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Breaking

When trauma snaps the strongest bone in the body, life-threatening trouble can ensue. Are you prepared?

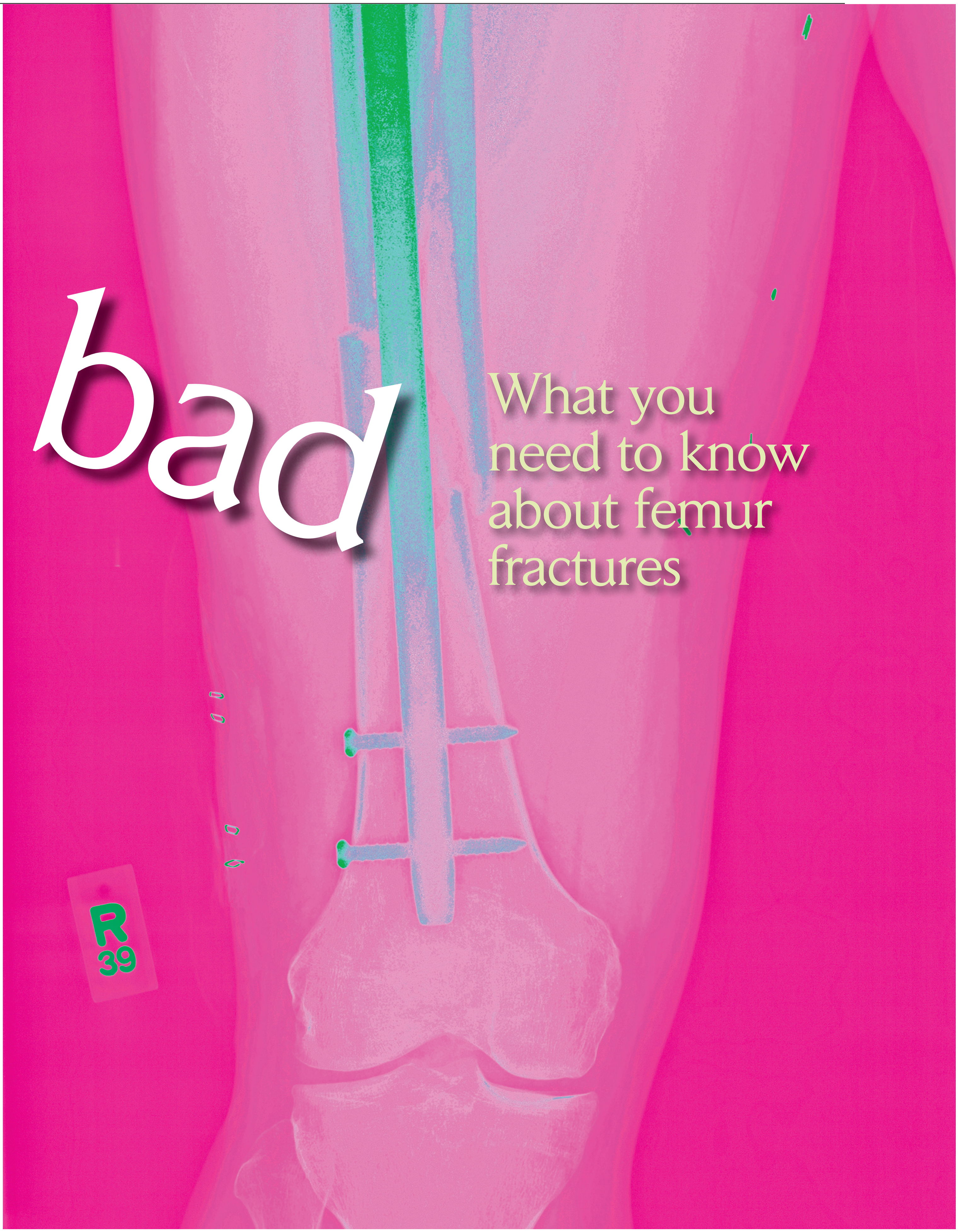
By Colleen Walsh, MSN, RN, ACNP-BC, CS, ONP-C

Nineteen-year-old Matt Lewis was riding his motorcycle at a high rate of speed when he lost control and crashed into a guardrail. He was wearing a helmet and protective leather pants and gloves. A bystander who witnessed the crash called 911. When the emergency medical services (EMS) arrive, Matt is awake, alert, and oriented to time, place, and person, and his only complaint is severe pain in his right thigh, where he has an obvious open femur fracture accompanied by brisk bleeding. The EMS personnel apply a portable traction splint to the right femur, place sterile saline-soaked gauze over the fracture site, and apply direct pressure to stop the bleeding.

