Chapter 21

EXTREMITY INJURIES

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INTRODUCTION

Combat casualties with orthopedic and soft-tissue injuries of the extremities constitute the majority of soldiers who require operative procedures in the corps and communications zones.[1] These injuries are usually not life threatening but are the major source of man-days lost by combat casualties. Extremity injuries are usually found in the secondary survey of the American College of Surgeons' Advanced Trauma Life Support (ATLS) examination unless the casualty has an obvious external hemorrhage associated with the injury.[2] In corps zone hospitals, casualties with extremity injuries undergo resuscitative surgery that is designed to control hemorrhage, decontaminate wounds, and stabilize fractures so the casualty can safely and comfortably be evacuated to hospitals in the communication zone or the continental United States (CONUS). Corrective surgery is undertaken in the higher-echelon hospitals for the purpose of closing wounds and restoring function. Providing anesthetic care in the corps support zone to casualties with extremity wounds may be difficult because such injuries commonly coexist with much more life-threatening injuries to the head or trunk. Multiple or simultaneous procedures that involve multiple surgical teams attending the casualty together or in succession may be required.

INJURY PATTERNS

The nature and severity of extremity injuries depend on the mechanism of injury. In general, the magnitude of injury depends on the magnitude of energy transferred to the tissue. High-energy blunt trauma (eg, a high-speed motor vehicle accident) is associated with extensive soft-tissue destruction and severely comminuted fractures.[3] These generally need to be reduced early to control bleeding and infection; early treatment also allows early mobilization to enhance pulmonary toilet. Frequently there are coexisting injuries to the cardiac, pulmonary, abdominal, and renal organ systems that complicate the injury. Fat emboli, thromboemboli, deep venous thrombosis, and adult respiratory distress syndrome often complicate the long-term care of the casualty.[3,4]

Low-energy blunt trauma (eg, skiing injuries and some falls) cause relatively simple fractures with minimal soft-tissue destruction. These usually require less-complex treatment and are not usually associated with severe coexisting injuries.

As with injuries caused by blunt trauma, the nature of penetrating injuries depends on the amount of kinetic energy transferred to the tissues. However, because the rate of energy transfer is much greater with penetrating injuries from, say, a bullet than it is with blunt trauma from, say, a vehicular bump, the same amount of energy transferred by the former mechanism will cause more damage.

With penetrating injuries, the kinetic energy is related to the mass of an object multiplied by its velocity squared. Therefore, doubling the velocity increases the kinetic energy 4-fold. The kinetic energy determines the maximum amount of energy that can be transferred, but whether the energy transfer actually occurs is determined by other factors, of which shape, stability, and construction of the missile are especially important. The small M16 bullet—even though its muzzle velocity is high—possesses less than half the kinetic energy of typical rifle bullets used during World War II; yet the M16 bullet is notorious for causing severe damage. Because of its propensity to fragment, a much greater fraction of the kinetic energy of the M16 bullet is usually transferred to the target tissue than occurred with bullets fired by earlier rifles. The surrounding tissue is damaged extensively by both the fragments and the temporary cavitation that is caused by the massive energy transfer. Skeletal muscle will be injured (*a*) by direct cutting and laceration caused by the bullet or fragments and (*b*) by being ripped apart by the stretch of cavitation. Several millimeters of necrotic muscle characteristically line the wound tract, although the major threat arises from infection in the surrounding tissue because it is both contused and contaminated with foreign material brought into the wound.[5]

In contrast, a low-velocity, penetrating injury (eg, a knife wound) is associated with only the local injury directly imposed by the object itself. A shotgun blast from a distance imparts only comparatively little energy to the body; when the wound is made at point-blank distance, however, all of the shell's energy—which is equal to that of an assault rifle bullet and is therefore much more dangerous—is imparted.

THE NATURE OF EXTREMITY INJURIES SEEN IN COMBAT CASUALTIES

The database compiled during the Vietnam War by the Wound Data and Munitions Effectiveness Team (WDMET) is a unique, underutilized repository of first-hand information on war wounds and the mechanisms of injury. (Chapter 1, Combat Trauma Overview, discusses WDMET data in some detail.) Analysis of the WDMET data reveals that about one half of combat casualties who survive long enough to be evacuated to the hospital level have injuries that involve only the extremities. In addition, a sizable number of the remaining casualties have injuries of the extremities in addition to more-serious injuries of the head or trunk (ie, combined injuries). About one third of extremity operations will be to treat injuries that involve only soft tissue (ie, skin, fat, and skeletal muscle). The remaining two thirds of extremity operations will be for injuries that involve soft tissue in addition to injuries to deeper structures such as long bones and neurovascular structures.[6] It is these latter injuries that may especially challenge the military anesthesiologist.

The prevalence of purely soft-tissue injuries has increased in recent wars due to the more frequent use of improved fragmentation munitions. These characteristically cause multiple, small fragment wounds (Figure 21-1). Another form of purely soft-

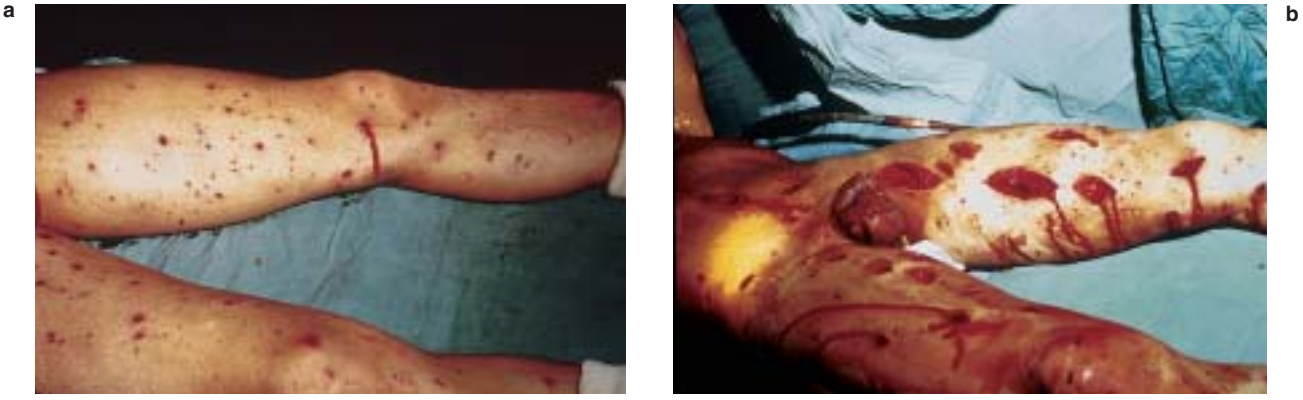


Fig. 21-1. (**a**) This casualty has multiple, tiny, fragment wounds of his lower extremities that were caused by hand-grenade fragments. (**b**) The surgeons have elected to excise some of the larger wounds, although there was probably little tissue damage or contamination. Photographs: Swan Vietnam Surgical Slide Collection.

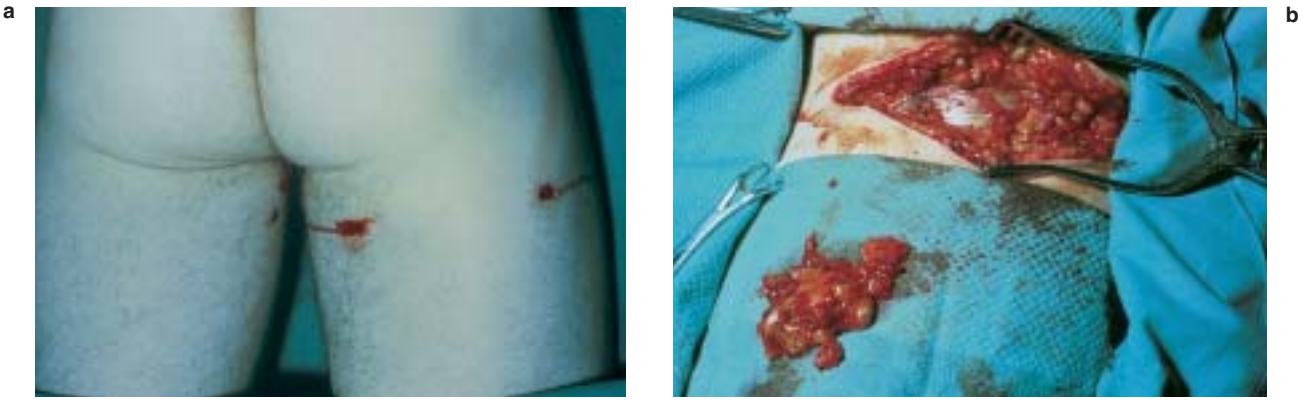
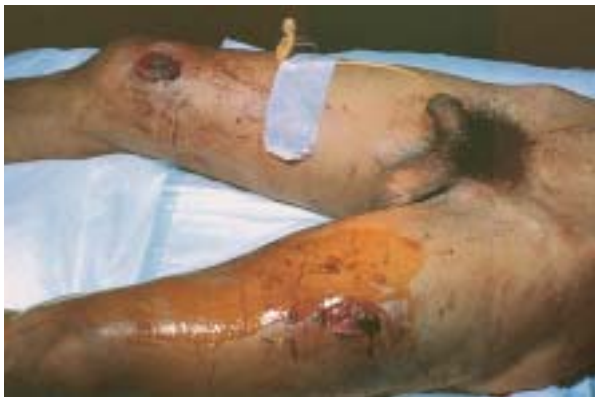


Fig. 21-2. (**a**) This casualty's through-and-through wound in the posterior thigh was made by a bullet fired by a .38-caliber pistol at close range. (**b**) The wound has been incised and the subcutaneous tissue superficial to the muscle fascia has been excised. It is possible that this wound could have been treated nonsurgically. Photographs: Swan Vietnam Surgical Slide Collection.

