Muscles

35

Alicia Gustafson and Cory A. Newman

Key Points
- Muscle injuries represent over 30% of all sports-related injuries.
- Sickle cell trait is a risk factor for muscle cramps, compartment syndrome, and rhabdomyolysis.
- There is no clear return-to-play protocol after a muscle strain, so clinical judgment is warranted.
- A quadriceps hematoma should be immobilized in at least 120 degrees of flexion for the first 24 hours after injury.

Table 35.1 Types of muscle injuries and the commonly associated sport

Injury	Most common sport
Hamstring strain	Sprinting, football, rugby
Adductor strain	Soccer, hockey
Gastrocnemius strain	Tennis, football, basketball
Chronic exertional compartment syndrome	Running
Hematoma	Football, rugby, hockey
Rhabdomyolysis	Weight lifting, endurance sports

Introduction

Muscle injuries are common in all ages and different types of sports. They represent over 30% of all sports-related injuries and are a major reason for missed playing time [19–21]. This chapter discusses the diagnosis, acute and long-term management, and prevention of some of the more common muscle injuries encountered in sports medicine. It also addresses rehabilitation and return-to-play guidelines following a muscular injury (Table 35.1).

Muscle Strain

- Acute muscle strain is one of the most common problems encountered in athletics. A muscle strain is the over-stretching or tearing of muscle fibers that usually occurs near the myotendinous junction [16, 27]. The degree of tearing can range from partial to full thickness and is given a grade I–III. A grade I strain is considered mild

and consists of stretching of the muscle fibers. Grade II strains are moderate strains with partial tearing of muscle fibers. Grade III strains, which are the most severe, are a full-thickness tear or complete muscle rupture [16, 17, 19]. The mechanism of injury is often either due to trauma, such as a fall or direct blow, or due to a sudden, forceful eccentric contraction as seen with the quick starts in track and field events [1, 27].
- *Risk factors:* Reduced cross-sectional area of the multifidus at L5 predicted 85% of muscular injuries of the hip, groin, and thigh in 83% of elite Australian football participants [56]. Other factors include poor lumbopelvic control, difference in muscle size when comparing right to left extremities, repetitive use of the dominant limb during sports, asymmetries in flexibility and strength between agonists and antagonist muscle groups, previous history of injury, improper training ramp up between off-season and pre-season, and insufficient warm-up prior to practice or game.
- *Clinical presentation:* The clinical presentation varies depending on the grade of the strain. The athlete will sometimes feel a pop or snap at the area of injury. The presentation for each grade of strain is listed below [16, 19, 27].
  - Grade I – pain with localized tenderness, possible mild swelling and bruising. The nerve and blood vessels are still intact; therefore, healing is accelerated. Examples include blunt trauma, minor surgical procedures, and

A. Gustafson (✉)
Northwest Allied Physicians, Tucson, AZ, USA

C. A. Newman
Proactive Physical Therapy, Tucson, AZ, USA

eccentric/concentric muscle injuries. The athlete may be able to continue playing.

- Grade II – more severe pain and tenderness with hemorrhage, swelling, weakness, and impaired function. Injury includes the soft tissue and nerve, but vasculature is intact. The extracellular matrix is also intact, so healing is accelerated in these instances as well.
- Grade III – severe and diffuse pain often causing the athlete to collapse after injury. There will likely be a palpable defect and significant loss of strength and function. The muscle, extracellular matrix, nerve, and blood vessels are damaged, thus prolonging the healing time and recovery. In grade III strains, the muscle has lost the ability to regenerate; thus, histological findings demonstrate a predominance of collagenous scar formation rather than muscle tissue.

- *Diagnosis:* A muscle strain is a clinical diagnosis determined by a thorough history of the injury and complete physical examination. Inspection, palpation, range of motion (ROM), and strength testing can often give you a sense of the severity [21]. Grade I strains do not require imaging unless there is no improvement. Imaging is helpful in grade II and III strains to assess the severity of the damage and the degree of tearing. Ultrasound is a very efficient and effective means to acquire post-injury information either on the sideline or in the training room. Direct visualization of edema, hemorrhage, and muscle fiber disruption can determine the grade and dictate management. Ultrasound can also be used to monitor resolution of the injury and functional dynamics. An MRI may also be obtained for finer details and better structural assessment of the involved muscle(s), tendons, and bone [21, 27].

- *Management:* The timing, speed, and intensity for applying movement and resistance exercises are individualized and dependent on age, severity of injury, and pain tolerance. In the acute stage, the acronym PRICEM "(P for protect, prevent, and promote healing; R for relative rest; I for ice or cryotherapy; C for compression; E for elevation; and M for modalities, medication, massage, mobilization, and movement)" should be followed [31]. There is conflicting evidence about the use of anti-inflammatory medications due to the possibility of slowing the healing process; therefore, the use of NSAIDs is situational and clinician-dependent [16, 18, 22, 27]. Restricted movement of the injured muscle is encouraged, as excessive stretching of injured muscles has been proven to result in excessive scar tissue formation and impaired muscle fiber regeneration [35]. Initial exercises performed should not elicit pain [27], but as pain and muscle function improve, rehabilitation can progress accordingly. Autologous blood product injections like platelet-rich plasma (PRP) are another possible management option. Results of PRP injections are anecdotal as there is insufficient human data to support the use at this time [23]. Most strains can be managed conservatively; however, surgical intervention is considered for cases that fail conservative management, most notably hamstring tears and complete ruptures [28, 29].