Matt's vital signs are: pulse, 126 and thready; respirations, 22; SpO_2, 96%; and BP, 112/70. His breath sounds are clear bilaterally on auscultation, he has no tracheal deviation or jugular vein distension, and his heart sounds are normal on auscultation. The EMS personnel administer 100% oxygen via nonrebreather mask and establish two large-bore peripheral I.V. access sites to infuse 0.9% sodium chloride solution for fluid replacement.

When Matt arrives at the ED, his BP has dropped to 90/60, his pulse is 144, and his skin is cool and clammy. His hemoglobin is 9.1 g/dL (normal for men, 14 to 18 g/dL) and his hematocrit is 27.2% (normal for men, 40% to 52%). His other lab results, including arterial blood gas analysis results, are within normal limits. Rapid trauma assessment reveals no other serious injuries, and imaging studies revealed no head, intrathoracic, intra-abdominal, spinal column, or spinal cord injuries. Right femur X-rays reveal an open, midshaft, comminuted right femur fracture. Despite manual compression, the fracture site is still bleeding and the healthcare provider can't palpate a distal pedal pulse. An emergent arteriogram reveals a tear in the superficial femoral artery.

The femur is the longest, strongest, and heaviest tubular bone in the human body, and is one of the major weight-bearing bones in the leg (see *Boning up on the femur*).[1,2] Fractures of the femoral shaft often result from high-energy forces such as motor vehicle crashes.[3-5] Complications and injuries associated with midshaft femur fractures in the adult can be life-threatening and may include hemorrhage, internal organ injury, wound infection, fat embolism, and acute respiratory distress syndrome.[4-6]



bad

What you need to know about femur fractures

The annual incidence of midshaft femur fractures is about 10 per 100,000 person-years.[3] The incidence peaks among the young, decreasing after age 20, and then increases again in older adults. Femoral shaft fractures are among the most common diaphyseal fractures in children, with an estimated annual incidence of 19 fractures per 100,000 children in the United States.[7,8] Femoral shaft fractures are also the most common pediatric fracture of the femur, accounting for up to 62% of all femur fractures.[9,10]

In adults over age 75, most fractures occur in the proximal third of the femur.[7] More than 90% of hip fractures in older adults result from trauma or torsion associated with a minor fall or occasionally in the absence of any obvious traumatic event. Osteoporosis is the leading cause of hip fracture.[11]

This article will focus on traumatic fractures of the mid and distal portions of the femur.

Classifying fractures

Fractures are classified based on the location of the fracture, whether the fracture is open or closed, and the fracture pattern (see *Those are the breaks*). Fracture locations include proximal, midshaft, and distal. Other factors to consider when looking at closed fractures are the amount of comminution or bone fragments, whether the fracture is intra-articular (through the joint), the degree of angulation, and distal vascular status.[12]

Open fractures are more complex, and are classified using the Gustilo classification, which is based on the amount of energy needed to produce the fracture, the degree of soft tissue injury, and the presence or absence of vascular injury. Under this system:

- Type I fractures are low-energy, simple fractures with a wound less than 1 cm (0.39 in).
- Type II fractures are higher-energy, with moderate comminution and a wound size greater than 1 cm.
- Type III fractures are high-energy with moderate to large tissue loss. These fractures are divided into three subsets: Type IIIA has limited periosteal stripping, type IIIB has extensive soft tissue and bone loss and periosteal stripping, and type IIIC has all of above with vascular injury that requires surgery.[13]

The most common system of classifying pediatric fractures involving the growth plate is the Salter-Harris classification (see *Salter-Harris fractures*).