Fig. 21-3. This soldier was struck by several fragments from an exploding booby trap. (**a**) Wounds of entrance are seen in the left lateral thigh and just proximal to the right knee. There were no wounds of exit. (**b**) The radiograph shows a grossly comminuted fracture of the middle portion of the left femur. Several metallic foreign bodies are seen. (**c**) The radiograph shows intact bones in the casualty's right knee. Several small metallic foreign bodies are seen. (**d**) The casualty's appearance after wound debridement. The extensive nature of the operation carried out on the left thigh is justified by the need to excise injured soft tissue in proximity to the femur fracture. The extensive nature of the operation carried out on the casualty's right thigh is justified by the need to preclude an injury to the superficial femoral artery. (**e**) The casualty had delayed primary closure on the 6th day after the injury. Photographs: Swan Vietnam Surgical Slide Collection.



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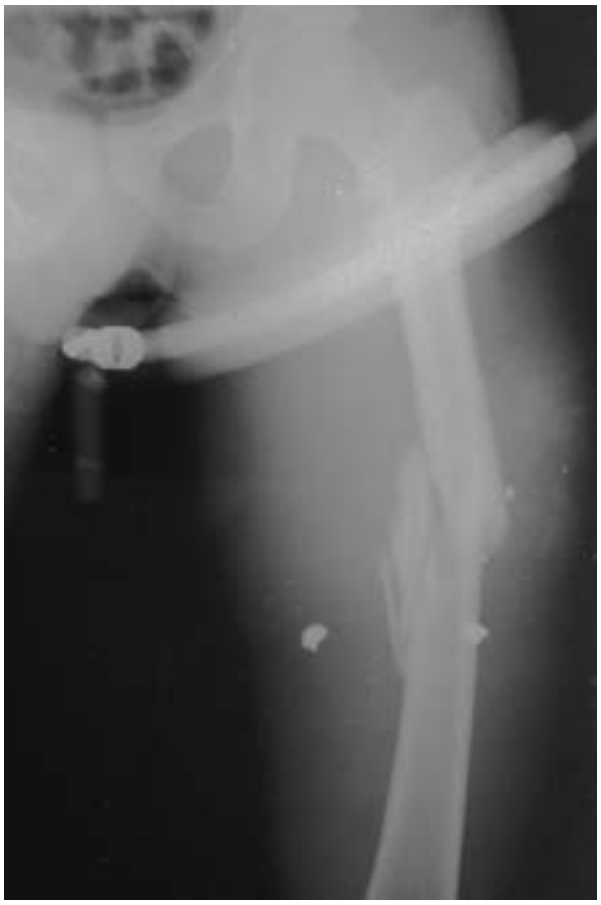

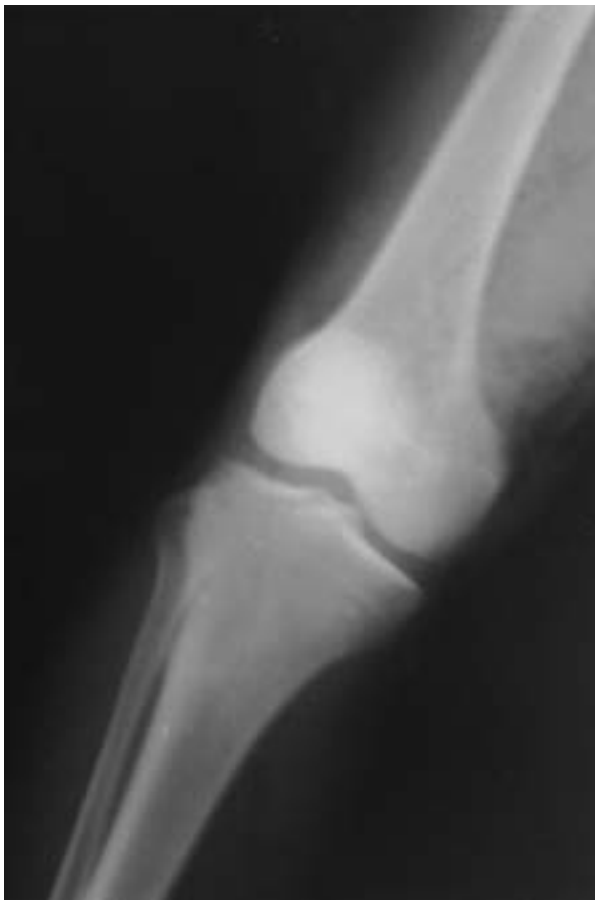

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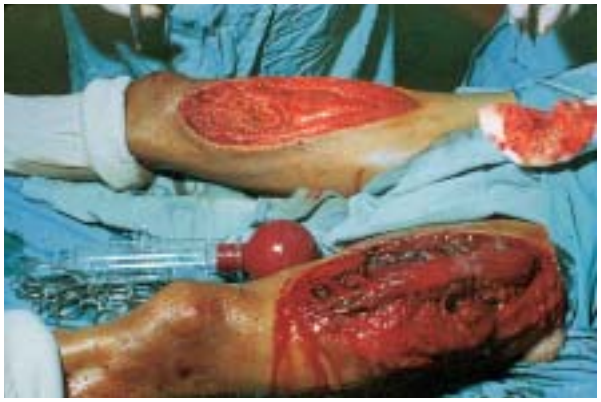

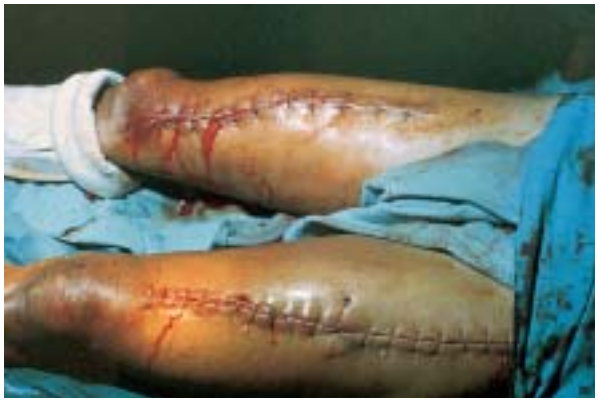

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tissue injury is the *en seton* (ie, through-and-through) gunshot wound (Figure 21-2). Although en seton wounds may be made by high-velocity bullets, the tissue damage is surprisingly small because energy transfer has been minimized by the failure of the bullet to yaw, tumble, fragment, or deform. The treatment of an en seton wound should be no different from the treatment given to a casualty with a wound made by a low-velocity bullet fired from a civilian hand gun.

By way of contrast, very extensive soft-tissue wounds are frequently seen in conjunction with open, comminuted, long-bone fractures (Figures 21-3 and 21-4). In these wounds, the missile is stopped by the bone with consequent maximal energy transfer, which, together with secondary missiles arising from bone fragments, causes massive tissue damage.

Even more massive tissue damage to the extremities is found in casualties who detonate antipersonnel mines or who are injured by shaped-charge warheads. Such injuries almost always result in traumatic amputations (Figure 21-5).

The increasing likelihood of involvement by the U.S. military in operations other than war (OOTW) makes it likely that military anesthesiologists will be confronted by casualties, many of whom are civilians, who have neglected extremity injuries. Figure 21-6 shows such a patient.

In the WDMET database, about 35% of the extremity injuries involve an isolated fracture of an extremity long bone. Lower-extremity fractures were 2.5-fold more common than those of the upper extremity. About 12% of casualties with extremity wounds had fractures of the bones of the hands or feet. About 7% of casualties with extremity wounds had a major extremity amputation (arm or leg) and about 5% had amputations of the hands, feet, fingers, or toes. Of casualties with extremity wounds, 12% had an isolated vascular injury (the femoral artery the most common), and 5% had an isolated nerve injury (the sciatic nerve the most common).

Of casualties who survived to reach the hospital level, 6% had an injury that involved both a fractured bone and a vascular or nerve injury. The most common combinations were a fractured femur and a femoral arterial injury, and a fractured humerus with an injury to the radial nerve.

