular coagulation [125]. Clinically, the coagulopathy manifests as nonsurgical bleeding from mucosal lesions, serosal surfaces, wounds, and vascular access sites, which continues even after control of identifiable vascular bleeding. Although post-injury coagulopathy has long been recognized, the pathophysiology remains poorly understood, and both clinical and laboratory parameters remain feeble predictors of coagulopathy. In 1982, our group described the "bloody vicious cycle," also referred to as "the lethal triad," in which the synergistic effects of acidosis, hypothermia, and coagulopathy combined create an irreversible clinical deterioration in patients receiving massive transfusions resulting in death by exsanguination despite surgical control of bleeding (Fig. 12.5) [126]. This was later correlated with decreased clotting factor concentrations and a corresponding prolongation of traditional measures of coagulopathy, such as prothrombin time (PT) and activated partial thromboplastin time (aPTT) suggesting either a consumption or dilution of clotting factors [127]. In spite of this, development of coagulopathy following massive transfusion still could not be adequately predicted with clinical and laboratory parameters [128]. However, recent evidence suggests an endogenous coagulopathy associated with severe trauma, termed the acute coagulopathy of trauma (ACOT), which occurs early and is independent of the secondary effects of body temperature, acidosis, and clotting factor levels [129].

Cell-Based Model of Coagulation

Effective management of post-injury coagulopathy requires a fundamental understanding of the coagulation process, which depends on an intricate balance between the anticoagulant, procoagulant, and fibrinolytic systems. Major proteins involved in these systems are listed in Table 12.6. Traditionally, coagulation was described as a cascade model composed of a sequential series of steps in which activation of one clotting factor led to the activation of another, resulting in thrombin generation. This model reflects two distinct pathways, the intrinsic and extrinsic pathways, which converge on a common pathway with the conversion of factor X to factor Xa. Although this model improved our understanding of key physiological events in vivo, it was clear from early studies that cells were important participants in coagulation. Hemostasis is not possible without platelets, and tissue factor (TF) is an integral membrane protein. However, cells were viewed only as donors of an anionic phospholipid surface for procoagulant complex assembly, and this model was supported by traditional plasma-based laboratory tests of isolated coagulation in a test tube, which do not correlate with clinical hemostasis.

Subsequently, the cascade model has been supplanted by the cell-based model (CBM) of coagulation, which proposes hemostasis occurs in a stepwise process, regulated by cellular

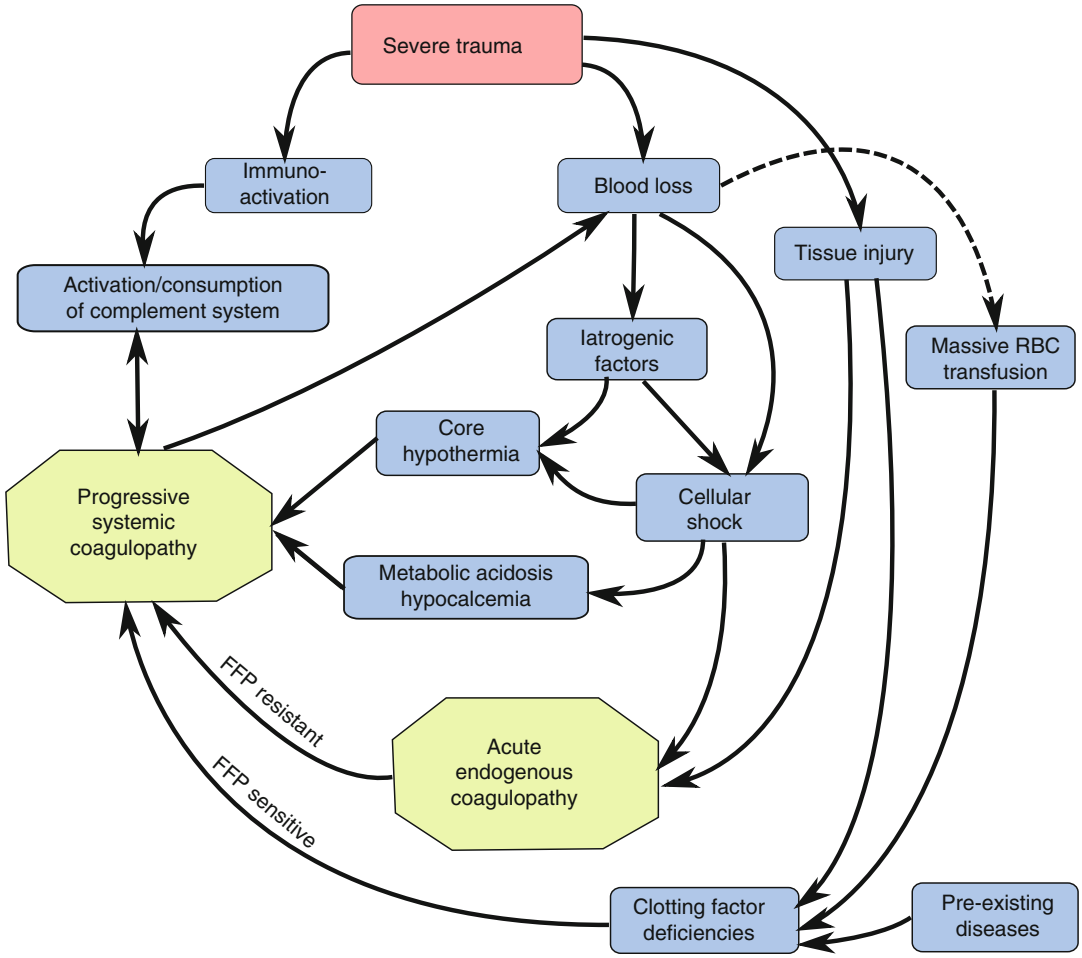


Fig. 12.5 The "bloody vicious cycle" of the acute coagulopathy of trauma. This updated cycle incorporates both the early acute endogenous coagulopathy of trauma, which is resistant to clotting factor replacement with fresh frozen plasma (FFP resistant), and a subsequent secondary coagulopathy that may be due to hypothermia, acidosis, clotting factor deficiency (FFP sensitive), or any combination of factors

Table 12.6 Proteins involved in coagulation, anticoagulation, and fibrinolysis

Protein	Source	Activated by	Function
Procoagulant			
Tissue factor	Subendothelium, monocytes in response to IL-6	Exposure to circulating platelets	Complex with VII to initiate clot formation
Fibrinogen (factor I)	Liver, activated platelets	Thrombin	Clot formation
Factor V	Liver, activated platelets	Thrombin	Cofactor that accelerates conversion of prothrombin to thrombin; Leiden mutation renders it resistant to inactivation by APC
Factor VII	Liver	Thrombin, Xa, XIa, XIIa	Complexes with TF to convert X to Xa
rFVIIa	N/A	N/A	Complexes with TF → activate X Binds to activated plts → activate X (bypassing VIII and IX) Activate TAFI

Table 12.6 (continued)

Protein	Source	Activated by	Function
Factor VIII	Endothelial cell Liver sinusoidal cells	Thrombin	Cofactor, activates factor X
Factor IX	Liver	Factor XI	Cofactor, activates factor X
Factor X	Liver	Factor VIII, factor IX	Converts prothrombin to thrombin
Factor XI	Liver	Thrombin	Activates IX
Factor XIII	Liver	Thrombin	Cross-links fibrin
Anticoagulants			
Heparin sulfates	Endothelial cells	Ischemia, hypoxia	Activation of ATIII
Antithrombin III	Liver	Thrombin	Inhibition of thrombin, Xa, Xia, XIIa
Protein C	Liver	Thrombin-TM-EPCR complex	Irreversibly inactivates Va and VIIIa
Protein S	Liver		Cofactor for protein C
Thrombomodulin (TM)	Endothelial cell	Tissue hypoperfusion (shock) Thrombin	Complexes with thrombin to activate protein C, reduces thrombin's procoagulant activity Inhibits TAFI, leading to fibrinolysis
Endothelial protein C receptor (EPCR)	Endothelial cell	N/A	Complexes with thrombin and TM to activate protein C
Tissue factor pathway inhibitor (TFPI)	Liver	Thrombin	Inhibits TF-VII complex from converting X → Xa, thereby inhibiting coagulation cascade
Fibrinolytic system			
Plasminogen	Eosinophiles	tPA	Converted to plasmin, leading to fibrinolysis
tPA	Endothelial cell	Ischemia, hypoxia, thrombin	Converts plasminogen to plasmin
PAI-1	Endothelium	Inflammation	Inhibits tPA, resulting in inhibition of fibrinolysis
Protein C	Liver	Thrombin/ thrombomodulin complex	Inhibits PAI-1, leading to fibrinolysis
Thrombin-activatable fibrinolysis inhibitor (TAFI)	Liver	Thrombin/ thrombomodulin complex	Inhibits fibrinolysis
Platelet activators/ inhibitors			
vWF	Subendothelium, platelets	Platelets, collagen	Binds to plt surface protein Ib-V-IX to cause adhesion
NO	Endothelial cells	Ischemia	Inhibit platelet activation
Prostacyclin	Endothelial cells	Ischemia	Inhibit platelet activation

components in vivo [130]. Additionally, this model allows for improved understanding and potential mechanistic links with cross talk between inflammation and coagulation. The first step is the initiation of coagulation on TF-bearing cells, followed by the amplification of the procoagulant signal by thrombin generated on the TF-bearing cell, and then the propagation of thrombin generation on the platelet surface (Fig. 12.6). Platelets are a crucial component of hemostasis following injury, and localization and activation are mediated by vWF, collagen, thrombin, platelet receptors, and factors within the vessel wall [131]. Once activated, platelets rapidly localize cofactors Va and VIIIa on their surface, as well as IXa and XIa, and subsequently, factor X is activated to Xa on the platelet surface. Therefore, there is a large amount of thrombin generated on the platelet surface, which has many other functions beyond promoting fibrin polymerization since most of the thrombin generated is produced after the initial fibrin clot is formed [130]. The platelet-produced thrombin activates factor XIII and thrombin-activatable

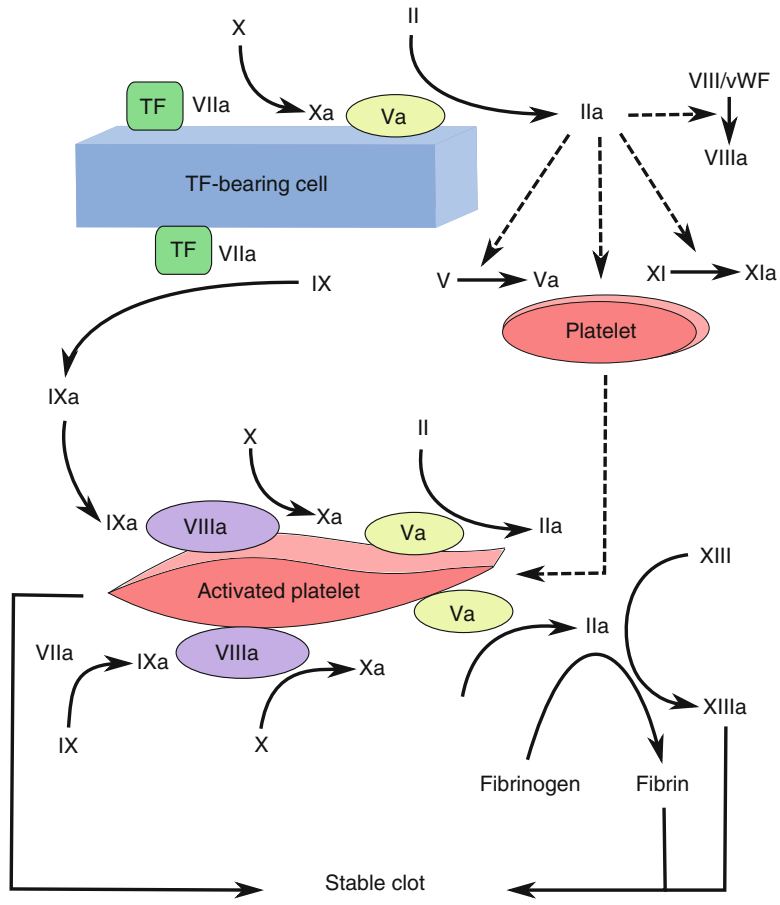


Fig. 12.6 Cell-based model of hemostasis, which occurs in a stepwise fashion and is regulated by tissue factor (TF)-bearing cells and platelets. A majority of thrombin generation occurs on the surface of activated platelets. *vWF* von Willebrand factor

fibrinolysis inhibitor (TAFI), cleaves the platelet PAR-4 receptor, and is incorporated into the structure of the clot [132–134].

Acute Coagulopathy of Trauma

Post-injury coagulation disturbances follow a trimodal pattern in severely injured trauma patients, with an immediate hypercoagulable state, followed quickly by a hypocoagulable state, and ending with a return to a hypercoagulable state [135]. Traditionally, post-injury coagulopathy was considered to be the consequence of clotting factor depletion from hemorrhage and consumption, dilution secondary to massive resuscitation, and dysfunction due to both acidosis and hypothermia. However, several recent reports have detailed that many trauma patients present with a coagulopathy prior to fluid resuscitation and clotting factor depletion [123, 124, 129, 136]. These patients consistently have evidence of protracted hypoperfusion and significant base deficits. Collectively, these data indicate an acute endogenous coagulopathy of trauma, which occurs early after injury, is independent of clotting factor levels, and is correlated closely with hypoperfusion and tissue injury.

In their recent study, Brohi and colleagues noted an increasing base deficit was directly correlated with thrombomodulin concentration (an anticoagulant protein expressed by the endothelium in response to ischemia) and inversely correlated to protein C concentration, suggesting protein C activation via the thrombomodulin/thrombin complex [129]. Activated protein C (APC), in turn, inhibits factors Va and VIIIa and promotes fibrinolysis through irreversible inhibition of plasminogen activator inhibitor-1 (PAI-1). Decreased protein C concentrations

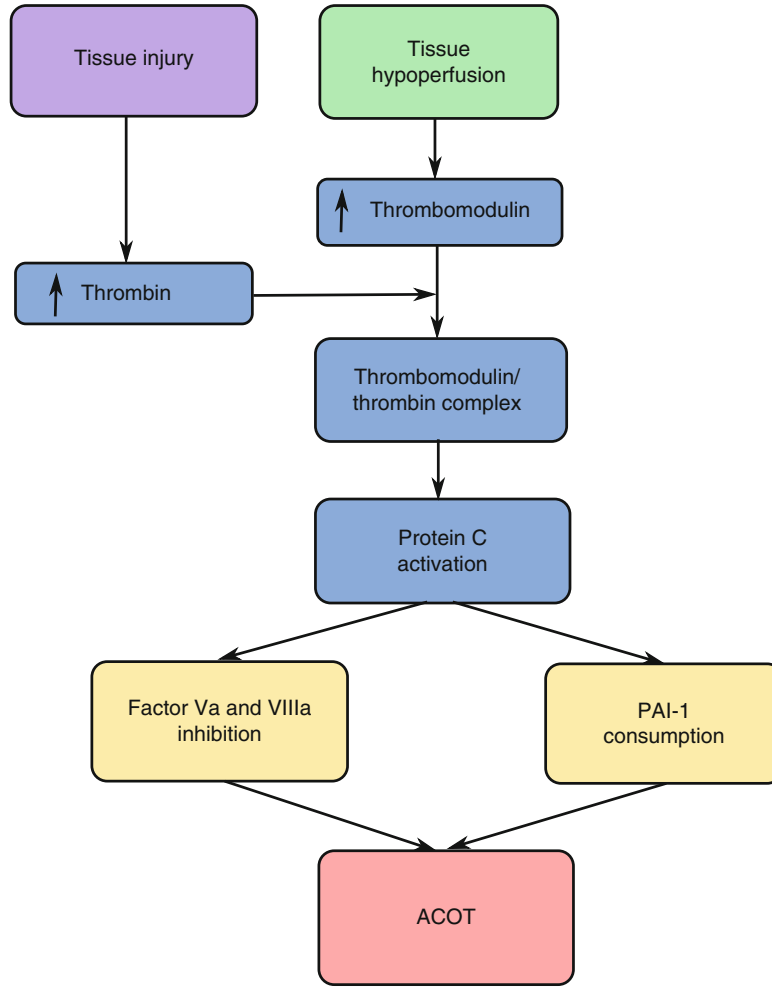


Fig. 12.7 Activated protein C hypothesis for the acute endogenous coagulopathy of trauma. Both significant tissue injury and tissue hypoperfusion are required to increase thrombin and thrombomodulin, resulting in the formation of the thrombomodulin/thrombin complex. This complex activates protein C and inhibits factors Va and VIIIa as well as plasminogen activator inhibitor-1 (PAI-1), resulting in an acute endogenous coagulopathy of trauma (*ACOT*)

were correlated with prolongation of the aPTT as well as with decreased concentrations of PAI-1. Moreover, this decrease in protein C was also correlated with increases in tissue plasminogen activator (tPA) and D-dimer concentrations, suggesting a protein C-mediated hyperfibrinolysis through consumption of PAI-1 (Fig. 12.7). Following this study, Cohen and colleagues were able to directly measure APC in trauma patients and confirmed the aforementioned correlations that APC was elevated following severe tissue injury and hypoperfusion and inhibited factors Va and VIIIa [137].

Others have proposed that the early coagulopathic changes following severe injury simply reflect the traditional concepts of disseminated intravascular coagulation (DIC) [138]. As seen in other insults that induce widespread inflammation (e.g., sepsis, toxins, cancer), trauma and ischemia/reperfusion may also illicit the same generic response by (1) release of tissue factor with massive thrombin generation and subsequent clotting factor consumption and (2) hyperfibrinolysis due to upregulation of tPA from the endothelium (Fig. 12.8). This argument is supported by longstanding reports of diffuse intravascular microthrombi in uninjured organs following hemorrhagic shock [139]. Furthermore, cytokine patterns in both trauma and sepsis patients are nearly identical, suggesting a potential common pathophysiological mechanism [140]. However, this view of post-injury coagulopathy is limited by the finding that clotting factor levels are relatively preserved in trauma patients early on when

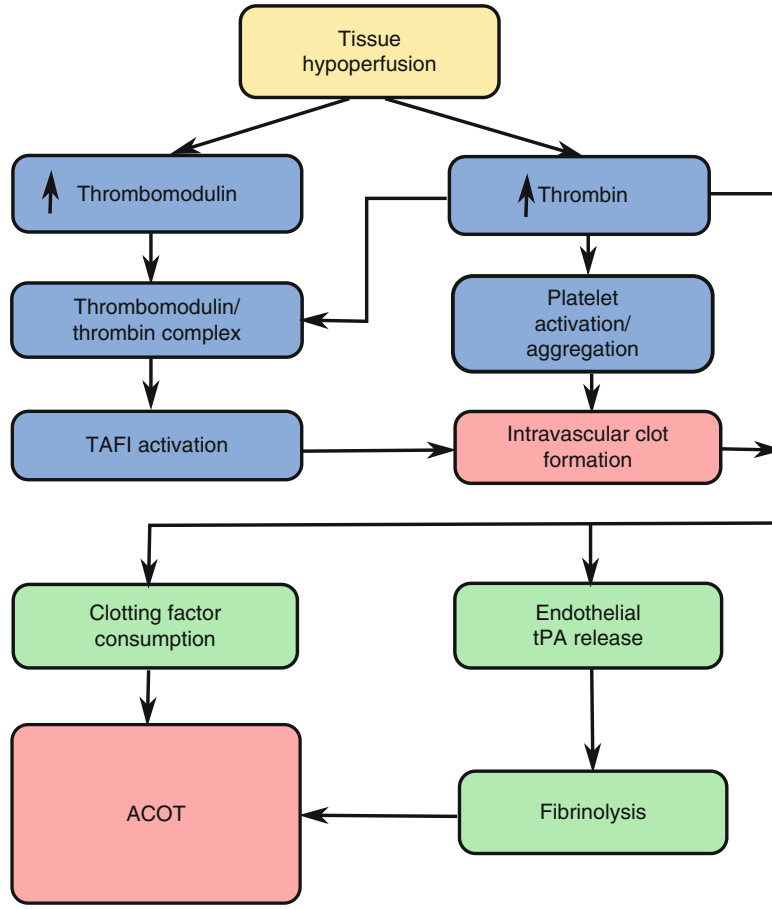


Fig. 12.8 Disseminated intravascular coagulation hypothesis for the acute coagulopathy of trauma. Tissue hypoperfusion is sufficient to increase both thrombomodulin and thrombin, resulting in thrombin-activatable fibrinolysis inhibitor (*TAFI*) and platelet activation leading to an initial hypercoagulable state. In severe trauma, this may consume clotting factors and, along with tissue plasminogen activator (*tPA*) released from injured endothelium, result in an acute coagulopathy of trauma (*ACOT*)

the diagnosis of coagulopathy is made [129]. Additionally, the degree of fibrinolysis appears substantially higher in trauma patients with an endogenous coagulopathy compared to patients with known DIC [136]. Moreover, DIC occurs in the setting of an underlying hypercoagulable state (e.g., malignancy, septic shock) and is associated with an upregulation of PAI-1, as opposed to the early hypocoagulable state observed in the severely injured patient, which reflects a predominance of both tPA upregulation and PAI-1 inhibition [141].