Preoperative and perioperative care

Emergency situations require quick and careful assessment with collection of as much data as possible. Follow the Association of periOperative Registered Nurses' *Perioperative Standards and Recommended Practice*, being sure to obtain the following information[14]:

- patient age, language, and language comprehension
- any neurosensory deficits that may impede recognition of surgical complications
- skin integrity before surgical procedures
- neurovascular status
- circulatory status
- confirmation of the operative site as outlined by the American Academy of Orthopaedic Surgeons and The Joint Commission.[15]

Carefully document these assessment findings and alert the healthcare provider about any abnormal data.[16,17]

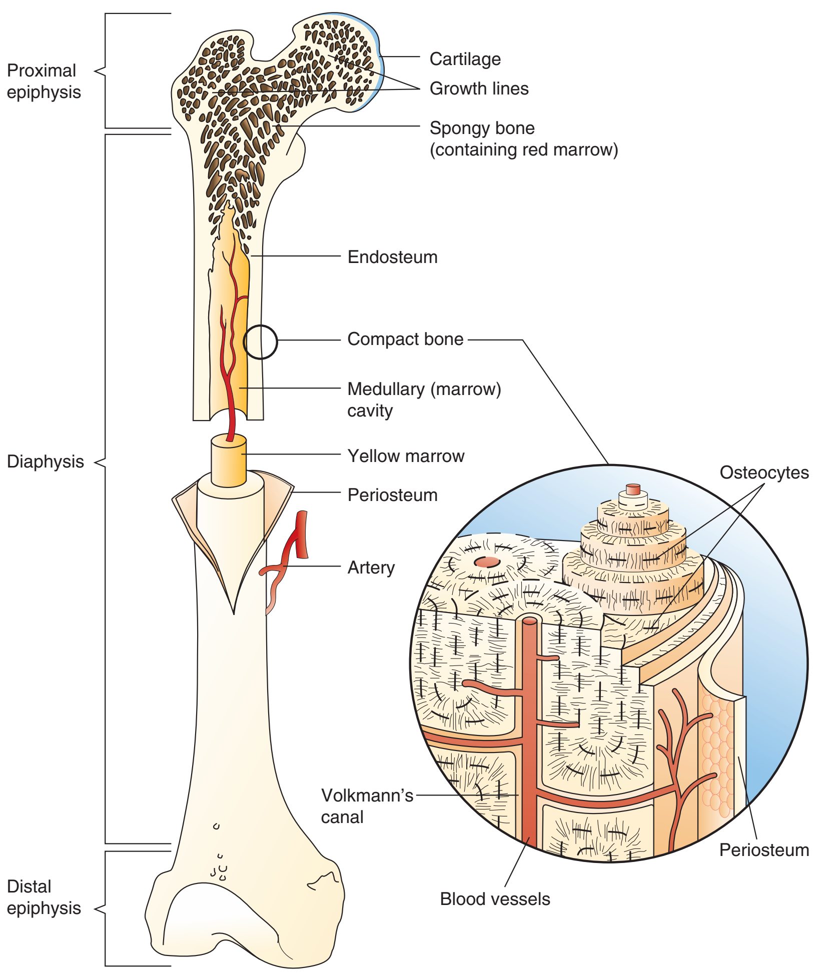
The anesthesiologist usually determines when to administer surgical antibiotic prophylaxis, but the OR nurse must ensure that the appropriate antibiotics are available for administration. Multiple studies have demonstrated that the optimal timing of antibiotic administration, especially in orthopedic procedures, is 30 to 60 minutes before skin incision.[18] This allows for adequate tissue concentration of the antibiotic. Administration of all prophylactic antibiotics should be completed within 24 hours, in accordance with the Surgical Care Improvement Project.[19]

The neurologic and musculoskeletal systems are most vulnerable to injury during surgical procedures, and because of the relatively superficial nature of nerve distribution, the brachial plexus, sciatic, saphenous, peroneal, tibial, and sural nerves are at highest risk for injury. Patients with fractures often sustain peripheral nerve injuries, so document any neurologic deficits before surgery.[20]

Preparing the OR

Be sure adequate padding has been applied to the OR table. Depending on the patient's injury and the surgical procedure being performed, the surgeon may use one of a variety of specialized orthopedic tables. Various positions and positioning devices may also be used to facilitate the surgical procedure; be sure you're familiar with how to use these devices safely.[17] Don't extend the patient's arms more than 90 degrees because overextension can cause brachial plexus injuries.[16] Document the position, positioning device use, location of the positioning device, and

Boning up on the femur



Bone is classified as either compact (cortical) or spongy (cancellous).[2] Each type of bone serves different functions in the skeletal system. Cortical bone is very organized, strong and dense, and can withstand a certain amount of force before it fractures. Cancellous bone is less organized and consists of irregular networks called trabeculae.[2] The spaces between the trabeculae contain red bone marrow, where hematopoiesis (blood cell formation) takes place.

