Chapter 10

RHABDOMYOLYSIS AND COMPARTMENT SYNDROME IN MILITARY TRAINEES

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INTRODUCTION

Extremity trauma is a common occurrence during military training. All basic trainees, to some degree, experience muscle injury below the threshold of permanent damage. However, the likelihood of a trainee developing a musculoskeletal injury that seriously threatens life or limb is quite low. Physicians who treat personnel undergoing basic military training need to be aware of factors that can push the level of injury to a dangerous point.

The purpose of this chapter is to highlight two closely related problems that occur within the training environment: rhabdomyolysis and compartment syndrome. These conditions develop as a result of a physiological cascade of metabolic abnormalities that occurs when the body is no longer able to compensate for the demands placed upon it.

RHABDOMYOLYSIS

Pathophysiology

Rhabdomyolysis is the breakdown of skeletal muscle as a result of injury. Injury causes the release of cellular contents—including myoglobin, serum creatine kinase, and aldolase—into the bloodstream. Some of these muscle fiber contents, most notably myoglobin, are toxic to the kidney and can frequently result in severe kidney damage.

Exhibit 10-1 presents various causes of rhabdomyolysis. Etiologies can coexist, especially in a military environment, yet also have negative synergistic effects. For example, a dehydrated soldier in Iraq is pinned for a brief period between two trucks and sustains major lower extremity crush injuries. That soldier's physiological response to muscle tissue death will be quantitatively different from that of a well-hydrated construction worker whose legs are similarly injured in a ditch collapse.[1]

The resulting cascade of metabolic abnormalities that develops after release of cellular proteins into the general circulation includes hyperkalemia, hypernatremia, hyperphosphatemia, hypocalcemia, lactic acidosis, and hyperuricemia.[2] Marked elevation of serum creatine kinase can develop, with values of more than 600,000 U/L reported. (Normal values range from 50 to 200 U/L.[3]) Severe cases can develop into disseminated intravascular coagulation and renal failure, and can result in death.[4]

Normally, myoglobin is not detected in the blood until levels exceed 1.5 mg/dL, an amount equal to the dissolution of approximately 100 g of skeletal muscle.[5] After injury, however, myoglobin concentrations rise in the plasma. Myoglobin is then excreted in the urine as concentration exceeds 21 mg/dL. When plasma concentration rises above 100 mg/dL, urine quickly turns a dark color. In acidic urine, myoglobin polymerizes with anionic mucoproteins in the ascending limb of the renal tubules. Therefore, acute renal failure results from direct myoglobin toxicity or precipitation within the nephron.[2,5] The false-positive presence of heme on a urine dipstick and a negative microscopic examination for red blood cells are classic indicators of myoglobinuria.

EXHIBIT 10-1

CAUSES OF RHABDOMYOLYSIS

- Acute exertion
- Massive crush injury
- Prolonged pressure
- Vascular ischemia
- Gangrenous infection
- Drug intoxication
- Status epilepticus
- Delirium tremens

Acute Exertional Rhabdomyolysis

Current studies recognize that, among a wide range of individuals, acute exertional rhabdomyolysis is a fairly common complication of strenuous physical activity. This condition has been documented in military recruits, professional and amateur athletes, weight lifters, firefighter trainees, and law enforcement trainees. Olerud et al[6] found that, during the early training phase, 40% of 337 military recruits had myoglobin in their urine, which is evidence of rhabdomyolysis. Line and Rust[4] described a study of 50 law enforcement trainees who had levels of creatine phosphokinase consistent with rhabdomyolysis. In a study by Sinert et al,[7] nearly half of the hospital admissions for rhabdomyolysis were exercise induced. The common theme present in these studies is that the participants exerted effort above their usual maximum effort. For physically unconditioned recruits entering training, the increased level of activity may actually be quite modest. There are numerous examples, how-

ever, of well-conditioned athletes (eg, marathon runners) who developed some degree of rhabdomyolysis after a supramaximal exercise session.[7] The history of a patient with acute exertional rhabdomyolysis typically includes some form of significant exertion. Muscle pain, weakness, swelling, and burning in the involved extremity are common complaints, as well as a history of voiding dark urine. Critical information to elicit from the patient includes noting the environment and circumstances when the injury occurred; the hydration status; a history of sickle cell trait; and use of any medication, laxative, or supplement. If an herbal medication has been used, a sample is needed by the physician to check for ingredients such as caffeine, aspirin, ephedrine.