Our conceptualization of the acute endogenous coagulopathy of trauma includes the roles of fibrinolysis, hypocoagulability, and platelet dysfunction. Specifically, severe tissue injury and systemic hypoperfusion lead to diffuse endothelial injury and massive thrombin generation resulting in the widespread release of tPA. Both injury and ischemia are well-known effectors of tPA release [142]. Furthermore, severe injury and hypoperfusion increase APC, which decreases PAI-1 concentrations and further promotes fibrinolysis. Moreover, APC inhibits factors Va and VIIIa, promoting further hemorrhage and hypoperfusion and creating a perpetual cycle of worsening coagulopathy. Recently, we have documented platelet dysfunction following severe injury, despite adequate platelet counts, which correlated with markers of hypoperfusion (base deficit and systolic blood pressure <70 mmHg) [143]. Although the mechanism remains unclear, data suggests early platelet hyperactivation, which renders the platelet unresponsive to subsequent stimulation and hypocoagulability. The proposed pathway of the endogenous coagulopathy of trauma is depicted in Fig. 12.9.

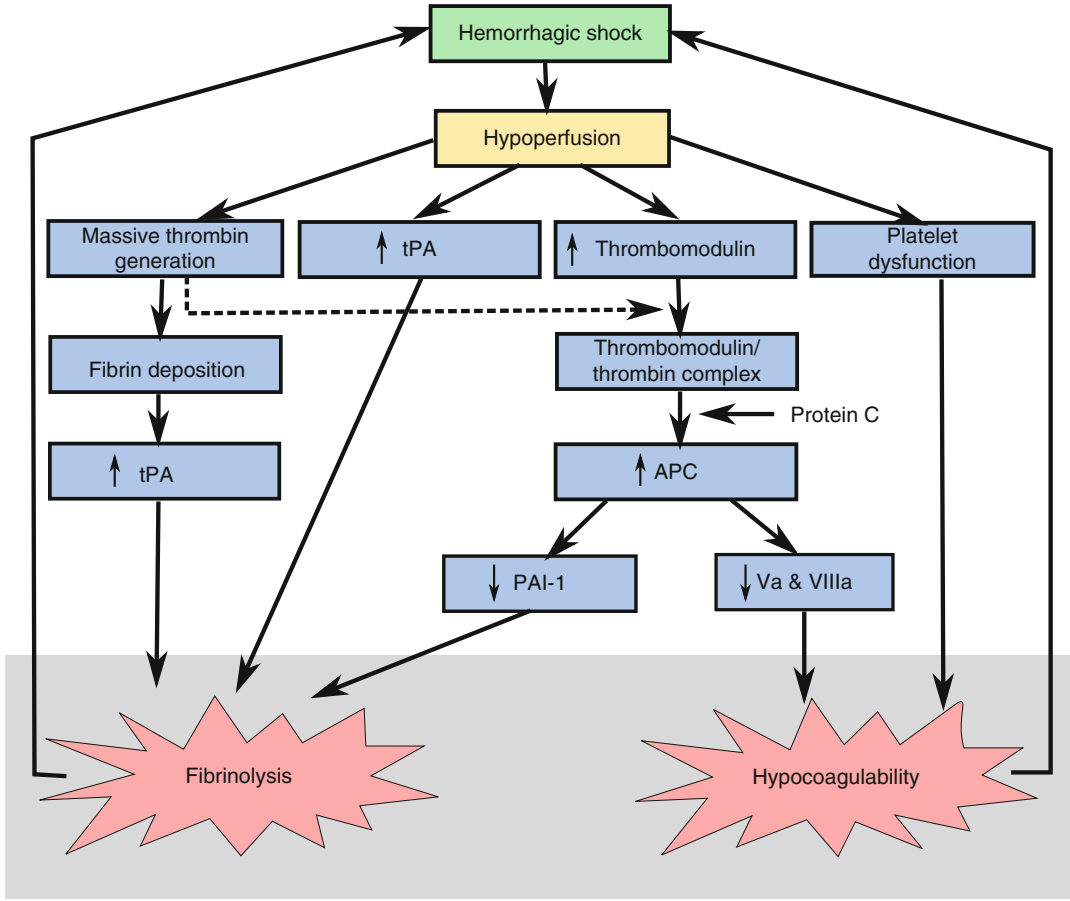


Fig. 12.9 Proposed pathways for the acute endogenous coagulopathy of trauma reflecting the role of fibrinolysis via multiple mechanisms, the necessary thrombin substrate, and the positive feedback cycle that perpetuates the coagulopathy. *tPA* tissue plasminogen activator, *APC* activated protein C, *PAI-1* plasminogen activator inhibitor-1

Elucidation of an endogenous coagulopathy of trauma has important therapeutic implications regardless of the inciting mechanism. Given that hypoperfusion appears to be the driving force of early coagulopathy rather than clotting factor consumption, replacement of clotting factors at this time would be ineffective and may serve to exacerbate the coagulopathy via generation of additional thrombin substrate for thrombomodulin. Therefore, we have noted the endogenous coagulopathy of trauma to be "fresh frozen plasma (FFP) resistant" and suggest that antifibrinolytic drugs may possibly mitigate this coagulopathy. In contrast, the development of a secondary coagulopathy due to the complications of massive resuscitation results in a clotting factor deficiency, thus making this coagulopathy "FFP responsive" (Fig. 12.5) [144].

Currently, neither a standardized definition nor diagnostic criteria for ACOT exists, and refinements of the mechanisms underlying this coagulopathy are needed. Furthermore, little is known about the mediators of thrombomodulin during traumatic shock, and there has been little work showing causality between the proposed mediators of coagulopathy (APC, clotting factor expression profiles, and biomarkers of shock) and the endogenous coagulopathy of trauma. In spite of these limitations, description of the endogenous coagulopathy of trauma represents a major turning point in understanding of the hemostatic derangements following trauma.

Massive Transfusion Protocols

Current Advanced Trauma Life Support guidelines emphasize 2 L of crystalloid followed by transfusion of RBCs in the case of persistent hemodynamic instability, and clotting factor and platelet replacement are indicated only in the presence of laboratory derangements (PT and platelet count, respectively) [145]. This approach is reasonable for patients who have sustained relatively minor hemorrhage (<30 % of circulating blood volume), but in the case of ongoing hemorrhage and massive transfusion, large volume isolated erythrocyte transfusion will result in dilution coagulopathy. The most commonly accepted definition of massive transfusion is the need for greater than 10 units of RBCs within the first 24 h of injury. Since over 80 % of blood component therapy transfused to patients who require massive transfusion is administered within the first 6 h of injury, we believe this to be a more appropriate time period for analysis [146]. Recognizing the danger with isolated RBC transfusion, many have adopted preemptive clotting factor (FFP) and platelet component therapy in patients requiring massive transfusions (Fig. 12.10). Although the transfusion ratios of RBCs, FFP, and platelets continue to be debated,

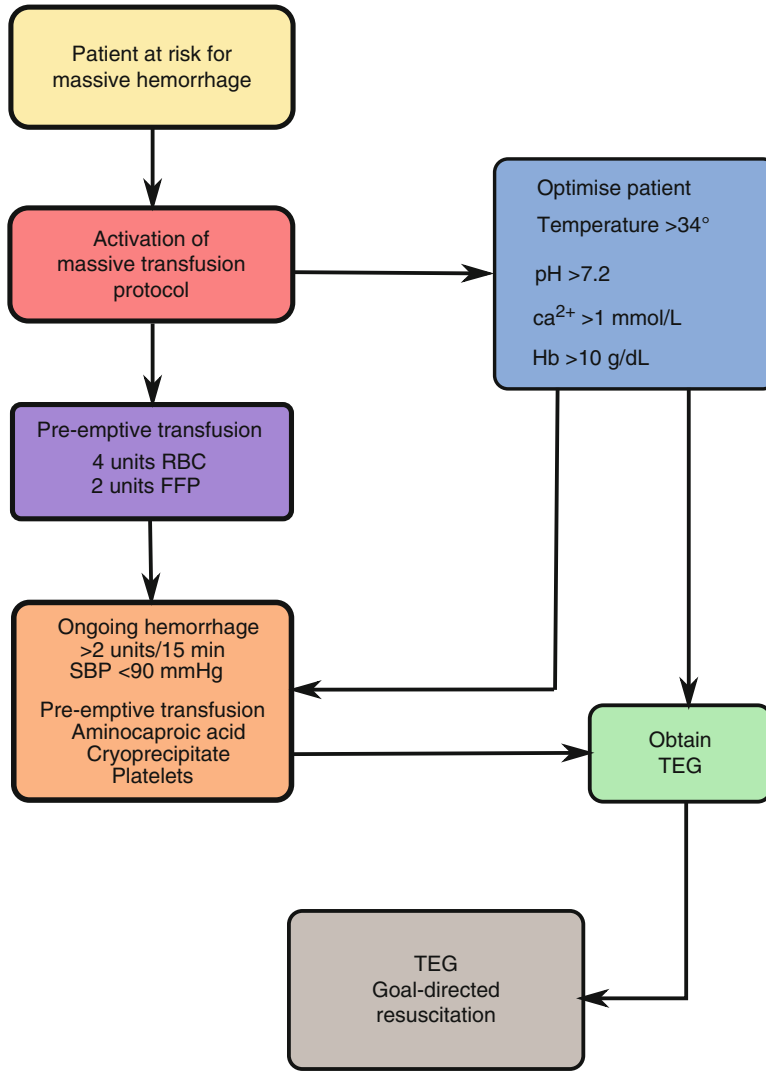


Fig. 12.10 Denver massive transfusion protocol. *RBC* red blood cells, *FFP* fresh frozen plasma, *Hb* hemoglobin, *TEG* thromboelastometry

there is substantial retrospective evidence that preemptive component therapy improves survival [147–151]. In fact, we first recommended a presumptive FFP to RBC transfusion ratio of 1:4 in 1981 [146]. However, no prospective studies have identified optimal timing and ratios of blood component transfusion.

Viscoelastic Hemostatic Assays

Lack of an accurate tool to identify and track coagulopathy remains a major limitation of the literature involving post-injury coagulopathy and therapeutic interventions. Traditional laboratory tests of coagulation, such as PT and aPTT, only evaluate the individual plasma components of coagulation, since these tests were originally developed for the assessment of anticoagulation function of hemophiliacs [152]. To date, the performance characteristics of these tests in the trauma patient remain unproven and have many limitations since these tests are performed on platelet-poor plasma. Greater than 95 % of thrombin generation occurs after the initial polymerization of fibrinogen and requires the surface of platelets for the localization of clotting factor and cofactor complexes. Furthermore, these tests are performed in an artificial environment, irrespective of the patient's core body temperature and pH, and require approximately 45 min to perform, making them almost prohibitive in the management of trauma patients. Other markers of endogenous coagulopathy, such as protein C, APC, PAI-1, and thrombomodulin, are both costly and time consuming. Diagnosing fibrinolysis is also problematic since euglobulin lysis time is complex and can take more than 90 min to perform, and D-dimers are not specific following trauma. Thus, all major aspects of the hemostatic system are inadequately measured using conventional plasma-based laboratory tests in trauma patients [153, 154].

In response to the shortcomings of conventional measurements of coagulopathy, point-of-care, rapid viscoelastic hemostatic assays are emerging as the standard of care for both the diagnosis and treatment of post-injury coagulopathy at many US and European trauma centers. These assays, also referred to as thrombelastography (TEG), or rotational thromboelastometry (ROTEM), were pioneered by the German physician Dr. Hellmut Hartert in 1948 and were brought to the United States by a fellow German, Dr. Kurt von Kaulla, who along with Dr. Henry Swan at the University of Colorado used thrombelastography to manage coagulation derangements during the application of hypothermic arrest in cardiac surgery [155]. A decade later, thrombelastography was instrumental in guiding blood component therapy during the birth of Dr. Thomas Starzl's liver transplant program in Denver [156]. Since this time, viscoelastic hemostatic assays have evolved to become more rapid and efficient, and currently there are only two devices on the market: the TEG® 5000, which is manufactured by Haemoscope Corporation (Niles, Illinois), and the ROTEM® delta manufactured by Tem Innovations GmbH (Munich, Germany). These devices provide a rapid, comprehensive assessment of in vivo coagulation status using whole blood, including the dynamics of clot formation and breakdown. Recent data continue to support the superiority of these devices in detecting coagulopathies and predicting massive transfusions in trauma patients compared to both aPTT and PT/INR [157–161]. These findings emphasize the limitations of classic coagulation tests and their lack of efficacy in post-injury coagulopathy.

Thrombelastography (TEG)

TEG uses a device composed of two mechanical parts separated by a blood specimen: a plastic cup, into which a 0.36 mL blood specimen is pipetted, and a plastic pin attached to a torsion wire and suspended with the specimen (Fig. 12.11). Once the sample within the cup is placed on the analyzer, the temperature may be adjusted to that of the patient. The cup then oscillates slowly through a 4.45° angle. Initially, movement of the cup does not affect the pin, but as clot develops, resistance from the developing fibrin strands couples the pin to the motion of the cup. In turn the torsion wire generates a

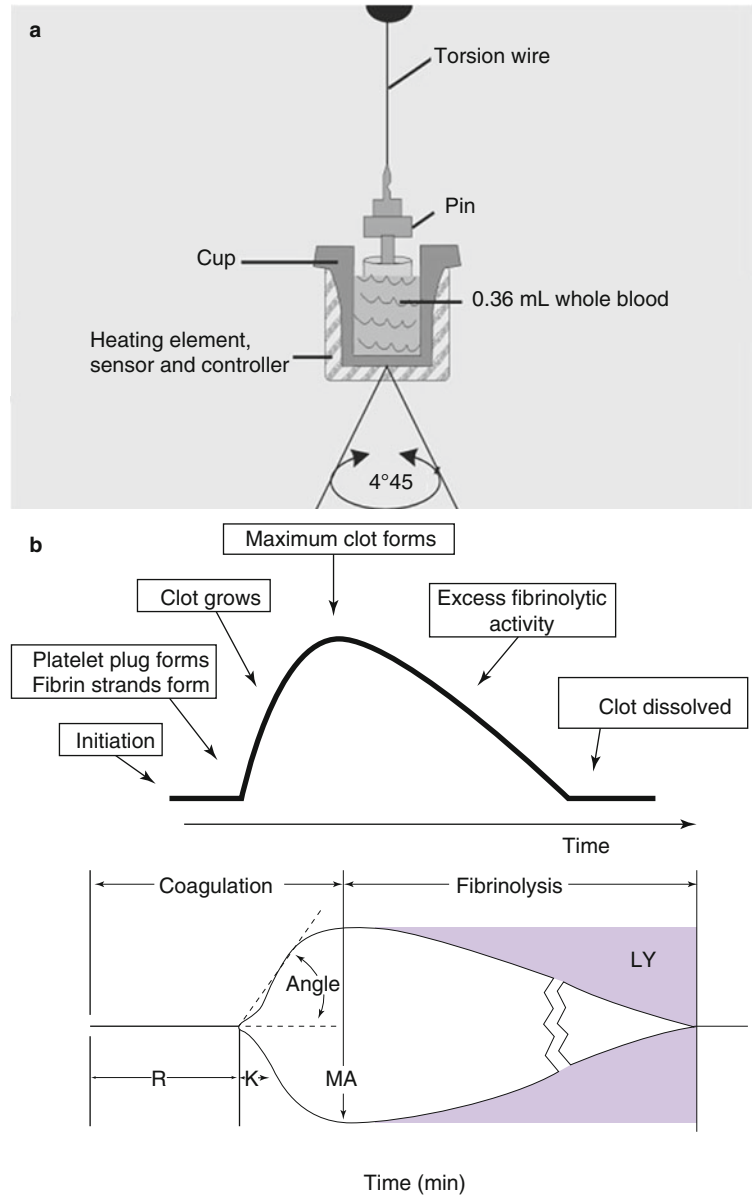


Fig. 12.11 (**a**) The TEG analyzer is composed of two main mechanical parts, which include a cup and a pin, and is separated by a whole-blood specimen. The cup rotates around the pin, and as clot formation ensues, the torsion wire attached to the pin detects the change in resistance, and a tracing is generated. (**b**) TEG tracing corresponding to clot kinetics from the initiation of clot formation to complete clot dissolution

signal that is amplified and records the characteristic tracing seen in Fig. 12.11. To initiate blood coagulation, TEG requires an activating solution consisting of kaolin (intrinsic pathway activator), phospholipids, and buffered stabilizers, which require an activation phase of several minutes before coagulation starts. The rapid-TEG assay, which uses an activating solution primarily including tissue factor (extrinsic pathway activator), further expedites the time to generate results, and clinically useful data concerning clot strength can be obtained as early as 10 min.

Other TEG assays also help diagnose coagulopathies attributed to platelet function as well as functional fibrinogen. Platelet mapping was designed to monitor the effects of platelet inhibitors (aspirin and $P2Y_{12}$ antagonists) on platelet function and works by measuring thrombin-independent coagulation following stimulation with either adenosine diphosphate

(ADP) or arachidonic acid (AA). This is achieved by the collection of blood in heparin and the addition of reptilase (which cleaves fibrinogen into fibrin in the absence of thrombin), allowing one to discern the individual effects of ADP and AA on platelet function. Platelet mapping has been employed to evaluate platelet function following trauma and demonstrated early significant platelet inhibition in severely injured patients [143]. The TEG-based functional fibrinogen (FF) assay allows for the discernment of fibrin and platelet components to clot strength. For this assay, platelets are inhibited by a GPIIb/IIIa antagonist, and the resultant thrombelastogram is reflective of the fibrin contribution to clot strength.