- *Return to play:* There is no clear consensus for when a player can return to play safely after a muscle strain. Ideally, an athlete would return to sports once they are pain-free and have regained full muscle strength and function [1, 16, 18, 27], but this is often not realistic as complete healing can take several months following grade II strains and possibly up to 1 year in complete tears. Therefore, the timing of when an athlete is safe to return to play is a group decision between the athlete, athletic trainers, therapists, and physicians. Muscles strains have a high rate of recurrence, and the exact cause is unclear due to the multifactorial nature of the injury. Some possible contributing factors are a reduction of muscle strength secondary to the injury [55], inappropriate rehabilitation methods such as not emphasizing eccentric strengthening and/or noncompliance of the patient, scar tissue reducing the extensibility of the musculotendinous unit resulting in decreased flexibility of the injured muscle, and possible alteration in biomechanics and neuromotor firing patterns [18, 27]. Also worthy of consideration is the athlete not completing a full rehabilitation program due to external pressures to return to the field prematurely.

- Some of the more common muscle strains, the rationale for increased occurrence, diagnosis, and appropriate rehabilitation methods for recovery, are described below.
  - *Hamstring* – Hamstring strains are some of the most common soft tissue injuries in athletes and the third most common hip injury in the sports medicine population, behind only gluteus medius tendinitis and trochanteric bursitis [31]. The hamstring is a two-joint muscle originating at the ischial tuberosity and inserting distal to the knee joint on both the tibia and fibula (Fig. 35.1). The hamstring is responsible for eccentrically controlling knee extension and hip flexion. The hamstring is comprised of three muscles: biceps femoris, semitendinosus, and semimembranosus, the former being the most commonly injured of the three due to the varying nerve supply. The short head of the biceps femoris is innervated by the peroneal division of the sciatic nerve, while the long head of the biceps femoris is innervated by the tibial division of the sciatic nerve. This varying nerve supply results in poor neuromuscular control ultimately leading to increased injury occurrence [27, 31]. Clinical presentation typically demonstrates a sub-optimal length-tension relationship due to postural asymmetries whereby the
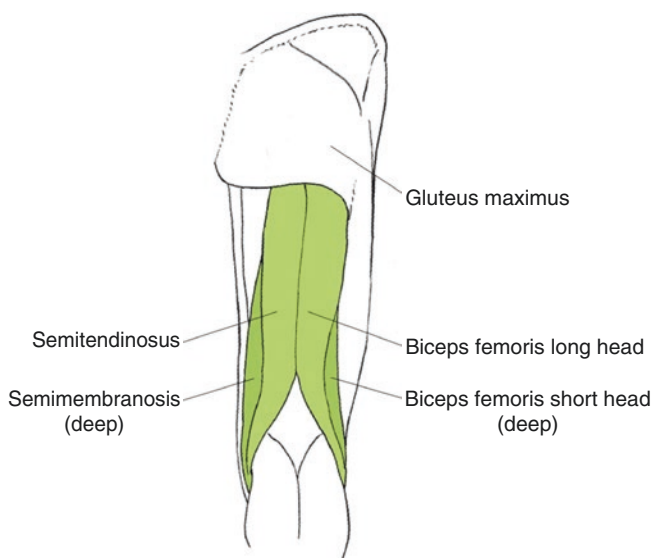
Fig. 35.1 Anatomy of the hamstring

hamstring is overly shortened and the quadriceps, antagonist, is overly lengthened. As a result of the shortened position, the hamstring cannot attenuate the eccentric forces of decelerating the leg during the terminal swing phase of gait, as is required during quick powerful movements (i.e., sprinting track events), and is consequently strained or torn.

Diagnosis – As noted, the biarticular nature of the hamstring requires multiple leg positions when testing strength and performing pain provocative tests. With the patient prone, and in 0 degrees of hip extension, knee flexion resistance can be applied at both 15 and 90 degrees of knee flexion. This resistance can be applied, while the leg is externally rotated to isolate the biceps femoris and internally rotated to bias the semimembranosus and semitendinosus muscles.

Rehabilitation guidelines are divided into three phases with specific goals and criteria in place before advancement to the next phase is indicated:

- During phase I (days 1–5), the goal is to minimize pain and edema and allow appropriate soft tissue healing with progression to phase II beginning when the following criteria are met: (1) non-antalgic walking pattern, (2) non-painful slow jog, and (3) pain-free submaximal isometric contraction (50–70%) with knee flexion at 90 degrees [27].
- During phase II gentle stretching in a pain-free range is incrementally increased, while also introducing higher-level core, lumbopelvic strengthening, and sports-specific agility drills that can be performed at higher speeds and intensities once neuromotor control is observed. Progression to phase III is achieved once the following criteria are met: (1) no pain during manual muscle testing of the injured muscle during maximal isometric contraction performed at 90 degrees knee flexion with a score of 5/5 and (2) able to run forward and backward at 50% maximum speed [27].
- Phase III, sports-specific drills incorporating functional movement patterns are emphasized in this stage. Explosive plyometric, ballistic, and quick change of direction activities should be avoided until the athlete attains back to sports criteria.

Return to sports criteria:

- Time frame for recovery is multifactorial and is dependent on the severity of the injury based upon MRI findings and clinical presentation (bruising, ROM, strength). Askling et al.. demonstrated that proximity of the injury to the ischial tuberosity is correlated to prolonged recovery time [34].
- The athlete should have full ROM and strength and be able to perform running, cutting, twisting, and jumping activities without pain or tightness. If isokinetic testing is available, there should be a less than 5% difference when comparing the involved and uninvolved sides [27].
- Brooks et al. have found there may be a link between lower incidences and severities of hamstring injury in athletes during training and competition if compliant with the Nordic hamstring protocol [36] (Table 35.2).

