Skeletal muscle is the single largest tissue in the body, making up 25-50% of one’s total body weight. As radiologists, we image many of the body’s 434 muscles each day -- both intentionally and incidentally. When evaluating the health of muscle, our most sophisticated radiological technique is MRI [1-2]. In this session, we review four topics that aid in understanding and diagnosing injuries of muscle: [I] normal anatomy; [II] MRI indications and technique; [III] pathological conditions; and [IV] differential diagnosis and diagnostic pitfalls.

I. NORMAL ANATOMY

ORGANIZATION OF MUSCLE
Muscle is a complex organ. The basic structural element of skeletal muscle is the muscle fiber. These fibers are grouped into fascicles, and the fascicles are grouped into muscles. Muscles, in turn, are arranged into compartments that are bounded by connective tissue termed fascia. Fascia plays a fundamental role in the pathogenesis of certain muscle disorders (e.g., compartment syndrome, fascial herniation) and influences the extent of others (e.g., spread of tumor and infection).

COMPARTMENTS
Given that there are hundreds of muscles in the body, the easiest way to conceptualize the location and function of muscles is to become familiar with compartmental anatomy. For example, in the mid-thigh, three compartments are present: anterior (quadriceps and sartorius); posterior (hamstrings); and medial (adductors and gracilis). Four compartments are present in the leg: anterior; lateral; superficial posterior; and deep posterior.

II. MRI INDICATIONS & TECHNIQUE

INDICATIONS
Muscle disorders have a wide variety of causes, treatments, and prognoses. Given that the cause and severity of musculoskeletal disorders may be difficult to determine clinically, MRI commonly is used to target the location, severity, and extent of the pain generator. Specifically, MRI of muscle also may be indicated to: aid in a prompt diagnosis of high performance athletes; exclude superimposed derangements (e.g., stress fracture); narrow the clinical differential diagnosis; predict the prognosis or possible complications associated with a disorder; direct the type and location of an intervention (such as biopsy or surgery, when indicated); and monitor treatment response or complications objectively.

MRI is the imaging test of choice for evaluating muscle and tendon disorders. Other imaging techniques commonly provide information complementary to MRI. Radiography is a relatively inexpensive means of screening patients for heterotopic ossification, avulsion fractures, and other osseous injuries. CT provides for cross-sectional assessment of these abnormalities, and may improve diagnostic confidence in some cases (e.g., myositis ossificans). However, soft-tissue contrast resolution is inferior to MRI. Sonography allows assessment of myotendinous disorders during dynamic maneuvers that may elicit symptoms and clarify the diagnosis. However, ultrasound remains an operator-dependent technique.

TECHNIQUE
While recognizing that each patient is unique, certain generalizations may be helpful in designing an appropriate MRI protocol.

- **Marker.** Placing a skin marker over the area of the patient’s symptoms allows you to correlate clinical complaints with imaging abnormalities.
- **Coil.** The choice of a coil will be dictated by the desired field of view and spatial resolution. With experience (and a good anatomic atlas!), scanning of both extremities usually is not necessary.
Imaging planes. The sagittal and coronal planes generally approximate the long axes of limb muscles. These “longitudinal” imaging planes are useful for depicting the longitudinal extent of myotendinous abnormalities (e.g., the size of a retracted tear).

Sagittal images are especially helpful for evaluating abnormalities at the anterior and posterior aspects of an extremity, while including the adjacent bone as an anatomic reference. Similarly, coronal images are particularly useful for assessing medial and lateral abnormalities.

Pulse Sequences

T1-weighted images provide a favorable ratio of signal-to-noise, while aiding in characterization of hemorrhagic lesions (e.g., hematoma, hemorrhagic neoplasm) or abnormal accumulations of fat (e.g., muscular atrophy, mature myositis ossificans, lipoma).

Fat-suppressed T2-weighted and inversion recovery (IR) fast spin-echo (FSE) images are more sensitive to the presence of edema and hemorrhage than long TE images that are not fat suppressed. However, compared to T2-weighted images, the altered dynamic range on fat-suppressed T2 and IR-FSE images may make specific diagnosis of soft-tissue masses more difficult [3].

Gradient-echo sequences accentuate paramagnetic effects. This “blooming” effect may indicate the presence of hemosiderin, foreign bodies, postoperative changes, or gas, and thus help in honing a differential diagnosis.