The various parameters of the TEG tracing are depicted in Fig. 12.12. The split point (SP, minutes) is a measure of the time to initial clot formation, interpreted from the earliest resistance detected by the analyzer causing the tracing to split. The reaction time (R, minutes) is defined as the time elapsed from the initiation of the test until the point where the onset of clotting provides enough resistance to produce a 2 mm amplitude reading. Of note, in the rapid-TEG (R-TEG) assay, due to the acceleration of clotting initiation, the R-time is represented by a TEG-derived activated clotting time (TEG-ACT). The R-time and TEG-ACT are most representative of the initiation phase of enzymatic clotting factors. Prolonged R-time or TEG-ACT is diagnostic of hypocoagulability, and decreased values may suggest hypercoagulability. The coagulation or kinetic time (K, minutes) is a measurement of the time interval from the R-time to the point where fibrin cross-linking provides enough clot resistance to produce a 20 mm amplitude reading. The alpha-angle (α[alpha], degrees) is the angle formed by the slope of a tangent line traced from the R-time to the K-time and reflects the kinetics of clot development. Both the K-time and the alpha-angle denote the rate at which the clot strengthens and are most representative of thrombin's cleaving of available fibrinogen to fibrin and the beginning of fibrin-platelet interactions. The maximum amplitude (MA, millimeters) indicates the point at which clot strength reaches its maximum measure in millimeters and reflects the end result of maximal platelet-fibrin interactions via GPIIb/IIIa receptors. The clot strength (G, dynes/cm^2) is a calculated measure of shear stress derived from amplitude (A, mm) making it the best measure for overall clot strength: $G = (5{,}000 \times A)/(100 \times A)$. Clot stability is determined by the percent of lysis 30 min following MA (LY30, %). An estimated percent lysis (EPL) value is given at any time point following MA until the actual LY30 is reached. The various TEG parameters and their significance are summarized in Table 12.7.

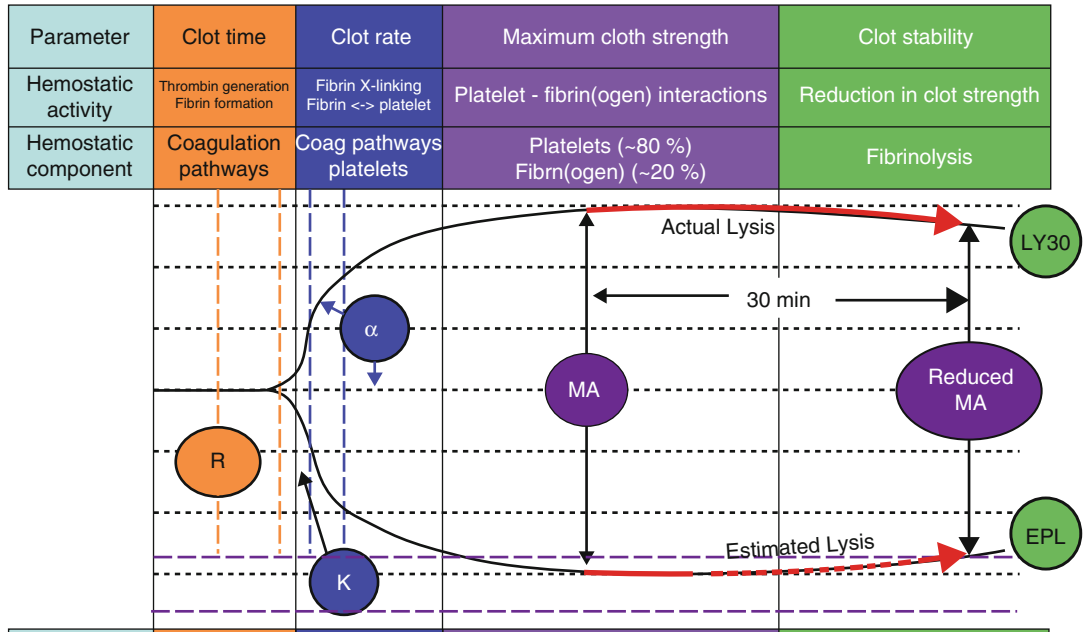
One of the benefits of viscoelastic hemostatic assays is the ability to take a complex and often abstract view of coagulation and generate specific tracing profiles that can be easily interpreted. Figure 12.13 depicts a normal TEG tracing and compares it to deranged TEG profiles with specific coagulation abnormalities. Patients may be hypocoagulable and prone to hemorrhage by having either low clotting factor function, low platelet function, low fibrinogen levels, or a combination of all three. Patients with low clotting factor function will have a prolonged R-time and K-time as well as a decreased α[alpha]-angle. Isolated low platelet function presents with a normal R-time and a decreased MA or G value. Low levels of fibrinogen may modestly prolong R-time and K-time, decrease α[alpha]-angle, as well as have a modest decrease in MA or G. Clinically significant fibrinolysis occurs when EPL or LY30 values exceed 15 %, resulting in a characteristic tapering of the TEG tracing immediately after the MA is reached. On the other hand, patients may become hypercoagulable by having increased platelet function and/or enzymatic activity and have TEG tracings with increased MA or G values and shortened R-times, respectively.

Rotational Thromboelastometry (ROTEM)

The ROTEM device is similar to TEG, except for a few key differences in machine technology and the reagents used. ROTEM uses a fixed cylindrical cup with an oscillating pin rotating through an

a

Parameter	Clot time	Clot rate	Maximum cloth strength	Clot stability
Hemostatic activity	Thrombin generation Fibrin formation	Fibrin X-linking Fibrin <-> platelet	Platelet - fibrin(ogen) interactions	Reduction in clot strength
Hemostatic component	Coagulation pathways	Coag pathways platelets	Platelets (~80 %) Fibrn(ogen) (~20 %)	Fibrinolysis
Hypo-coagulable	↑ R (min)	↑ K (min) ↓ a (deg)	↓ MA	LY30 > 7.5 % EPL > 15 %
Hyper-coagulable	↓ R (min)	↑ K (min) ↓ a (deg)	↑ MA	N/A



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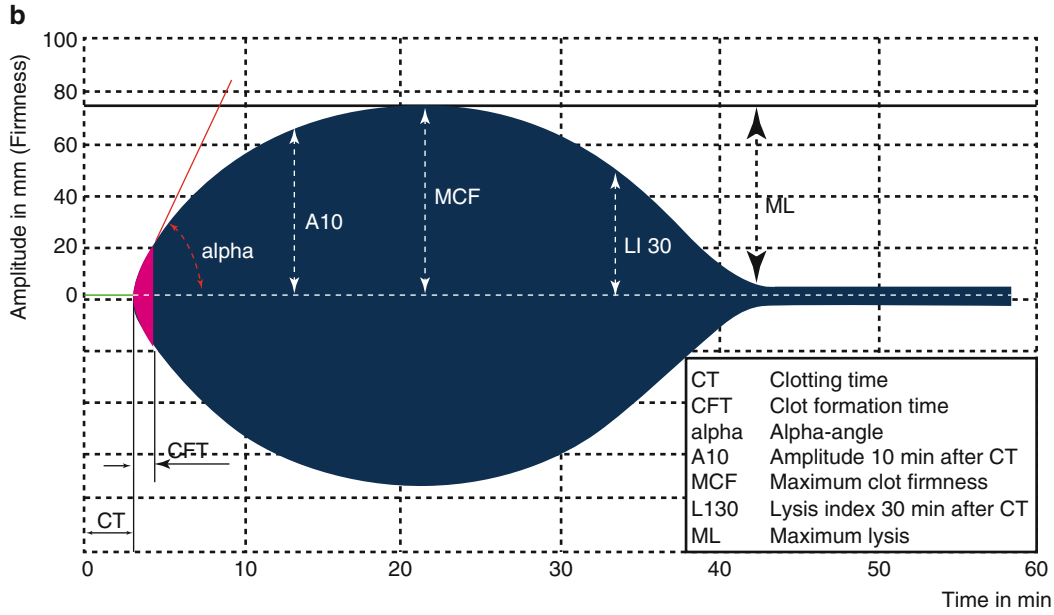

Fig. 12.12 (**a**) TEG parameters. (**b**) ROTEM parameters

Table 12.7 TEG/ROTEM parameters

TEG	ROTEM	Definition
SP (min)	N/A	Split point, earliest activity of enzymatic factors, causing tracing to split
R (min)	CT (s)	Reaction time, earliest measurable clot formation with 2 mm of amplitude
K (min)	CFT (s)	Coagulation time, potentiation phase of enzymatic factors achieving a certain clot firmness at 20 mm of amplitude
α[alpha]-angle (°)	α[alpha]-angle (°)	Kinetics of clot development reflecting the rate of clot development through fibrin cross-linking and the beginning of fibrin-platelet interactions
MA (mm)	MCF (mm)	Maximum amplitude, fibrin, and platelet contribution to clot strength through GPIIb/IIIa receptor
G (dynes/cm^2)	G (dynes/cm^2)	Maximum strength of clot, measured as shear stress
EPL (%)	N/A	Estimated percent lysis following MA
LY30 (%)	LI30 (%)	Percent lysis 30 min after MA (CT for ROTEM) is reached

angle of 4.75° rather than the cup rotating around the pin (Fig. 12.14). This rotation is detected optically via a mirror plate from a diode light source to a light-sensitive sensor. Therefore, when a clot is formed and attaches itself between the pin and cup surface, the movement is obstructed, which is detected by the sensor, and then converted to a representative tracing. In addition, slightly less blood is required for each ROTEM test (0.30 mL) compared to TEG (0.36 mL), and the ROTEM device is equipped with an electronic automated pipette to reduce pipetting error. As with TEG, ROTEM can also assess coagulation factor activity, fibrin/fibrinogen polymerization, anticoagulants, fibrinolysis, and platelet contribution to guide management. One limitation to ROTEM is the lack of a platelet function assay. However, an advantage of ROTEM is an assay, which employs aprotinin, to determine if fibrinolysis may be adequately reversed with a fibrinolysis inhibitor and may ultimately distinguish an acute endogenous coagulopathy from other coagulopathies. The commonly used ROTEM assays are the EXTEM (extrinsic pathway activator similar

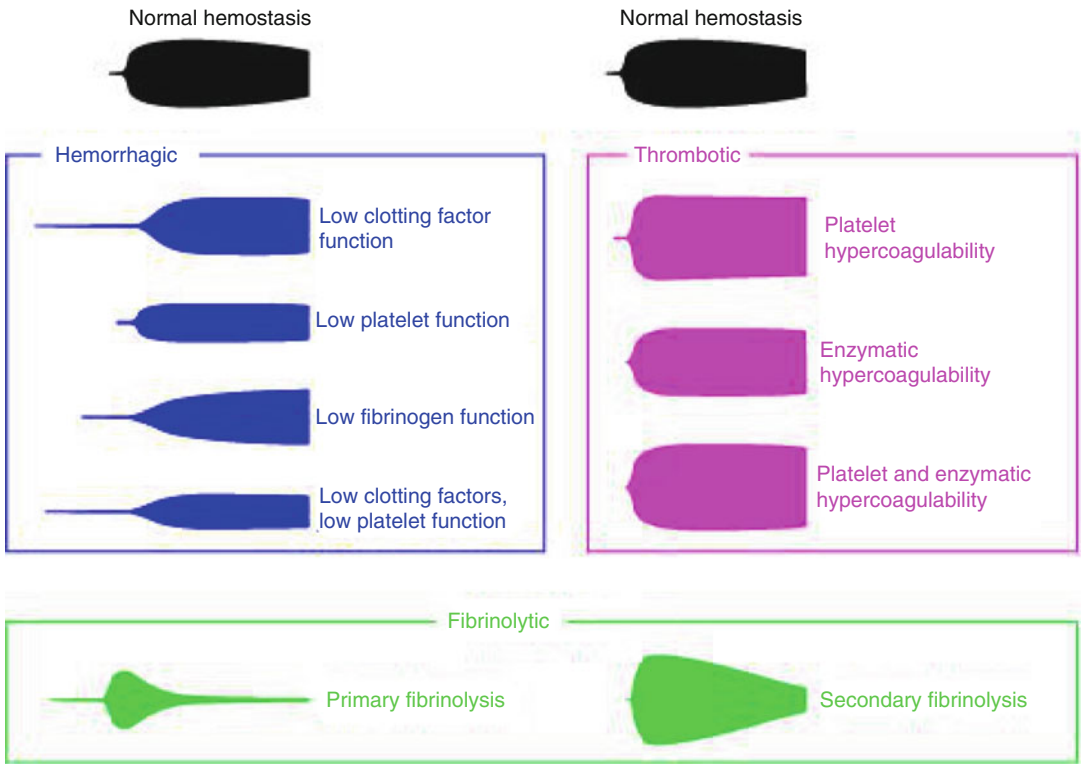

Fig. 12.13 Abnormal TEG tracings observed in hemorrhagic, thrombotic, and fibrinolytic states

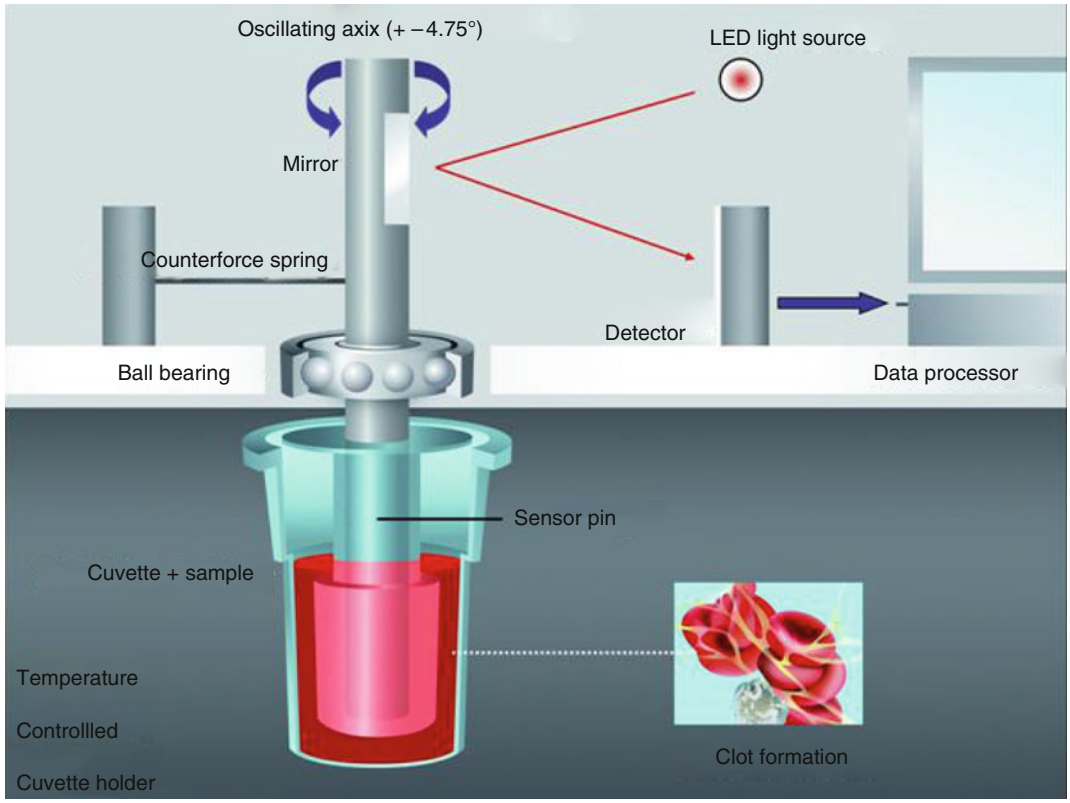

Fig. 12.14 ROTEM uses a fixed cylindrical cup with an oscillating pin rotating, as opposed to TEG in which the cup rotates around the pin. This rotation is detected optically via a mirror plate from a diode light source to a light-sensitive sensor

to the rapid-TEG), INTEM (intrinsic pathway activator similar to the kaolin TEG), FIBTEM (inhibits platelet function to determine the fibrinogen contribution to clot strength, which is similar to the TEG FF assay), and APTEM (fibrinolysis inhibitor, which determines if coagulopathy is reversible with aprotinin).

The various ROTEM parameters are similar to TEG parameters but with different nomenclature and different units of measurement (Fig. 12.12 and Table 12.7). The clotting time (CT, seconds) is the time from the beginning of the test until the time when an amplitude of 2 mm is achieved, reflecting the activity of clotting factors in the initiation of fibrin formation. Clot formation time (CFT, seconds) is the time between the 2 mm amplitude and the 20 mm amplitude points. The alpha-angle (α[alpha], degrees) is the angle between the middle axis and the tangent to the clotting curve through the 2 mm amplitude point. Both the CFT and α[alpha]-angle represent the kinetics of the formation of a stable clot through both activated platelets and fibrin. Maximum clot firmness (MCF, millimeters) is the maximum amplitude that is reached reflecting platelet-fibrinogen interactions. In addition, amplitude at 10 and 20 min may also be measured (A10 and A20) prior to MCF and is used for some resuscitation algorithms. The lysis index at 30 min (LI30) represents the percent of fibrinolysis 30 min following CT. This is different from TEG, which measures lysis 30 min following MA (LY30).

Goal-Directed Transfusion Therapy

Prior to the implementation of viscoelastic hemostatic assays in the trauma setting, much debate remained regarding empiric transfusion ratios of fresh frozen plasma (FFP), packed red blood

cells (PRBCs), and platelets. The literature varies greatly with reported improved outcomes of FFP:PRBC ratios greater than 1:1, 1:2, or 1:3 [143, 146, 162]. However, there were no comprehensive studies to evaluate the in vivo physiological changes in coagulation following the transfusion of these different ratios, and studies had to rely solely on patient outcomes without a great understanding on why these outcomes were better or worse. With the current understanding of acute endogenous coagulopathies, and subsequent secondary coagulopathies, and the limitations of traditional coagulation tests, both TEG and ROTEM assays have provided a tool to evaluate in vivo physiological changes in coagulation as a point-of-care test, which can provide rapid feedback to transfusion therapy and can ultimately guide transfusion therapy. Therefore, goal-directed transfusion therapy was established to adapt treatment to the individual patient based on viscoelastic hemostatic assays. Preliminary validation studies of these potential benefits have been encouraging. Retrospective data and pilot studies support a reduction in massive transfusion rates, decreased need for multiple and repeated classic coagulation tests, and decreased morbidity and mortality after implementation of viscoelastic hemostatic assays in trauma care [163–165].

Although viscoelastic hemostatic assay parameters provide detailed insight to the physiology of coagulation, there still remains some debate regarding therapeutic interventions for correcting abnormal parameters. This debate stems from the interpretation of the cell-based model of hemostasis, pathophysiology of the acute coagulopathy of trauma, and the availability and safety of therapeutic interventions. Philosophies in the management of coagulopathies have been sharply divided by the Atlantic Ocean, where many European countries take one approach and the United States has adopted, in large part, the other. European institutions have developed a stepwise algorithm, which has a primary emphasis on rapidly treating fibrinolysis through the use of tranexamic acid (TXA), then addressing the fibrin deficit with the use of fibrinogen concentrate, the thrombin generation deficit with prothrombin complex concentrate (PCC), and finally the platelet deficit with platelet transfusion (Fig. 12.15) [166, 167]. ROTEM is predominantly used in Europe, and consequently, most algorithms use ROTEM parameters.