In summary, the most common extremity injuries—exclusive of those of soft tissue only—likely to be seen by the military anesthesiologist are

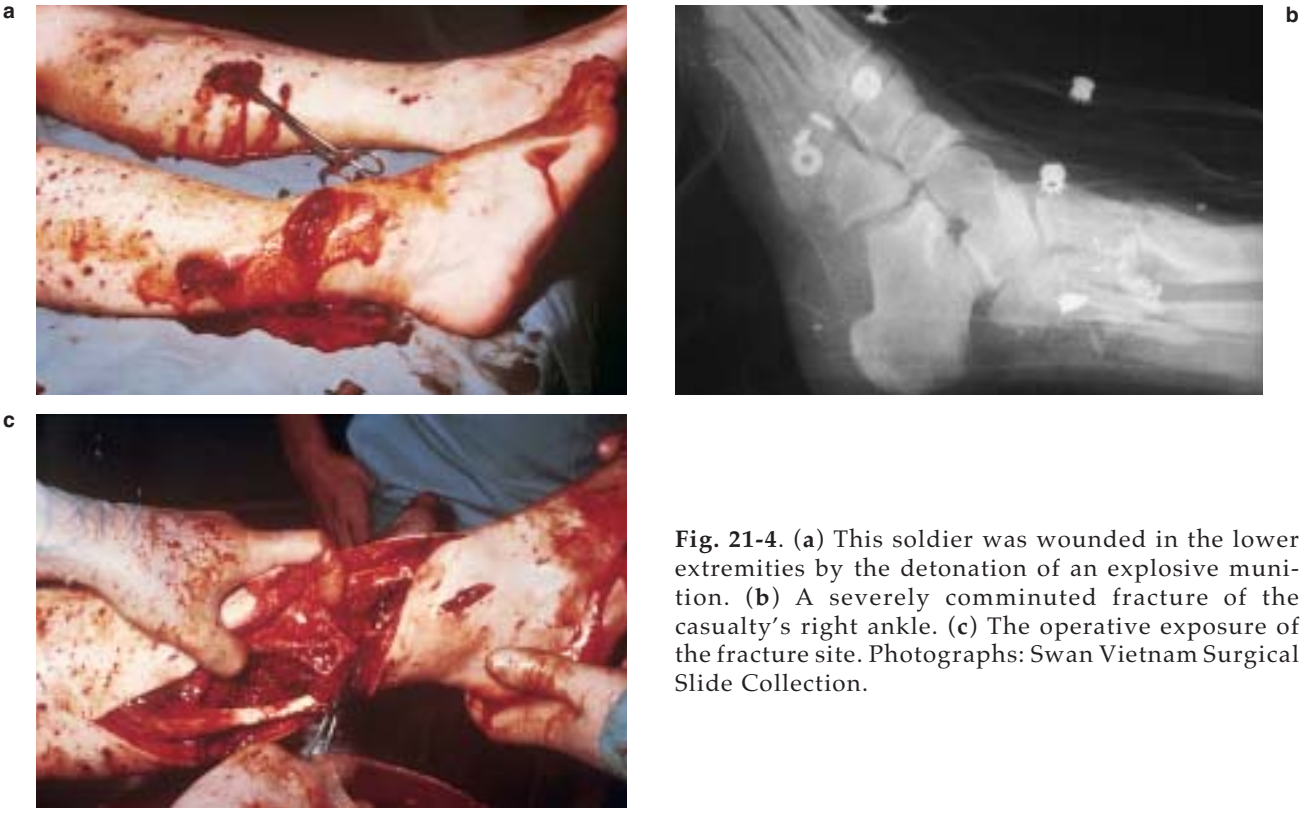

Fig. 21-4. (**a**) This soldier was wounded in the lower extremities by the detonation of an explosive munition. (**b**) A severely comminuted fracture of the casualty's right ankle. (**c**) The operative exposure of the fracture site. Photographs: Swan Vietnam Surgical Slide Collection.

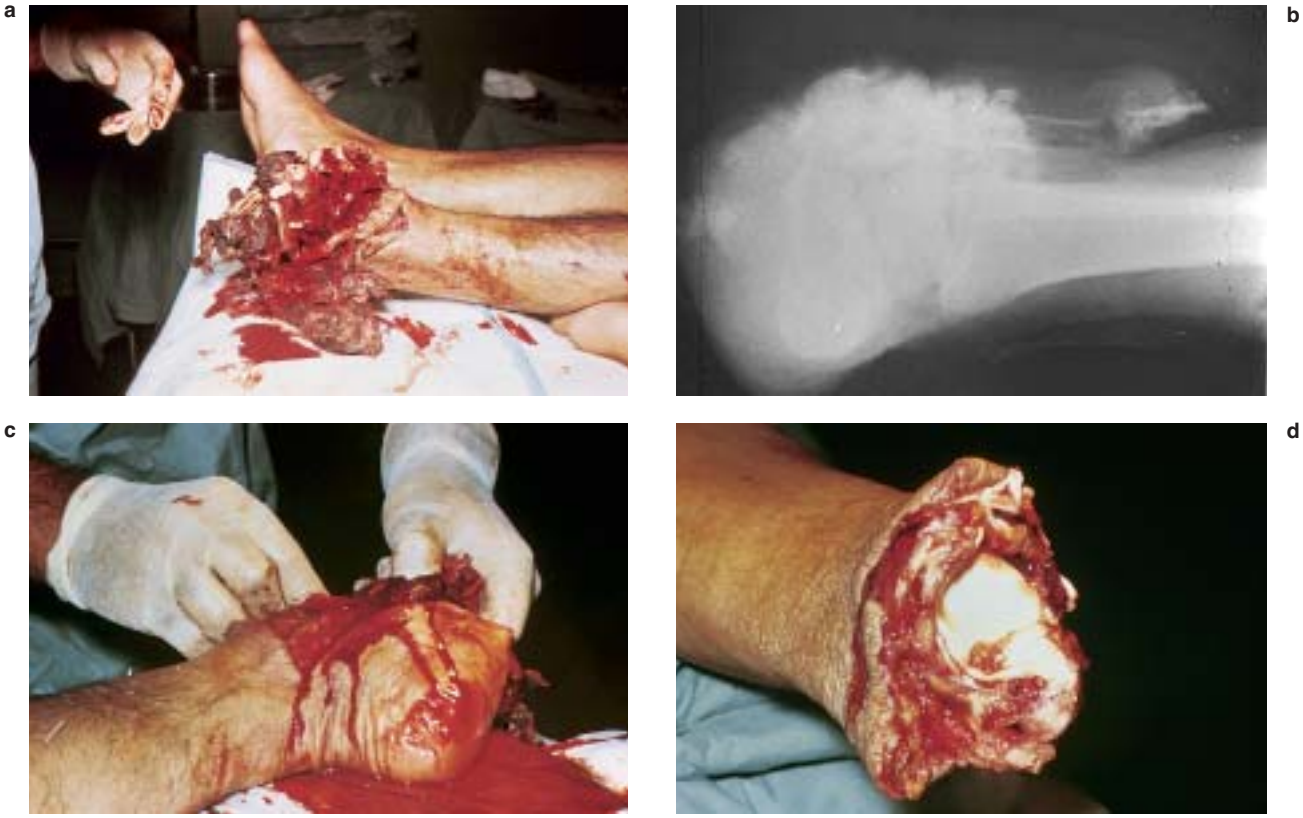

Fig. 21-5. (**a**) This soldier detonated an antipersonnel mine; a traumatic amputation of the distal portion of his foot (ie, the forefoot) has occurred. (**b**) The radiograph shows the extent of bony injury. (**c**) A formal forefoot amputation is in progress. (**d**) The finished amputation. Note that the forefoot has been removed at the talus. This is known as a Syme's amputation. Retaining sufficient skin to cover the stump is sometimes difficult to achieve with this procedure. Photographs: Swan Vietnam Surgical Slide Collection.

- an open, comminuted fracture of a long bone, the femoral shaft being the most common site;
- an amputation of the leg, arm, hand, or foot; and
- an isolated vascular injury, the superficial femoral artery being the most common site.

Management

Shock and sepsis are the two most serious medical treatment problems that may arise in combat casualties with extremity trauma. The relative importance of these two causes of death has been changed by modern military surgery. During World War I and earlier wars such as the American Civil War, sepsis in the injured extremity was the most frequent cause of death in hospitalized casualties. With the use of wound excision and antibiotics, life-threatening sepsis has become much less common.

Shock

Extremity wounds can be fatal. The WDMET database indicates that about 9% of casualties with extremity wounds that involved more than just soft-tissue damage died. Most casualties with injured extremities who were fatally wounded were killed in action; the most common cause of death was exsanguination—from amputation of an arm or a leg, or from lacerations of the femoral artery. The few casualties with extremity injuries who are at risk of dying at the hospital level have sustained either massive blood loss or have developed life-threatening sepsis, usually from an anaerobic wound infection.