Supplemental Scans

Gadolinium. Generally, contrast material is not necessary. Areas affected by trauma or inflammation generally are well-demonstrated with fat-suppressed T2-weighted or IR-FSE images. Occasionally, fat-suppressed T1-weighted images after gadolinium administration can be helpful in difficult cases (e.g., when evaluating for a cystic vs. a solid mass or a necrotic neoplasm).

Muscle contraction during the MR examination rarely is used to demonstrate retraction of a torn muscle, herniation of a muscle through a fascial defect, or nerve entrapment that may occur dynamically during muscle activation (e.g., tarsal tunnel syndrome caused by a hypertrophied accessory soleus muscle). Pulse sequences (e.g., gradient-echo) with short acquisition times must be used to provide high temporal resolution images.

Exercise Enhancement. Exercise enhancement has been used for studying muscle recruitment during sports training programs. In patients with suspected chronic exertional compartment syndrome, MRI before and after exercise may be helpful. Of greatest practical importance to radiologists, however, is that spurious increased signal intensity may occur on fat-suppressed T2-weighted or IR-FSE images if MRI is performed within 30 minutes after exercise!

III. MRI OF PATHOLOGICAL CONDITIONS
INJURIES IN THE BONE-TENDON-MUSCLE COMPLEX

In athletes, the most common injuries to the bone-tendon-muscle complex are strains, contusions, and avulsions.

Strain

Mechanism & Sites of Injury. Strain injuries result from excessive stretch or tension on myotendinous fibers. Strains tend to occur in muscles that cross two joints, have a high proportion of fast twitch fibers, and undergo eccentric contraction (i.e., stretch during contraction). Eccentric contraction of certain muscles that do not cross two joints may result in strain injury (e.g., the hip adductors, especially the adductor longus muscle). The most commonly strained muscles in the extremities include the hamstrings, rectus femoris, and gastrocnemius muscles.

Spectrum of Injury. The degree of strain may be graded along a spectrum of injury -- from mild (grade I, microscopic injury) to moderate (grade II, partial-thickness tear) to severe (grade III, complete tear). These grades are used to facilitate communication and to influence patient management. (Speaking of communication … experts commonly use the term “strain” to refer to an injury of muscle or musculotendinous fibers, while “sprain” refers to ligamentous injury.)
Strain Injury of Specific Muscles

Calf Muscles
Several muscles and tendons at the posterior aspect of the knee and calf may be subjected to strain injuries, including the gastrocnemius, soleus, plantaris, and popliteus muscles.

Tennis Leg. “Tennis leg” has been defined clinically as the sudden onset of sharp pain in the middle portion of the calf while participating in athletics (e.g., racket sports, running). This condition typically occurs in middle-aged persons, and most commonly involves strain of the medial head of the gastrocnemius. (Isolated rupture of the plantaris tendon and soleus muscle are uncommon [4].) Clinical differential diagnosis may include a ruptured Baker’s cyst, overuse “tendinitis”, stress fracture, chronic exertional compartment syndrome, fascial herniation, venous thrombosis, nerve entrapment, and popliteal artery entrapment syndrome.

MRI may be used to help determine an accurate diagnosis and determine its severity. In one MRI study of 23 injuries to the gastrocnemius, the myotendinous junction was involved in 96% of cases [5]. The medial head was more frequently involved than the lateral head (86% and 14%, respectively). In another study of 65 patients with “tennis leg”, 51 partial and 14 complete tears were diagnosed [6]. Treatment is conservative, typically with relief of pain within two weeks and return to sports after at least three weeks.

Hamstring Muscles
The hamstrings are the most commonly injured muscles in sprinting and jumping athletes. For example, 10% of 180 soccer players suffered hamstring injuries during a single season in one prospective study [7]. In young adults, the majority of hamstring injuries are partial tears; complete hamstring tears or avulsions are relatively uncommon. Of the three hamstring components, the biceps femoris is the most commonly injured. Injury to more than one component of the hamstrings is not uncommon (prevalence: 25%-33%).

MRI. In one study of 15 college athletes, acute hamstring injuries occurred at the myotendinous junction in diverse locations: the proximal myotendinous junction (33%); the intramuscular myotendinous junction (53%); and the distal myotendinous junction (13%) [8]. In older adults, hamstring injuries also may occur at other sites, particularly at the tendinous attachments, owing to underlying tendinosis.

Prognosis. Rehabilitation time may be predicted by the extent of hamstring injury seen by MRI [9]. Convalescence periods reportedly vary from 2 weeks to 1.5 years before patients can return to vigorous activities. Recurrent injuries are common, occurring in one-fourth of athletes. Even minor hamstring injuries may double the risk of a more severe injury within 2 months.