Fibrinogen is addressed early in this algorithm, since fibrinogen levels are critically low in severely injured trauma patients [136, 168, 169]. The use of fibrinogen concentrate has several advantages to FFP and cryoprecipitate including a known, high concentration of fibrinogen, easy storage, and immediate use since it does not require thawing. Although used extensively in the treatment of post-injury coagulopathies in European countries, there is limited evidence that fibrinogen administration improves outcomes. Only one retrospective study suggests a survival benefit in patients who received a high fibrinogen/RBC ratio [170]. In the United States, fibrinogen concentrate is not available for use in trauma, and FFP and cryoprecipitate are used for fibrinogen deficiencies.

Decreased thrombin generation is uncommon in early post-injury coagulopathy and may actually be increased in severely injured patients [171, 172]. However, if present, the deficit should be corrected early by clotting factor administration. Current options for clotting factor replacement include FFP, PCC (which contains clotting factors II, VII, IX, and X as well as proteins C and S), and recombinant activated factor VII (rFVIIa). However, rFVIIa failed to show any survival benefit in two randomized controlled trials. In addition, PCC only contains a portion of the essential clotting factors, and there is little evidence supporting the use of PCC in trauma patients. One retrospective study suggested improved survival benefits in patients receiving a combination of fibrinogen concentrate and PCC, and another showed decreased transfusion rates of PRBCs and platelets in patients receiving coagulation factor concentrates [173, 174].

Platelets are addressed last in this algorithm and are only transfused if the platelet count is less than 50,000/μ[mu]L, and all other ROTEM parameters have been normalized. It is important to note that platelet counts only provide quantitative data on platelet number and do not

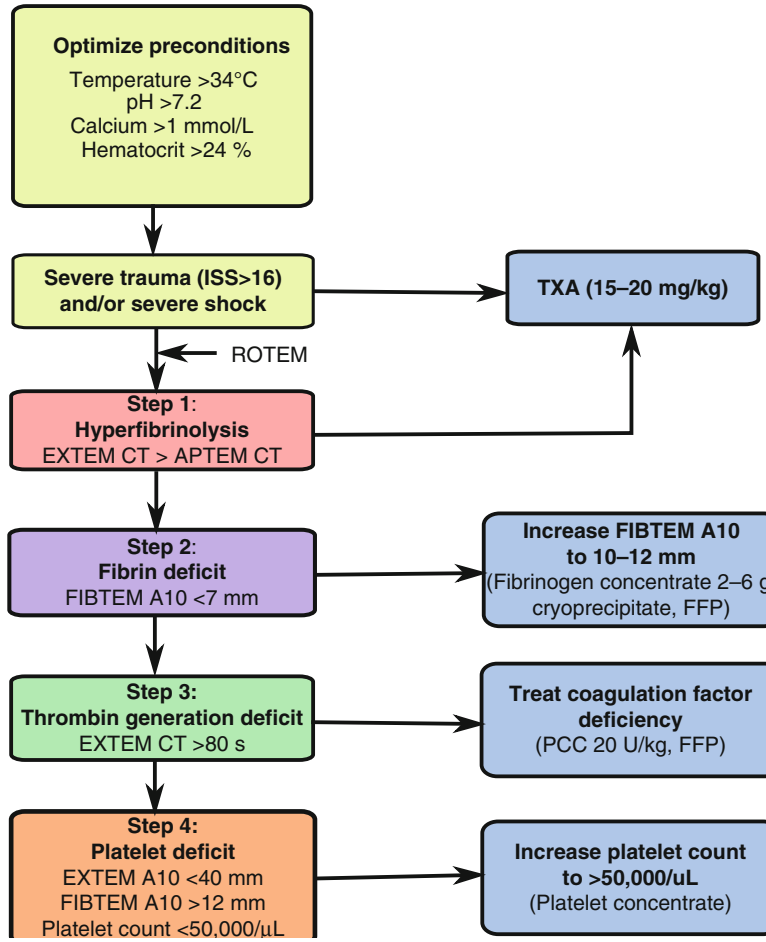

Fig. 12.15 European goal-directed transfusion algorithm

provide information regarding platelet function. It remains unclear if platelet dysfunction warrants platelet transfusion and can be used to ultimately guide platelet transfusion. Although few retrospective studies suggest a higher platelet/PRBC ratio improves survival, there are others that show no survival benefit [149, 175–177]. With the lack of definitive evidence, and the increased risk of MOF with FFP and platelet transfusions, many prefer the judicial use of these products.

In the United States, algorithms are primarily based on TEG parameters and reflect a different philosophy compared to the European algorithm (Fig. 12.16). A patient with post-injury coagulopathy is first identified by having evidence of clinical bleeding and confirmation of abnormal coagulation parameters by a rapid-TEG. In general, a sequential approach is taken. If a patient is identified as having hyperfibrinolysis (EPL >7.5 % on rapid-TEG), anti-fibrinolytics are immediately given. If there is no fibrinolysis, the enzymatic portion of thrombin generation is assessed next by the TEG-ACT. If the TEG-ACT is greater than 110 s, FFP is given, and the rapid-TEG reassessed. Once the TEG-ACT normalizes, the α[alpha]-angle (representing the rate of fibrin cross-linking and the beginning of fibrin-platelet interactions) is assessed, and if less than 66°, cryoprecipitate is given to supply additional fibrinogen. After the α[alpha]-angle exceeds 66°, attention is then turned to the MA, and if less than 54 mm, platelets will be given to improve overall clot strength regardless of platelet count. Although patients may have normal platelet counts following severe injury, these platelets become nonfunctional, have less correlation to

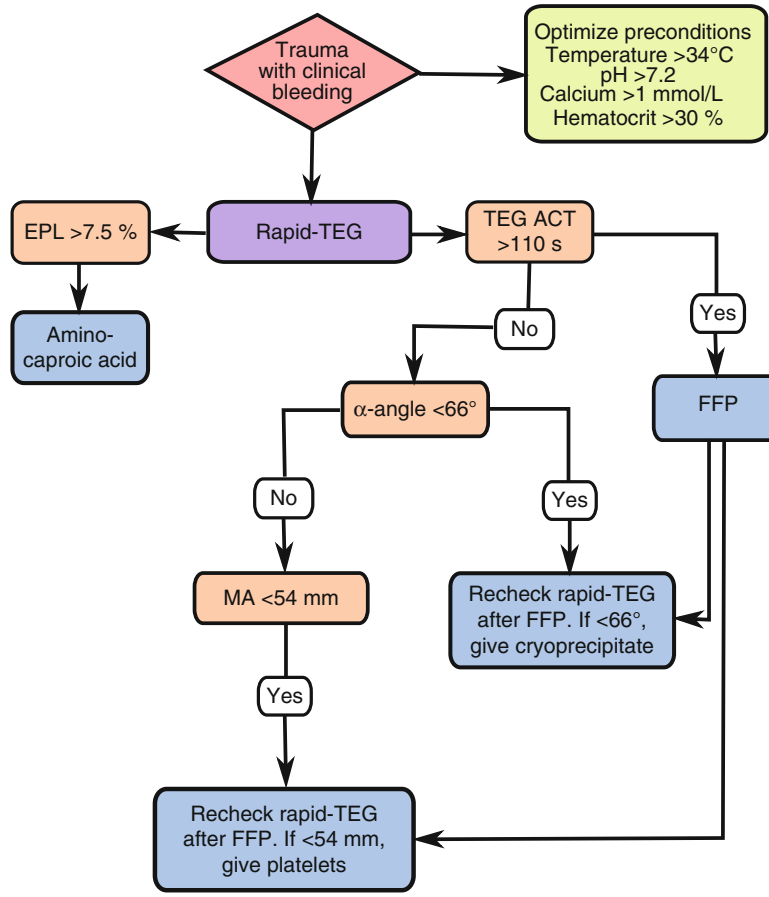

Fig. 12.16 The US goal-directed transfusion algorithm

clot strength, and may promote coagulopathy. In addition, the surfaces of functional platelets are necessary for the localization of clotting factors and, ultimately, thrombin generation. Although not used clinically at this time, the TEG functional fibrinogen assay may ultimately be used to assess both the fibrinogen and platelet contribution to clot strength and may further guide specific transfusion therapies. Ongoing studies are evaluating if the functional fibrinogen assay better reflects fibrinogen function, rather than α[alpha]-angle or k-time, and if the derived platelet contribution to clot strength will better reflect platelet function in the trauma patient. Therefore, the addition of this test may further augment the US goal-directed algorithm by giving cryoprecipitate or FFP earlier to patients with low functional fibrinogen levels or platelets to those with decreased platelet function and normal functional fibrinogen.

A limitation to these algorithms is that both have been established primarily on retrospective studies and limited by interpretations of the current data on the pathogenesis of post-injury coagulopathies. Consequently, randomized, clinically controlled trials are needed. Unfortunately, randomized trials are constrained in the United States by the availability of products approved by the Food and Drug Administration and the costs of factor concentrates. In spite of this, viscoelastic hemostatic assays have changed the paradigm of trauma resuscitation, are improving trauma outcomes, and are emerging as the standard of care in many countries.

Conclusion

The care of bleeding trauma patients with associated orthopedic injuries remains challenging. The combination of shock and tissue injury in these severely injured patients results

in multiple proinflammatory factors being released and/or produced. Unfortunately, these responses to severe injury lead to excessive inflammation resulting in worse outcomes, including multiple organ failure and death. As our understandings of post-injury physiological responses evolve, we are finding that there are multiple intersections between both the inflammatory and coagulation pathways, which explain the close and frequent association of shock with coagulopathy. However, the current evaluation and diagnosis of coagulopathies, as determined by plasma-based laboratory tests, have been limited in identifying both hypocoagulable and hypercoagulable states in post-injury trauma patients. Consequently, viscoelastic hemostatic assays are now the standard of care in identifying post-injury coagulopathies and have further elucidated links between inflammation and coagulation. Moreover, viscoelastic hemostatic assays may also prove to be the optimal devices to guide component resuscitation. Extensive research in resuscitation and the use of rapid point-of-care assays are necessary to further understand the complex pathophysiological responses to shock, especially in the trauma setting. Earlier, and appropriate, interventions, which minimize inflammation and decrease coagulopathies, may ultimately reduce transfusions and improve outcomes.

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Closed Head Injury

13

Philip F. Stahel and Michael A. Flierl

Background

Closed head injury (CHI) represents the leading cause of death in the trauma patient, ranking higher than mortality related to exsanguinating traumatic hemorrhagic shock [1–3]. One of the central aspects of our current understanding of the pathophysiology of CHI is that the extent of neurological injury is not solely determined by the traumatic impact itself, but rather evolves over time [4, 5]. The evolution of "secondary brain injury" is characterized by a complex cascade of molecular and biochemical reactions to the initial trauma which occur as a consequence of complicating processes initiated by the primary traumatic impact [6]. These events trigger an acute inflammatory response within the injured brain, leading to development of cerebral edema, breakdown of the blood-brain barrier (BBB), and leakage of neurotoxic molecules from the peripheral bloodstream into the subarachnoid space of the injured brain [7–10]. Ultimately, the extent of secondary brain injury, characterized by neuroinflammation, ischemia/reperfusion injuries, cerebral edema, intracranial hemorrhage, and intracranial hypertension, represents the main determinant for the poor outcome of head-injured patients [11]. In addition, iatrogenic factors, such as permissive hypotension, prophylactic hyperventilation, overzealous volume resuscitation, and inappropriate timing and technique of associated fracture fixation, may contribute to a deterioration of secondary brain injury [12, 13]. Despite recent advances in basic and clinical research and improved neurointensive care, no specific pharmacological therapy is currently available which may attenuate or prevent the development of secondary brain injuries [14]. Due to the complex underlying pathophysiology and the high vulnerability of the injured brain to "2nd hit" insults, it is imperative to closely coordinate the timing and surgical priorities for the management of associated injuries in head-injured patients.

The "Lethal Duo": Hypoxia and Hypotension

Episodes of hypoxia and hypotension represent the main independent predictive factors for poor outcome after severe brain injury [5, 15]. In a landmark article published in 1993, Chestnut et al. analyzed the impact of hypotension, as defined as a systolic blood pressure (SBP) <90 mmHg, either during the resuscitation phase ("early") or in the ICU ("late"), on the outcome of head-injured patients prospectively entered into the Traumatic

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Coma Data Bank (TCDB) [16]. Early hypotension occurred in 248 of 717 patients (34.6 %) and was associated with a doubling of post-injury mortality from 27 to 55 % [16]. Late hypotension occurred in 156 of 493 patients (31.6 %), of which 39 patients (7.9 %) had combined early and late hypotensive episodes. For 117 patients with an exclusive hypotensive episode in the ICU, 66 % either died or survived in a vegetative state, as defined by a Glasgow Outcome Scale (GOS) score of 1 or 2 points [16]. The authors furthermore determined that mortality is drastically increased in combination with hypotension (SBP <90 mmHg) and hypoxia (PaO_2 ≤60 mmHg) [17]. A more recent study by Elf et al. from 2003 confirmed the notion that severe secondary insults occur during the neurointensive care period in more than 35 % of all head-injured patients, including episodes of hypoxia, hypotension, elevated intracranial pressure (ICP), and decreased cerebral perfusion pressure (CPP) [11].

The prevention of hypoxemia and hypotension represents the "key" parameter for avoiding secondary insults to the injured brain and improving outcomes of CHI patients [15, 18]. National guidelines by the *Brain Trauma Foundation* mandate that blood pressure and oxygenation be monitored in all head-injured patients and advocate to maintain a systolic blood pressure >90 mmHg and a PaO_2 >60 mmHg, respectively [19]. This notion is of particular importance in view of the ongoing debate on the controversial concept of "permissive hypotension" in patients with traumatic hemorrhage from penetrating or blunt torso injuries [20, 21]. The strategy of "permissive hypotension" is mainly based on a landmark article from the 1990s advocating a modified prehospital resuscitation concept for hypotensive patients with penetrating torso injuries, by delaying fluid resuscitation until arrival in the operating room [22]. This proactive concept is certainly intuitive from the perspective that traditional resuscitation with aggressive fluid administration may lead to increased hydrostatic pressure and displacement of blood clots, a dilution of coagulation factors, and an undesirable hypothermia in critically injured patients [23]. However, in light of the vulnerability of the injured brain to secondary insults mediated by hypoxia and hypotension during the early post-injury period, the concept of hypotensive resuscitation, which has seen an unjustified expansion from penetrating to blunt trauma, in absence of high-level evidence [20, 24], appears contraindicated for patients with traumatic brain injuries [21, 25].

Initial Management Strategies

Head-injured patients are initially assessed and resuscitated according to the American College of Surgeons' *Advanced Trauma Life Support* (ATLS®) protocol [23]. The severity of head injury is diagnosed by the combination of (1) mechanism of trauma, the (2) clinical/neurological status, and (3) imaging by computed tomography (CT) scan. The neurologic status is assessed after stabilization of vital functions [26]. The level of consciousness is rapidly evaluated by the Glasgow Coma Scale (GCS), which grades the severity of TBI as mild (GCS 14/15), moderate (GCS 9–13), and severe (GCS 3–8) [4]. The post-resuscitation GCS score is of clinical importance due to the significant correlation with patient outcome [4]. A head CT should be obtained under the following circumstances: (1) altered level of consciousness with GCS <14 (moderate or severe brain injury), (2) abnormal neurological status, (3) differences in pupil size or reactivity, (4) suspected skull fracture, (5) intoxicated patients, and should be repeated whenever the patient's neurologic status deteriorates [4].

Elevated intracranial pressure (ICP) above 15–20 mmHg has been associated with poor outcomes after severe CHI [27]. Monitoring of ICP by indwelling catheters is recommended under the following conditions [28–31]:

- S*evere* CHI (GCS ≤8) and abnormal admission CT scan
- S*evere* CHI (GCS ≤8) with normal CT scan, and prolonged coma >6 h
- Surgical evacuation of intracranial hematomas
- Neurological deterioration (GCS ≤8) in patients with initially mild or moderate extent of CHI
- Head-injured patients requiring prolonged mechanical ventilation, e.g., for management

of associated extracranial injuries, unless the initial CT scan is normal

The indications and benefits of emergency craniotomy or decompressive craniectomy are beyond the scope of this book chapter, and the reader is deferred to the pertinent peer-reviewed literature [32–34].

Maintenance of an adequate cerebral perfusion pressure (CPP) is recommended above 70–80 mmHg, which is calculated as the mean arterial pressure (MAP) minus ICP [27, 29, 35]. This notion reflects on the imperative not to allow any period of hypotension in head-injured patients, as discussed above [15, 25]. In addition to the outlined dangers of hypoxemia and hypotension, hypercarbia and hypoglycemia should be strictly avoided or rapidly corrected to minimize the risk of developing secondary brain injuries [11]. Hyperosmolar therapy with mannitol or hypertonic saline is recommended for reduction of cerebral edema and increased ICP and in patients displaying clinical signs of transtentorial herniation, progressive neurological deterioration, or bilaterally dilated and nonreactive pupils [36]. However, the routine use of osmotherapy for management of brain edema represents a topic of heavy debate [37–39]. Similarly, the concept of therapeutic hypothermia for patients with severe head injuries remains controversial [34, 39, 40]. This noninvasive modality of neuroprotection has been investigated for decades in patients with head injuries, cerebrovascular stroke, cardiac arrest, and spinal cord injury [41]. The underlying rationale of moderately lowering the patient's body temperature is aimed at slowing down the acute inflammatory processes in the injured CNS and to reduce the extent of traumatic and ischemic tissue injury [42]. Interestingly, the historic euphoria in the 1990s for applying therapeutic hypothermia to patients with severe head injuries [43] was revoked later in additional validation studies, and the debate on the appropriateness of cooling down the injured brain remains unresolved until present [40, 44]. Despite increased understanding of the pathophysiology of secondary brain injury, the pharmacological "golden bullet" for treating CHI patients and preventing or reducing incidence of secondary cerebral insults has not yet been identified [14]. However, there is unequivocal consensus that the use of steroids is considered obsolete and contraindicated for patients with traumatic brain injuries, since the failure of the large-scale "CRASH" trial was published in 2004 [45, 46].