Blunt injuries to the extremities may also be associated with hypovolemic shock secondary to hemorrhage, but this occurrence is due not to massive hemorrhage but to prolonged, slow bleeding secondary to rupture of intraosseous blood vessels or lacerations of blood vessels near the site of frac-

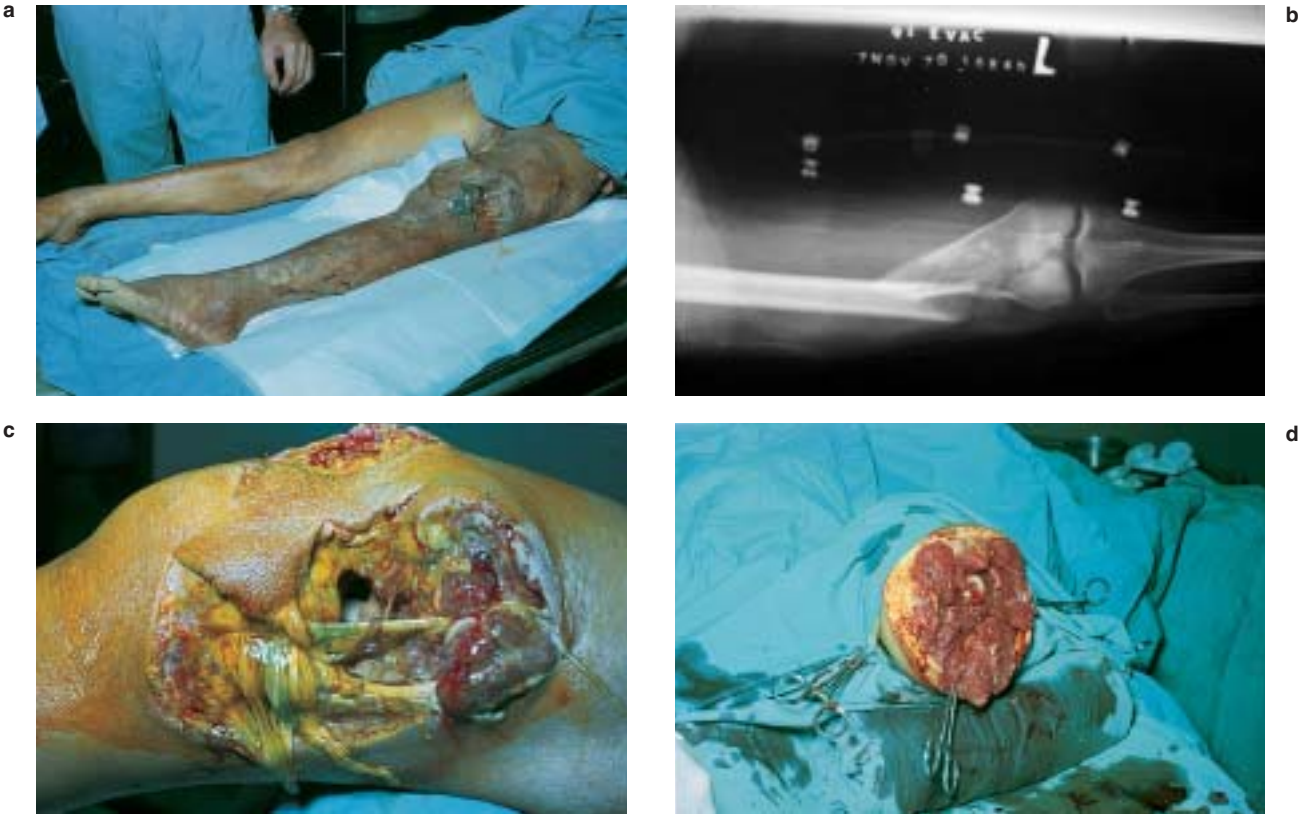

Fig. 21-6. (**a**) This woman has a 3-day-old open comminuted fracture of the left femur that was caused by a perforating gunshot wound. (**b**) The radiograph shows the comminuted fracture. (**c**) A close-up view of the wound of entrance. The wound shows signs of anaerobic sepsis. (**d**) An above-the-knee amputation of the guillotine type has been performed. Such an operation may be lifesaving when the patient has an infected open fracture. Photographs: Swan Vietnam Surgical Slide Collection.

ture.[4,7] Large volumes of blood can be sequestered in limbs even without significantly changing limb size.[8] Therefore, refractory shock in the casualty with blunt trauma to an extremity requires that a search be made for occult fractures. Large volumes of blood and blood products may be required, and coagulopathies should be anticipated (Table 21-1).

Pneumatic antishock garments can be beneficial in stabilizing fractures. These trousers have limited value in correcting hypovolemia, as the intravascular volume returned to the central circulation is minimal. They are beneficial in that they temporarily stabilize fractures of the pelvis and lower extremity and decrease the bleeding.[9]

Sepsis

The goal of military medical management is to return the injured soldier to full duty. This is accomplished by promoting rapid and complete

TABLE 21-1

POTENTIAL BLOOD LOSS FROM CLOSED FRACTURES

Expected Blood Loss (mL)	Bone Fractured
500	Forearm: radius and/or ulna
750	Elbow Tibia Ankle
1,000	Humerus Femoral shaft
1,250	Hip
> 1,500	Pelvis

Adapted with permission from Shumaik GM. Extremity trauma. In: Baxt WG, ed. *Trauma: The First Hour*. Norwalk, Conn: Appleton-Century-Crofts; 1985: 227.

healing of the wound. Sometimes, as in the case of an amputation, this is not possible. Return to duty is also unlikely for many casualties with severe fractures or nerve injuries. In most casualties, the major factor delaying wound healing and return to duty is sepsis, which occurs in about 5% of casualties with pure soft-tissue injuries but in as many as 30% or more of casualties with open, comminuted fractures of the femoral shaft.[10,11]

Wound sepsis is prevented by surgical excision of the contaminated and damaged tissue lining the wound tract. The operation is commonly known as *debridement*, although, strictly speaking, debridement means only wound incision. In general, the amount of tissue to be excised is related to the size of the wound. Tiny extremity wounds can be left alone, but this is rarely the case with the large wounds of exit that frequently accompany extremity fractures. Even in casualties with only soft-tissue wounds, debridement can be an extensive operation (see Figure 21-1). Perioperative administration of antibiotics—especially if they suppress the growth of clostridial bacteria and *Streptococcus pyogenes*—may be a useful adjunct. Wound sepsis that occurs weeks after injury is usually the result of the growth of staphylococci or Gram-negative organisms in open wounds that contain dead tissue. Such wounds require redebridement.

Surgical Treatment

The most common operative procedure performed by military surgeons is wound debridement, and the most common sites for this operation are the extremities. The goal of debridement is to decontaminate injured tissue so that healing is not impeded by local infection. Following debridement, a surgical intervention is usually required to bring about wound closure. In contrast to civilian practice, the military surgeon rarely closes the wound at the time of initial debridement. The most common approach, with the patient anesthetized, is to inspect the wound 4 to 6 days after wounding and to close the wound at that time if there is (*a*) no evidence of other than superficial infection and (*b*) little necrotic tissue that cannot be easily removed. This procedure is known as *delayed primary closure.* The delay in closure is a consequence of both the inability of the surgeon to be certain that all damaged and contaminated tissue has been removed and the provision of military medical care by echelons. Primary closure by a surgeon at one echelon, followed by the patient's evacuation to a higher echelon, would remove the casualty from observation just when the most lethal form of sepsis—gas gangrene—is most likely to appear.

Secondary closure is the name applied to the operation performed on extremity wounds that cannot undergo delayed primary closure but are closed after the appearance of granulation tissue—7 to 10 days after wounding. Some amputation stumps undergo secondary closure; however, most are not closed surgically but are allowed to close by scaring, a process known as healing by *secondary intention*. Reamputation of the stump is a common operation: the soft tissue retracts so much that the bone is relatively too long.

The most common vascular injuries involve the femoral artery, brachial artery, and femoral vein. Arterial reconstruction usually necessitates the insertion of a vein segment, which is usually taken from the saphenous vein of the opposite leg. Reoperation is a common occurrence following extremity arterial reconstructions in combat casualties: thrombosis, delayed hemorrhage, and wound sepsis seem to be more common than in civilian patients who have peripheral vascular operation. If the vascular injury coexists with a fracture, it is essential that the fracture first be stabilized. External fixation is especially useful for this purpose. It is desirable to reconstruct an injured femoral vein, especially when there is a coexisting injury to the femoral artery. In most venous reconstructions, thrombosis probably occurs in the repaired vein within 2 to 3 days; however, the vein stays open long enough to keep the arterial reconstruction open during the critical first 2 or 3 days. Venous ligations may be an acceptable alternative to reconstruction in the casualty with multiple injuries.