– *Gastrocnemius* – Strains of this muscle are referred to as "tennis leg" and frequently seen in sports that require rapid quick cutting movements in all directions. The most common mechanism of injury is forced ankle dorsiflexion while the knee is extended [31]. The triceps surae, i.e., calf muscle, is responsible for deceleration of the tibia and fibula in terminal swing phase of gait and plantar flexion of the ankle. The triceps surae is composed of the gastrocnemius and soleus muscles. The gastrocnemius, like the hamstring, is a two-joint muscle comprised of a medial and lateral head, the former originating on the medial condyle of the femur, the latter with an origin on the lateral condyle of the femur. Both medial and lateral heads conjoin to form the gastroc-soleus complex which inserts into the calcaneus via the Achilles tendon. The gastrocnemius is innervated by the tibial nerve.

Table 35.2 Nordic hamstring protocol

State of injury	Duration	Intervention	Progression criteria
Acute	1–3 days	Protect injured tissue Rest in lengthened position Non-antalgic gait pattern Ice, compression, and elevation	Passive SLR to 80 degrees with <3/10 pain
Subacute	3 days to >3 weeks	Gentle static stretching Isometric and concentric exercise begin 30% 1 rep max (RM) of uninjured leg, 3–4 sets × 10 reps Massage Cardio (UBE, stationary bike, etc.)	Perform 60% 1 RM of non-injured leg
Remodeling phase	1–6 weeks	Begin more aggressive concentric (seated hamstring curls 60–80% 1 RM) Eccentric hamstring curls and/or isokinetics from high to low speeds in pain-free range of motion Nordic hamstring exercise Plyometric progression (high knees, butt kicks, leg swings, box jumps)	Completion of all activities with <3/10 pain Perform 10 pain-free Nordic hamstring exercises Complete running progression
Return to activity	2 weeks to 6 months	17-level progression from walking to jogging to running and finally sprinting with presence or absence of soreness being indicator for regression or progression to next level High-intensity plyometrics Agility and sports-/position-specific drills	

Diagnosis – There is conflicting evidence concerning the ideal knee position when differentiating which muscles of the triceps surae are injured. Hebert-Loiser et al. demonstrate that the standing heel raise test should be performed for at least 40 times with the knee positioned between 0 and 5 degrees of knee extension to assess the strength of the gastrocnemius [45]. However, the same author concluded that heel raise testing at 0 degrees and 45 degrees of knee flexion was not enough to differentiate the varying muscles of the triceps surae [46]. Anecdotally, it has been observed that having the patient perform a single-leg heel raise in standing at 0–5 degrees of knee extension is sufficient to isolate the gastrocnemius. Resistive testing of ankle plantar flexion while seated with the knee flexed at 90 degrees is sufficient for isolating the soleus muscle. During the test, the clinician needs to be aware of signs for fatigue such as change in knee angle, reduced heel height, pace of performing a heel raise, loss of balance, or forward weight shift.

Return-to-play guidelines:

- The circumferential measurement 10 cm distal to the tibial tubercle is within 10% of the uninjured leg.
- The maximum number of single-leg heel raises is within 10% of the uninjured leg.

– *Adductor strain* – Adductor injuries are common among athletes due to the physical nature of the sports they participate in, especially contact sports requiring excessive cutting, twisting, and explosive movement patterns that involve sudden stopping and starting, as seen in football, hockey, and soccer. Also, sports that involve repetitive rotational forces such as ballet and martial arts have a high incidence of injury. Factors that contribute to hip injuries include female gender, previous injury, age, and generalized hip weakness. Arnanson et al.. demonstrated that athletes with increased fat content and reduced adductor flexibility were at risk for injury and those suffering from previous groin injury were 5× more likely to injure the same area [60].

The hip adductors are composed of five muscles that originate along the pubic symphysis, pubic crest, and inferior pubic ramus. The pectineus inserts on the posterior-medial surface of the femur just distal to the lesser trochanter and is innervated by the femoral nerve. The other four muscles are innervated by the obturator nerve: adductor brevis, adductor magnus, gracilis, and adductor longus, the latter being the most commonly injured [59]. The adductor brevis, longus, and magnus have insertion points along the linea aspera of the femur. The gracilis is a two-joint muscle inserting on the pes anserine (Fig. 35.2).

Diagnosis – The anatomical nature of the pelvis and the biomechanical relationship between the abdomen and hip make differential diagnosis challenging. Athletic pubalgia needs to be in the differential when diagnosing a groin strain. Kachingwe et al. have identified five signs for athletic pubalgia [61]:

1. Subjective complaint of deep groin pain.
2. Pain is exacerbated with exertion and relieved with rest.
3. Tenderness to palpation along the pubic ramus at site of rectus abdominis insertion.
4. Pain with resisted hip adduction at 0, 45, and 90 degrees of hip flexion.