Head Injury and Long-Bone Fractures

Head-injured patients with associated orthopedic injuries represent a vulnerable population due to the high risk of "2nd hit" insults, particularly in presence of femur shaft fractures [13]. The benefits of early definitive fracture stabilization in multiply injured patients are well described and include early unrestricted mobility in conjunction with a decreased "antigenic load" related to stress, pain, and systemic inflammation [10, 47, 48]. Clearly, the question regarding the "optimal" timing and modality of long-bone fracture fixation in patients with associated head injuries remains a topic of ongoing discussion and debate [49–54]. Even though the benefits of early femur fracture stabilization have been unequivocally demonstrated in Larry Bone's landmark study more than 20 years ago [55], not all multiply injured patients are able to tolerate early definitive fracture fixation due to hemodynamic instability, refractory hypoxemia, or intracranial hypertension [53]. Impressively, experimental studies in sheep showed that femoral reaming and nailing leads to increased ICP levels above 15 mmHg in models of hemorrhagic shock/resuscitation with or without associated traumatic brain injury [56, 57]. A clinical study in 33 blunt trauma patients with CHI revealed that early definitive fracture fixation within 24 h was associated with adverse neurological outcomes and increased mortality, associated with early episodes of hypoxia and hypotension, compared to CHI patients whose orthopedic injuries were fixed at later time points (>24 h) [58]. A larger 10-year study on 61 patients with severe CHI revealed that early femur fracture fixation within <24 h is associated with an increased incidence of secondary brain injury, related to

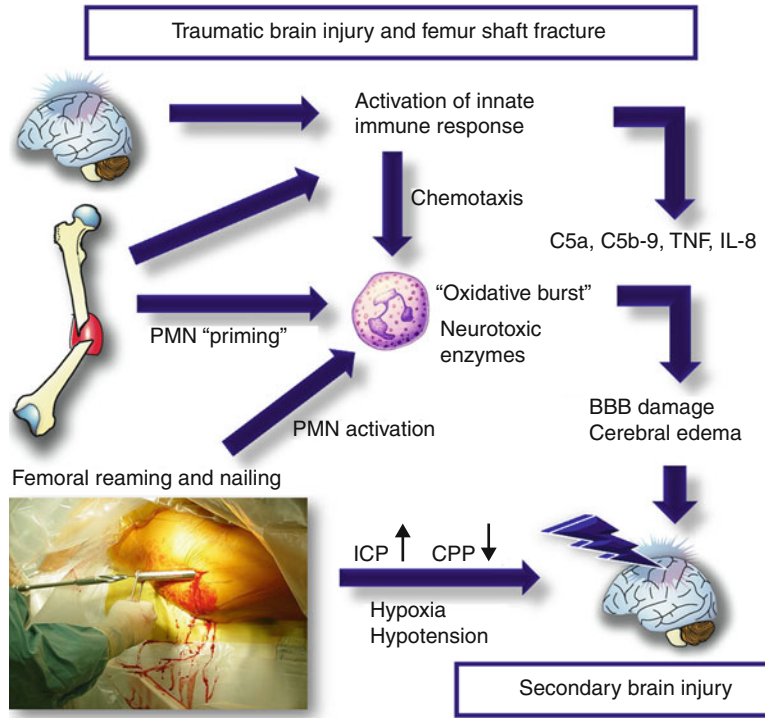

Fig. 13.1 Pathophysiology of the inflammatory cascade and iatrogenic "2nd hit" insults leading to secondary brain injury

significantly increased rates of hypotension and decreased CPP <70 mmHg [59]. These data were corroborated by a different study analyzing changes in ICP and CPP in 17 patients with severe head injuries undergoing reamed intramedullary nailing of associated femur fractures [60]. The authors showed that the CPP dropped below a minimal threshold of 75 mmHg intraoperatively during the fracture fixation in all patients, with an average decrease in CPP of Δ18 mmHg [60]. The decrease in CPP was attributed to intraoperative episodes of systemic hypotension, and patients with early femoral nailing within 24 h had statistically significant lower CPP values than the rest of the cohort [60].

Overall, there is unequivocal evidence—both from experimental animal studies and from clinical trials in patients with severe CHI—that the early (<24 h) definitive fixation of associated femur shaft fractures in head-injured patients leads to significant adverse effects, including intraoperative episodes of hypotension, increases in ICP, and critical decreases in CPP, all of which ultimately constitute preventable "2nd hits" and contribute to secondary brain injury and poor long-term outcomes (Fig. 13.1).

Consequently, alternative strategies to provide early fracture stabilization of long bones, while avoiding the risk of "early total care," have been proposed, including skeletal traction and "damage control" external fixation [61]. The concept of "damage control" surgery was extended beyond its initial applications in abdominal and thoracic trauma, to the acute management of major fractures in the severely injured, particularly in presence of associated head injuries [53, 62]. The principle is to provide early fracture stabilization by external fixation as a bridge to definitive fracture care once the patient is physiologically stable and the injured brain less vulnerable to iatrogenic "2nd hit" insults [13]. The delayed conversion from external fixation to intramedullary nailing of femur shaft fractures is considered safe once the ICP has normalized and/or patients are awake, oriented, and fully resuscitated [23]. In other words, the second procedure-related intramedullary reaming and nailing of long-bone fractures should be performed outside of the "priming" window, once

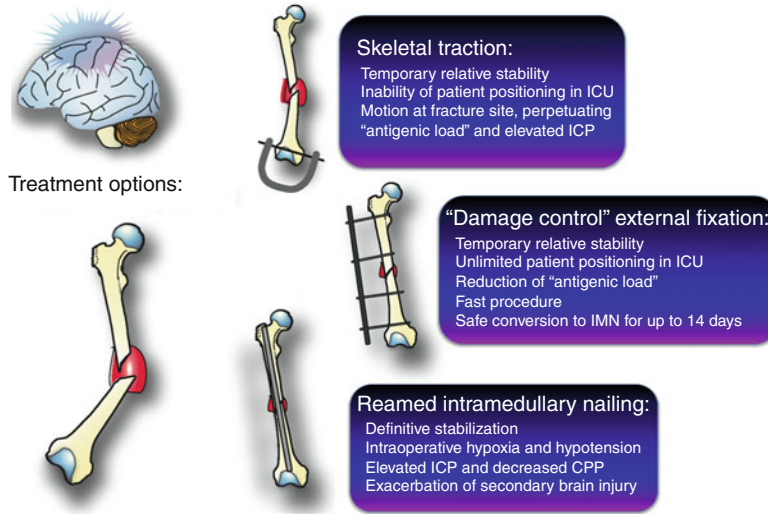

Fig. 13.2 Risks and benefits of different acute management strategies for immobilizing femoral shaft fractures in head-injured patients

the post-injury hyperinflammatory response has subsided (Fig. 13.1). When compared to early total care, the "damage control" approach with delayed conversion to definitive care has been shown to decrease the initial operative time and intraoperative blood loss without increasing the risk of procedure-related complications such as infection and nonunion [63, 64].

The pros and cons of the three main modalities for acute management of femur shaft fractures in head-injured patients, namely, (1) skeletal traction [61], (2) "damage control" external fixation [62, 63], and (3) "early total care" by reamed intramedullary nail fixation [60] are depicted in Fig. 13.2.

Conclusion

Head-injured patients with associated long-bone fractures represent a very vulnerable patient population [49]. These patients have a high risk of sustaining secondary cerebral insults related to hypotension, increased ICP, and decreased CPP, all of which contribute to increased mortality and adverse neurological outcomes [56–60]. The involved specialties in the early management of multiply injured patients with head injuries and associated long-bone fractures, including ED physicians, trauma surgeons, neurosurgeons, and orthopedic surgeons, must "speak the same language" in terms of understanding the underlying pathophysiology of CHI and the time-dependent vulnerability of the injured brain to iatrogenic "2nd hit" insults [4, 13]. Despite recent advances from basic research and clinical studies, which improved our current understanding of the pathophysiology of CHI, the current literature remains conflicting in terms of identifying a clear-cut management strategy for timing and modality of fracture fixation in severely head-injured patients [13, 50, 51, 54, 58, 59]. This notion emphasizes the pressing need for well-designed, prospective, controlled multicenter trails aimed at comparing the standard treatment strategies for initial management of long-bone fractures in patients with severe head injuries (Fig. 13.2). Until higher-level evidence-based recommendations are available, the clinical approach for the management of this vulnerable cohort of patients must be based on the basic principle of *do not further harm* by applying simple measures of "damage control"—when in doubt—which respects the underlying pathophysiology of traumatic brain injury and the hyperinflammatory response of the combination of multiple critical injuries [10]. We recommend the following specific management strategy for

associated long bone fractures in head-injured patients, based on a combination of empiric experience and review of the available pertinent literature in the field [13, 49]:

1. *Damage control orthopedics*" by spanning external fixation in all patients with *severe* CHI (GCS ≤8 and intracranial pathology on CT scan, including cerebral edema, midline shift, sub/epidural bleeding, or open head injuries)
2. Optional *damage control orthopedics* in all patients with *moderate* CHI (GCS 9–13) or patients with GCS of 14/15 with "minor" intracranial pathology on CT scan (e.g., traumatic subarachnoid hemorrhage that warrants observation only)
3. *Conversion from external to internal fixation* in CHI patients who are awake and alert (GCS 13–15) or comatose patients with a stable ICP (<15 mmHg) and CPP in a normal range (>80 mmHg) for more than 48 consecutive hours
4. *Early total care* for long-bone fractures all patients with *mild* CHI (GCS 14/15) and normal initial craniocerebral CT scan
5. *Temporary skeletal traction* as a valid adjunct for patients "in extremis", i.e., in severe protracted traumatic hemorrhagic shock and coagulopathy, who are unsafe to be taken to the operating room until adequately resuscitated

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Chest Trauma and Lung Contusions

14

Jeffrey L. Johnson

Introduction

Injury to the thorax contributes uniquely to outcome in the multitrauma patient with orthopedic injuries. It has long been recognized that chest injury is an independent predictor of morbidity and mortality in this setting [1]. That being said, the precise factors in thoracic trauma and extrathoracic bony injury that combine to put patients at risk remain an important area of investigation. Two themes emerge. First, primary pulmonary injury from an injured chest wall and lung makes operative management of bony injuries potentially unsafe. For example, a severe pulmonary contusion resulting in marked hypoxemia is a pivotal organ dysfunction that may increase the risk of any planned intervention. Second, the systemic response effected by blunt multitrauma, and exacerbated by bony injury, puts the lung at risk for a secondary inflammatory injury manifesting as the acute respiratory distress syndrome (ARDS). This second phenomenon, though incompletely understood, has substantial implications about the proper timing and nature of our interventions.

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Injury Severity Scoring

The most common descriptive tool in use today remains the *Injury Severity Score* (ISS). This provides a framework for description of the anatomic extent of injuries. For each of six defined body regions, individual injuries are assigned an *Abbreviated Injury Scale* (AIS) graded from zero (no injury) to six (unsurvivable). The highest AIS for each of three regions is squared and added to calculate the overall ISS. The AIS assigned to common chest injuries is shown in Table 14.1. While this system provides a common language to describe individual patients and allows for comparisons between groups, it does have some drawbacks for initial decision making. First, it largely ignores the underlying physiology of the patient. For example, while the number of rib fractures certainly has some correlation with the physiology of the patient, the ability of any individual patient to execute the work of breathing cannot reliably be predicted based on number of fractures alone. Second, not every injury is

Table 14.1 Abbreviated injury scale for some common chest injuries

Injury description	AIS
Pulmonary contusion	3
Fracture <3 ribs	1–2
Flail chest, unilateral	3
Blunt cardiac injury, minor ECG changes	3
Torn descending thoracic aorta	4–5

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immediately recognized and categorized; so many times, the "final" AIS/ISS is not appreciated until further imaging, assessment, or repair is done. Other scoring systems specific to chest injuries may have more utility for early decision making and are discussed later in this chapter.

Epidemiology

Thoracic injury is the primary cause of death in about 1 of 4 patients who succumb to trauma and contributes to the death of another 1 out of those 4 [2]. Most mortality directly attributable to chest trauma occurs very early (within minutes of injury) due to major cardiovascular disruption or major lacerations of the tracheobronchial tree. Excluding these early deaths, less than 5 % of patients with blunt thoracic injuries will require an operative intervention in the chest. For example, in a study of over 1,500 patients with combined blunt thoracoabdominal injuries, only 4.3 % of patients underwent thoracotomy (excluding resuscitative thoracotomies) [3]. For practical purposes, tube thoracostomy is the most invasive thoracic procedure needed in the vast majority of patients. Despite the rarity of operative intervention, patients with major chest injuries frequently have major cardiopulmonary dysfunction.

Blunt mechanisms of thoracic injury predominate in most centers, with motor vehicle accidents accounting for the vast majority. Penetrating injuries are less common and are likely to be of limited interest to those clinicians enjoying this book; they will not be discussed further. Blunt chest injuries usually occur in association with multiple injuries to other anatomic regions. Indeed—patients with major thoracic injuries typify the multiply injured patient. For example, in a 1987 study of over 500 patients admitted to the Maryland Institute of Emergency Medical Services with blunt chest injuries, only 16 % of patients had injuries limited to the chest [4]. In the Quebec trauma registry, approximately 25 % of patients with chest trauma had concomitant abdominal injuries [5]. The structures most commonly injured in the chest are the ribs, the pleura, and the lung. Major cardiac and vascular injuries, while certainly important, are in fact uncommon in patients who survive the initial insult. Blunt esophageal injuries are vanishingly rare. A summary of the distribution of injuries from three large studies is provided in Table 14.2.

Table 14.2 Relative frequency of injuries in the chest in three large published series

Injury	Cited frequency (%)
Rib fractures	35–64
Pulmonary contusion	16–30
Hemo-/pneumothorax	11–50
Flail chest	5–10
Heart/great vessels	2–6

Given that thoracic injuries are rarely an immediate threat to life in the patient who survives the initial insult, the primary challenge for the clinician is optimizing supportive care of the cardiopulmonary system and preventing pulmonary complications such as pneumonia, ARDS, fat emboli syndrome, and prolonged ventilator dependence. The sequelae of both direct and indirect cardiopulmonary injury can substantially complicate the care of multiply injured patients. In the Hannover experience, outcome was described in 278 multiply injured patients with chest trauma (ISS > 15 and Chest AIS >2, excluding severe brain injuries) [6]. They found that length of stay averaged 33 days and rates of pneumonia and ARDS and multiple organ failure (MOF) were 22 and 13 %, respectively. In general, then, given the likelihood of prolonged and complex hospital course, the optimal care of a patient with orthopedic injuries and concomitant thoracic injuries involves proper risk assessment (is it safe to take this patient to the operating room?) and planning interventions (is the secondary insult from an orthopedic procedure likely to worsen cardiopulmonary physiology?).

Pathophysiology of Pulmonary Dysfunction

Multiply injured patients are at risk for major pulmonary dysfunction because of disruption of three key elements. First, brain injury is common

in patients with chest injuries, resulting in inadequate respiratory drive, or inability to maintain patent proximal airways. Second, injury to the torso can produce changes in compliance, ineffective respiratory effort, and pain that impact the patient's ability to complete the work of breathing. Third, insults to the lungs themselves result in ineffective gas exchange and hypoxemia. In polytrauma patients, it is likely the clinician must consider simultaneous insults affecting all three elements. Impaired airway patency (e.g., diminished level of consciousness), increased work of breathing (e.g., multiple rib fractures), and impaired gas exchange (e.g., pulmonary contusion, fat emboli syndrome) often coexist.

At the same time that these patients experience impaired gas exchange, they actually have a marked increase in respiratory demand because the neurohormonal response to injury results in increased cellular metabolism. This creates a substantial increase in CO_2 production that must be matched by increased elimination from the lungs. While a resting adult eliminates 200 cc/kg/min of CO_2, postinjury hypermetabolism results in CO_2 production in the range of 425 cc/kg/min [7]. Thus, the minute ventilation required to maintain a normal pH may rise from a resting rate of approximately 5 L/min to more than 10 L/min. This represents a 100 % increase in ventilation simply to meet metabolic demands. To make matters worse, injured patients typically have an increase in physiologic and anatomic pulmonary dead space—ventilated regions of the lung that do not participate in gas exchange. In a normal adult, the proportion of each breath that is dead space (V_d/V_t) is approximately 0.35. For injured patients with pulmonary failure, the V_d/V_t often exceeds 0.6. Simply put, extra dead space means each breath is less effective at eliminating CO_2. Therefore minute ventilation requirements in the 12–20 L/min range are not uncommon in the postinjury setting.

In this light, secondary insults that further impair gas exchange or further increase metabolic rate may cause a stable patient to decompensate; as discussed below, orthopedic interventions are uniformly associated with worsening gas exchange. This makes timing of bony fixation a challenging puzzle. If we do not definitively repair fractures, we impair the respiratory system by immobilizing the patient (impaired work of breathing, increasing dead space, ineffective cough). Alternatively, If we opt for definitive fixation in a tenuous patient, we may impair the pulmonary system by worsening gas exchange and increasing metabolic demand.

The patient with chest injuries faces hurdles in meeting increased respiratory demand. Respiratory drive may be impaired by brain injury and by medications routinely used for sedation and analgesia. The energy required to complete a respiratory cycle is increased by chest wall edema and recumbent positioning, which is often prolonged in patients with major bony injuries. Muscular weakness from impaired energetics (acidosis, cardiovascular failure, mitochondrial dysfunction, oxidant stress) or fatigue may be an insurmountable challenge. Decreased pulmonary compliance from an increase in extravascular lung water and pleural collections (effusions/hemothorax) also contributes. Lastly, and perhaps most importantly, pain from torso injuries or operative interventions make the increased ventilatory demand a substantial burden to the patient.

Primary Injury Patterns

Rib Fractures and Flail Chest

Rib fractures are the most commonly identified chest injury in the multiply injured patient. Crude rates of morbidity and mortality are consistently associated with the number of broken ribs, particularly in elderly patients [8, 9]. Patients with multiple rib fractures are thought to be at high risk for pulmonary failure and pneumonia—likely from impaired cough, atelectasis from splinting, and inability to execute the work of breathing if pain control is poor. A recently published analysis of over 40,000 patients queries this association a bit more closely [10]. This work by Jones et al. highlights that in patients whose only injuries are rib fractures, mortality is less than 6 %. Further, when early (<24 h) deaths

are excluded, crude mortality, while still related to number of fractures, is less than 10 % across all groups. The most powerful predictor of mortality was the abbreviated injury score for the chest region, reflecting the potential importance of flail chest (below) and injuries involving the pleural space, lung, and mediastinal structures. The theme again here is that patients who surviving long enough to warrant orthopedic interventions are unlikely to die from their thoracic injury, and thus minimizing secondary insults becomes pivotal in achieving excellent outcomes.

Flail chest is a pattern of injury wherein a portion of the chest wall loses bony continuity with the rest of the respiratory pump. This most commonly occurs when multiple adjacent ribs are fractured in more than one location. It can also occur in association with sternal fractures or disruptions of costochondral junctions. When the patient expands their chest to take a breath, creating negative intrathoracic pressure, the disconnected area ("flail segment") moves inward in a paradoxical fashion. Particularly when this injury occurs in concert with a major loss of thoracic volume ("caved in chest"), the expansion of the underlying lung is attenuated, and there may be decreased effective tidal volume and therefore impaired ventilation.

Acutely, flail chest injuries per se are not frequently an early threat to life, with mortality reported as less than 10 % in modern series [11, 12]. In isolated flail chest, for example, most patients will not need mechanical ventilation. The major initial challenge is pain control and pulmonary hygiene as ineffective cough and ability to execute increased ventilatory demands are common. In long-term follow-up [13], this is a morbid injury pattern to be sure, as chronic pain, chronic dyspnea, and disability are a common outcome. The significance of a flail chest in the acute setting largely relates to the fact that it denotes major energy transfer to the thorax. This is particularly true in younger patients, where ribs are relatively elastic—more likely to transiently deform than to fracture. Major bony injuries to the chest wall in a young patient with a major mechanism of injury (e.g., high-velocity motor vehicle crash) signify a high likelihood of underlying pulmonary contusion (see below) and extrathoracic injuries [14].

Supportive care remains the mainstay of treatment in rib injuries, with or without flail chest, though there is renewed and justified interest in rib fixation. Acute mortality for chest wall injury is low, but long-term morbidity is substantial and is largely related to malunion—which would appear eminently preventable. Design of rib-specific hardware permits a more practical approach, and newer techniques involving plates with some elasticity as well as minimally invasive approaches may continue to fuel enthusiasm for operative treatment. A number of small published series suggest improved short-term outcomes [15, 16]. The current challenge is identifying patients who are likely to substantially benefit. For example, the patient with other major injuries that may result in prolonged ventilator dependence (brain injury, open abdomen, spinal cord injury) may not benefit acutely or long term from chest wall fixation.