Immobilization

Because fractures are very common in soldiers with extremity wounds, immobilization is a necessary part of most extremity operations. In general, three approaches to fracture immobilization can be employed in preparing the casualty for evacuation from the combat or communication zones:

1. Internal fixation, wherein a metal rod is inserted longitudinally through the marrow cavity, or metal screws and plates are used to hold the fracture together. This approach is rarely desirable in combat casualties with open, comminuted fractures because of the strong possibility of sepsis.
2. External fixation, wherein long, threaded rods are inserted transversely into the bone

fragments above and below the fracture site and are held in place externally by an adjustable frame. This is an increasingly favored means of immobilizing fractures such as those of the tibia, but the approach depends on the availability of the necessary equipment.

3. Plaster of paris cast or splint, which is the traditional approach to immobilizing fractures, and is still the best approach to high femoral-shaft and hip fractures. The drawbacks to this approach are the discomfort of the casualty and the difficulty of exposing the soft-tissue component of the wound.

COMPLICATIONS

Injuries to other parts of the body frequently coexist with extremity injuries. The most common combinations are abdomen and legs, especially when the wounds are made by buried explosive munitions; and head, upper torso, and arms, when the wounds result from the detonation of a shaped-charge warhead. Wounds to the abdomen are discussed in Chapter 20, Abdominal Injuries; to the head in Chapter 16, Neurological Injuries; and to the upper torso in Chapter 19, Thoracic Injuries.

In addition to managing combined injuries, the military anesthesiologist needs to be prepared to manage complications that are unique to extremity injuries, of which compartment syndrome and thrombolic and embolic phenomena are the most serious.

Compartment Syndrome

Although compartment syndromes are not common in combat casualties for the simple reason that the wound tract usually decompresses the compartment via the wounds of entrance or exit, it may be seen following vascular reconstructions, especially when there has been a delay in operating.[12] Compartment syndromes are orthopedic emergencies that require early treatment to prevent long-term disability.[4,13] The myofascial compartments are of a fixed volume, and after injury, edema and hemorrhage cause increased pressure in this fixed space. The increased pressure causes decreased venous outflow and decreased microvascular perfusion of the tissues. As muscle dies, it losses its normal ability to regulate intracellular water and electrolyte concentrations. The result is a vicious cycle: swelling of the cells, further increase in compartment pressure, and decreased arterial flow.[3,14] If the cycle is not interrupted by early fasciotomy to decrease tissue pressure, severe muscle necrosis and peripheral nerve damage will follow, and a severely disabled, fibrotic myofascial compartment with severe disability will be the result (Figure 21-7).

Pain is the most frequent symptom of increased compartment pressures.[3] Decreased pulses distal to a fracture site should be a warning of an impending compartment syndrome or vascular injury. While full evaluations of motor and sensory function are difficult to perform early in the management of an extremity injury, function distal to a fracture site suggests that peripheral nerve damage, if any, is incomplete. Early diagnosis is important to decrease damage to the tissue. The clinical picture can be confusing, but pain on passive range of motion of the affected muscle and distal sensory deficits are the most reliable indicators. The patient will complain of subjective pain out of proportion to the degree of apparent injury, and may note a tense feeling in the tissue. Physical examination can reveal tense compartments with warm, tense skin overlying the area. Pain with passive range of motion of the joint is an important indicator, but can be confusing in the presence of other injuries.[15] Laboratory determinations are of little help in combat zone hospitals because the needed tests are not available, but elevated levels of creatine kinase and myoglobinuria are consistent with muscle destruction.

Compartment pressures can be evaluated in fractured limbs or in nonfractured limbs where the mechanism of injury suggests ischemia (ie, crush injury).[3] These pressure determinations can be helpful when the patient is unable to answer questions (eg, the patient is under anesthesia, uncooperative, or has proximal nerve injury and sensory nerve damage). The most common method of determining compartment pressure is to insert an 18-gauge needle into the compartment in question, and then connect the needle to a pressure transducer. There are no absolute pressure ranges to allow a definitive diagnosis. The normal intramuscular pressure is 0 to 8 mm Hg.[16] Various investigators recommend fasciotomy at various pressures: from 30 to 35 mm Hg,[17] to greater than 45 mm Hg,[18] to decompression when the intramuscular pressure is within 10 to 30 mm Hg of the diastolic pressure.[19]

Although the preceding discussion has emphasized the deleterious local effects of compression syndrome, dire systemic effects may also occur.

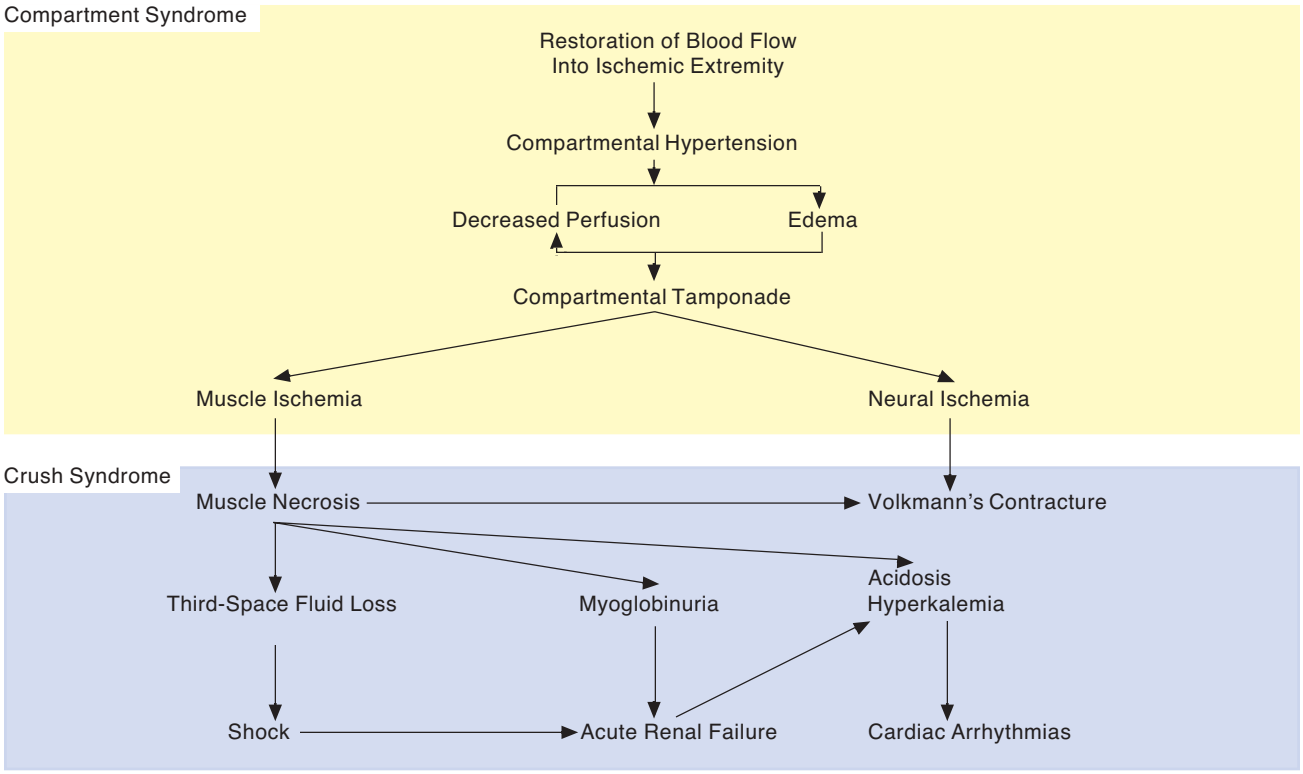

Fig. 21-7. The pathophysiology of compartment and crush syndromes. Adapted with permission from Kitka MJ, Meyer JP, Bishara RA, Goodson SF, Schuler JJ, Flanigan P. Crush syndrome due to limb compression. *Arch Surg*. 1987;122:1078–1081.

When systemic effects are present, the condition is known as crush syndrome. When the ischemic region is reperfused—either when arterial inflow is restored following a vascular reconstruction or when external compression is relieved, untreated or inadequately treated—the injured area may swell explosively due to fluid sequestered there. In addition, myoglobin from necrotic muscle is washed into the systemic circulation, which, in conjunction with hypovolemia, can cause acute renal failure. The prudent military anesthesiologist will see that a prophylactic approach is taken to prevent compartment syndrome by assuring that fasciotomies are performed in all casualties with extremity wounds whose arterial reconstruction has been delayed.