Pectineus

Adductor brevis (deep)

Adductor longus

Adductor magnus (deep)

Gracilis
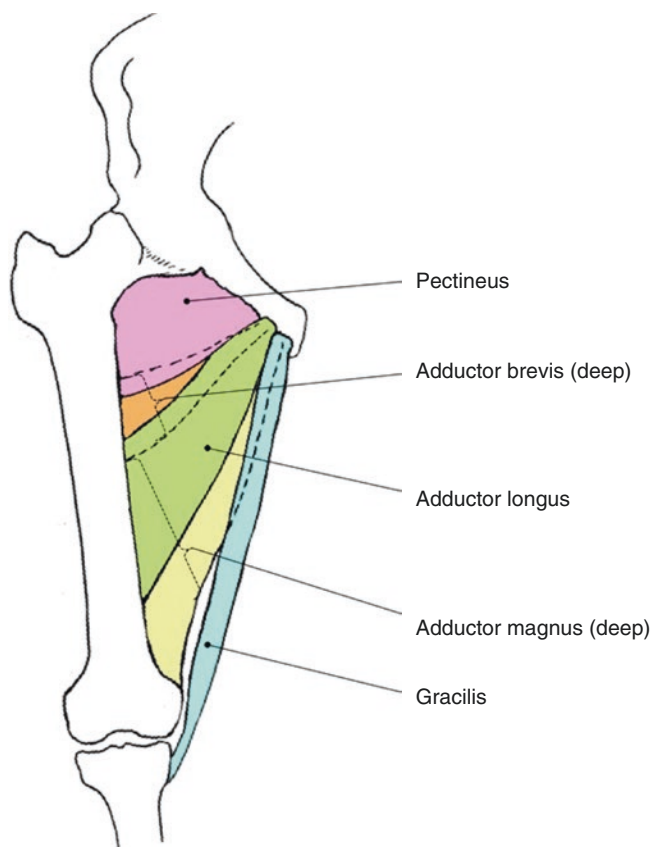
Fig. 35.2 Anatomy of the hip adductors

5. Pain with resisted abdominal crunch.

A dull aching pain is more indicative of an adductor strain compared to a deep-seated groin pain being a more reflective sign of athletic pubalgia.

Rehabilitation – Generalized rehabilitation methods mentioned previously can be implemented in the instances of an adductor strain. A broad range of return to sports time frames exist in the literature ranging from 5 weeks for surgical repair of the adductor longus in an elite athlete [62] to 10–52 weeks for adductor longus rupture managed conservatively in a college soccer player [63]. Basic biomechanical principles should be implemented when designing a return to activity progression. Each activity must be performed pain-free prior to initiating the next stage of higher-intensity activity. The progression follows the "walk before you can run" mantra and begins with pain-free walking and then progresses to the following activities in order of intensity: jog, run, sprint, cutting/pivoting, kicking, and finally return to normal practice session.

Other rehabilitation milestones that should be considered with return to field readiness include hip adduction strength deficits no greater than 10%

compared to the uninvolved leg [64, 65] and hip adduction to abduction strength ratio of at least 90% [65]. These measures are best obtained with a hand-held dynamometer as it has been indicated that even small changes in strength can be monitored [57].

Return to play – There are three return-to-play indicators [8]:

1. The athlete completes a running distance based on average running performed in a game.
2. Five minutes of skating on a 3 m slide board.
3. Three sets of 12 repetitions of hip adduction with 6 kg of resistance.

During the rehabilitation process, it is imperative to bear in mind the continued assessment of lumbar spine mobility, hip flexibility, ability to maintain appropriate posture during static and dynamic sports movements, and especially overall core strength as a delayed activation in the transverse abdominis muscle was frequently observed in athletes suffering from adductor strains [56].

Contusion/Hematoma

- A contusion is a bruise that is the result of a direct force or a repetitive force often seen in contact sports. The underlying blood vessels and muscle fibers at the area of impact are disrupted without damage to the overlying skin. Most contusions are subcutaneous. If significant damage to the deep vessels and muscle fibers occurs, an intramuscular hematoma can develop. A hematoma is an abnormal collection of coagulated blood outside of a blood vessel [2]. Another diagnosis to consider if there is persistent or worsening subcutaneous fluid collection at the site of injury is a Morel-Lavallee lesion which is often mistaken for a hematoma or contusion. A Morel-Lavallee lesion is generally due to a shearing force that causes the skin and cutaneous tissue to separate from the underlying fascia which leads to disruption of the lymphatics and blood vessels. It is also referred to as internal degloving [24–26]. Clinical presentation of a Morel-Lavallee lesion is a palpable, soft, fluctuant mass at the site of injury. Ultrasound will show hypoechoic/anechoic fluid with blood, lymph, and fat [24–26]. Management consists of aspiration, compression, and surgical intervention.
- *Risk factors:* The major risk factor for obtaining a contusion or hematoma is participation in a contact sport. Athletes who have a bleeding disorder such as hemophilia or are taking a blood thinner are also at a much higher risk.
- *Clinical presentation:* Contusions present with pain, tenderness, discoloration, and possible swelling at the site of injury. A hematoma will present similarly but may have

redness and warmth at the site of trauma [2]. Signs and symptoms can range from mild to severe. Mild contusions will have minimal pain and normal to slightly diminished range of motion [54]. If the lower extremity is mildly injured, that athlete's gait will still be normal. A severe contusion would be a hematoma with extreme pain and significant loss of range of motion [54]. Again, if the injury was to the leg, the athlete would have a severely antalgic gait. Moderate contusions fall in between [54].

- *Diagnosis:* Contusions and hematomas are a clinical diagnosis based on the mechanism of injury and the athlete's physical exam findings. Imaging generally is not needed in the acute setting unless an underlying fracture is suspected. In the subacute setting, ultrasound can be useful to further diagnose a hematoma and determine the size. A hematoma in the early phase will show hypoechoic fluid throughout the injured muscle that is generally well demarcated. Subacute and chronic hematomas will show more lobulations within the fluid as the blood coagulates. Ultrasound will also likely show calcifications if heterotopic bone formation is occurring within the hematoma. An MRI may be warranted to rule out other muscle pathology such as an avulsion or strain [54].