All bones are covered with a double-layered sheath of connective tissue called the periosteum.[2] The outer layer of the periosteum contains blood vessels and nerves, some of which penetrate into the inner portions of bone.[2] The inner layer of the periosteum is attached to the bone via connective tissue structures called Sharpey fibers, which help attach ligaments and tendons to the bone.[2]

The femur is part of the appendicular skeleton, which consists of the 126 bones of the upper shoulder girdle and lower extremities.[2] The femur is classified as a long bone, or a bone that's longer than it is wide. Long bones consist of a narrow tubular midportion called the diaphysis, which broadens proximally into the proximal metaphysis.[2] The distal end of long bone is also broader than the diaphysis, which distributes weight over a larger area.[2] The inner portion of the diaphysis is known as the medullary canal and contains yellow bone marrow. The yellow marrow helps the red marrow to produce red blood cells during periods of physiologic stress.[2]

A critical bone structure in children and adolescents is the epiphysis, which consists of spongy bone covered by a thin layer of compact bone. The epiphysis is separated from the metaphysis in children by a cartilaginous growth plate, also called the epiphyseal plate.[2] Longitudinal bone growth occurs at the growth plate, and the growth plate usually ossifies during late puberty. Any injury to the growth plate before it ossifies can impair the bone's ability to achieve maximum length.[46]

Source for art: Smeltzer SC, Bare BG, Hinkle JL, Cheever KH. *Brunner & Suddarth's Textbook of Medical-Surgical Nursing.* 11th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:2338.

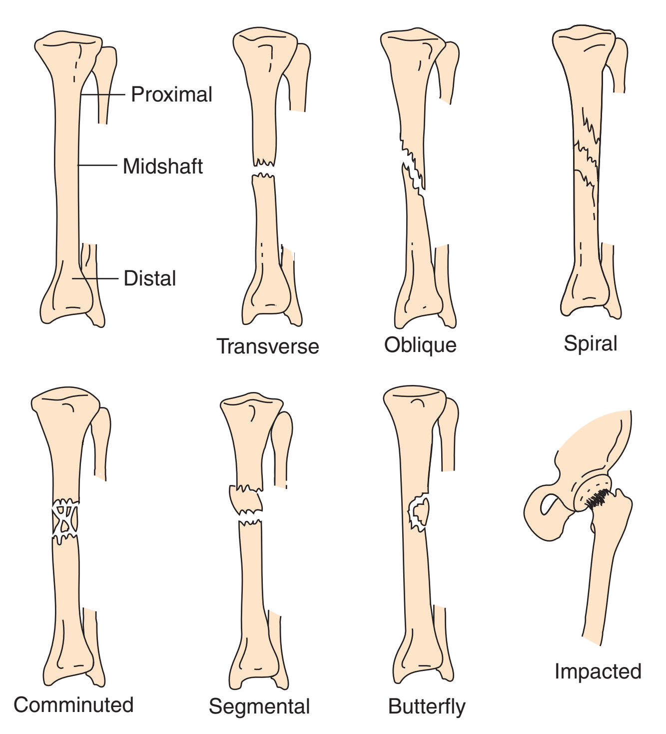
the personnel who facilitated the positioning in the patient's record.[17]

Patients who have longitudinal leg traction during fracture surgery are at risk for pudendal neuropathy, especially after static interlocking nailing of the femur. The mechanism of neurologic injury is thought to be a combination of localized ischemia and direct compression of the perineum against the counter-traction post of the fracture table.[21] Be sure that the fracture-table post is adequately padded to minimize the patient's risk of pudendal nerve injury.