Pectoralis Major Muscle
The pectoralis major is the largest, most superficial muscle in the anterior chest wall. This fan-shaped muscle originates primarily from the medial half of the clavicle, the sternum, and the first six costal cartilages. The clavicular and sternal heads converge as they pass laterally, generally producing a bilaminar tendon that inserts into the lateral lip of the humeral bicipital groove. The pectoralis major muscle functions to adduct, flex, and internally rotate the humerus.

MRI. Pectoralis major tears most commonly occur while the arm is abducted during eccentric contraction (e.g., in weight-lifters) or during a direct blow (e.g., in a motor vehicle accident). Partial tears of the pectoralis major are generally more common than complete tears [10]. Partial tears tend to occur at the myotendinous junction, and are usually managed non-operatively. Complete tears usually occur more distally at the enthesis. With avulsion of the tendon from its insertion site, high T2 signal intensity may be seen superficial to the humeral cortex due to periosteal stripping. Complete tears, particularly avulsion injuries from the humerus, are treated optimally in active individuals with prompt surgical repair in order to hasten rehabilitation and improve functional outcome.

Contusion
Contusion of muscle is produced by direct trauma, usually by a blunt object. Interstitial edema and hemorrhage results in varying degrees of pain, swelling, and spasm. Although contusion injuries often appear larger in size than
strain injuries, the recovery time for contusions tends to be significantly shorter. A recognized complication of muscular contusion is myositis ossificans.

MRI. Fat-suppressed T2-weighted and IR-FSE images provide a conspicuous display of high signal intensity that often has a feathery, infiltrative appearance. Although the girth of the muscle typically is increased, no fiber discontinuity or laxity is observed (allowing it to be distinguished from high-grade strain injuries).

Avulsion
The age of a patient influences the location of injuries in the bone-tendon-muscle complex. In children and adolescents, the weakest link in this chain tends to be the physis. Given the many apophyses in the pelvis and hip, it comes as no surprise that these are common sites of avulsion injuries. The single most common site of apophyseal avulsion is at the ischial tuberosity; this injury tends to occur between puberty and 25 years of age.

In the non-acute setting, avulsion injuries may resemble a neoplastic or infectious process, especially when no history of trauma is provided. Knowledge of the major tendinous attachments to bone is indispensable in arriving at a correct diagnosis -- and avoiding misdiagnosis of an osteochondroma or osteosarcoma. Avulsions generally are treated conservatively and have a good prognosis, although non-unions can occur.

SEQUELAE OF MUSCULOTENDINOUS INJURY
Several sequelae of musculotendinous injury may be observed, including atrophy, fibrosis, and hematoma.

Atrophy
Muscle atrophy may occur after certain musculotendinous injuries, disuse, or other insults. The cardinal features of muscle atrophy seen with MRI are fatty degeneration and/or decreased muscle volume.

The most frequently sought site of muscle atrophy using MRI is perhaps in the shoulder girdle after a rotator cuff tear. After a supraspinatus tendon tear, adjacent muscle atrophy is recognized as a negative prognostic factor for patients undergoing cuff repair. MR spectroscopy of the supraspinatus muscle to assess muscle fat content now can be performed routinely [11-12]. Interestingly, after a supraspinatus tendon tear, infraspinatus muscle atrophy can occur in isolation even when the infraspinatus tendon is intact [13]!

Muscle atrophy begins to develop within 10 days after immobilization. After bed rest for 20 days, the muscle cross-sectional area decreases approximately 10% in healthy men [14]. Similar degrees of muscle atrophy also can be measured by MRI after short (e.g., 2 week) spaceflights [15]. Muscle atrophy may be partially irreversible by 4 months.

Fibrosis
Fibrosis is characteristically displayed as low signal intensity tissue in muscle on T2-weighted images after a non-acute insult. Recognized sites of muscle fibrosis include the deltoid muscle (associated with scapular winging) and the vastus lateralis muscle (associated with non-traumatic patellar dislocation) [16].

Hematoma
Hematomas are common after a myotendinous injury, and may be predominantly intramuscular or intermuscular in location. Intramuscular hematomas often resorb spontaneously over a period of 6 to 8 weeks. Most of the intramuscular hematomas that have been evaluated with MRI between 5 days and 5 months after injury display characteristics of methemoglobin, with increased signal intensity on both T1- and T2-weighted images. Occasionally, serous-appearing fluid from a hematoma may linger within a connective tissue sheath, creating an intramuscular pseudocyst.