Pulmonary Contusion

Pulmonary contusion, simply put, is a bruise of the lung. The most common presentation is a young passenger struck on the nearside compartment; rapid deceleration and frontal crashes into fixed objects are frequent contributors [17]. Three different types of forces combine to produce injury to the lung. First, direct transmission of energy through the chest wall can bruise the lung. Secondly, the lung can be bruised by shearing forces, for example, when a high-energy missile passes through the lung parenchyma, there is a zone of contusion around the tract of the missile. Thirdly, blast or concussive injury can produce significant lung contusion without obvious chest wall damage. An isolated pulmonary contusion is pathologically similar to bruises elsewhere. The initial response is edema and hemorrhage. This is followed by inflammation, recruitment of cellular elements to the zone of injury, and then by repair. The clinical course follows a similar pattern. As the swelling and inflammation evolves, there is worsening of

pulmonary compliance and gas exchange. This continues for 48–72 h after which improvement should be expected. Some mild hemoptysis can be expected as hemorrhagic secretions are cleared from the distal airways.

An initial chest x-ray is diagnostic in patients with large contusions. Smaller injuries may not become evident until later—when swelling and inflammation occur. Approximately one third of patients with blunt chest injuries will have evidence of pulmonary contusion on Computed Tomography (CT) that was not appreciated on initial plain radiographs [18]. CT has thus been promulgated as a more sensitive tool for diagnosis, and a number of scoring systems have been developed. Strumwasser et al. [19] analyzed 106 consecutive patients undergoing CT of the chest for blunt multitrauma. They observed that a computed tomography volume index (estimating the fraction of total lung involved by contusion) was an independent predictor of ICU length of stay. Additionally, pts with a CT volume index >0.2 had, on aggregate, a higher risk of pneumonia, ARDS, and death. In a larger retrospective series from Boston (almost 400 pts), a score of 1–6 was used, based on presence or absence of contusion in three zones of each lung [20]. They observed that mechanical ventilation was required more often in patients with a score >2, and this was an independent risk factor for the need for ventilation (odds ratio = 13); this can be thought of as 50 % or more of lung zones involved with contusion. That being said, only 35 % of patients with BPC6 >2 required mechanical ventilation. Additional factors also predictive of mechanical ventilation included diminished Glasgow Coma Scale (GCS) score and >4 rib fractures. Wang et al. observed that PC volume predicted chest trauma patients who would later meet criteria for adult respiratory distress syndrome [21].

It would seem logical, given these studies, that patients with large pulmonary contusions evident on radiographs should be recognized as at increased risk for secondary pulmonary insults. A different and as yet unanswered question is whether broad application of CT scanning for blunt chest injuries is cost effective for pulmonary contusion, as management is entirely expectant and treatment entirely supportive. Some studies strongly suggest that contusions identified only on CT scanning are of limited clinical significance [22]. Further, areas of dependent edema, consolidation, or aspiration pneumonitis may be mistaken for contusion. Certainly, if imaging is already done and available, it should be used to guide decision making, but in the tenuous patient, a trip to CT scan may represent an unnecessary risk.

Early evolution of the patient's gas exchange must be taken into account. Patients with early (<6 h) impairment in oxygenation should be approached with caution. The most common tool for describing impairment in oxygenation is the ratio of arterial partial pressure of oxygen (paO_2—the tension of oxygen in the blood) to the percentage (fraction) of oxygen the patient is inhaling (FiO_2—how much oxygen is the patient on). This is commonly referred to as the P/F ratio. At sea level, a normal P/F is about 400. Impairment in gas exchange results in progressively lower values, with mild impairment being <300, moderate <200, and severe <100.

Blunt Cardiovascular Injuries

Like the lung, the heart may be bruised by direct, shear, or blast forces. Since the true "gold standard" for myocardial contusion would be direct examination or biopsy, it is difficult to assess any particular diagnostic approach for sensitivity and specificity. Thus, for practical purposes, one should consider that there are only two common sequelae of blunt cardiac injury: arrhythmia and pump failure. Many of the arrhythmias associated with blunt trauma are relatively benign (sinus tachycardia, atrial fibrillation), and an initial EKG that is normal is associated with a very low chance of a malignant arrhythmia [23]. Thus, an early EKG can be advocated to identify patients at risk. With respect to pump failure, the most common cause is a major contusion of the right ventricle (which lies more anterior), and this typically presents early as hypotension refractory to volume replacement. In these rare cases, early echocardiography can be recommended to confirm the

diagnosis. In patients without clinical evidence of pump failure, the utility and clinical significance of cardiac enzyme measurement, while advocated by some, is a matter of some debate.

Other cardiovascular injuries such as great vessel injury, pericardial rupture, and cardiac rupture are remarkably rare and are largely beyond the scope of this chapter. One injury worth mentioning both in terms of incidence and significance is the torn descending thoracic aorta. While historically described as an immediate threat to life, many of these injuries can be safely observed in the stable patient, and intervention planned for a time when the patient is physiologically well enough to sustain an additional insult [24–26]. Minimally invasive approaches using stent grafts are rapidly replacing operative repair, though long-term follow-up is far from complete. There are some concerns about the ultimate fate of stent grafts placed in young patients, as graft collapse and migration have been described, and it is unclear what will evolve as the young aorta gradually dilates with age and a fixed stent graft remains in place. With respect to operative repair of the torn aorta, many of the risk factors that make any secondary procedure potentially unwise apply: major pulmonary contusion, poor gas exchange, and injuries that would make anticoagulation contraindicated.

Secondary Pulmonary Injury

Acute Respiratory Distress Syndrome (ARDS)

The syndrome of ARDS was outlined in a small series of patients by Ashbaugh and Petty in 1967 [27], and the essence of this description remains today. The main components in the clinical setting include (1) hypoxemia refractory to oxygen administration; (2) diffuse, bilateral infiltrates on imaging of the lungs; and (3) decreased lung compliance. A standard definition of ARDS has been in use for almost 20 years. The Consensus Conference of North American and European investigators (NAECC) agreed that ARDS should be viewed as the most severe end of a spectrum of an acute lung injury (ALI) [28]. The diagnostic criteria for ARDS include acute onset, the PaO_2/F_iO_2 200 mmHg or less (<300 for ALI), bilateral infiltrates on chest radiograph, and no evidence of left atrial hypertension (either clinical or with direct measurement). During the last two decades, some limitations of this definition have been apparent, including an unclear meaning of "acute," the unclear role of transient changes in the P/F ratio in establishing the diagnosis, and potential inclusion of a broad array of patients with hypoxemia. Additionally, recruitment of collapsed lung tissue (predominantly with positive end-expiratory pressure, PEEP) may result in a remarkable improvement in P/F ratio in a short period of time—does this patient no longer have the syndrome ARDS? Lastly, while the NAECC definition excludes patients with left atrial pressure (LAP) >18, Ferguson et al. [29] showed that patients with no risk factors for congestive heart failure but with a clinical syndrome of ARDS commonly had LAP >18.

Table 14.3 Summary of the proposed "Berlin" definition of ARDS

Factor	Description
Onset	Within 1 week of known risk factor
Imaging	Bilateral opacities not explained by effusion, collapse, or nodules
Type of pulmonary edema	Not explained by cardiac failure or fluid overload. If no clinical risk factor identified, objective assessment required
Severity (with PEEP≥5)	Mild: P/F ≤ 300 Moderate: P/F ≤ 200 Severe: P/F ≤ 100

Recently proposed changes to the ARDS definition have been developed using consensus methodology in a series of meetings in Germany. This new "Berlin definition" of ARDS is likely to be widely embraced and offers significant advantages over the 1994 definition [30]. In particular, it defines "acute" more precisely, drops the term ALI (which may be confused as a separate entity), and provides for a larger consideration of precipitating factors when CHF and ARDS may coexist. This modern definition of ARDS is shown in Table 14.3.

Clinical risk factors for ARDS can be broadly categorized into direct and indirect groups. Direct

Table 14.4 Commonly observed risk factors for the adult respiratory distress syndrome

Direct	Indirect
Pulmonary contusion	Severe sepsis
Aspiration	Severe trauma
Pneumonia	Pancreatitis
Pulmonary ischemia/reperfusion	Extrapulmonary (e.g., splanchnic) Ischemia/reperfusion
Fat emboli syndrome	Transfusion

factors are those primarily associated with local pulmonary parenchymal injury and include pulmonary contusion, aspiration, and pulmonary infection. Indirect factors are those thought to be associated with systemic inflammation and resultant lung injury. These include severe sepsis, transfusion of banked red cells, transfusion of FFP, and multiple long bone fractures [31]. Unless shock is associated with significant tissue injury or other known risk factors (e.g., transfusion), it is generally not known to precipitate ARDS. Orthopedic injuries are consistently found to be an independent risk factor for ARDS, particularly in the case of femur fractures [32–35]. Commonly observed risk factors for ARDS are shown in Table 14.4.

We currently understand ARDS as an immuno-inflammatory injury to lung tissue which produces markedly impaired gas exchange [36]. This paradigm posits that both infectious and noninfectious insults initiate a generalized inflammatory response that subsequently injures the lung in an autotoxic fashion. Mediators proposed to initiate this response include danger-associated molecular patterns (DAMPs, released from soft tissue injury), leukocytes/lipid/protein mediators from stored blood components and leukotrienes elaborated from gut lymph. Most relevant to the current discussion is the observation that several components of bone marrow and fracture serum can initiate or exacerbate this phenomenon, including particulate matter, arachidonic acid metabolites, and proinflammatory cytokines [34, 37, 38]. These mediators can initiate indiscriminate activation of effector cells (predominantly macrophages and neutrophils) that subsequently release oxidants, proteinases, and other factors that promote tissue injury.

If the initial insult is severe enough, early organ dysfunction results ("one-hit" or single insult model). More often, a less severe insult results in a systemic inflammatory response syndrome (SIRS) that may not be injurious. These patients appear, however, to be "primed" such that they have an exaggerated response to a second insult, which leads to an augmented/amplified systemic inflammatory response and multiple-organ dysfunction [39]. Fixation of fractures, which may represent additional soft tissue injury, blood loss, and release of mediators from bone marrow/fracture sites, is often thought to be a second insult. In that light, understanding which patients are at risk for ARDS from insults which can be planned may be pivotal in the care of the multiply injured patient.

The characteristic lesion of ARDS affects the interface between alveoli and pulmonary capillaries, with both epithelial and endothelial damage, resulting in a high permeability pulmonary edema. Changes in this lesion—and in the patient's physiology—follow a typical pattern, usually divided into three overlapping phases: (1) the exudative phase, with edema and hemorrhage; (2) the proliferative phase, with organization and repair; and (3) the fibrotic phase [40]. The exudative phase is apparent in the first 3–7 days. Histologic changes include proteinaceous alveolar edema, interstitial edema, and intra-alveolar hemorrhage. The exudative phase is characterized by the appearance of hyaline membranes, which are composed of cellular debris and plasma proteins. Loss of the alveolar epithelial barrier results in alveolar edema, as the remaining cells are unable to drive sodium from the alveolar into the interstitial compartment.

During the proliferative phase, type II cells divide and re-cover the lining of the alveolar wall, beginning about 3 days after the onset of clinical ARDS. Fibroblasts and myofibroblasts proliferate and migrate into the alveolar space in the third phase. Fibroblasts change the alveolar exudate into granulation tissue, which subsequently organizes and forms dense fibrous tissue. Eventually, epithelial cells cover the granulation

tissue. The fibrotic stage is characterized by thickened, collagenous connective tissue in the alveolar septa and walls. Pulmonary vascular changes occur as well, with intimal thickening and medial hypertrophy of the pulmonary arterioles. Complete obliteration of portions of the pulmonary vascular bed can result.

Clinically, ARDS is characterized by tachypnea, hypoxemia refractory to oxygen, and then the development of diffuse, patchy, panlobar pulmonary infiltrates on plain chest radiograph. Computed tomography of the chest will demonstrate that the parenchymal changes are inhomogeneous with the dependent lung regions most affected. Thus, in management of the patient with ARDS, it must be recognized that overall the lung should be considered small (many alveolar spaces are flooded); in fact, the aerated lung volume able to participate in gas exchange may be markedly reduced to one third of the original volume [41]. Further, though the overall lung compliance is diminished, there are actually a variety of airway units ranging from normally compliant to completely collapsed. The inhomogeneous distribution of parenchymal densities led to the concept of a four-compartment model of the lung in ARDS [42]. One compartment is substantially normal (healthy zone), one is fully diseased without any possibility of recruitment (diseased zone), a third compartment is composed of collapsed alveoli potentially recruitable with increasing pressure (recruitable zone), and, finally, a fourth compartment contains overdistended airway units.

Because there is no proven specific treatment for ARDS, therapy primarily involves supportive measures to maintain life while the lung injury resolves. Such measures include identifying and treating predisposing conditions, mechanical ventilatory support with oxygen, nutritional support, nonpulmonary organ support, and hemodynamic monitoring as necessary. Attention to detail is necessary to avoid nosocomial infection and iatrogenic complications. Increased airway pressure is necessary to recruit collapsed alveoli, and thus application of positive pressure ventilation is key in supporting patients with severe ARDS. As early as the 1990s, however, it was recognized that in the heterogeneously injured lung, airway pressure or stretch may be damaging to the healthy zone. This ventilator-induced lung injury is now thought to be responsible for severe protracted ARDS, as well as perpetuation of systemic inflammation and multiple organ failure. This is thought to be why ventilator strategies that minimize volume and pressure are associated with decreased mortality in ARDS.

Currently, ventilator-induced lung injury can be thought of in terms of stress-related injury and strain-related injury [43, 44]. Stress can be thought of as tension on the lung skeleton related to static distension (transpulmonary pressures); higher pressures produce injury by overdistending normally compliant units. Strain can be thought of deformation of lung units through the respiratory cycle, including the potential for repetitive "opening" and "closing" of alveoli. This is related to tidal volumes used and the end-expiratory volume. Of note, since lung units share walls, and one unit may not have the same compliance as its neighbor, strain can result from the interaction of two or more adjacent units. Both stress and strain are thought to potentiate ongoing lung inflammation.

ARDS network trials published in 2006 addressed appropriate fluid management in patients with ARDS. In this study, a total of 1,000 patients was randomized to either liberal or conservative fluid strategies over a period of 7 days [45]. The conservative group received approximately a net one liter less per day and spent 2.5 fewer days on the ventilator. There was no mortality difference and no increase in other organ failures in the conservative group. While this may not be considered a profound effect related to fluid, it is one of very few "positive" trials in the ICU setting; it can be concluded that a conservative approach to volume administration is safe and associated with some improvement in outcome. The modest effect observed may relate to the fact that pressure-limited ventilation trumps any major effect of fluid balance in this patient population. With respect to colloids, while conceptually attractive, there is little evidence that their routine use for acute resuscitation improves outcome.

Fat Emboli Syndrome

Extravasation of bone marrow into the venous system can result in a striking syndrome that includes severe pulmonary failure. Approximately 75 % with this clinical syndrome will have acute or subacute impairment of oxygenation, presumably due to microparticles of fat that obstruct pulmonary vessels or produce vasomotor dysfunction. Global CNS dysfunction mimicking encephalopathy is common; while it can be dramatic, it generally resolves without permanent sequelae. Petechial rash of the upper torso, axillae, oral mucosa, or other sites is present in a minority of described cases. Hematologic changes, including acute unexplained anemia and thrombocytopenia, are relatively common. Fever, tachycardia, and tachypnea are common but nonspecific findings. While no diagnostic test has adequate sensitivity, the finding of fat globules in urine (present in a minority of cases) is considered confirmatory. Like ARDS, this syndrome is a constellation of symptoms and signs without a "gold standard" test, and there may be considerable overlap between these two conditions in the multiply injured patient. While three different sets of diagnostic criteria have been proposed, the 1974 description by Gurd and Wilson is the most commonly cited. In this composite, one major and four minor findings can be used to make the diagnosis [46].

Definitive fixation of long bone fractures is the most common risk factor for fat emboli syndrome. Prospective evaluation of patients at risk identifies the syndrome in as many as 10 % of patients [47, 48]. Interestingly, observation by transesophageal echocardiography suggests that particulate matter in the right heart is quite common in this scenario, yet only a fraction of patients with this finding go on to have clinical manifestations. While the involvement of the pulmonary circulation is somewhat intuitive, the mechanism by which fat in the pulmonary circulation creates systemic manifestations attributable to the systemic circulation (brain, skin, kidneys) is unclear. Some patients have an identifiable anatomic right to left shunt (such as patent foramen ovale), yet in others, fat—or biologically active catabolites—presumably reaches the system circulation by moving through pulmonary capillary beds or around those beds via anatomic intrapulmonary shunts.

Implications on Timing of Fracture Fixation

Given that the patient with the combination of chest and nonthoracic bony injuries is at high risk for pulmonary failure, the matter of fracture fixation timing has been an area of intense scrutiny. Conceptually, fracture fixation may represent a "second hit" that could convert a patient's systemic inflammatory response into an autotoxic state of ARDS and multiple organ failure. On the other hand, fracture fixation may reduce bleeding, bone marrow release, pain-associated systemic manifestations, and further tissue injury. Concerns about the second insult phenomenon appear warranted based on both animal models and human observations of increased circulating proinflammatory cytokines, increased SIRS, and increased pulmonary dysfunction after fracture fixation. Furthermore, the inflammatory response to external fixators applied in a damage control fashion is markedly blunted compared to intramedullary fixation [38, 49, 50]. This would suggest that a damage control approach should be safer in patients at risk for ARDS and multiple organ failure.

Despite these findings, comparative studies of early versus late fracture fixation in multiply injured patients have produced divergent results, ranging from studies suggesting that early fixation is beneficial to studies suggesting it is harmful [51, 52]. In the case of femur fractures, a recent review of the literature captures the lack of clarity quite nicely. In the eight high-quality studies identified where incidence of periprocedural ARDS was reported, exactly half favored early definitive treatment, whereas half suggested no difference. In the studies reporting length of stay and mortality data, the majority favor early definitive treatment [53]. Thus, the debate continues over which patients should receive "early total care" for fractures and which should undergo a "damage control" procedure.

Given the lack of clarity regarding timing of fracture fixation in *populations* of multiply injured patients, a selective approach to the *individual* patient appears highly advisable. Understanding which patients with chest injury are most at risk is pivotal to this concept. In that light, a number of factors and scoring systems have been reported to predict outcome. Mommsen et al. [6] evaluated 278 patients with chest injuries and an ISS >15 admitted to a single regional trauma center. They studied whether abbreviated injury scores for the chest, scores related to pulmonary contusion, or the thoracic trauma score (TTS, which combines both anatomic and physiologic parameters) best predicted clinical outcomes in this set of patients. The anatomic score (AIS) was least predictive of clinical outcomes, whereas the TTS, using a cut-off of 9, was most predictive. For example, TTS >9 was associated with an almost fivefold increase in the result of ARDS, sixfold increase in the likelihood of MOF, and a fourfold increase in the likelihood of death. The components of the TTS are P/F ratio, severity of pulmonary contusion, injury involving the pleurae, number of rib fractures, and age of the patient.

Battle et al. [54] published a meta-analysis in 2012 attempting to describe which factors in chest wall injury were best predictive of mortality. These authors made a concerted effort to include both mild and severe injuries, with the goal of reliably predicting patients who might be considered on a safe clinical trajectory (and might be managed as outpatients) and those who might be considered at risk. While this is an imperfect parallel to the multiply injured patient, it may help inform our decisions. They observed that the following predicted mortality: patients older than 65 (odds ratio = 2), patients with three or more rib fractures (odds ratio = 2), and presence of preexisting conditions (odds ratio = 2.3). Pneumonia was the best predictor of mortality, though for practical purposes this is a late occurrence and thus can rarely inform our decision about fracture care.