The OR should have all the equipment required for the procedure already bundled into the instrument pack, and the internal fixation hardware should be in stock. This prevents delays in treatment that could affect patient outcomes.

Those are the breaks

Fractures are classified according to location (proximal, midshaft, or distal), the pattern or direction of the fracture line (transverse, oblique, spiral), and the type (comminuted, segmental, butterfly, or impacted).



Source: Porth CM, Matfin G. *Pathophysiology Concepts of Altered Health States.* 8th ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2009:1472.

Complications of femur fractures

Femur fractures are especially dangerous as the forces required to fracture the femur often result in multisystem injuries.[22] Early complications of femur fracture include hemorrhage, pneumonia, compartment syndrome, fat embolus syndrome, venous thromboembolism (VTE), multisystem organ failure, peripheral nerve injuries, and wound infections.[23] Late complications include hardware failure, refracture, malunion, nonunion, and chronic osteomyelitis.[24]

Isolated femur fractures can result in significant blood loss leading to hemodynamic instability.[22] Fluid resuscitation efforts should be geared toward restoring intravascular and interstitial fluid volume and maintaining normal peripheral perfusion.[25] Adequate fluid resuscitation results in urine outputs of 50 mL/hour in adults and 1 mL/kg/hour in children.[26] Trauma patients may need invasive monitoring (such as a pulmonary artery catheter) to help with accurate assessment and calculation of fluid replacement and to guide the use of vasoactive medications.[27] Capillary refill time, normally less than 2 seconds, is an indirect measure of peripheral perfusion, but can be affected by other variables, such as cardiac output and ambient air temperature.[28]

Compartment syndrome is a potentially life-threatening complication of fractures, and occurs when increased tissue pressure within a body compartment compromises the circulation in that compartment.[5,29,30] A compartment consists of bone, muscle, nerves, arteries, veins, and soft tissue surrounded by inelastic fascia that prevents compartment expansion; 38 of the body's 46 compartments are in the arms and legs.[5,29]

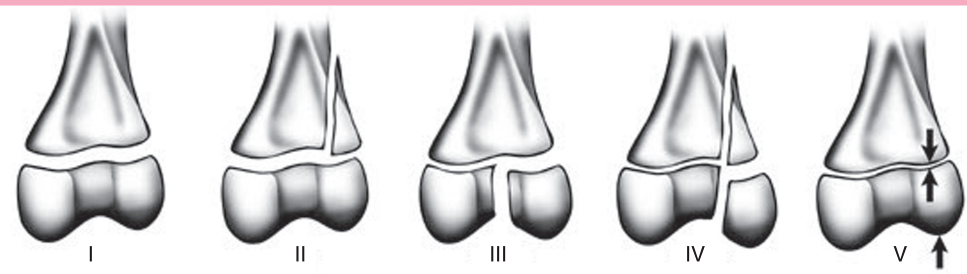
Two major predisposing factors increase the risk of compartment syndrome—space-limiting envelopes (such as circumferential dressings, splints, and burn eschar) and increased intracompartmental contents, caused by bleeding or hemorrhage into the compartment, coagulation disorders, infiltrated

Salter-Harris fractures

The five main types of pediatric fractures involving the growth plate are:

- **Type I,** a transverse fracture through the growth plate that separates the epiphysis from the metaphysis.
- **Type II,** a fracture through the growth plate and the metaphysis, sparing the epiphysis. About 75% of femur fractures fall into this type.
- **Type III,** a fracture through growth plate and epiphysis, sparing the metaphysis.
- **Type IV,** a fracture through all three elements of the bone, the growth plate, metaphysis, and epiphysis.
- **Type V,** a compression fracture of the growth plate, resulting in a decrease in the perceived space between the epiphysis and diaphysis on X-ray.[47,48]

Accurate diagnosis of Salter-Harris fractures is essential so that the fracture can be anatomically reduced (realigned) and the growth plate can continue to grow. Failure to diagnose and treat these fractures can result in abnormal or arrested bone growth in children and adolescents.[47]



I.V. fluids, improper positioning, venous pooling, or increased capillary filtration.[5,29] Patients at high risk for compartment syndrome include trauma patients, those experiencing periods of hypoperfusion, and patients with crush injuries.[5,29]