Findings on physical examination consist of muscle swelling, tenderness, and edema. The muscles involved may be quite isolated, such as the pectoralis major or the triceps. Neurological function is usually normal.

Laboratory studies include a comprehensive metabolic panel, serum creatine kinase levels, clotting parameters, and urinalysis. Sequential, close monitoring is necessary because of the rapid progress of electrolyte changes (occurring over 4 to 6 hours) and the possible need for emergency dialysis.

Treatment of rhabdomyolysis involves rapid volume loading and ongoing replacement to maintain urine output. Mannitol and furosemide (both diuretic medications) have been shown to dilute myoglobin concentration and reduce nephrotoxicity.[4] The two cases described below, which were treated at a local MEDDAC (medical department activity), further amplify the dramatic clinical scenarios that can rapidly develop and result in devastating outcomes.

Case Study 10-1: A 32-year-old African American female Army recruit collapsed near the finish line of a timed 2-mile run that was part of her physical fitness test. She was transported to the base hospital. During transport, the patient remained awake, but became obtunded. On arrival at the hospital, she complained of bilateral leg pain and remained lethargic. Examination revealed bilateral leg and thigh tenderness and swelling, red urine, and an initial temperature of 102.8°F. She had multiple electrolyte abnormalities and was significantly acidemic. At this point, she was given aggressive fluid resuscitation and transferred to a local referral hospital. On admission, she had elevated creatine phosphokinase, metabolic acidosis, hyperkalemia, hypocalcemia, myoglobinuria, acute renal failure, and disseminated intravascular coagulopathy—all consistent with a diagnosis of rhabdomyolysis. A history obtained from the patient and accompanying US Army medical personnel revealed that she took an over-the-counter diet pill containing ephedrine, aspirin, and caffeine, and had lost 15 pounds over the past few weeks in preparation for the physical fitness test. She did not smoke and only drank alcohol occasionally before entering basic training. Her medical history was significant for the sickle cell trait.

An orthopedic consultant evaluated the patient's lower extremity pain and swelling. On initial examination, her leg and thigh compartments were soft. Her foot pulses were faintly palpable and symmetric. She had intact sensation in both lower extremities. Her thigh and leg muscles were tender on palpation, but she had no significant increase in pain with passive motion of her ankles or toes. The overall picture at the initial examination did not suggest muscle ischemia. However, repeat examinations over the next few hours revealed increasing swelling and tenseness in the thigh and leg compartments, with decreasing bilateral sensation. Compartment pressures, measured with a handheld pressure monitoring device, were elevated, ranging from 40 to 77 mm Hg (normal, 10–12 mm Hg) in all compartments of the leg and thigh.

At this time, bilateral lower extremity fasciotomies were planned; however, the patient required emergent hemodialysis for life-threatening hyperkalemia. With hemodialysis, she received multiple units of fresh frozen plasma to correct her coagulopathy. The patient complained of abdominal pain and had diarrhea. A duplex scan then showed possible nonocclusive mesenteric ischemia. Therefore, after hemodialysis, she was taken to the operating room for emergent exploratory laparotomy to evaluate the viability of her bowel.

After administration of anesthesia, the patient developed ventricular tachycardia with depressed ST segments and an undetectable pulse. Aggressive resuscitation was initiated; defibrillation was administered; and epinephrine, atropine, and lidocaine were given. She returned to sinus tachycardia with a systolic blood pressure of 200 mm Hg. The patient required multiple units of blood and fresh frozen plasma for her coagulopathy. Exploratory laparotomy revealed viability of the bowel with evidence of some mucosal ischemia.

Attention was then directed to the patient's lower extremity compartment syndrome. Fasciotomies were performed bilaterally in all leg and thigh compartments, using two incisions in the legs and one lateral incision in the thighs (Figure 10-1). When the fascia was incised, bulging, edematous muscles in all compartments were noted. The muscles appeared hemorrhagic and dusky colored, but were contractile without obvious necrosis. The wounds were packed open with saline gauze.

After completion of the fasciotomies, the patient again developed ventricular tachycardia. Emergency resuscitation was reinitiated and sinus tachycardia was restored. The patient was then transferred to the intensive care unit. However, although the patient was administered large amounts of blood products, fluids, and fresh frozen plasma, severe coagulopathy and electrolyte abnormalities persisted. Refractory hypotension and bradycardia were followed by bradysystolic arrest. Despite resuscitative efforts, the patient died.

