Anatomic, Vascular, and Mechanical Overview of the Achilles Tendon

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KEYWORDS

- Achilles • Anatomy • Tendon • Biomechanics

KEY POINTS

- The anatomic and histologic properties of the Achilles tendon are directly related to resistance to mechanical damage and subsequent pathologic processes as well as tendon healing.
- Gastrocnemius and soleus anatomy have a direct effect on many pathologic processes in the foot and ankle.
- Experimental data indicating uniform hemodynamic flow throughout the Achilles tendon has challenged the widespread notion that ischemia is a primary etiology of pathology and rupture in the central portion of the tendon, which is based on a circumstantial association with an anatomic watershed.

GROSS ANATOMY

The Achilles is composed of the conjoined tendons of the gastrocnemius, soleus, and occasionally plantaris muscles.[1,2] These associated muscles and the Achilles tendon make up the superficial posterior compartment of the leg. The gastrocnemius muscle is the most superficial component of the triceps surae. The medial and lateral heads of the gastrocnemius arise from the femoral condyles. The medial head originates from behind the medial supracondylar line and adductor tubercle, superior to the medial femoral condyle. The lateral head originates from the posterior aspect of the lateral femoral condyle superior and posterior to the lateral epicondyle, as well as from a portion of the lateral lip of the linea aspera superior to the lateral condyle. The medial head is larger, longer, and extends farther distally than the lateral head. Both heads share an additional origin from the posterior aspect of the knee capsule termed the popliteal ligament. The deep surface of the muscle is tendinous and intimately approximated to the soleus muscle. Deep to both heads may lie bursae. The gastrocnemius

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heads are large, fusiform bodies that join in the midcalf to form a wide aponeurosis at the muscle's distal aspect. The heads form a tendinous raphe where they meet midline and communicate with the deep, anterior aspect of the aponeurosis. This aponeurosis continues distally as a component of the Achilles tendon.

The soleus muscle is a broad, flat, pennate muscle that arises entirely from below the knee. The muscle lies deep to the gastrocnemius muscle within the superficial posterior compartment of the leg and is housed between 2 aponeurotic lamellae with the posterior aponeurosis beginning more proximally than that of the gastrocnemius. Muscle origins include the head and proximal fourth of the posterior fibula, the oblique line and middle third of the medial tibial border, and the fibrous arch between the 2. The gross muscle is wider and extends more distally than the gastrocnemius with fibers joining centrally in a posterior aponeurosis or intramuscular tendon, which promotes a bipennate arrangement of muscle fibers. This central intramuscular tendon merges distally to form a component of the Achilles tendon. Between gastrocnemius and soleus muscles lies a layer of dense fibrous connective tissue with a film of loose connective tissue between the each of the layers. The posterior aponeurosis of the soleus is the largest contributory component of the Achilles tendon.

The plantaris muscle is variable in size and absent in 6% to 8% of individuals.[2] The plantaris originates from the superior aspect of the lateral femoral condyle. The muscle belly runs medially and continues as a long tendon that extends distally between the gastrocnemius and soleus to insert on the medial border of the Achilles tendon.

The nerve supply to the gastrocsoleus is derived from tibial branches proximally. The Achilles tendon has sparse innervation from several sources arising from the paratendinous soft tissues. The sural nerve is a main contributor to tendon and peritendon structures and is at risk for injury during surgical procedures because of its proximity to the tendon and aponeurosis posteriorly.

The Achilles tendon begins at the musculotendinous junction of the gastrocnemius and soleus muscles with typical full incorporation occurring approximately 8 to 10 cm above the calcaneal insertion site.[1] In total, the Achilles tendon is approximately 15 cm in length and begins flattened at the musculotendinous junction and becomes rounded approximately 4 cm from the calcaneus. The anterior and medial aspects of the tendon receive fibers from the soleus, and the posterior aspect is derived from gastrocnemius fibers. The contributions and proportions from both the gastrocnemius and soleus are variable. As fibers travel distally, they rotate 90° such that gastrocnemius fibers attach laterally and posteriorly, whereas the fibers of the soleus attach medially and anteriorly. This spiraling has been shown to result in less fiber buckling when the tendon is lax and less deformation when tension is applied to the tendon.[3] At its insertion site, the tendon flattens and broadens into a deltoid-type of attachment and develops an anterior concavity before inserting along the middle third of the posterior aspect of the calcaneus. The surface of the distal tendon that overlies the calcaneus is composed of fibrocartilage. Underneath the tendon lies the retrocalcaneal bursa, which is interpositioned between the tendon and the posterior calcaneal tuberosity. At the distal-most insertion of the tendon, some collagen fibers form Sharpey fibers and become continuous with fibrous tissue overlying the calcaneus.