Thrombolic and Embolic Phenomena

Deep Venous Thrombosis and Pulmonary Embolism

The multiply injured combat casualty is at risk for deep venous thrombosis and pulmonary embolism. While soldiers usually have no underlying cardiopulmonary disease, those who have multiple injuries have multiple risk factors to develop a hypercoagulable state. Trauma to femoral or pelvic veins is associated with a high incidence of deep venous thrombosis and subsequent pulmonary embolism.[20,21] Additionally, surgery, venous stasis, trauma, immobilization, vascular damage, disseminated intravascular coagulation, thrombocytopenia, and the administration of heparin are associated with deep venous thrombosis.[22,23] Peripheral thrombosis below the knee is common in surgical patients, with an incidence approaching 45%, and with 10% of these patients having symptoms of pulmonary embolism.[24] Casualties having major lower-limb orthopedic reconstruction are at high risk for two reasons: (1) there is extensive vascular damage and (2) they will be immobilized for an extended time. It seems unlikely that combat casualties who have had extremity venous reconstructions are at a higher risk of deep venous thrombosis and pulmonary embolism than are casualties with isolated arterial reconstructions and casualties in whom the venous injury has been treated by ligation. Patients with thrombophlebitis and patients older than 40 years of age with extensive abdominal

surgery have an incidence of calf-vein thrombosis of 40% to 80%, proximal vein thrombosis of 10% to 20%, and fatal pulmonary embolism of 1% to 5%.[25] Clearly, pulmonary embolism is a major risk factor in this surgical group, and recognition, treatment, and prophylaxis are mandatory.

Intraoperative presentations are quite dramatic and have been associated with leg manipulation, wrapping the leg with Esmarch bandage, and realigning of the proximal femoral fracture component; they have been seen days after the initial trauma.[26,27] The hemodynamic picture is one with increased central venous and pulmonary artery pressures, hypotension, and tachycardia.[28] The pulmonary capillary wedge pressure is unchanged unless there is underlying cardiac disease.[29] The cardiac output does not correlate to the degree of obstruction of the pulmonary vasculature.[28] The right ventricle is prone to failure, as it is a highly compliant ventricle that decompensates with acute increases in right ventricular afterload.[30–32] The degree of pulmonary hypertension correlates with the acute degree of obstruction. The mean pulmonary artery pressure never exceeds 40 mm Hg unless there is coexisting cardiac disease. A pulmonary artery pressure of 22 mm Hg correlates with a 30% obstruction of pulmonary vasculature, while 36 mm Hg correlates with 50% obstruction.[28] Further decompensation occurs when a coexisting patent foramen ovale allows right-to-left shunting of blood when pressure on the right side of the heart exceeds pressure on the left.[33]

Bilateral wheezing and arterial blood gas evidence of hypoxemia and hypercarbia, together with nonspecific roentgenographic chest findings, should alert the prudent practitioner to the possibility of pulmonary embolism.[34,35] As dead-space ventilation increases secondary to decreased perfusion, atelectasis ensues, with loss of surfactant in the involved alveoli. Platelet-mediated vasoconstriction and bronchospasm also contribute to the clinical picture.

Diagnosis. The diagnosis of pulmonary embolism needs to be made early because empirical treatment with heparin can lessen the disease process.[34] Pulmonary angiography is the standard for diagnosis but is very invasive and may be difficult to perform in combat zone hospitals. Ventilation–perfusion scans attempt to demonstrate lack of perfusion to ventilated lung; however, these are associated with a 25% to 40% false-negative rate in low-probability scans, and a 29% false-positive rate in high-probability scans. As most emboli arise in the proximal veins, impedance plethysmography is a highly specific, noninvasive test that can diminish the need for angiograms.[36] When needed, a pulmonary artery catheter can be used for pulmonary arteriography.

Treatment. The aim of treatment is to maximize the cardiac output and oxygen delivery to the body tissues. In the early treatment of the patient with right ventricular failure secondary to pulmonary embolism, the arterial partial pressure of oxygen decreases due to increased shunting of blood in the pulmonary bed. But oxygen delivery may actually be increased to tissues, since the mixed venous oxygen content is increased.[37]

Judicious volume infusion is indicated to increase central venous pressure to 12 to 15 mm Hg. Increasing the central venous pressure can shift the interventricular septum and compromise left ventricular filling because the noncompliant pericardium produces tamponade-like hemodynamics.[38]

Inotropic drugs are used to improve both the cardiac output and coronary perfusion. When the right ventricle is failing, there is a decrease in right ventricular perfusion and a loss of the continuous perfusion that is seen on the right side of the normal heart. This leads to a vicious cycle: decreased cardiac output leads to decreased perfusion, which leads to further decreased cardiac output, which leads to further decreased perfusion, and so on. Isoproterenol has been tried, but hypotension, tachycardia, and dysrhythmias have detracted from its usefulness.[39] After volume loading to central venous pressure of 12 to 15 mm Hg, dobutamine[40] and dopamine[39] have been used successfully to increase cardiac output, maintain blood pressure, and decrease pulmonary vascular resistance. In dog models, norepinephrine has increased cardiac output with improvement of right ventricle perfusion.[41]

Pulmonary vasodilators such as prostaglandin E_1 are nonspecific systemic vasodilators and can decrease right ventricular perfusion, and have been combined with norepinephrine.[34]

Heparin has been shown to reverse pulmonary vasoconstriction and bronchoconstriction from thrombin activation and platelet aggregation. Administering a bolus of heparin has been recommended when the clinical picture is highly suggestive of pulmonary embolus (even before definitive diagnosis).[33,42–45] The possibility of increasing a surgical patient's risk of hemorrhage must be weighed against heparin's vasodilating and bronchodilating actions.

Positive end-expiratory pressure (PEEP) needs to be used judiciously, if at all. Venous return to the heart is impeded, and stress to the right ventricular wall and

oxygen consumption are increased. Small tidal volumes at rapid rates with no PEEP will help maximize cardiac output and mixed venous oxygen.[46–48]

Prophylaxis. Along with maintaining a high index of suspicion, military trauma anesthesiologists need to consider prophylaxis against deep venous thrombosis in all patients with orthopedic injuries. Low-dose heparin (5,000 units administered subcutaneously 2 h before surgery and every 8 h after, for several days) has been shown to decrease deep venous thrombosis, but it increases intraoperative bleeding, and there is no overall decrease in mortality.[49] Another concern with administering heparin is bleeding when regional anesthesia is used. External pneumatic compression stockings decrease venous stasis, induce fibrinolysis, are as effective as low-dose heparin,[50,51] and are especially effective when heparin is contraindicated. There may be decreased thrombosis when regional anesthesia has been used in hip and prostate surgery. The most effective prophylaxis is patient ambulation.

Fat Embolism Syndrome

Death as a result of fat embolism syndrome was first recognized in 1862. The syndrome has usually been diagnosed when there are fat globules demonstrated in the blood with predisposing conditions such as long-bone fracture. The classic triad (neurological dysfunction, respiratory insufficiency, and petechial skin rash) is only seen in 1% to 5% of patients with fat emboli. In contrast, respiratory insufficiency is seen in as many of 29% of patients with fat emboli, as demonstrated by arterial blood-gas monitoring.[52]

Pathophysiology. The pathophysiology of fat embolism syndrome is complex and not fully elucidated. The consensus is that fat globules gain access to the venous circulation through torn venules.[53] Long-bone fractures are commonly the source of fat emboli, but the syndrome is also seen with joint replacements, liposuction, bone marrow transplants, acute hemorrhagic pancreatitis, carbon tetrachloride poisoning, and external cardiac massage.[54]

An especially dramatic presentation of the fat embolism syndrome was reported in 1994, occurring in a patient who was undergoing intramedullary fixation of a femoral-shaft fracture. A two-dimensional transesophageal Doppler probe had been inserted as part of the patient's monitoring. Every time the femur was manipulated, a swarm of emboli appeared in the right side of the heart. Pulmonary hypertension soon developed and culminated in the opening of a probe patent foramen ovale, through which an ultimately fatal paradoxical embolization occurred.[55] It seems reasonable to speculate that fat emboli frequently occur when long-bone fractures are manipulated.

Studies with dogs have demonstrated that when the fat embolism syndrome is induced by fracturing the femurs, the fat found in the lung parenchyma has the same lipid profile as that seen in the bone marrow.[56] As there are only small quantities of femoral fat, other mechanisms must be at work to account for both the severe damage that is seen in the lung parenchyma and the neurological dysfunction, as they cannot be explained by simple vascular occlusion alone.[54]

Free fatty acids and platelet-mediated factors are postulated to increase the damage to the lung parenchyma. Multiple studies have demonstrated that free fatty acids damage lung tissue.[57–59] Because free fatty acids are increased as part of the stress response to injury, it is postulated that they work synergistically with the fat emboli to induce tissue damage (Figure 21-8).[60] This concept is difficult to demonstrate in the laboratory because free fatty acids caused by the trauma cannot be separated from those that are caused by local tissue injury.

Platelet aggregation around the fat microemboli can also cause lung damage. The platelets release a variety of mediators to cause vasospasm and

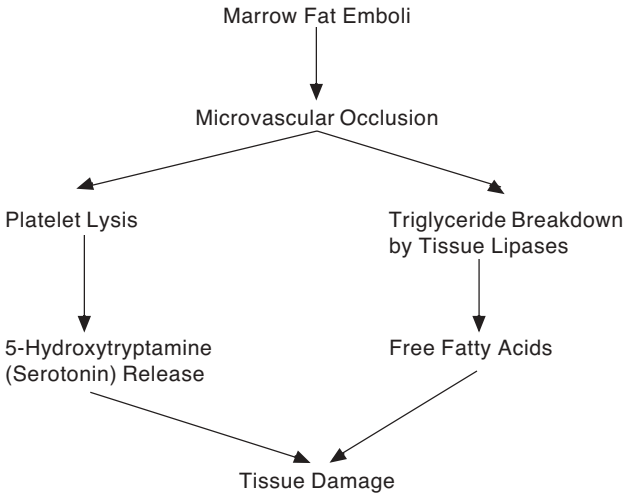

Fig. 21-8. Pathophysiology of the fat embolism syndrome. Reprinted with permission from Van Besouw J-P, Hinds CJ. Fat embolism syndrome. *Br J Hosp Med*. 1989:42:304.