D/Dx. Differentiation between a simple hematoma and a hemorrhagic neoplasm may be difficult in some patients both clinically and with imaging. Administration of contrast material aids in excluding a neoplasm when the lesion in question shows no enhancement. Conversely, the presence of an enhancing nodule in a muscle lesion may suggest the possible diagnosis of a neoplasm rather than a hematoma. When the diagnosis of a probably benign
hematoma is in doubt, clinical correlation and a followup MRI examination may be indicated to confirm lesion stability or resolution.

**Myositis Ossificans**

The most common type of heterotropic ossification occurs in muscle, and commonly is referred to as myositis ossificans. Well-recognized precursors are observed in up to 75% of patients. Predisposing factors most commonly include: traumatic insults (e.g., contusion, surgery, burns); neurological insults (e.g., paraplegia, traumatic brain injury, stroke); or bleeding dyscrasias (e.g., hemophilia).

In the clinical and radiological arenas, three typical phases of evolution occur: (1) an acute or pseudo-inflammatory phase; (2) a subacute or pseudo-tumoral phase; and (3) a chronic, self-limited phase that may undergo spontaneous healing:

- In the acute and subacute stages of myositis ossificans, imaging examinations have a notoriously nonspecific appearance. CT is the single best imaging exam.
- In the final stage, the essential imaging findings that permit confident differentiation of myositis ossificans from neoplasm are three-fold: (1) the ossific mass is well-defined, sharply marginated, and appears more mature peripherally than centrally; (2) the lesion generally decreases in size with the passage of time; and (3) there is no destruction in the underlying bone.
- In contradistinction to calcification that has an amorphous appearance, the *sine qua non* of mature myositis ossificans is its recognizable architecture that approximates native bone: an area of cancellous bone centrally surrounded by compact bone peripherally.

**MRI.** MRI findings also evolve over time. In the acute and subacute stages, MRI findings are nonspecific. The involved muscle is enlarged and exhibits intermediate T1 and high T2 signal intensity. As the lesion matures, T2 hyperintensity and contrast enhancement progressively decrease. The signal intensity of the lesion may remain inhomogeneous, although areas of signal intensity equivalent to fat and cortical bone increase [17].

**Treatment.** Management of myositis ossificans may include nonsteroidal antiinflammatory agents (e.g., indomethacin), diphosphonates, low-dose irradiation therapy, physical therapy, and in uncommon cases, surgical resection. Surgical resection of myositis ossificans traditionally has been performed after the mass “matures” in the hopes of minimizing the risk of recurrence. The surgical excision of myositis ossificans occasionally may be indicated for purposes of a histopathologic diagnosis, unremitting pain, a bulky area of ossification that limits range of motion, or nerve entrapment. Myositis ossificans has been designated a “don’t touch” lesion that should not be biopsied injudiciously.

**Muscle Herniation**

**Pathogenesis.** Muscle herniation refers to protrusion of muscle tissue through a focal fascial defect. These fascial defects most commonly occur secondary to muscle hypertrophy and increased intracompartmental pressure, with subsequent herniation of muscle through relatively weak areas in the fascia, such as those traversed by blood vessels and nerves.

**Location.** Muscle hernias characteristically occur in the middle to lower portions of the leg. The tibialis anterior is the most commonly involved muscle. Virtually any muscle can be affected after an episode of penetrating trauma.

**Clinical.** Patients typically present with a small, superficial, soft-tissue bulge that becomes more prominent and firm with muscle contraction. Although most muscle herniations are asymptomatic, they can cause substantial pain, cramping, and tenderness. Fascial defects also may enlarge over time, thus resulting in cosmetic complaints. Rarely, herniated muscle may become incarcerated or result in nerve entrapment. For example, herniation of the gastrocnemius muscle can compress the peroneal nerve and result in a clinical presentation that resembles sciatica. Muscle herniation also may be observed in patients with compartment syndrome owing to intracompartmental hypertension.

**MRI.** MRI can be used to document herniation of muscle, as well as discontinuity in the overlying fascia. When routine MRI is negative, imaging during muscle contraction may be helpful in eliciting this derangement.
**Treatment.** Treatment of asymptomatic muscle hernias is usually conservative. For symptoms that are recalcitrant or severe, management may include local injection of botulinum toxin, fasciotomy, or fascial repair.