Signs and symptoms of compartment syndrome include pain that's out of proportion to the injury and isn't relieved by opioid analgesics. The "6 Ps" of compartment syndrome are:

- pain on passive stretch of the compartment
- paresthesia along dermatomal patterns
- pulselessness
- pallor of the extremity
- polar or poikilothermia
- paralysis of the affected limb.[5,29]

Remember that irreversible ischemia can occur even in the absence of these symptoms.[30] Definitive diagnosis of compartment syndrome requires intracompartmental pressure readings.[5,29,30] Most experts consider pressure of 30 to 45 mm Hg diagnostic of compartment syndrome and the need for surgical fasciotomy, although the required pressure level is controversial.[30]

Treatment of compartment syndrome depends on the severity of the vascular compromise. The limb should never be elevated above the level of the heart because blood flow is further compromised as an effect of gravity.[5,29,30] Surgical fasciotomy to relieve the pressure is the definitive treatment.[5,29,30]

Fat embolus syndrome (FES) may occur after long-bone fractures, joint reconstruction, cardiopulmonary bypass, liposuction, and burns, and also occurs in patients with sepsis, diabetes, and acute pancreatitis.[31] The mechanical theory of FES pathogenesis states that large fat droplets are released into the venous system, deposited in the pulmonary capillary beds, and travel through arteriovenous shunts to the brain. Local ischemia and inflammation develop as a result of microvascular lodging of droplets, with subsequent release of inflammatory mediators and vasoactive amines.[31]

The biochemical theory states that trauma or sepsis causes hormonal changes that induce systemic release of free fatty acids as chylomicrons. Acute-phase reactants, such as C-reactive proteins, cause chylomicrons to form, triggering inflammation. This theory helps explain nontraumatic forms of FES.[31] A patient with FES usually has the classic triad of tachypnea, mental status changes, and a nonblanching petechial rash on the anterior chest, axilla, and mucous membranes.[31] No lab or imaging tests are specific to FES, but it should be suspected in patients with long-bone fractures who develop hypoxia, bilateral pulmonary infiltrates, and a rash.[31] Treatment is supportive and is aimed at maintaining adequate oxygenation and hemodynamic status. Early fixation of long-bone fractures (within 24 hours of injury) can reduce the patient's risk of developing acute respiratory distress syndrome.[31]

VTE is often seen in patients who sustain femur fractures, especially in those who require prolonged bedrest.[5,13,32] The formation of a deep-vein thrombus is more likely in patients with venous stasis, endothelial injury, and a hypercoagulable state (Virchow triad).[33] In patients with a femur fracture, damage to the vascular endothelium releases tissue factor (tissue thromboplastin) that initiates the clotting cascade.[34] Hypercoagulability (an abnormal level of procoagulation factors) is often caused by hemoconcentration and release of inflammatory cytokines initiated by the injury.[34]

Follow the guidelines for anticoagulation therapy for the prevention and management of VTE.[35] In patients with contraindications to anticoagulation therapy, such as patients with concurrent spinal cord injury or closed head injury (which raises the risk of bleeding), mechanical methods such as sequential compression devices, venous foot pumps, or graduated compression stockings can be used.[33] Mechanical prophylaxis for VTE can be used in combination with pharmacologic agents such as low-molecular-weight heparin or warfarin.[36] If the patient suffers from polytrauma that makes anticoagulation therapy impossible, an inferior vena cava filter can be inserted to prevent pulmonary emboli (PE).[37]

Symptoms of thrombophlebitis include warmth and swelling of the leg or legs. Homans sign (pain in the calf on forced dorsiflexion of the foot) isn't diagnostic of thrombophlebitis, but is often used at the bedside as a screening tool.[5] Symptoms of a PE include pleuritic chest pain that increases with inspiration, dyspnea, tachycardia, and anxiety.[33] Patients will describe a "feeling of impending doom."[5]

Treatment of VTE involves infusing I.V. unfractionated heparin, then transitioning the patient to oral warfarin, with dosage adjustments until the patient's international normalized ratio is between 2.0 and 3.0.[35]