Case Study 10-2: A 21-year-old African American male recruit collapsed during the running portion of his physical training entrance test. He was taken to the base hospital where he complained of numbness and pain in his legs and buttocks. Vital signs at initial evaluation included the fol-

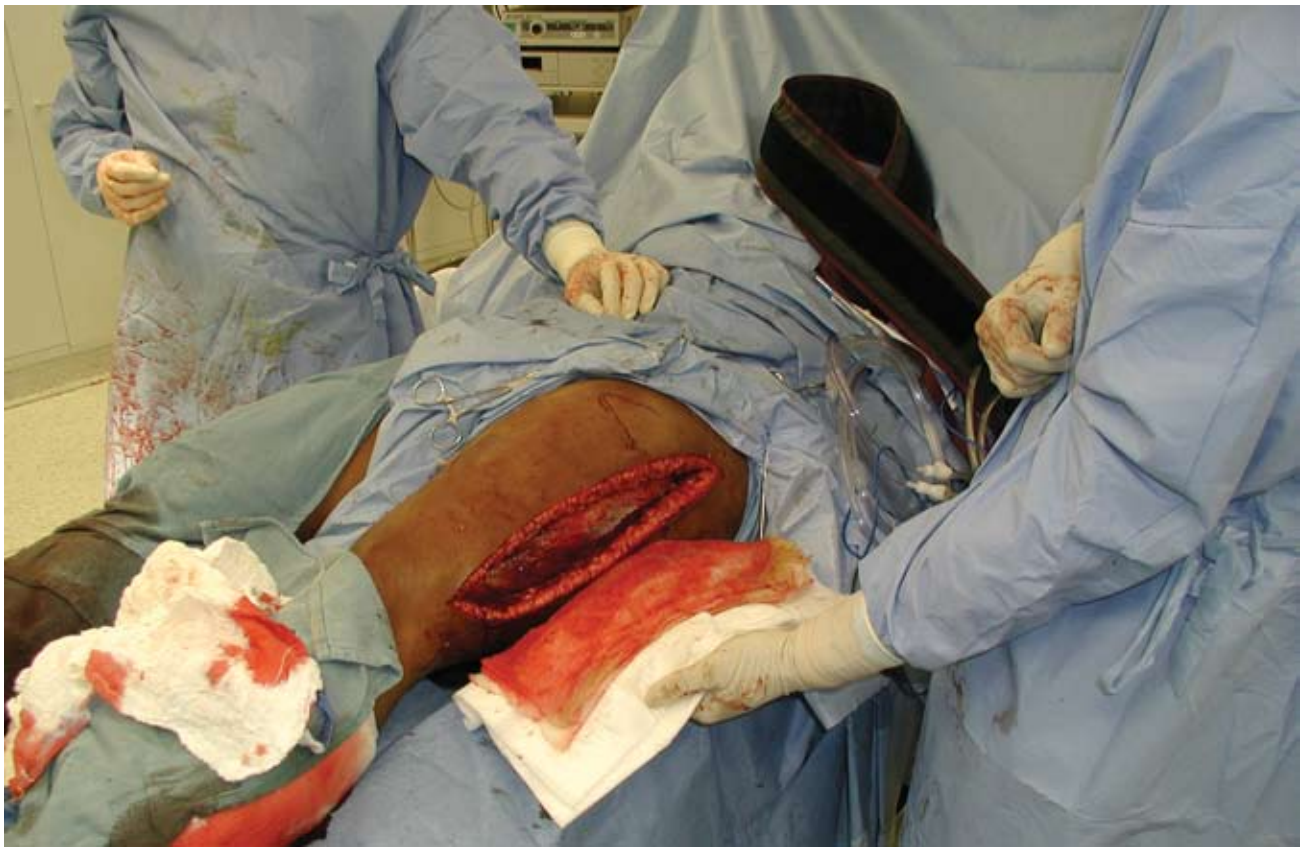

Fig. 10-1. Bulging thigh musculature after compartment release. Copious drainage from the leg faciotomies is present as a result of coagulopathy. Note incisions covered by towels.

lowing: temperature, 99.2°F; heart rate, 143 beats/minute; and blood pressure, 125/25 mm Hg. He denied specifically using any medication with ephedrine, but did report that he had used an appetite suppressant 3 months before and was currently using magnesium citrate laxatives to aid in passing his weight requirement. He had already lost 10 pounds the week before admission. Physical examination revealed an obese male in mild distress, with diffuse bilateral lower extremity tenderness but no clinical signs consistent with compartment syndrome. Serum creatinine kinase level was elevated to 2.2, creatine phosphokinase level was 749, and serum potassium level was 5.8. Sickle cell trait was positive. Urinalysis was also positive for myoglobin.