- *Management:* The initial management of contusions and hematomas is the same as for most other injuries: cryotherapy, rest, and compression. Resting the injured muscle helps to limit the size of the hematoma which will prevent further gaping within the muscle and reduce surrounding scar tissue [51]. Compression reduces intramuscular blood flow [51]. In the case of a quadriceps hematoma, the injured extremity should also be immobilized as soon as possible with the knee flexed to at least 120 degrees for the first 24 hours [54]. Keeping the knee flexed allows the quadriceps to remain elongated which helps prevent further hematoma formation and decreases the risk of developing myositis ossificans. Analgesics, such as NSAIDs, can be used in the acute setting but should not be used long-term due to their unknown effect on muscle healing. The use of a NSAID for at least the first 7 days in an athlete with a hematoma is encouraged to prevent the formation of myositis ossificans [54]. Most hematomas will resolve with conservative treatment; however, larger hematomas may require more invasive interventions such as percutaneous drainage or debridement [2, 24, 51, 52].

- *Return to play:* There is no clear return-to-play protocol after a contusion or hematoma. General guidelines include returning to play once an athlete has regained near normalization of the anatomic or physiologic deficit due to the injury and is able to perform sports-specific skills. The athlete should also feel comfortable returning [32]. An athlete may be able to return the same day after sustaining a contusion if it is not too severe. The use of protective padding may help to prevent recurrence [54].

- *Complications:* Some potential complications of a severe hematoma are myositis ossificans, compartment syndrome, and infection. Myositis ossificans, also known as heterotopic ossification, is generally seen with a quadriceps or gluteus hematoma. It is a tumorlike, non-neoplastic lesion consisting of proliferated bone and cartilage that develops at the site of the hematoma [53, 54]. It should be suspected in an athlete who has a hard, more painful mass at the hematoma site a few weeks post-injury. Early in its course, myositis ossificans may be visualized on radiography. Ultrasound will show shadowing of the cortical bone as it forms [53]. The diagnostic test of choice is a CT scan to evaluate mineralization and rule out any other etiologies such as malignancy or infection [53, 54]. Athletes can participate with a heterotopic ossified lesion but may experience flare-ups of pain and some loss of range of motion. Most cases ultimately require surgical intervention. This occurs once the lesion is completely mature, which is around 12 months on average [53, 54]. If the ossified lesion is removed before it is matured, recurrence is likely.

If there is a significant amount of intramuscular edema and swelling following a contusion or hematoma, the risk for developing compartment syndrome arises. The increase in compartment pressure leads to severe pain often out of proportion to exam. A hematoma is also potentially a nidus for infection, so close monitoring for increased redness, warmth, and fever is warranted.

Muscle Cramps

- Skeletal muscle cramping can be the result of numerous underlying medical conditions. The most common reason for cramping among athletes is exercise-associated muscle cramps (EAMC) [3, 7]. It is a painful, involuntary muscle contraction that can occur during or immediately after exercise. The exact etiology is unclear and controversial, but it is hypothesized that electrolyte disturbances, dehydration, and muscle fatigue causing altered neuromuscular control are amid the main contributing factors [3, 4, 7, 15]. In more recent years, it is believed that the electrolyte losses and dehydration theories are actually less likely causes of EAMC and that the more likely etiology is an imbalance between the inhibitory and excitatory drives at the spinal level [3–7, 15]. However, more conclusive research needs to be conducted in order to determine the primary pathophysiology.

Because various medical conditions and medications can make athletes more susceptible to cramping, obtain-

ing a thorough medical history is vital. It is important to consider a workup for an underlying etiology such as a metabolic or neurologic disorder in athletes with recurrent episodes of muscle cramping. Some examples of causes are diabetes, thyroid dysfunction, radiculopathy, or plexopathy [5–7]. Athletes with sickle cell trait and disease are also more prone to cramping. Exercise leads to hyperthermia, hypoxemia, and an acidotic state in the muscle which can provoke sickling of red blood cells [9–11, 48]. Sickled RBCs cause vaso-occlusion in small vessels leading to muscle pain and cramping. Athletes with sickle cell trait or disease who develop cramping need to cease physical activity and notify medical personnel immediately to prevent progression to a life-threatening condition [9–13].

- *Risk factors:* Multiple risk factors are believed to contribute to exercise-associated muscle cramps. Previous episode of EAMC, increased exercise intensity and duration, previous muscle injury, hot and humid environment, and physical deconditioning are just a few examples [3, 7, 15].
- *Management:* Static stretching is the most effective treatment for EAMC [5–7] and can be done immediately on the sideline. Hydration and electrolyte replenishment are still mainstays of management. Oral hydration is preferred over intravenous. Medications such as benzodiazepines, magnesium, and calcium can be used for severe cramping although these are not usually recommended due to the potential complications [7, 15]. In general, transfer to a tertiary care center should be considered in any athlete with persistent or worsening muscle cramping due to the risk of developing rhabdomyolysis. The threshold for transferring those with sickle cell trait should be low because of the numerous potential serious complications.
- *Return to play:* Many athletes with EAMC can return to sports the same day once the cramping has been alleviated with rest, stretching, and fluids. However, ultimately return to play requires clinical judgment based on the severity and cause of cramping [15].
- *Prevention:* Prevention consists of identifying and addressing the risk factors for every athlete. This includes adequate, regular stretching and muscle conditioning (plyometric exercises); appropriate nutrition and hydration before, during, and after exercise with modifications based on extrinsic factors (i.e., heat, humidity, altitude); and monitoring the duration and intensity of physical activity [5, 7, 15]. In athletes with recurrent EAMC, keeping a journal may be beneficial to identify and avoid triggers [4]. It is also important to educate athletes with sickle cell trait and disease, about their condition and warning signs during exercise.