Multiple organ dysfunction syndrome (MODS), another complication of multiple trauma, is a leading cause of death in trauma patients.[38,39] Massive injury leads to a systemic inflammatory response syndrome that can spiral into tissue destruction in organs not originally affected by the initial trauma. The proinflammatory response is followed by an anti-inflammatory response that can result in immune suppression with an increased risk of infection and sepsis.[38,39] Sepsis alters the fragile balance between oxygen delivery and consumption as well as the coagulation pathways, leading to cellular hypoxia and organ death.[38,39] Preventing hypoxia and promptly treating infection can decrease the incidence of MODS.[38,39]

Infection rates of soft tissue and bone are higher in patients with open fractures, so surgical debridement and antibiotic administration should be started as soon as possible.[40] Removing devitalized bone and tissue prevents continuing inflammation with fluid shifts within the wound. Negative-pressure wound therapy can be used over open wounds and has been shown to reduce the number of superficial and deep-wound infections after severe open fractures.[41]

Osteomyelitis, or bone infection, is a late complication of open fractures that can linger for years. The risk of infection can be significantly lowered by administering a short course of first-generation cephalosporins as soon as possible after injury, in combination with prompt, orthopedic fracture wound management.[42] High-grade open fractures result in extensive skin and soft tissue loss, and free muscle flaps and skin grafts are sometimes needed to provide wound and bone coverage to promote healing.[43]

Hardware failure, a late complication of fractures, can occur before or after fracture healing, and is defined as a break in the rod, plate, or screws used for internal or external fixation. Micromotion across the hardware at the fracture site is commonly cited as a major factor in hardware failure.[44]

Malunion of a fracture occurs when the bone doesn't heal in an anatomically correct position. *Nonunion* is defined as a fracture that remains unhealed 6 months after injury.[45] Failure of the immobilization device and the amount of wound contamination and bone loss contribute to malunion and nonunion of fractures. Patient nutrition, adequate immobilization, patient comorbidities, medications, social factors, and type of fracture also influence the outcome of fracture healing.[12] Normally, a fracture heals in four stages after the initial inflammatory phase and hematoma formation:

- granulation tissue formation
- callus (immature bone) formation
- lamellar bone deposition and ossification
- remodeling, in which the bone returns to its normal shape and architecture.

Mending Matt

Matt Lewis, whose femur was fractured in the motorcycle crash, is transferred to the OR immediately. About 20 minutes before the start of surgery, he receives I.V. cefazolin and gentamicin. During surgery, two units of packed red blood cells are infused, raising his BP to 130/78 and reducing his pulse to 92. The orthopedic surgeon performs a careful irrigation and debridement of the devitalized muscle, and inserts an unreamed intramedullary rod with proximal interlocking screw into the right femur. The vascular surgeon repairs the superficial femoral artery, and Matt's distal pulses are now palpable and his foot is warm and pink with normal capillary refill time. The wound was left open and negative-pressure wound therapy is started. Matt is placed in a padded posterior splint with elastic wrap and transferred to the postanesthesia care unit.

After an uneventful recovery, Matt was transferred to the surgical ICU for closer monitoring of his neurovascular status. He'll continue receiving antibiotics and will receive morphine via a patient-controlled analgesia pump and enoxaparin (a low-molecular-weight heparin) via subcutaneous injections. On postoperative day 2, he returns to the OR for further irrigation and debridement of his open thigh wound, which remains clean, but has delayed primary closure. The next day, the I.V. antibiotics are discontinued and he's transferred to the general orthopedic unit.

Seven days after his injury, Matt is discharged home. He'll continue to receive outpatient physical therapy for general strengthening exercises, and within 6 months, his femur fracture has healed and he's returned to college and resumed all his previous activities. Although he had a major open fracture, it was an isolated injury without other body systems involved, and he didn't have significant soft tissue or bone loss. Although his vascular injury with hemorrhage placed him at higher risk for infection and other complications, prompt fluid resuscitation prevented serious hypoperfusion, and the healthcare team's quick, appropriate interventions all helped to promote a positive outcome.

By understanding your role in managing femur fractures, you can help other patients avoid life-threatening injuries. **OR**

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Colleen Walsh is a faculty member in graduate nursing at the University of Southern Indiana's College of Nursing and Health Professions in Evansville, Ind.

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