The patient underwent aggressive fluid management and frequent laboratory assessment for electrolyte abnormalities. However, his clinical condition slowly worsened over the next 24 hours. Serum potassium and creatine phosphokinase levels rose steadily, requiring hemodialysis. Systemic coagulopathy soon developed.

An orthopedic consult was obtained 1 day after admission. Examination showed a patient in minimal distress. A focused examination showed tense anterior leg compartments. Pain on passive motion was present, and poor active control of toe and ankle dorsiflexion was noted. Bedside compartment

EXHIBIT 10-2

RISK FACTORS FOR EXERTIONAL RHABDOMYOLYSIS

- Elevated heat and humidity
- Poor conditioning
- Dehydration
- Sickle cell trait
- Recent weight loss
- Prior history of heat injury
- Altitude
- Stimulant use (ephedrine, caffeine)
- High-intensity, load-bearing exercise

Data sources: (1) Line RL, Rust GS. Acute exertional rhabdomyolysis. *Am Fam Physician.* 1995;52:502–506. (2) Walsworth M, Kessler T. Diagnosing exertional rhabdomyolysis: a brief review and report of two cases. *Mil Med*. 2001;166(3):275–227.

pressures in both legs showed anterior leg compartment pressures between 55 and 60 mm Hg. No additional pressure measurements were obtained at bedside. The patient was taken to the operating room, where anesthesia was administered and further compartment pressure testing was completed. Pressures ranging from 45 to 60 mm Hg were noted throughout both glutei, quadriceps, and anterior and lateral leg compartments. Fasciotomies were performed on all involved compartments. Both the thigh and the right gluteal musculature were abnormal in color but appeared well perfused and viable. Lateral leg compartment findings were similar. The left gluteus and both anterior leg compartments were found to have gray, necrotic, noncontractile muscle. All three areas required significant debridement.

Forty-eight hours later, the patient was returned to the operating room. Closure was possible in the thighs and glutei, with healthy muscle noted. Further necrosis was present in the anterior leg compartments and had developed in the lateral leg compartments. Additional debridement was performed, and the wounds were packed open.

Despite aggressive medical management of the patient throughout his hospital stay—including intubation, mechanical ventilation, emergency hemodialysis, and arterial pressure support—the patient's condition deteriorated. He died 6 hours after his last debridement.

Many risk factors can have an effect on the degree of systemic involvement that develops after exertional muscle injury[4,8] (Exhibit 10-2). It is important to determine factors that predict the development of rhabdomyolysis and its severe forms. For example, in their 1995 study, Line and Rust[4] described several risk factors for rhabdomyolysis, including high temperature, humidity, poor conditioning, dehydration, renal insufficiency, and sickle cell trait. Kuklo et al[3] felt that in their patient, rapid weight loss and dehydration from dieting probably contributed to the development of rhabdomyolysis. They also suggested that the use of dietary supplements, including creatine and ephedrine, might have been a factor. Ward[9] found that dehydration and the degree of creatine phosphokinase, potassium, and phosphorus elevation at presentation were predictive of renal failure.

In Case Study 10-1, multiple factors probably led to the development of rhabdomyolysis and renal failure. In preparation for the physical fitness test, the patient had lost approximately 15 pounds within a few weeks. Weight loss this rapid is usually caused, at least in part, by total body water loss; therefore, most likely she was volume depleted. She was taking not only weight-loss supplements, but also aspirin, ephedrine, and caffeine. Aspirin has been linked to rhabdomyolysis. Ephedrine has many known side effects—including tachycardia and hypertension—and, as suggested by Kuklo et al,[3] can increase hyperthermia. The diuretic action of caffeine can exacerbate dehydration and hypovolemia. In addition, the combination of caffeine and ephedrine has been shown to increase lactate levels. These actions could have exacerbated acidosis and nephrotoxicity. This combination also causes central nervous system stimulation and increases time-to-perceived exhaustion. Subsequently, this may have allowed for greater muscle injury than would have otherwise occurred.