Compartment Syndrome

- Compartment syndrome is a condition where the pressure inside the compartments of the extremities increases causing pain and symptoms of neurovascular compromise such as muscle weakness or claudication. The condition can be acute or chronic. Acute compartment syndrome (ACS) is a medical emergency requiring immediate recognition and intervention. ACS usually occurs after a trauma or prolonged immobilization. The injury to the extremity causes increased fluid accumulation within a compartment raising the pressure [38, 39]. It can be a complication of a fracture, muscle rupture, or hematoma. Nontraumatic causes of ACS are rhabdomyolysis and sickle cell trait. Acute compartment syndrome should be suspected in patients who are being treated for rhabdomyolysis where a second spike in their creatine kinase is observed [32, 33].

  Chronic exertional compartment syndrome (CECS) is more of an insidious onset with an unclear etiology causing it to often be misdiagnosed [37]. A few possible hypotheses are decreased fascial compliance, muscle hypertrophy, abnormally thickened fascia, or inappropriate muscle swelling during activity [50]. The incidence is equal between males and females and usually occurs in athletes in their early 20s. Ninety-five percent of cases involve the lower extremity [37]. The majority of cases are bilateral and occur with running [37]. Acute exertional compartment syndrome is also possible but is extremely rare.
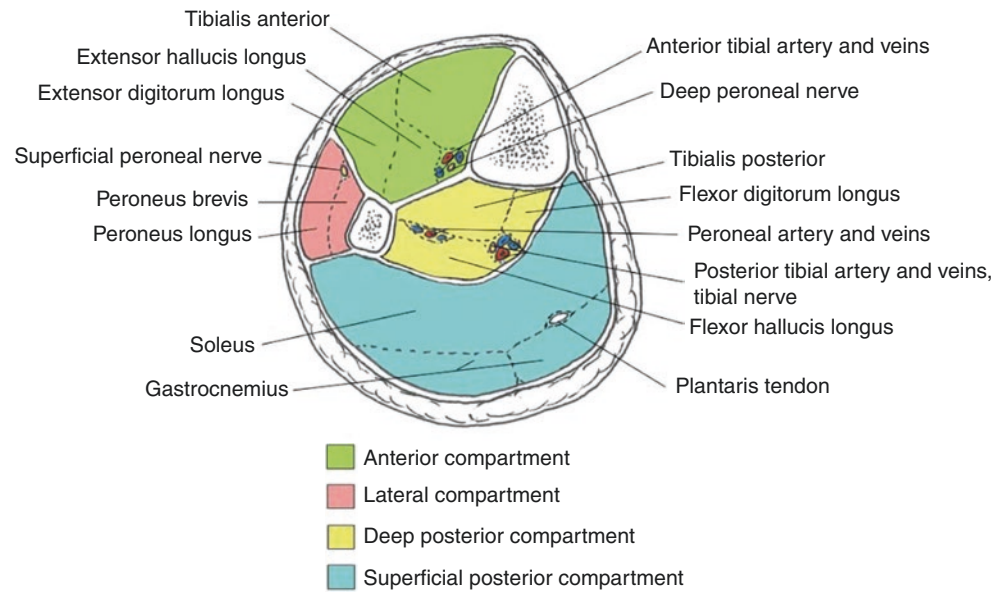- *Clinical presentation:* With chronic exertional compartment syndrome, the onset of pain is often predictable with a certain intensity and duration of exercise. There can also be sensory changes and muscle weakness [37]. Symptoms will improve or completely resolve with rest [43]. The clinical presentation of acute compartment syndrome will depend on the affected compartment. Pain, often out of proportion with the exam, will be present in almost all cases. Listed below are the major compartments of the upper and lower extremities and their contents.
  – *Lower leg* (Fig. 35.3)
    Anterior compartment – most common site for ACS. It contains the tibialis anterior, extensor hallucis longus, extensor digitorum longus, tibial artery and veins, and deep peroneal nerve [38–40]. Signs of compartment syndrome in this compartment would be decreased sensation at the first web space, pain with passive plantar flexion, and weakness of dorsiflexion. Late sequelae are foot drop and claw foot [38].
    Lateral – peroneus brevis and longus, peroneal artery, superficial peroneal nerve, and common

Fig. 35.3 Cross-sectional anatomy of the lower extremity

Tibialis anterior
Extensor hallucis longus
Extensor digitorum longus
Superficial peroneal nerve
Peroneus brevis
Peroneus longus
Soleus
Gastrocnemius

Anterior tibial artery and veins
Deep peroneal nerve
Tibialis posterior
Flexor digitorum longus
Peroneal artery and veins
Posterior tibial artery and veins, tibial nerve
Flexor hallucis longus
Plantaris tendon

- Anterior compartment
- Lateral compartment
- Deep posterior compartment
- Superficial posterior compartment

peroneal nerve [38–40]. Signs include weakness of eversion, inversion, dorsi- and plantar flexion, and decreased first web space sensation [38].

Superficial posterior – least likely to develop ACS. It contains the gastrocnemius, plantaris, and soleus which are palpable and painful on exam [38–40].