EXHIBIT 21-1

DIAGNOSTIC FEATURES OF FAT EMBOLISM SYNDROME

Major Features

Respiratory insufficiency occurs 2 to 3 days after the injury in 75% of patients with fat emboli syndrome.[1] Tachypnea, dyspnea, and fine inspiratory rales are the usual clinical manifestations. Initial chest radiography is usually normal. As the disease progresses, bilateral, fluffy shadows and obliteration of the lung fields develop (appearing like adult respiratory distress syndrome). Respiratory failure occurs in 10% of cases.[2] Hypoxemia generally precedes respiratory distress by several hours and is considered to be a sensitive marker for the initiation of therapy.[3]

Central nervous system signs and symptoms can be early, often preceding respiratory symptoms by 6 to 12 hours and can be the primary cause of death.[4] Patients usually develop an encephalopathy manifested by a confused state that is exacerbated by hypoxia and not resolved by supplemental oxygen. Associated focal neurologic signs include hemiplegia, aphasia, apraxia, scotoma, and anisocoria.[3,5] These focal symptoms are secondary to the local effects of vascular occlusion. These are seen as widespread destruction in the white matter of the cortex, brainstem, and spinal cord and are secondary to platelet aggregation and free fatty acid mechanisms of injury.

Dermatological manifestations are seen as a petechial rash in the oral mucous membranes, conjunctiva, and skin folds of the upper half of the body (especially the neck and axilla). This rash, which is seen in 60% of those affected, is secondary to the occlusion by fat globules of the dermal capillary network.[1,6] The petechiae are seen on the nondependent body aspects, and are probably secondary to the embolization within the nondependent portions of the subclavian and carotid artery (analogous to oil floating on water). The rash is self-limited and resolves completely in 7 to 10 days.[7,8]

Minor Features

Pyrexia and tachycardia, which are related to the fat embolism itself or to secondary infection[9,10]

Electrocardiographic changes, which are associated with right ventricular strain (S wave in V1, Q wave in III, and nonspecific ST segment changes)

Retinal changes, which are manifested by soft, fluffy exudates; macular edema; retinal hemorrhages; and fat droplets

Renal changes of oliguria, lipuria, proteinuria, and hematuria, which are transient with the fat embolus syndrome and are unrelated to subsequent renal failure[4]

Hepatic changes, which are manifested by rare, self-limited jaundice[1]

(1) Gurd AR, Wilson RI. The fat embolism syndrome. *J Bone and Joint Surg*. 1974;56(B):408–416. (2) Guenter CA, Braun TE. Fat embolism syndrome: Changing prognosis. *Chest*. 1981;79:143–145. (3) Gosling HR, Donohue TA. The fat embolism syndrome. *JAMA*. 1979;241:2740–2742. (4) Sevitt S. The significance and pathology of fat embolism. *Ann Clin Res*. 1977;9:173–180. (5) Jacobson DM, Terrence CF, Reinmuth OM. The neurologic manifestations of fat embolism. *Neurology*. 1986;36:847–851. (6) Alho A. Fat embolism syndrome, etiology, pathogenesis and treatment. *Acta Chir Scand*. 1980;49:75–85. (7) Tachakra SS. Distribution of skin petechiae in fat embolism rash. *Lancet*. 1976;1:284–285. (8) Stephens JH, Fred HL. Petechiae associated with systemic fat embolism. *Arch Dermatol*. 1962;86:515–517. (9) Murray DG, Racz GB. Fat embolism syndrome (respiratory insufficiency syndrome). A rationale for treatment. *J Bone and Joint Surg*. 1974;56(A):1338–1349. (10) Wildsmith JAW, Masson AHB. Severe fat embolism: A review of 24 cases. *Scott Med J*. 1978;23:141–148.

bronchospasm with subsequent ventilation–perfusion mismatching.[61] Hypoperfusion causes secondary cellular disruption, which then causes the release of secondary tissue lipases. This then causes further increases in free fatty acids and further lung injury.[54]

Fat emboli that bypass the filtering capacity of the lung and gain access to the arterial circulation (ie, right-to-left shunt) are responsible for the dermatological and neurological manifestations of the syndrome. Normal shunting occurs through the bronchial and thebesian veins and allows small emboli to bypass the lung to the arterial circulation. As the amount of emboli increases and damage is occurring, the pulmonary artery pressure increases, and the opening of precapillary shunts increases

the right-to-left shunt. This is associated with larger emboli and more-severe systemic manifestations.[62]

Diagnosis. Fat embolism syndrome is difficult to diagnose,[63–65] although respiratory insufficiency, central nervous system dysfunction, and dermatological manifestations are major diagnostic features; fever, tachycardia, and electrocardiographic, retinal, renal, and hepatic changes are minor, nonspecific diagnostic features not specific to the fat embolism syndrome per se (Exhibit 21-1). In 1987, four criteria (sustained $Pao_2 < 60$ mm Hg; $Paco_2 > 55$ or pH <7.3; respiratory rate > 35; and increased work of breathing manifested by dyspnea, use of accessory muscles, and tachycardia) in the presence of a long-bone fracture were suggested as being diagnostic.[66] These criteria probably lead to overdiagnosis, which illustrates the difficulties of studying the syndrome, its pathophysiology, diagnosis, and treatment.

Laboratory Changes. The following hematological and biochemical changes can usually be seen in patients with fat embolism syndrome.

- Hematological changes:
  - Hematocrit usually decreases even in the face of adequate blood-loss replacement. This is usually due to intrapulmonary hemorrhage, lung parenchymal damage, and to increased blood aggregation and hemolysis.[54]
  - Platelet count is decreased secondary to the platelets' adherence to fat emboli and raw edges of bone. In addition, disseminated intravascular coagulation will consume platelets.[54]
  - Erythrocyte sedimentation rate is increased.
  - Coagulation times (prothrombin and thrombin) are increased.
- Biochemical changes:
  - Fat globules can be detected in blood, urine, and sputum but are not diagnostic in the absence of other clinical signs.[64]
  - Free fatty acids, cortisol, glucagon, and catechols increase as part of the stress response.[62]
  - Calcium is decreased secondary to binding to free fatty acids.[67]

Treatment. The fat embolism syndrome is difficult to diagnose because universal agreement on diagnostic criteria is lacking. This lack, coupled with the relatively low incidence of the syndrome, creates difficulties in prospectively evaluating treatment.

Treatment is supportive in nature. Because movement of the fracture increases the incidence of fat embolism syndrome, early immobilization is advocated.[68] Early immobilization will also allow early patient mobilization to increase pulmonary toilet, and decrease blood loss and platelet consumption by stopping the constant movement at the fracture site. Internal fixation decreases the incidence of the syndrome.[69,70]

Adequate fluid resuscitation and maintenance of colloid oncotic pressure with blood or colloid fluids help to absorb circulating free fatty acids. Studies with animals in hypovolemic shock have demonstrated their increased susceptibility to fat embolism syndrome.[71] Albumin is thought to bind free fatty acids, but this needs further investigation as to the appropriate levels of albumin required.[72] The patient should be given adequate analgesia to ablate the sympathetic responses that will cause increased free fatty acids and susceptibility to fat embolism syndrome.[54]

Respiratory care is of paramount importance in the treatment of fat embolism syndrome. Oxygen requirements range from supplemental oxygen provided via nasal catheter to intubation and PEEP required for frank respiratory failure. The pulse oximeter allows for noninvasive determination of saturation and permits continuous monitoring of pulmonary function.[52]

Neurological function must be evaluated serially to determine early deterioration of function. There is no information at present as to the incidence of increased intracerebral pressure, or if measures usually employed to control intracranial hypertension alter patient outcome.[54]

As alcohol is known to inhibit lipase, it was postulated, but never proven, that inhibiting lipase will limit the lipolysis of neutral fat emboli and decrease free fatty acid damage to the lung.[73] A resurgence of interest in alcohol has developed since researchers noted a decreased incidence of fat emboli in patients with femoral fractures and elevated blood alcohol levels.[74] However, no biochemical relationship has been demonstrated among blood alcohol levels, free fatty acid level, and fat embolism syndrome.[75]

Heparin, a stimulator of lipase, has been used to decrease the number of circulating fat globules, thereby preventing them from reaching the lung.[74,75] Heparin will also cause an increase in free fatty acids, which can cause increased damage. As exThis

pected, the data conflict.[76,77] Before administering heparin, the medical officer must weigh the concern of causing further bleeding against the continued circulation of fat.