The best predictor of severe rhabdomyolysis might have been the patient's sickle cell trait. Kerle and Nishimura[10] reported on a soldier with sickle cell trait who developed sudden cardiac death during physical exertion. LeGallais et al[11] suggested that rhabdomyolysis, dehydration, and acidosis might initiate a sickle cell crisis, along with disseminated intravascular coagulopathy and renal failure. A statistical study of deaths among military recruits concluded that recruits in basic training who had the sickle cell trait have a risk of exercise-related sudden cardiac death that is 40 times greater than other recruits.[1,12,13]

COMPARTMENT SYNDROME

Compartment syndrome is a painful condition in which increased pressure within a muscle compartment causes a decrease in blood supply to the affected muscles, and it can present in a variety of ways. Because of the potentially catastrophic consequences if left untreated, it is important to recognize it promptly. Compartment syndrome reflects ischemia at the cellular level. Basically, it occurs when an inciting event produces edema within a muscle compartment. Causes include blunt trauma, fractures, burns, muscular exertion, or muscle swelling and edema (such as that occurring with rhabdomyolysis). When swelling within the closed space of an unyielding fascial compartment is sufficient to cause local capillary collapse (pressures from 25 to 40 mm Hg), the tissue becomes ischemic. Because the pressure is insufficient to collapse major arteries (pressures from 80 to 120 mm Hg), distal flow is preserved.[14,15] This is a common source of confusion. Distal capillary refill, tissue color, and turgor are usually normal, even in a florid compartment syndrome. This is in marked contrast to the findings of major vessel arterial occlusion, in which an ischemic injury occurs; however, distal flow is *not* preserved in this case.

Diagnosis

Acute compartment syndrome presents with marked pain not relieved by narcotics, tense myofascial compartments, pain on passive motion of the muscles within the compartments, and elevated local tissue

Fig. 10-2. Fasciotomy for compartment syndrome. Note severe lower leg swelling and blackened, necrotic muscle.

pressures. The pressures can be measured by a number of different techniques (including the Whitesides infusion technique, the wick catheter technique, the continuous infusion technique, and the STIC technique). The compartments most commonly affected include those of the leg (Figure 10-2) and forearm (Figure 10-3). Less commonly involved are those of the hand (Figure 10-4), foot, and thigh.[3,5,12,14-20] If the process goes on long enough and presents in a delayed or subacute fashion, it might be too late to salvage muscle injured by pressure ischemia. In this delayed presentation, some or all of the previously described physical findings might be present, and compartment pressures may return to normal. This probably explains the operative findings in Case Study 10-2. Recognition of the process can be difficult for a number of reasons. For example, a concomitant fracture or soft-tissue injury can cause enough discomfort that the pain from muscle ischemia is not clear. It is an evolving condition that can present at different stages, resulting in missed diagnosis, particularly when multiple providers are involved.[14] This commonly occurs when patients are transferred to higher echelons of care. It is important to reinforce the need for repeated, complete extremity evaluation. Often, the patient is assigned a diagnosis and is then not reevaluated. However, when tissue is at risk, the level of urgency is considerably higher to reevaluate that patient.

Serial physical examinations should consist of a new assessment of the patient's vital signs, followed by a distal neurovascular examination of the involved extremities. The evaluation labeled as "pain out of proportion with physical findings" is by definition a subjective assessment. Careful consideration of the patient's stoicism, the physician's stoicism, cultural norms for pain expression, sedation using pain medication, concomitant injuries to other extremities, and secondary gain issues affecting training status all need to be factored into the decision making. One useful technique for detecting clinical clues is to obtain a recent medical history from hospital personnel. Another useful technique involves performing undetected observation of the patient, which can also reveal useful information about patient behavior patterns. Although most patients verbalize their pain, markedly stoic individuals can give a false impression to medical personnel that all is well. Subsequently, the compartments should be palpated for tenderness and relative degree of firmness. Palpation of the contralateral limb can give a general idea of the patient's baseline, assuming it is uninvolved. A considerable degree of variation in tissue turgor requires careful clinical judgment and experience. A muscular, athletic trainee might have firm muscle compartments as a normal baseline finding. However, a thin, ligamentously lax trainee will have an entirely different baseline examination finding in an uninjured limb. If a focal injury is present (such as a fracture or gunshot wound),

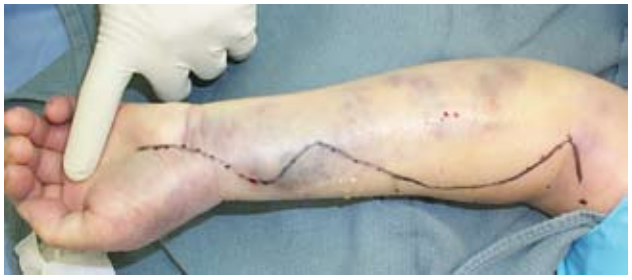

Fig. 10-3. Rigid swelling of forearm from compartment syndrome. Skin shows surface blistering, a common finding in severely swollen limbs.