Wutzler et al. [55], utilizing the German trauma registry, analyzed 5,892 patients with pulmonary contusions or lacerations and ISS >15 admitted to the ICU. Using the lung component of a standard organ failure score as an end point, they sought to identify patients most at risk for severe pulmonary failure. In multivariate analysis, age, ISS, male gender, and >1 surgical intervention independently predicted severe pulmonary failure. While this is a large retrospective study, it might be criticized for including patients with severe head injuries; in this population of patients, prolonged coma and high incidence of pneumonia may dominate many other considered variables.

The task of identifying patients at the highest risk for perioperative complications has been uniquely championed through a series of outstanding works by Pape and associated investigators over the last 20 years. These investigators have matured a set of criteria that define the "at risk" or "borderline patient" in whom the chance of exacerbating systemic inflammation appeared unacceptably high for definitive orthopedic care and wherein a damage control approach is preferred. Sometimes referred to as the "Hannover criteria," one iteration of the components are as follows:(1) polytrauma with thoracic trauma (ISS >20 and chest AIS >2), (2) polytrauma with abdominopelvic injury (AIS >3 for this region) and shock (SBP <90), (3) ISS >40, (4) bilateral lung contusion, (5) pulmonary hypertension (mean pulmonary artery pressure >24), or (6) increase in mean pulmonary artery pressure of >6 during intramedullary nailing. These have substantial face validity, yet suffer from some practical drawbacks, including the fact that ISS is often undetermined early in the patient's course and pulmonary artery catheters have all but disappeared from routine use in the trauma setting. Using a modification of these criteria, a multi-institutional randomized study of damage control versus early definitive care was published in 2007 [49]. Interpretation of this "EPOFF" study is hampered somewhat by disparities in injury severity and concomitant brain injury in the (randomized) groups, yet it was observed that for patients in "borderline" condition, there was an increase in morbidity in the group undergoing an early definitive care approach. Specifically, the risk of acute lung injury in the early total care group was sixfold higher.

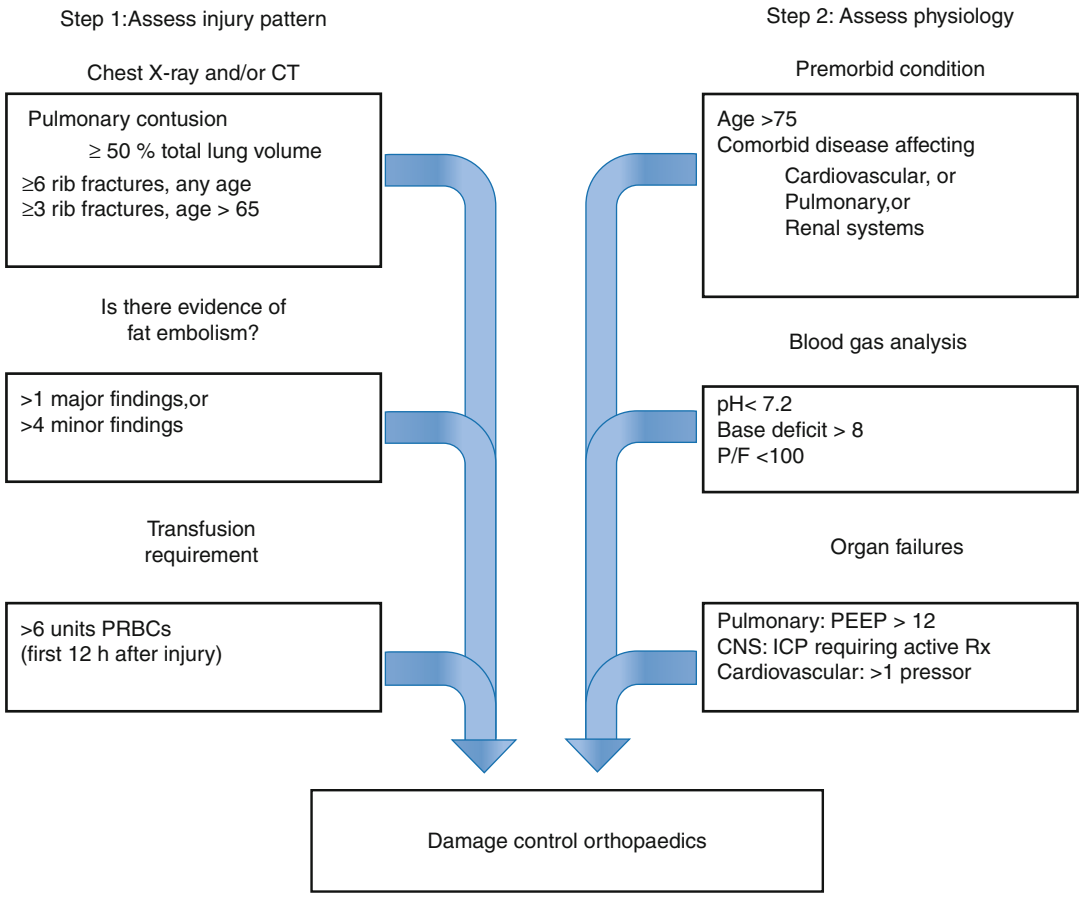

Fig. 14.1 Factors known to increase the risk of cardiopulmonary morbidity in the multiply injured patient with orthopedic injuries. It is proposed that the presence of any one of these factors should warrant consideration of a damage control approach to fracture care

Now that the reader understands the physiology of chest injury and the patients most at risk for secondary pulmonary injury, an algorithm-based approach can be proposed (Fig. 14.1). This will include our current understanding of the role of rib fractures, pulmonary contusion, ARDS risk factors, and physiologic parameters that might make major operative intervention unsuitable. Synthesizing the material up to this point, we can combine known risk factors to define the at risk patient. The following approach is proposed, concentrating on clinical variables that are in routine use or can be easily and rapidly calculated. The risk factors are subdivided into injury pattern and patient factors known in the early (first 6 h) phase of resuscitation. Based on current understanding of the literature, it can be stated that the presence of any one of these factors should prompt strong consideration of a damage control approach to fracture fixation in the patient with chest injuries. To be sure, this is not an exhaustive list of factors that should delay definitive fixation, yet it provides an approach that captures the majority of common events in the multiply injured patient.

The first consideration should be the pattern of injury, specifically evidence of pulmonary contusion, major chest wall injury, major hemorrhage, or fat emboli syndrome. With respect to contusion, those involving 50 % or more of the lung parenchyma suggest the patient will go on to have major gas exchange abnormalities in the ensuing

48 h and would therefore be unsuitable for early total fracture care. It is reasonable to place more weight on this finding when the contusion is evident on a plain radiograph as opposed to chest CT as the latter is clearly more sensitive. Pulmonary contusions evident on later (>24 h postinjury) radiographs may have less clinical importance as they may reflect the development of dependent atelectasis, aspiration pneumonitis, or the blossoming of an earlier smaller contusion.

Major chest wall injuries are a risk factor for early pulmonary dysfunction, in part because they are frequently associated with underlying pulmonary contusions and lacerations. Factors to take into consideration include age, number of rib fractures, and the likelihood of pulmonary failure. In aggregate, 3 or more rib fractures in a patient >65 or 6 or more fractures in any patient should warrant a damage control approach to orthopedic injuries.

In regard to hemorrhage, early (first 12 h) red cell transfusion is an important marker; furthermore, there is a linear relationship between number of units transfused in this time period and the risk for ARDS and multiple organ failure [56]. A precise threshold effect is not evident, yet for practical purposes a working "cutoff" for high risk of transfusion-associated ARDS would be useful; in this author's opinion, a 6-unit transfusion (first 12 h) is indicative of a substantial risk of subsequent organ failure; thus, delay of definitive orthopedic repair is the optimal approach. This is not to say that fracture fixation should not be pursued—some form of stabilization is likely to minimize ongoing blood loss. Finally, an injury pattern that produces the syndrome associated with fat embolization early in the patient's course should warrant fracture fixation with external devices as opposed to an intramedullary approach.

The second consideration in making a decision about early total care versus a damage control approach is patient factors. The emphasis should be on those that affect the cardiopulmonary reserve an individual patient. As previously described, the metabolic demand of postinjury physiology requires dramatic increases in carbon dioxide excretion. Furthermore, patients who fail to mount a hyperdynamic cardiovascular response appear to be at substantially increased risk of later organ failure [57]. This is likely a combination of diminished reserve and cardiovascular depressant factors present in the cytokine milieu in the critically injured patient. In any event, elderly patients and/or those with preinjury pulmonary, cardiac, or renal insufficiency are at higher risk and may tolerate major operative interventions poorly in the early postinjury period. To assess the patient at risk, pay particular attention to the early physiology—this is highly predictive of subsequent trajectory. This is both because a "second hit" may be injurious and because some patients are placed at risk simply from trying to transport them to an operating room environment. Five factors are proposed for consideration here: current pH, base deficit, P/F ratio, PEEP requirement, intracranial hypertension, and cardiovascular failure.

In the "at-risk" patient, arterial blood gas analysis is an invaluable insight into the current physiology of the patient. The particular elements of interest include the current pH, the base deficit, and the current pO_2 (and thus P/F ratio). With respect to pH, a patient who remains acidemic despite resuscitation is unlikely to tolerate further insults; $pH < 7.2$ suggests a damage control approach is warranted. All organ systems exhibit dysfunction at deranged pH and cardiopulmonary compromise; renal dysfunction and hepatic insufficiency can be expected in this scenario.

Base deficit can be thought of as the amount of base that would need to be given to regain a normal pH with normal pCO_2. It is a surrogate for the depth and duration of cellular shock. An elevated base deficit in the trauma setting implies impaired oxygen delivery/utilization in tissue beds, with resultant anaerobic production of lactate. Certainly other coexistent factors can produce a base deficit, and other measures such as lactate/pyruvate ratios or near-infrared spectroscopy can be used. Since blood gas analysis is so rapid and so widely available, in many ways it is the single best test to determine the patient's current state and likelihood of subsequent morbidity and mortality. An early (<6 h) base deficit of eight or more is independently associated with

organ failure and death and should prompt consideration of a damage control approach. Again, this is not to suggest that fracture fixation should not be pursued—merely that it should be done in a way that puts the patient at minimal risk.

The remaining patient factors that warrant a damage control approach include evidence of early organ failure. With respect to pulmonary failure, the arterial pO_2 gives us a measure of gas exchange and must be strongly considered as well. Patients' meeting criteria for severe ARDS (Berlin criteria, P/F < 100) are unlikely to tolerate additional insults and should be approached with caution. For practical purposes, a patient requiring an FiO_2 >0.6 to maintain oxygen saturations >90 % will fall in this category. Additionally, patients who require moderate or high levels of PEEP are likely to have very limited pulmonary reserve; a threshold of 12 is proposed. In most centers, it is not practicable to maintain such levels of PEEP during transport and anesthesia. While bag mask ventilators may have PEEP valves, and some transport and anesthesia ventilators can deliver advanced modes and pressures, attempts at transport to the operating room are likely to be met with de-recruitment of alveoli to a point where maintaining oxygenation is problematic. Creative strategies such as bedside external fixators in the ICU should be considered.

For CNS organ dysfunction, patients with intracranial hypertension requiring active treatment also fall into this category of transport risk as head elevation, maintenance of eucapnia, and judicious fluid administration are necessary. Lastly patients with severe cardiovascular instability (as measured by the requirement of more than one pressor) are poor candidates for definitive fixation.

Conclusion

In conclusion, patients with the combination of major chest injuries and fractures requiring orthopedic fixation represent some of the most challenging multitrauma patients. This overview may provide the reader with an improved understanding and approach. With our understanding of the interplay between the physiology of chest injury and the response of individual patients, the above guidelines can serve to guide clinicians on critical decision in individual patients. The author suggests a stepwise approach, considering injury pattern and early (patho) physiology.

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Abdominal Compartment Syndrome and the Open Abdomen

15

Clay Cothren Burlew

Introduction

In the late 1800s, several physicians identified the entity of increased abdominal pressure, due to either extremes of normal physiology or due to particular disease states, and recognized that this increase in abdominal pressure has systemic effects. These concepts were largely ignored until the 1940s when Dr. Gross and his colleagues recognized that early forced closure of omphalocele defects, with reduction of the abdominal contents under extreme pressure, would lead to the baby's cardiovascular collapse [1]. It took another 35 years before the concept of intra-abdominal hypertension (IAH) and its associated end-organ sequelae were discussed in the literature again with regularity. This reported the concept that IAH, caused by postoperative bleeding, resulted in anuria that could be reversed with reoperation and decompression of the abdomen [2].

It was Kron et al. who made the leap to a significant clinical application that abdominal pressure, itself, was a criterion that could be used to determine need for decompression and that this intervention could be lifesaving [3]. Although many report it was this group that coined the term abdominal compartment syndrome (ACS), the first report of this term in the literature was not until 1989 [4]. The original description was in four patients who had undergone ruptured abdominal aortic aneurysm repair. Postoperatively the patients developed IAH and end-organ sequelae, namely, abdominal distension, increased airway pressures, increased central venous pressure, and oliguria. With reopening of the abdomen, the end-organ derangements resolved. These particular parameters are what define ACS today.

Etiology

The etiology of ACS is multifactorial and may be associated with a variety of clinical situations. ACS is typified by intra-abdominal hypertension (IAH) due to either intra-abdominal injury (primary) or following massive resuscitation (secondary) [5–16]. Some causes of primary ACS include solid organ injuries, pelvic or retroperitoneal hematomas, bowel perforations or obstruction, ruptured vasculature, and postoperative hemorrhage. Secondary ACS may be due to any etiology requiring large volume resuscitation, including both crystalloid and blood products. The most common scenario for ACS is the multiply injured trauma patient who requires such a large volume resuscitation. In these cases, intra-abdominal hypertension is due to resuscitation-associated bowel edema, retroperitoneal edema, and large quantities of ascitic fluid combined with an associated intra-abdominal pathology. Increasing abdominal pressure may develop before, during, or after surgery, typically

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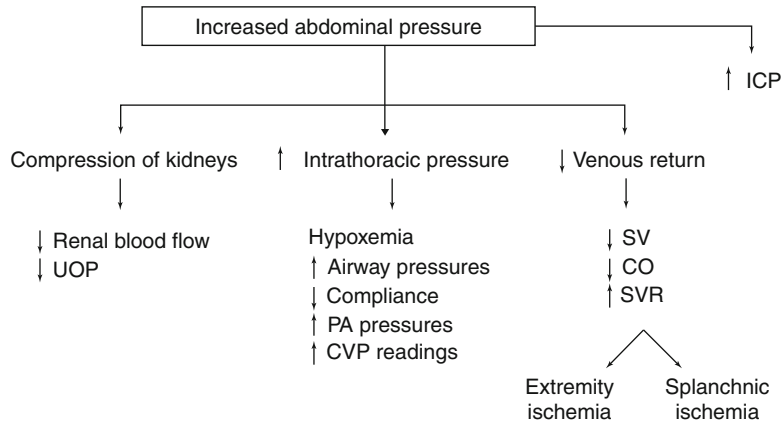

Fig. 15.1 Increased abdominal pressure affects multiple organ systems

within the first 24–48 h after injury. Trauma patients that develop the lethal triad of hypothermia, acidosis, and coagulopathy [17] are particularly susceptible to ACS, and damage control operative techniques for the chest, abdomen, and bony elements should be considered [18–20].

Physiology

Increased abdominal pressure affects multiple organ systems (Fig. 15.1) [13, 21–25]. The intra-abdominal pressure causes direct compression of the retroperitoneal vascular structures, resulting in decreased venous return to the right heart via the inferior vena cava; in turn, this decreases preload and subsequent effective cardiac output. An increase in systemic vascular resistance can diminish stroke volume. Intra-abdominal hypertension and swollen viscera push the diaphragm cephalad; this causes a restrictive pattern of pulmonary physiology with increased intrathoracic pressures and decreased thoracic compliance. Clinically, the patient manifests elevated airway pressures and hypoxemia. Elevated intrathoracic pressures may also worsen venous return, diminish ventricular end-diastolic volume, and diminish cardiovascular function.

Intra-abdominal hypertension also has a direct compressive effect on the renal system. This results in a relative obstruction to renal venous drainage plus an increase in renal vascular resistance. With a consequent reduction in blood flow, there is a decrease in urine output. There is a similar impact on the vascular system causing a reduction in hepatic and intestinal perfusion. Finally, there is an apparent association between ACS and intracranial hypertension, postulated to be due to a decrease in venous return.

Diagnosis

Clinical indices of end-organ derangement such as decreased urine output, increased pulmonary pressures, decreased preload, cardiac dysfunction, and elevated intracranial pressure are fundamental to the identification of ACS [26]. In any critically injured patient, however, there are many etiologies that could cause a low urine output and cardiopulmonary woes. Therefore, the diagnosis of IAH and ACS has to remain in one's differential in the at-risk patient. Physical examination cannot definitively diagnose IAH or its severity [27]. Although the patient may have a markedly distended abdomen that is suggestive of the diagnosis, exam may reliable only about 40 % of the time. A diagnosis of IAH is obtained by measuring the patient's bladder pressure. The bladder is capable of transmitting intra-abdominal pressure without imparting any additional pressure from its own musculature, hence acting as a passive reservoir. To measure a patient's bladder pressure, 50 cc of saline is instilled into the bladder via the aspiration port of a 3-way Foley catheter with the drainage tube clamped; after waiting for 30–60 s to allow the detrusor musculature to relax, pressure measurement with a manometer at the pubic symphysis is performed [28]. Although this

technique is a single measurement, continuous monitoring is also an option [29]. There are several conditions in which the bladder pressure may not be reflective of the intra-abdominal pressure. These include patients with external compression on the bladder such as pelvic packing, those with bladder rupture, marked adhesive disease, and patients with a neurogenic bladder.

A grading system based on bladder pressure measurements was developed to aid in the diagnosis and subsequent treatment of ACS (Table 15.1) [6]. Measurements are obtained in either cmH_2O or mmHg depending upon the institution. Typically, intra-abdominal pressures over 20 cmH_2O become worrisome. More recently, abdominal perfusion pressure, defined as the mean arterial pressure minus the intra-abdominal pressure, has been advocated to diagnose IAH and ACS [30]; to date this has not been widely adopted in practice. Recognizing that ACS is a late event in the evolution of IAH, monitoring at-risk patients is advocated; this allows one to intervene in patients with IAH in an attempt to prevent the sequelae of ACS. Maxwell and colleagues report that monitoring for IAH should occur after 10 units of packed red cells or 10L of crystalloid [11]. Others have also reported on the need for a heightened awareness of IAH with early monitoring of high-risk patients [9, 12, 31].

Table 15.1 Acute compartment syndrome grading system based on bladder pressure measurements

ACS grade	Bladder pressure	
	mmHg	cmH_2O
I	10–15	13–20
II	16–25	21–35
III	26–35	36–47
IV	>35	>48

Intervention

There is not, unfortunately, a single pressure measurement that mandates intervention; rather, it is the combination of the bladder pressure measurement and end-organ sequelae (decreased urine output, increased pulmonary pressures, and decreased cardiac output) that is required for the diagnosis of ACS. Organ failure can occur over a wide range of recorded bladder pressures. If the patient has ACS, however, emergent decompression is indicated; mortality is directly affected by decompression [30].