Steroids, usually methylprednisolone, have been employed both prophylactically and therapeutically and may have beneficial effects, but all investigators in steroid studies note the need for adequate resuscitation and oxygen before using steroids.[54] The proposed mechanisms of action include stabilizing membranes, limiting the rise of free fatty acids, limiting complement, and inhibiting leukocyte aggregation. Steroids appear to attenuate the syndrome, and hypoxia has been shown to decrease after steroids were administered: in 1971, methylprednisolone in a first dose of 125 mg, then 80 mg every 6 hours for 3 days, was advocated[78]; in 1983, the dose was increased to 7.5 mg/kg every 6 hours for 12 doses (90 mg/kg total)[79]; and in 1987, 30 mg/kg was administered in one dose.[52]

Prognosis. The multiplicity of coexisting injuries makes the overall prognosis for patients with fat embolism syndrome difficult to assess; overall, the mortality is 5% to 15%, owing to the other injuries. As expected, if no mechanical ventilation is required to treat the patient, the mortality decreases.[52] Overall, the respiratory manifestations are self-limited, and if the physician maintains oxygenation, then the patient's respiratory function will return to normal.[80]

Neurological complications are responsible for most of the long-term morbidity and mortality, especially when focal deficits are present.[64,81]

ANESTHETIC MANAGEMENT OF COMBAT CASUALTIES

The anesthetic management of combat casualties with severe extremity wounds is demanding. The more common injuries, such as grossly comminuted femoral or tibial fractures, typically cause substantial blood loss before the casualty reaches the hospital level. By the time casualties with bilateral leg amputations have reached the hospital level, they have usually lost over half their blood volume and may be agonal. Even casualties who have multiple, small, soft-tissue extremity wounds may require extensive procedures, with consequent considerable blood loss. During the Vietnam War, 16% of casualties with extremity injuries received blood transfusions; the most common volume transfused ranged between two and five units.[82]

Depending on the severity of coexisting injuries, these procedures can be prolonged, and caring for the total patient, complex. Considerations include hypovolemia, shock, bleeding, and poor intravascular access. Medical officers must be aware that previously undiagnosed injuries may manifest intraoperatively (eg, pneumothorax and refractory hypovolemia secondary to occult fractures). Coexisting injuries include closed head injuries, cervical spine injuries, thoracoabdominal injuries, burns, and coagulopathies. Sepsis is always an impending risk, and patient transport requires expertise to avoid further injury.

Regional Anesthesia

Regional anesthesia is an attractive choice in the appropriate casualty whose injury is localized.[83,84] The advantages of regional anesthesia are (1) an awake patient is able to control his or her own airway; (2) the treatment team can perform sequential central nervous system examinations and further assess for occult injuries; and (3) the anesthesiologist can more accurately control the administered dose of postoperative analgesia. The disadvantages are (1) the patient may lose control of his or her airway and aspirate during surgery and (2) severe hypotension may develop in the intravascularly depleted patient secondary to the induced sympathectomy. Regional anesthesia is often not indicated for the initial surgery because the surgery tends to be prolonged and the patient will not tolerate the immobility required. In addition, many patients will require mechanical ventilation as part of their medical management.[85–89] A further consideration that applies to deployed hospitals (especially in OOTW when refugees are being treated) is the likelihood of a language barrier between the patient and the military anesthesiologist.

Intravenous regional anesthesia (ie, the Bier block) is acceptable for simple fractures and surgery to the forearm, wrist, and hand.[90–93] The simplicity of the technique, rapid onset, and minimal equipment needs make for an attractive field anesthetic. The disadvantages of brief duration and lack of postoperative analgesia detract from its use. Intravascular overdose can occur if the local anesthetic is injected too quickly near the tourniquet or if the tourniquet deflates prematurely.

Reduction of simple fractures can be facilitated by local infiltration of anesthetic into the hematoma.

is technically easy to perform. Sterility is mandatory: the hematoma is an excellent culture medium for bacteria that may cause a subsequent osteomyelitis.

Axillary block is simple and offers prolonged anesthesia and postoperative analgesia. Inserting a catheter into the axillary sheath allows subsequent redosing to permit prolonged anesthesia. This technique is appropriate for procedures distal to the elbow. The musculocutaneous nerve and the intercostobrachial nerve diverge after the brachial plexus is formed in at least 50% of the population and, therefore, will need to be separately blocked if a tourniquet is used. In addition, axillary blockade has an effect beyond providing analgesia: by interfering with sympathetic nerve transmission, vasodilation and increased blood flow to the injured extremity may be induced.

The management of the hypovolemic patient is challenging in the face of continued bleeding that will only be controlled by surgical hemostasis. The use of ketamine can be considered while hemostasis is achieved, but the anesthesia provider needs to remember that ketamine is a potent myocardial depressant in the patient whose sympathetic system is already maximally stressed. Some experienced military trauma surgeons consider ketamine to be the anesthetic agent of choice for extremity operations, especially when combined with a benzodiazepine.[94] The uses and limitations of ketamine are discussed in Chapter 10, Intravenous Anesthesia.

General Anesthesia

Overall, the military trauma anesthesia provider needs to consider the casualty case load and balance general anesthesia's increased risk to the patient but shorter induction time against other anesthetic modalities that may be safer for the patient but take longer to induce. A well-conducted general anesthetic utilizing whatever airway protection is re-

EXHIBIT 21-2

PURPOSES OF PULMONARY ARTERY CATHETERIZATION IN CASUALTIES WITH EXTREMITY INJURIES

1. To measure cardiac output
2. To measure oxygen content of mixed venous blood
3. To assess hemodynamic indices:
   - mean arterial pressure, with an arterial line
   - cardiac index, with a pulmonary artery line
   - stroke volume
   - stroke volume index
   - systemic vascular resistance
   - left ventricular stroke work index
4. To measure derived indices in conjunction with other monitors such as oxygen consumption (Vo_2) by the Fick method: cardiac output • (arterial oxygen content – venous oxygen content)
5. To measure filling pressures in the right side of the heart (CVP) and the left side of the heart (PAWP) to assess for
   - shock
   - expected large volume shifts
   - myocardial dysfunction
6. To continuously monitor mixed venous oxygen saturation

CVP: central venous pressure; PAWP: pulmonary artery wedge pressure

4.

quired can be preferable to the potential complications of delaying surgery. The actual incidence of gastric aspiration is small compared to the potential risks of loss of function of an extremity.[95] Early fixation of severe fractures will decrease the incidence of fat embolism syndrome, deep venous thrombosis, and continuing hemorrhage. The incidence of morbidity and mortality following femoral fracture has been decreased after early fixation, allowing mobilization of the patient. Aggressive treatment of these patients is warranted, as the risk of deep venous thrombosis approaches 40% to 60% in the immobilized patient, with pulmonary embolism at 5% to 10%.[69,85,87,89,96]

INTRAOPERATIVE MONITORING

Trauma patients may benefit from invasive monitoring to optimize their hemodynamic performance (Exhibit 21-2). These patients require continuous vigilance to detect new injuries, with simultaneous monitoring of their arterial blood gases, hematocrit, urinary output, electrolytes, and coagulation status. The anesthesiologist needs to monitor the surgical field to estimate coagulation and blood loss. Temperature monitoring is vital as it is related to coagulation, cardiac output, shock, and so forth. A normothermic environment and warming fluids and gases will help to maintain the patient's temperature. Additionally, the anesthesiologist must rely on clinical skills, as there is frequently is not enough time to institute technologically advanced monitoring. Wartime and civilian mass casualty situations are examples where clinical skills are of paramount importance.

Prolonged surgery should not be terminated on the grounds that the patient has had too much surgery without objective evidence of refractory shock or hypothermia. The patient should not be taken to the intensive care unit until the life-threatening injuries are corrected. In addition, the military trauma anesthesiologist should consider that triage will be ongoing in mass casualty situations during war or disaster. This is important, considering the previously mentioned decreases in morbidity and mortality seen with early fixation of fractures.

SUMMARY

Extremity injuries in combat casualties are generally not immediately life threatening but are the major cause of morbidity as measured in man-days lost. In addition, extremity injuries are a frequent reason for disability separation or retirement of combat casualties. Most combat casualties with extremity injuries will have either a wound involving only soft tissues or a wound that involves soft tissue in addition to bone or, less commonly, neurovascular structures. The principles of management involve control of hemorrhage, excision of dead or contaminated tissue or both, and fixation of a fracture, if present, in a position likely to lead to restoration of normal function when union occurs. Wound sepsis is by far the most common complication that occurs with extremity injuries. Less common complications are fat embolism syndrome, deep venous thrombosis, compartment syndrome, and pulmonary embolism. Because wounds of the extremities constitute so large a fraction of the operative case load, expert and expeditious anesthetic care of these casualties is necessary to maximize the use of operating room resources.

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