Deep posterior – posterior tibial artery/vein, peroneal artery/vein, tibial nerve, tibialis posterior, flexor hallucis longus, and flexor digitorum longus [38–40]. Presentation would be plantar hypesthesia, weakness of toe flexion, and pain with passive extension of toes [38].

– *Upper leg*

Anterior – rectus femoris, vastus medialis, vastus intermedius, vastus lateralis, and cutaneous branches of the femoral nerve and saphenous vein [40]. Signs would be weakness with knee extension, pain with passive knee flexion, and sensory changes of the thigh [38].

Posterior – biceps femoris, semitendinosus, and semimembranosus. Presentation would be pain with passive knee extension, weakness of knee flexion and plantar flexion of the ankle, and sensory deficits of the peroneal nerve [38].

Medial – adductor magnus, longus, brevis, and minimus; gracilis, pectineus, and obturator externus [40]. Signs may be weakness of hip adduction, pain with passive hip abduction, and sensory deficit of the obturator nerve [38].

– *Forearm*

Superficial volar – flexor carpi ulnaris, flexor digitorum superficialis, flexor carpi radialis, and pronator teres. The volar compartments also contain the median artery and nerve and the ulnar artery and

nerve [40]. These two compartments (superficial and deep volar) are at higher risk for developing ACS following trauma [38].

Deep volar – flexor digitorum profundus and flexor pollicis longus [38–40].

Dorsal – supinator, extensor carpi ulnaris, extensor digitorum, extensor pollicis longus and brevis, abductor pollicis longus, and radial and posterior interosseous nerve and artery [40].

Lateral – brachioradialis and extensor carpi radialis brevis and longus [40].

– *Upper arm*

Anterior – biceps brachii, brachialis, median, and ulnar nerves.

Posterior – triceps and radial nerve.

- *Diagnosis:* The diagnosis of compartment syndrome is based on history, physical exam, intracompartmental pressure testing, and a high clinical suspicion. The five Ps (pain, pallor, paresthesias, pulselessness, paralysis) that are classically associated with the diagnosis of compartment syndrome are a misconception [38]. Pain, sometimes out of proportion of exam, is the symptom most commonly associated with compartment syndrome. Paresthesias can sometimes occur in the early stages [39]. However, pallor, pulselessness, and paralysis are all late findings, and ACS usually is diagnosed before this time. A manometer is used to measure intracompartmental pressure. The normal pressure in a compartment is 0–8 mmHg. Pain will usually develop with a pressure >20 mmHg [38]. In the acute setting, if a pressure is within 30 mmHg of the diastolic pressure, then emergent fasciotomy is warranted [38, 39]. This equates to a delta pressure of <30 mmHg. There is some dispute about what the cutoff pressure should be [37–39]. Chronic exertional compartment syndrome is diagnosed

when symptoms are present and any of the following criteria are met: a pre-exercise pressure of greater than or equal to 15 mmHg, a 1 minute post-exercise pressure of 30 mmHg or greater, or a 5 minute post-exercise pressure of 20 mmHg or greater [37, 50].

- *Management:* Management of acute compartment syndrome first begins with having a high clinical suspicion in patients at risk [38]. The affected limb should not be elevated as blood flow to it is already compromised [39]. If the exam is indicative of ACS with an intracompartmental pressure within 30 mmHg of the diastolic pressure, then emergent fasciotomy is the definitive treatment [38, 39]. In some cases, the patient can be observed for several hours with serial examinations and pressure testing. If the exam is improving and the pressure readings are decreasing, then a fasciotomy may not be needed [38, 39]. The definitive treatment for CECS is also fasciotomy [37, 39, 43]. Many athletes will often discontinue any physical activity that causes symptoms in lieu of surgical intervention. Nonoperative management in CECS has been unsuccessful in the past, but potential newer treatments are being studied with promising results. Some examples are physical therapy that aims to reduce myofascial restrictions and improve neuromuscular function and motor control deficits [37], modifying running mechanics to avoid heel striking [43], and botulinum toxin injections [42].
- *Return to play:* There are no strict return-to-play guidelines after compartment syndrome. It is case dependent. The severity of the presentation and intervention dictate the recovery and rehabilitation. Acute compartment syndrome tends to have a longer recovery phase as the surgery is often more aggressive with more complications [39, 44]. An athlete may never return to their pre-injury level [39, 44]. Athletes with CECS can generally anticipate a gradual return to full sports in 6–12 weeks, on average, whether the treatment is conservative or surgical [49, 50]. Much like with returning after any injury, the athlete should have normal function and strength of the extremity and be able to participate in sports-specific drills. Recurrence of symptoms can occur regardless of the treatment but are more likely with nonoperative management. Recurrence rates range from 3% to 20% [37, 39, 47, 49, 50].
- *Complications:* Several serious complications can occur if there is a delay in diagnosis and treatment of acute compartment syndrome. Muscle necrosis occurs within the first 3–6 hours which leads to contractures and permanent deformity [39]. If the ischemic insult is severe enough, amputation may be required. Persistence of intracompartmental pressure also leads to rhabdomyolysis which causes myoglobinemia and potentially acute kidney injury [39]. Post-fasciotomy infection, particularly osteo-

myelitis, is another potentially serious complication [44]. Other complications after a fasciotomy are chronic pain, nerve damage, and muscle weakness.