Decompression is typically performed via a midline laparotomy incision performed in the operating room; this allows egress of peritoneal fluid or blood as well as evisceration of the edematous bowel (Fig. 15.2). In patients who are too

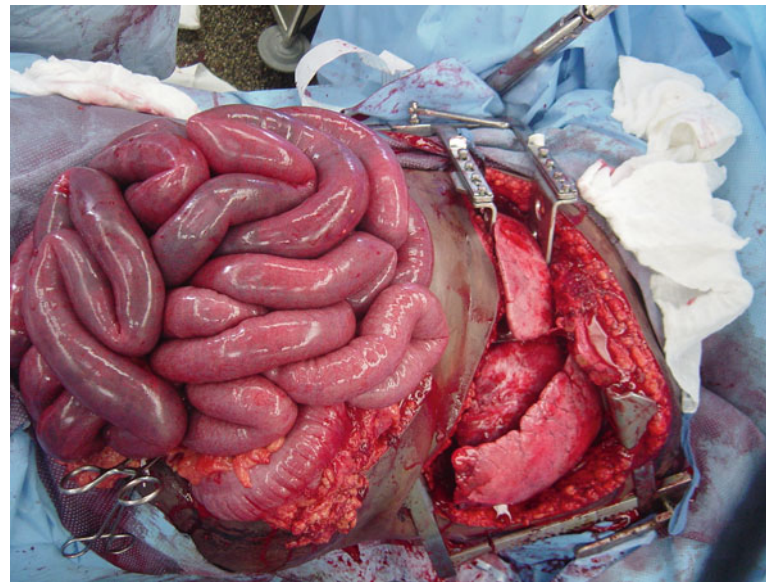

Fig. 15.2 Midline decompressive laparotomy performed for secondary abdominal compartment syndrome following massive resuscitation for a gunshot wound to the heart; peritoneal fluid or blood is evacuated, and the edematous bowel protrudes from the abdominal cavity

unstable for transport to the operating room, operating room personnel and equipment can be transported to the ICU for a bedside procedure. Bedside laparotomy is easily accomplished, precludes transport in hemodynamically compromised patients, and requires minimal equipment (scalpel, suction, cautery, and abdominal temporary closure dressings). Patients with significant intra-abdominal fluid as the primary component of their ACS may be candidates for decompression via a percutaneous drain [31–33]. Differentiation of those amenable to such drainage is determined by bedside ultrasound, hence obviating a trip to the operating room. Removing a significant amount of ascites can lower the intra-abdominal pressures enough to obviate laparotomy.

Following operative decompression, temporary coverage of the abdominal viscera is necessary. One option of temporary closure is "towel clipping" the abdomen; penetrating towel clips are placed through the skin 2–3 cm apart to approximate the abdominal wall over the length of the laparotomy incision (Fig. 15.3). Although this rapidly closes the abdomen, the closure does not allow egress of the edematous bowel which may promote recurrent ACS. Another option for temporary closure is the Bogota bag closure (Fig. 15.4). This temporary silo is constructed of a sterile 3L GU irrigation bag or X-ray cassette cover that is sutured to the skin; this contains the edematous bowel while providing excellent

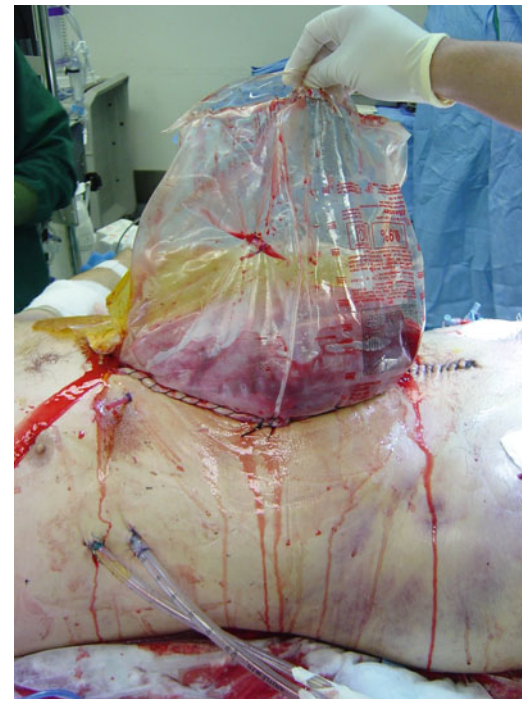

Fig. 15.4 A sterile 3L GU irrigation bag sutured to the skin, also termed the Bogota bag closure of the abdomen, permits egress of the edematous viscera

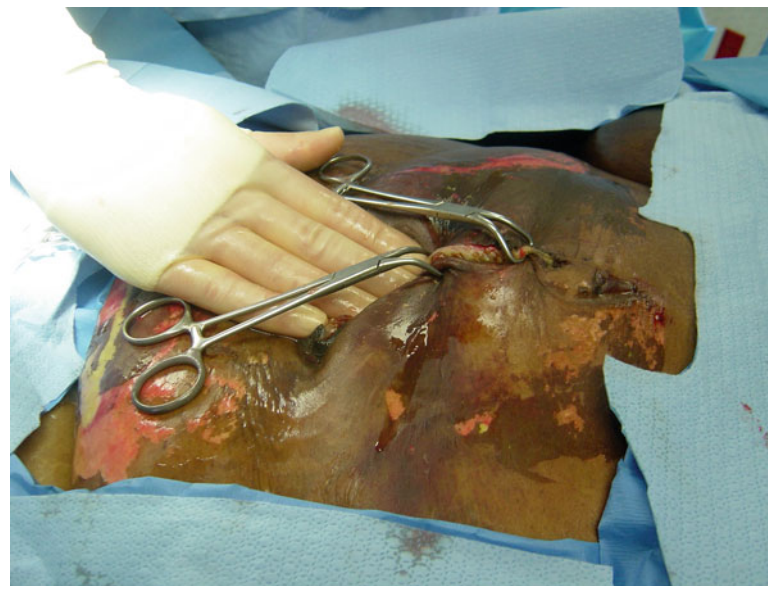

Fig. 15.3 Towel clip closure of the abdominal skin

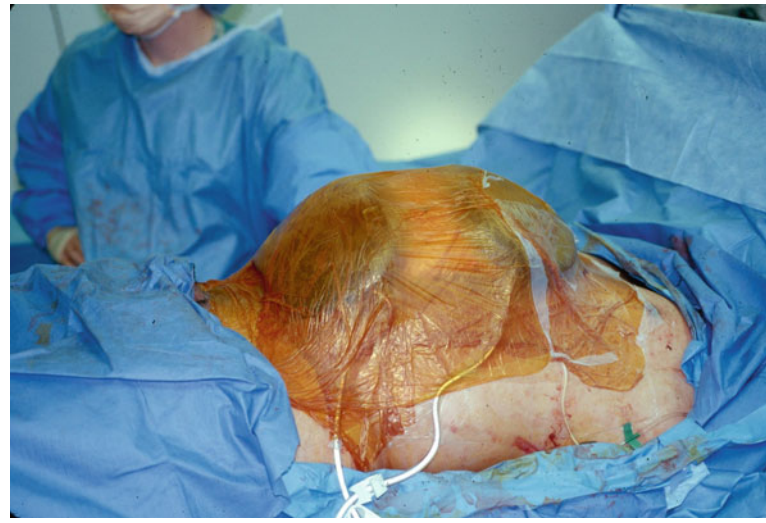

Fig. 15.5 Vacuum-assisted closure with applied suction may be used to cover the abdominal contents

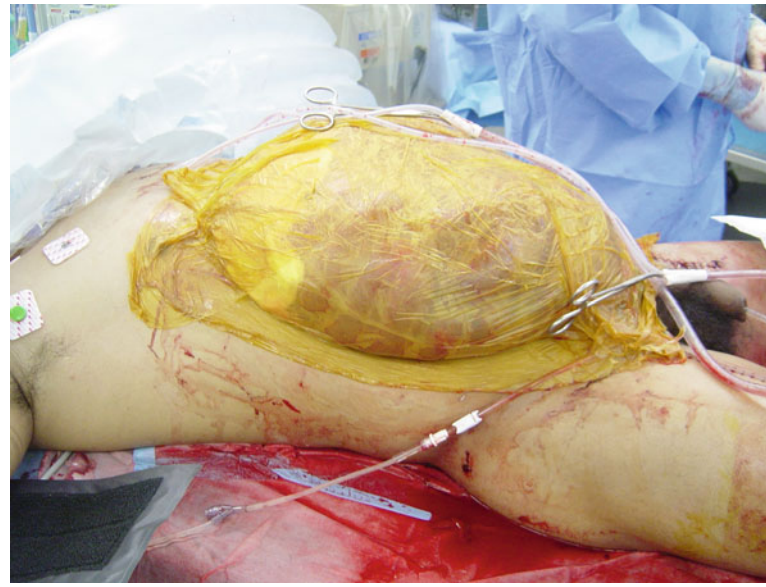

Fig. 15.6 1010 Steri-Drape and Ioban temporary closure of the abdomen

decompression. A third option for temporary coverage is a vacuum-assisted closure device (Fig. 15.5); the caveat, however, is to ensure that recurrent ACS does not occur due to the application of suction-related pressure on the viscera. Our preferred option for temporary closure is 1010 Steri-Drape and Ioban closure (Fig. 15.6). In this technique, the bowel is covered with a fenestrated subfascial 1010 Steri-Drape (3M Health Care, St. Paul, MN); small holes are cut in the plastic drape with a scalpel to allow fluid to pass through the drape while not allowing the Ioban to stick to the underlying bowel. The drape is placed over the bowel and tucked under the fascia. Two Jackson-Pratt drains, with the tubing running cephalad, are placed along the fascial edges to control reperfusion-related ascitic fluid. Everything is then covered using a large Ioban (3M Health Care, St. Paul, MN). In any temporary closure of the abdomen, one has to ensure that the patient does not develop recurrent ACS; one should not assume that because the patient has an open abdomen that they cannot develop IAH or ACS [34]. Therefore, leaving "expansion space"

for the bowel in your temporary covering is critical. Additionally, monitoring bladder pressures in patients who are unstable or have low urine output is an important adjunct.

ICU Management of the Open Abdomen

Management of the patient with an open abdomen is not markedly different from the care of any critically ill patient. The guidelines in such cases include appropriate resuscitation, rewarming, correction of acidosis and coagulopathy, lung protective ventilation strategies (once resuscitated), prevention of ventilator-associated pneumonia, treatment of adrenal suppression, and management of hyperglycemia. Issues specific to the open abdomen patient include balanced fluid administration, nutrition support, and management of enteric injuries.

During the acute resuscitation in the first 12–24 h following injury, initial volume loading attains adequate preload followed by judicious use of inotropic agents or vasopressors [35]. Optimizing fluid administration is a challenging aspect of early patient care, balancing cardiac performance versus generating marked visceral and retroperitoneal edema. Supranormal trauma resuscitation has been shown to require more crystalloid administration and to cause more cases of ACS [36]; therefore, goal-directed therapy should aim for an oxygen delivery index of >500 mL/min/m^2 [37]. Although early colloid administration may be appealing in these patients, evidence to date does not support this concept [38].

Despite studies illustrating the importance of enteral nutrition (EN) in the trauma population [39–44], there remains hesitancy about enteral feeding in post-injury patients with an open abdomen. This may relate to issues of enteral access, concerns about bowel edema, or questions of intestinal motility and enterocyte functionality. Three studies addressing EN in the open abdomen patient have conflicting findings regarding impact of EN on abdominal closure rates and septic complications [45–47]. The most recent evaluation of feeding patients with an open abdomen [48] demonstrated EN in patients with bowel injuries did not appear to alter fascial closure rates, complications, or mortality; hence, EN appears to be neither advantageous nor detrimental in these patients. For patients without a bowel injury, EN in the open abdomen was associated with a marked increase in successful fascial closure, a decrease in complications, and a decrease in mortality. That study concluded that EN in the post-injury open abdomen was feasible. Therefore, once resuscitation is complete, initiation of EN should be considered in all injured patients [49].

Abdominal Closure

Coverage of the enteric contents is the most critical step in the management of the open abdomen. Leaving the bowel exposed to the atmosphere can result in enteroatmospheric fistulas which are notoriously difficult to manage. The ideal coverage for the bowel is native fascia, so primary closure is the goal, either with early fascial closure or sequential fascial closure techniques. Other options for bowel coverage include prosthetic fascial closure with either mesh or biologics, or bowel coverage with skin grafts and planned ventral hernia.

Our preferred approach in Denver for those patients that are not closed at second laparotomy is the sequential fascial closure technique [50, 51], a modification of Miller et al.'s described VAC technique [52]. In our described technique, closure is sequentially performed with the combination of a Wound Vac™ as well as constant fascial tension with sutures (Fig. 15.7). Patients are returned to the operating room for sequential fascial closure and replacement of the sponge sandwich every 2 days until closure is accomplished. Gastrostomy and needle catheter jejunostomy tubes may be placed prior to complete closure; however, manipulation or marked movement of enteral access sites could cause injury with fistula formation. For this reason, operative gastrostomy and jejunostomy tubes should not be placed until closure of the fascia is well under

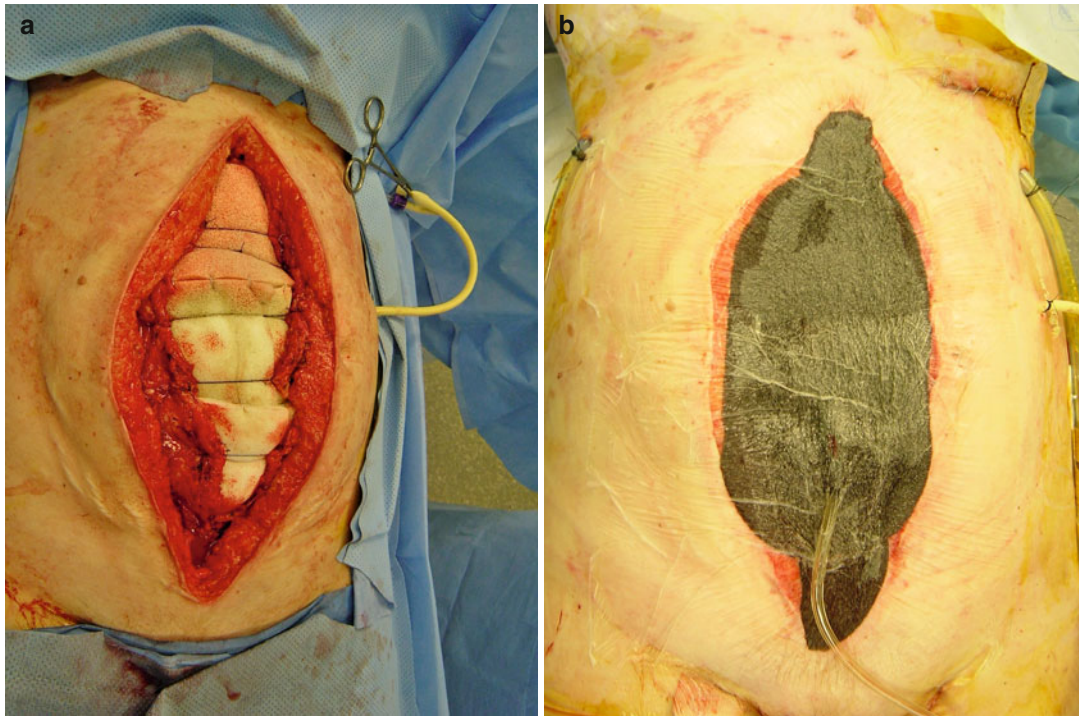

Fig. 15.7 Sequential fascial closure technique uses a sponge sandwich of white sponges over the bowel with #1 PDS sutures over the white sponges to prevent fascial retraction (**a**); black sponges, occlusive dressing and standard suction is applied on top of the white sponges (**b**)

way. There may be hesitancy to place an operative jejunostomy through the edematous bowel wall; however, this can be safely performed [49]. Alternatively, nasojejunal access is also a viable option for early enteral nutrition.

Summary

In summary, open abdomens are necessary sequelae after DCS or ACS, but they do save lives. In the multiply injured patient, management should include rapid fracture stabilization in concert with damage control techniques for thoracoabdominal injuries. Management of the patient in the ICU continues to evolve with considerations of fluid resuscitation, enteral nutrition, and supportive care. Management of the bowel incorporates several basic techniques and considerations: appropriate temporary covering, a consideration of bowel repair in the majority of patients, placement of the anastomosis in an area of the abdomen with minimal manipulation without exposure to the atmosphere, and a consideration of enteral access for initiation of enteral nutrition while the abdomen is still open. And finally, the importance of fascial closure cannot be overemphasized.

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Epilogue

Trauma care in developed countries is increasingly characterized by adherence to the basic principles of ATLS, multidisciplinary care, and a focus on improved patient outcomes. Much of yesterday's new information has become today's standard. ARDS rates are extremely low due to improvements in resuscitation, ventilatory strategies, and ICU care. Sepsis and infection rates have greatly improved due to evidence-based guidelines on rational antibiotic usage and standardized central venous access techniques, among many other improvements. Patients with musculoskeletal injuries appear to have better outcomes thanks to the increased number of specially trained orthopedic traumatologists who have increasingly replaced general orthopedic surgeons as the providers of complex fracture care. Trauma subspecialties in general surgery, neurosurgery, and orthopedics have fostered an awareness that trauma is not just something "everyone does" as part of call but is an important subspecialty requiring training, system building, and specialized resources. Overall, this evolution reflects systematic multidisciplinary progress in the care of the injured patient and manifests with better patient outcomes.

However, trauma patients continue to suffer potentially preventable disability and death despite access to modern state-of-the-art healthcare. It is disappointing that many hospitals do not consistently follow the best published practices. For example, the majority of trauma centers in the United States do not follow standardized guidelines for the management of pelvic trauma, multiple injuries with long bone fractures, or open fractures. Often, a focus on being a trauma center for commercial or competitive reasons supersedes the culture of safety and quality required to achieve best possible patient outcomes. The expert authors in this book have outlined the basic principles for modern multidisciplinary management of trauma patients in significant detail. These approaches should be available to the injured patient at every trauma center.

We hope that this new textbook provides guidance and insights for the "ideal" multidisciplinary management of trauma patients with associated musculoskeletal injuries.

The next great challenge for those who care for injured patients is to continue research and prospective data collection, ideally with multicenter registries. The goal should be to discover new options for clinical care by stepping beyond the laboratory and data processors to confront the current failures of our trauma systems. For example, the historically high mortality of 50–60 % in patients with hemodynamically unstable pelvic fractures was successfully reduced to around 20 % with new resuscitation techniques described in this book. This number should be in the same range at every hospital accredited or labeled as a trauma center. We must begin to work together in national societies to develop coherent data registries so that we can use objective scientific data instead of financial interest to drive policy and patient triage. Society believes that all our trauma centers deliver the highest level of care, and yet the disparities can be striking in terms of quality of practice and patient outcomes. Clearly, writing textbooks and teaching courses is not enough. We must focus less research on our "widgets" and techniques and more on our systems. As providers of care and advocates for the injured patient, our obligation is to look at our own institutions

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and systems, apply the highest level of quality metrics, and critically evaluate where we fall short of our promise to do the very best for our patients.

Hopefully, the perspectives of the authors in this book will assist in pointing the way toward best practices and further standardization of care. We are extremely grateful for their expert insights, experience, and many years of service in caring for the severely injured and often most vulnerable patient populations. Several of the authors have been influential mentors in our lives, and we are eternally grateful for their inspiration and work. We are also deeply indebted to all authors for being unique role models as free thinkers, talented surgeons, and hardworking traumatologists.

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Index

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