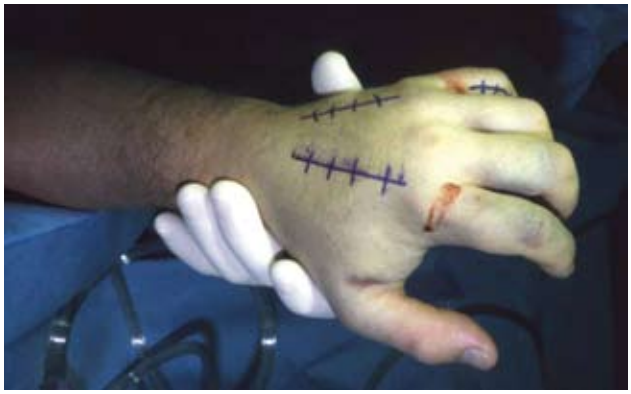

Fig. 10-4. Classic hand posture as a result of severe swelling from compartment syndrome. Fasciotomy incisions have been marked.

palpation should be performed some distance from the injury site to avoid confusion about the source of pain. Passive motion of the muscles that traverse the compartment yields further information on the status of that limb. For example, passive dorsiflexion of the toes assesses the deep posterior compartment of the leg, whereas ankle dorsiflexion and plantar flexion assess the superficial posterior and superficial anterior compartments, respectively. Ischemic muscle in the forearm is stretched by passive extension and flexion of the fingers. A patient who has marked, increasing pain at rest, rigid compartments, and severe pain on passive motion, and who shifts positions constantly in bed, has compartment syndrome by definition and should have an emergent fasciotomy.[14] If, however, the diagnosis is unclear, then an assessment of compartment pressure must be performed.

Measurement of Compartment Pressure

There are a number of methods used to measure compartment pressure.[14] Although measurement can be performed in a field setting with a manometer, tubing, syringe, and saline, this particular method is often impractical. The easiest method uses a commercial device known as a solid-state transducer intracompartment catheter (Stic catheter, Stryker Medical, Mississauga, Ontario, Canada) shown in Figure 10-5. This portable device is small, handheld, and gives an instant readout of compartment pressure. After cleansing the skin, a subcutaneous wheal of lidocaine is injected at the proposed puncture sites. Only a volume sufficient enough to anesthetize the skin is injected to avoid artificially increasing the tissue pressure. Puncture sites should be arranged so that the needle will not injure neurovascular structures as it is inserted below the fascia. Usually, a distinct decrease in resistance is felt when the needle is pushed through the fascia. To ensure that the side port portion of the needle is entirely within the compartment, the needle should be advanced another 3 to 4 mm. The device should be initialized at zero before reading the pressure. (There are, however, some drawbacks to using this instrument, including the relatively large, side-ported needle that causes significant pain on puncture of the deep fascia and the need to maintain a stock of disposable syringe inserts for it.)

Fig. 10-5. Stic catheter (Stryker, Mississauga, Ontario, Canada), a commercial device that measures compartment pressure.

There is not a specific numerical value that mandates surgical compartment release.[14,15] The primary goal is to prevent capillary collapse, which leads ultimately to tissue death and the resulting cascade of membrane dissolution, cellular leakage, edema, and increased pressure. However, frequently recommended compartment threshold pressures are between 25 and 40 mm Hg.[14,15] Pressure measurements should also be interpreted in light of the patient's systemic blood pressure, which directly affects the pressure gradient across the capillary bed. Less tissue pressure is required to collapse inflow arterioles in a hypotensive patient. Tissue pressure measurements higher than 25 mm Hg are worrisome. Measured pressures should be combined with other available clinical data when deciding the next step in patient management. If these pressures are marginal, close clinical monitoring is crucial, and clinical judgment is essential. Factors such as history and current examination, clinical trends, systemic health of the patient, and evacuation and support scenarios all need to be weighed carefully. Consider the following two scenarios:

Scenario 10-1: A 29-year-old white male Marine on shipboard deployment began an intensive exercise program, which included running all of the ship's stairs three times the morning of presentation. He presents to sick bay complaining of steadily increasing calf pain. Clinical examination reveals an anxious male in mild distress. Vital signs include the following: blood pressure, 110/60 mm Hg; heart rate, 85 beats/minute; and respirations, 16 breaths/minute (unlabored). Pulses, capillary refill, and sensation are normal and bilaterally symmetrical. Both calves are tender and firm. Passive dorsiflexion is painful but tolerable. His legs are iced and elevated, and no systemic pain medications are administered. Over the next 2 hours, he complains of increasing pain. Repeat examination is essentially identical to that at presentation, except the right leg is somewhat more symptomatic. Because of the increasing pain, compartment pressures are obtained from the right leg. Values for the compartments include the following: anterior, 22 mm Hg; lateral, 19 mm Hg; superficial posterior, 26 mm Hg; and deep posterior, 28 mm Hg.

The ship is 100 miles offshore from a hospital with surgical capability. No onboard operating facilities are available.

Preparations are made to transport the Marine to shore but must await the return of a helicopter to do so. One hour later, he seems more comfortable, and his pain has decreased. The helicopter is now available. Objective clinical findings are unchanged. The captain inquires whether the Marine can undergo further monitoring while in sick bay or if transportation to a mainland hospital is still necessary.

Scenario 10-2: A 19-year-old Hispanic female trainee is brought to a battalion aid station because of increasing right calf pain. She completed a 15-mile road march that morning. Clinical examination reveals an anxious female in moderate distress. She appears dehydrated and her mucous membranes are dry. Her vital signs are as follows: blood pressure, 90/50 mm Hg; heart rate, 100 beats/minute; and respirations, 16 breaths/minute. Both legs have symmetrical, bounding pulses, with normal light touch sensation and capillary refill. Palpation of the anterior and lateral compartments in the legs is unremarkable. The posterior calves are swollen and tender. Passive dorsiflexion is quite painful. Her legs are iced and elevated to the level of her heart. Intravenous fluids are administered. Within 30 minutes, her heart rate drops to 85 beats/minute, but her blood pressure remains 90/60 mm Hg. Extremity examination is unchanged; however, the medic reports that she seems more restless and uncomfortable. Compartment pressures include the following: anterior, 22 mm Hg; lateral, 19 mm Hg; superficial posterior, 26 mm Hg; and deep posterior, 28 mm Hg. She continues to shift position frequently.

The nearest medical facility with orthopedic coverage is 45 miles away from the training area. Transportation requires an ambulance that is scheduled to return trainees to another area. The patient voices her desire to complete her last day of training in the morning.

Both of these scenarios share similarities: recent history of beyond-average activity, initially increasing calf pain, and clinical examination suggesting the onset of compartment syndrome involving the posterior leg compartments. The compartment pressures are identical. However, significant differences are also present. The baseline level of conditioning is substantially different in these two individuals. The clinical course seems to be improving for the Marine yet worsening for the trainee. Her blood pressure has remained lower, causing a decreased inflow pressure into the capillary beds in her calves.

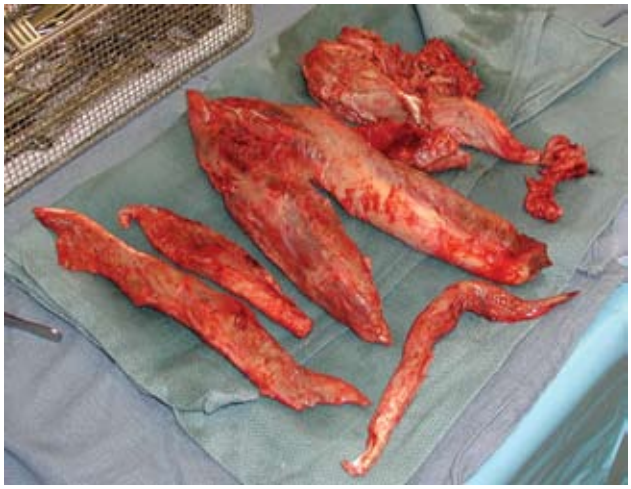

Fig. 10-6. Debrided necrotic muscle from an untreated case of compartment syndrome. All four compartments of the leg were involved (anterior, lateral, superficial posterior, and deep posterior).

Transport of both of these patients to facilities with surgical care would be highly recommended. The constellation of clinical findings suggests that the trainee should be evacuated immediately. It would be reasonable to continue observing the Marine for another 1 to 2 hours, given his subjective improvement. Clinical improvement with decreased pain in an awake, cooperative, and nonmedicated patient is reassuring and may warrant further observation.