**Compartment Syndrome**
Compartment syndrome refers to elevated pressure in a relatively noncompliant anatomic space that is associated with ischemia and may result in neuromuscular injury, including myonecrosis. Knowledge of compartmental anatomy is fundamental to accurate diagnosis and treatment of this potentially devastating condition. Factors that may predispose a compartment to this syndrome include a history of trauma, external compression, systemic hypotension, increased intracompartmental volume (e.g., hemorrhage, edema, poor venous return, muscle hypertrophy), and loss of compartment elasticity (e.g., fibrotic or constricted fascia). Patients initially complain of painful aching, tightness, or pressure that worsens with palpation and passive stretching of the affected muscles.

**Acute Compartment Syndrome.** Although most cases of acute compartment syndrome are associated with fractures, the second most common cause is injury to soft tissues (e.g., contusion) without fracture. Compartment syndrome occasionally may be caused by muscle rupture in athletes.

**Chronic Compartment Syndrome.** Chronic compartment syndrome may occur owing to exertional causes (e.g., exercise, occupational overuse) or “non-exertional” causes (e.g., a mass lesion, infection). In running athletes, for example, the most common site of chronic compartment syndrome is the leg. The thigh, forearm, and foot are the next most common sites in athletes.

**Diagnostic Examinations.** Diagnostic tests used to evaluate for compartment syndrome include direct compartment pressure measurements, near infrared spectroscopy, and MRI. Normally, intracompartmental pressures should be less than 15-20 mm Hg at rest and within 5 minutes after finishing exercise. Direct pressure measurements are the gold standard for objective diagnosis.

**MRI.** Although cross-sectional imaging is not the primary technique for diagnosing compartment syndrome, MRI can be used to clarify the location and extent of ischemic damage to muscle in the non-acute setting. Imaging also assists in evaluation for an underlying lesion (e.g., hematoma, neoplasm) that may contribute to compartment hypertension and need to be addressed at surgery. Familiarity with the imaging appearance of compartment syndrome is important, given that imaging may be performed for assessment of pain that initially is thought to be due to other causes (e.g., stress fracture, myotendinous strain, soft-tissue tumor).

In patients with compartment syndrome, the most common findings are T2 hyperintensity and increased muscle volume (that may be due to muscle hypertrophy, edema, or both). This increased intracompartmental volume and pressure occasionally may result in herniation of muscle through a tear in the surrounding fascia. Fascial thickening also may be observed.

**Gadolinium.** Although controversial, gadolinium-enhanced MRI may be helpful in evaluating patients with impending compartment syndrome by showing avid contrast enhancement in the affected muscles. This enhancement can be useful in distinguishing muscles that are still perfused from those with devitalized areas. The rate of contrast material “washout” may be prolonged in muscles affected by compartment syndrome.

**Post-exercise MRI.** The change of T2 signal intensity between pre-exercise and post-exercise images is significantly greater in compartments with post-exercise hypertension [18]. Furthermore, the magnitude of this change in signal intensity correlates significantly with the change in pre-exercise and post-exercise pressure measurements, as well as with the absolute post-exercise pressure. Diffusion-weighted echo-planar MRI potentially can depict alterations in the circulating blood volume in muscle induced by exercise and changes in compartment pressure.

**Differential Diagnosis.** Although MRI may be sensitive in the evaluation of compartment syndrome, it is not specific. Depending on the clinical context, the imaging differential diagnosis may include other causes of painful, swollen extremities, (e.g., delayed onset muscle soreness, muscle strain, deep venous thrombosis, cellulitis, lymphedema).
Fasciotomy generally is performed when acute compartment pressures reach 30 to 80 mm Hg. Findings favoring fascial release include increasing compartment pressures over time, paresthesia, and paresis. For chronic compartment syndrome, fasciotomy generally is recommended if symptoms persist more than 6 months despite conservative therapy.

IV. DIFFERENTIAL DIAGNOSIS & DIAGNOSTIC PITFALLS

DIFFERENTIAL DIAGNOSIS

MRI facilitates the diagnostic process primarily by detecting alterations in muscle size or signal intensity. Although these alterations may be diagnostic in the appropriate clinical setting, a wide array of focal and systemic pathological conditions affecting muscle may have a similar appearance. In addition to traumatic muscle injuries, common categories of disease affecting muscle include: ischemia and necrosis; inflammation and infection; congenital and inherited conditions; neoplasms; and various iatrogenic insults. Given that the potential causes for abnormal signal intensity in muscle are diverse, the differential diagnosis approach may be simplified by recognizing one of three basic patterns on MR images [19].