Rhabdomyolysis

- Exertional rhabdomyolysis (ER) is the breakdown of muscle tissue secondary to a metabolic or mechanical insult such as strenuous exercise or normal exercise under various circumstances such as dehydration, supplement/medication use, high environmental temperatures, or recent viral illness [15, 30, 32, 33]. When the myocyte breaks, its intracellular contents are released into circulation [32]. Exertional rhabdomyolysis may also be the initial manifestation of an underlying genetic disorder. A genetic cause such as RyR1 mutation (which is the same gene involved in malignant hyperthermia), myopathy, or sickle cell trait should be considered in athletes with recurrent episodes or when the severity of rhabdomyolysis exceeds the expected response to the exercise performed [30, 32, 33]. The hyperthermic, acidotic, and hypoxemic environment created during exercise leads to sickling of RBCs in athletes with sickle cell trait. The sickled cells cause vaso-occlusion of muscle leading to muscle breakdown and rhabdomyolysis [48].
- *Clinical presentation:* Exertional rhabdomyolysis can range from mild to severe. Athletes with mild cases may be unaware and believe they are experiencing normal post-exertion muscle soreness and not seek medical attention. These milder cases are physiologic. More severe cases, with a likely pathological cause, present with extreme myalgias, muscle swelling and weakness, and "cola-colored" urine within 36 hours after exercise [30, 32, 33]. Consider rhabdomyolysis in athletes complaining of myalgias after performing repetitive eccentric exercises (squats, pull-ups, push-ups, etc.) with minimal rest periods or in those who are unaccustomed to the physical activity performed [30, 33]. Other potential signs and symptoms are fatigue, nausea, vomiting, and fever [30].
- *Diagnosis:* Rhabdomyolysis is a diagnosis determined by degree of symptoms and associated laboratory findings. The definition of the two types of exertional rhabdomyolysis are listed below:
  - *Physiologic ER*: Creatine kinase (CK) elevated at least five times the upper limit of normal within 36 hours after exercise with none or minimal muscle pain, with a maximum CK level at 3–4 days, followed by normalization within 2 weeks of no physical activity. Myoglobinuria may or may not be present. A urine dipstick that is positive for blood but no red blood cells are seen on microscopy is an indirect marker of urinary myoglobin. Also, ER is likely to be

physiologic if a known inciting factor is present such as certain medications, supplements, or recent viral illness and there is no contributing personal or family history [30, 32, 33].

– *Pathologic ER*: Symptomatic elevated CK which would include any of the following features: severe myalgias, swelling, and/or weakness. There is presence of severe myoglobinemia and/or myoglobinuria either by urine inspection or by laboratory testing. If complications such as acute kidney injury, significant electrolyte abnormalities, and delayed recovery are present, it is more likely to be pathologic, and also, if the patient has a personal or family history of a potential genetic cause or myopathy [30, 32, 41].

Ethnicity and gender play a role in what the athlete's baseline CK value is. These variables should be considered when determining if an athlete's CK is elevated. African American men and athletic men have the highest baselines, and non-African American women have the lowest [33].

• *Management:* The primary goal in treating rhabdomyolysis is preserving intravascular volume in order to maintain renal blood flow and organ perfusion [15]. Mild cases can be managed in an outpatient setting with rest, aggressive oral hydration, and serial monitoring of CK levels. However, an athlete complaining of muscle pain out of proportion to the exercise performed, muscle weakness, and dark urine should be sent to the emergency department for evaluation. Hospitalization will likely be required if there is symptomatic elevated CK, electrolyte abnormalities, elevated myoglobin with evidence of renal injury, or minimal urine output [41]. Intravenous fluid hydration should be given at a rate that maintains at least 200–300 mL/h of urine output [30, 41]. Dialysis may be warranted if the athlete is severely acidotic and has significant renal failure, hyperkalemia with cardiac arrhythmias, or no urine output even with adequate hydration [30, 41]. The normalization of the CK level will often lag behind the resolution of symptoms [14].

• Another possible treatment is dantrolene which has an effect on the calcium channels and muscle contractility [15]. This is not routinely used on the sideline as there is not enough research to support its efficacy [15].

• *Return to play:* There is currently no evidence-based return-to-play protocol after an athlete develops exertional rhabdomyolysis. The most important thing is to determine if there is a risk for recurrence. Guidelines were created to identify athletes who are at high risk for recurrence of exertional rhabdomyolysis [32, 41]. The guidelines are as follows [32, 58]:

1. Greater than 1 week to recover while resting
2. CK persistently elevated 5 times the upper limit of normal after 2 weeks of rest
3. Acute kidney injury

4. History of sickle cell trait
5. Personal or family history of myopathy, recurrent muscle cramps, or complications after general anesthesia (malignant hyperthermia)

If an athlete has any one of these risk factors, a further workup is warranted before they can be either cleared to return with close monitoring or disqualified. This would consist of genetic testing and possibly a muscle biopsy [32].

Athletes who are at lower risk for recurrence and likely had physiologic exertional rhabdomyolysis can follow a return-to-play protocol. After the athlete has rested for 72 hours with aggressive oral hydration, a CK level should be rechecked. If it is less than five times the upper limit of normal and the athlete has normal urine output, then they can begin light activities. This should continue for 1 week. If they remain asymptomatic, gradual progression to sports-related activities can occur [14, 30, 32, 58].

• *Complications:* Many complications can arise from exertional rhabdomyolysis because of the contents that are released from the myocyte. A large quantity of myoglobin is toxic to the renal tubules causing acute kidney injury and potentially renal failure [30, 32]. Excess extracellular potassium can lead to cardiac arrhythmias and possibly cardiac arrest [15, 30, 32]. The release of free radicals may cause tissue edema increasing the risk for compartment syndrome [32]. Disseminated intravascular coagulation is another possible complication [15].

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