A delay in surgery when tissue is becoming more ischemic and pressures are high or climbing can be disastrous. Tissue necrosis can necessitate debridement of large volumes of muscle (Figure 10-6), and amputation may be necessary. It is important to remember that, although a compartment is a tight, unyielding space, it is not perfectly uniform. Pressure differences do exist within any compartment. The extent of damage is dependent on a number of factors, including the level of pressure within the compartment, the duration of applied pressure, changes in the patient's systemic blood pressure, and the presence of constrictive dressings or casts that can limit possible room for swelling.[14]

RELATIONSHIP BETWEEN RHABDOMYOLYSIS AND COMPARTMENT SYNDROME

Both rhabdomyolysis and compartment syndrome can result from marked increases in exertion. Systemic factors, such as baseline fitness and hydration status, affect both. The similarities raise questions. Which condition comes first? Does one condition cause the other? Does the presence of one condition mandate the presence of the other?

Both conditions can coexist but usually do not. Primary rhabdomyolysis has a number of possible etiologies and risk factors (as seen in the previously described case studies). The vast majority of cases do not require fasciotomy.[7] The extent of nephrotoxicity leading to dialysis appears to vary widely among reports and is clearly not related in a linear fashion

to levels of serum creatine kinase.[3,7,8,16,17,20] Prompt recognition, hydration, diuresis, and correction of any electrolyte abnormalities are usually sufficient treatment options. A negative synergistic effect among cofactors—such as sickle cell trait, ephedrine use, or preexisting dehydration—can have severe systemic consequences and possibly produce a fatal outcome. The amount of muscle swelling elevates pressures within a compartment enough to cause cellular ischemia. If this process continues unabated, continued muscle dissolution can worsen swelling and compartment syndrome can occur.[4]

In a primary compartment syndrome resulting from overexertion, the initial event is simple edema in a myofascial compartment. There is a subsequent increase in intracompartment myocyte ischemia as well as increased edema. The resulting cycle can end in cell death.

A soldier with heatstroke has a generalized seizure with severe muscle contractions. After the seizure, he is hypotensive from dehydration and is lying on an extremity that is compressed by his own body weight. Thus, this scenario could produce both rhabdomyolysis (heat injury, dehydration, tonic contractions with seizure) and compartment syndrome (hypotension, limb compression).

SUMMARY

Acute exertional rhabdomyolysis exists as a spectrum of physiological changes that might develop after excessive exercise. The extent to which systemic and renal toxicities develop is dependent on various cofactors, including baseline fitness, recent weight loss, hydration status, viral illness, stimulant use, and the presence of sickle cell trait. Prompt recognition and aggressive treatment usually prevent serious complications. Military personnel usually recover without ill effects after treatment, rest, and a gradual return to activity.[5,8,18]

Compartment syndrome is a condition characterized by elevated local tissue pressures within a tight fascial compartment. Ischemia at the cellular level ultimately results in tissue death, but can be prevented by emergent fasciotomy. The ischemic process evolves over time and requires repeated examination.

Close monitoring of military personnel undergoing physical training is mandatory. It is particularly important in those recruits who have just begun training. Their baseline level of fitness can be difficult to judge, and their general health habits before entering training are largely unknown. In addition, the use of stimulants, laxatives, and alternative medication with unknown ingredients can all adversely impact a basic trainee's response to military training demands and widely affect that individual's physiological responses. Compartment syndrome and rhabdomyolysis are just two of the problems that can result from overtraining, as depicted in Figure 10-7.

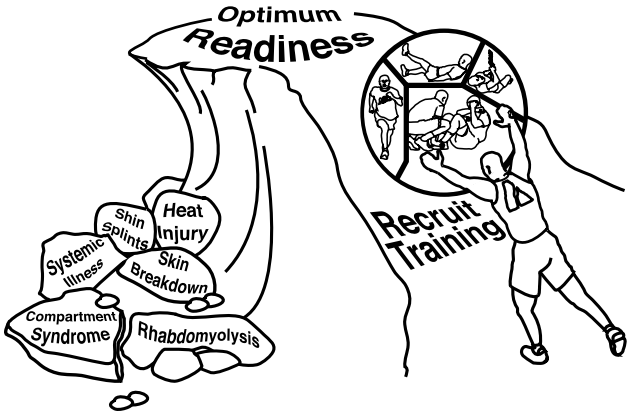

Fig. 10-7. A diagram representing the goal of recruit training: optimum readiness without injury. Training that exceeds physical limitations can result in a variety of injuries, some of which are depicted here.

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