Three Basic Patterns of Abnormal Signal Intensity in Muscle

- The “muscle edema pattern” may be seen with recent trauma (e.g., strain injury), as well as with subacute denervation, infectious or autoimmune myositis, rhabdomyolysis, vascular insult (e.g., diabetic muscle infarction, deep venous thrombosis) or recent iatrogenic insults (e.g., surgery, radiation therapy).
- The “fatty infiltration pattern” may be observed in the chronic setting after a high-grade myotendinous injury, as well as with other insults causing chronic muscle disuse or chronic denervation.
- The “mass lesion pattern” can be seen with traumatic injuries (e.g., myositis ossificans), as well as with neoplasm, infection (e.g., pyomyositis, parasitic infection), and muscular sarcoidosis.

Delayed Onset Muscle Soreness (DOMS)

DOMS refers to the muscular pain, soreness, and swelling that follows unaccustomed exertion. Activities that require eccentric muscle contractions are common culprits, such as hiking downhill or certain types of manual labor. DOMS is thought to occur from reversible microstructural muscle injury; no permanent damage to muscle function ensues. Patients with DOMS do not recall any one particular moment of trauma or experience an acute onset of pain. Rather, symptoms tend to begin within 1 to 2 days after exercise. Soreness often crescendos until it peaks 2 to 3 days after the inciting exercise, and then generally subsides within 1 week. This soreness may be associated with temporarily diminished muscle strength.

MRI. High signal intensity indicative of interstitial edema is observed on T2-weighted or IR images. Perifascial fluid/edema occasionally may be seen in the early phase. The MRI appearance of DOMS is reportedly similar to a first-degree muscle strain. Clinical history allows for easy differentiation between these two entities in most instances. However, the history of a provocative event may not be forthcoming in all cases, since abnormal signal intensity reportedly may remain for up to 80 days!

Muscle Denervation & Hypertrophy

Entrapment neuropathy and denervation can be a cause of pain and weakness that simulates a primary abnormality in skeletal muscle. MRI may be a useful adjunct to electromyography (EMG) in detecting muscle denervation and its causes. For example, in a study of 90 patients with clinical evidence of peripheral nerve injury or radiculopathy, the sensitivity and specificity of IR-FSE MRI relative to EMG were 84% and 100%, respectively. Although less sensitive than EMG, MRI may display the site and cause of nerve entrapment in many cases (e.g., intervertebral disc herniation, ganglion cyst, hematoma).

Subacute Denervation. With denervation, the signal intensity and morphology of muscle undergo characteristic changes with MRI. Although these denervation changes have been reported as early as 4 days after a nerve insult, hyperintense signal on T2-weighted or IR-FSE MR images usually are not detectable for 2 to 3 weeks. Three MRI features may help distinguish the hyperintense T2 signal in denervated muscles from that seen with strained muscles: [1] unlike strain injury, the hyperintense T2 signal in denervated muscles is not associated with perifascial edema; [2] the pattern of muscle involvement may suggest a specific nerve territory responsible for the denervation changes; and [3] abnormally hyperintense T2 signal in peripheral nerves is a hallmark of many neuropathies.
**Chronic Denervation.** With chronic denervation, diminished bulk and fatty infiltration occur in muscle. These atrophic changes are best displayed on T1-weighted MR images. Whereas the signal intensity changes of acute muscle denervation are reversible, profound atrophic changes seen late in the course of denervation may be irreversible. The atrophic changes from denervation are not specific, and may be seen with conditions as diverse as motor neuron diseases (e.g., poliomyelitis) and demyelination (e.g., hereditary motor and sensory neuropathies). Although chronic denervation usually results in atrophy, pseudohypertrophy and true hypertrophy have been reported. Both conditions may present clinically as a palpable soft-tissue mass that serves as an indication for MRI.

**DIAGNOSTIC PITFALLS**

**Anomalous Muscles**

Anomalous muscles are encountered relatively commonly. Although they usually are of no clinical significance *per se*, they can be mistaken for mass lesions or torn muscles that have retracted. In uncommon cases, these types of muscles may be injured or may result in symptoms owing to nerve entrapment, especially when these muscles become hypertrophied (e.g., in athletes).

**SUMMARY**

Muscle disorders have a wide variety of causes, treatments, and prognoses. Given that the cause and severity of these disorders may be difficult to determine clinically in some cases, MRI can be used to narrow the differential diagnosis and pinpoint the appropriate pain generator.

**SELECTED REFERENCES**