There is no true synovial sheath surrounding the Achilles tendon. Instead, a paratenon forms an elastic sleeve around the tendon to permit gliding. It is composed of sheets of dense connective tissue that separate the tendon from the deep fascia of the leg. Within this tendon lie numerous blood vessels and nerves. The peritendinous structure and the abundance of mucopolysaccharides within the sheath permits sliding of the tendon along the adjacent tissues. Proximally, the paratenon is continuous with the muscle fascia and distally it blends with the periosteum of the calcaneus.

HISTOLOGIC ANATOMY

Like most tendons, the Achilles tendon is composed of paralleled bundles of Type 1 collagen.[1] Collagen fibers average 60 μm in diameter. These fibers are organized into fibrils that range from 30 to 130 nm in diameter and assume a wavy pattern. Microfibrils are grouped into fibrils; fibrils are organized into fibers. A group of fibers is organized into fascicles, which are further grouped into bundles. Individual fibrils do not run the entire length of the tendon but rather are linked in succession, necessitating the transfer of stress between associated fibril units.[1] Within the midsubstance of the tendon are fibroblasts that are arranged in longitudinal rows.

Around each collagen bundle is an endotenon, an elastin-rich connective tissue envelope that maintains the bundle's integrity and permits independent bundle gliding in relation to other bundles. This endotenon contains vessels, lymphatics, and nerves. Surrounding the entire gross tendon lies a fine connective tissue sheath called the epitenon with mesotenon and paratenon overlying it.

In a study performed by Cutts,[4] cadaveric observation showed that sarcomere lengths for the muscles in the lower limb vary significantly. The length of soleus sarcomeres averaged 2.033 mm when in anatomic position, and ranged from 1.260 mm in its theoretic shortest position to 3.359 mm in its theoretic longest position. Soleus fibers are shorter Type I fibers and are involved in slow contractures and balance. As for the gastrocnemius muscle, its observed sarcomeres averaged 2.033 mm in length with a range from 1.012 mm at its theoretic shortest position to 4.413 mm at its longest position. Gastrocnemius Type II muscle fibers are responsible for explosive contractures of jumping and running. These sarcomeres are arranged in series, averaging 17,400 before forming the Achilles tendon.

As previously described, the soleus is a pennate muscle. Muscle fibers are arranged anterior to posterior, proximal to distal with an average pennation angle of 19.3°. This is similar to reported findings from Alexander and Vernon[5] that found a mean pennation angle of 20°. Wickiewicz and colleagues[6] described a small portion of highly (60°) pennated fibers along the ventral surface of the muscle.

The gastrocnemius is not regarded as a pennate muscle; however, the individual heads exhibit pennation in relation to one another. Cutts[4] found an average pennation angle of 10.7° of the lateral head. This lends to observations made by Wickiewicz and colleagues[6] that describe the lateral head of the gastrocnemius to be shorter than its medial counterpart, whereas individual fibers are 46% longer in the lateral head compared with the medial, and fibers of the lateral head have a smaller angle of pennation.

BLOOD SUPPLY AND HEMODYNAMICS

As with any tissue, the quality and quantity of vascularization directly affects a tissue's response to trauma and provides the basis for healing. The blood supply to the gastrocnemius and the soleus muscles is usually discussed in 3 separate regions: the musculotendinous junction, direct supply to the tendon, and the tendon-bone junction. Blood vessels from these regions originate from the perimysium, mesotenon-paratenon structure, and periosteum. At the musculotendinous junction, blood is supplied via superficial vessels from surrounding tissues. The main blood supply to the midsubstance of the tendon flows via the paratenon. Vessels within this connective tissue envelope run transversely toward the tendon on its anterior aspect, branch, and then continue longitudinally with the course of the tendon. The blood supply arises from the mesotenon on the anterior aspect of the tendon where the paratenon meets itself. The recurrent branch of the posterior tibial artery supplies

the proximal aspect of the tendon, whereas the distal tendon is supplied by the rete arteriosum calcaneare, fibular, and posterior tibial arteries. Vessels enter the tendon at the endoteon, and arterioles continue as capillaries that loop into venules without penetrating collagen bundles. Vessels that supply the bone-tendon junction also supply the lower third of the tendon. These vessels indirectly anastomose with vessels supplying the midsubstance of the tendon.[1,2]

The most commonly cited understanding of the Achilles tendon blood flow is based on cadaveric anatomic research done in the 1950s.[7] We have come to regard the midsubstance of the Achilles as relatively ischemic based on this historic cadaveric research. In 1958, a "watershed" area was identified in the midsubstance of the Achilles tendon through cadaveric injection and analysis of the Achilles peritendinous vessels.[7] It is this "watershed" that has been proposed as the etiology of an ischemic zone in the tendon and proposed a main determinant in weakness and rupture. The term watershed is a geographic concept identifying the point at which 2 bodies of water come together from 2 different directions, or a central zone of land that divides areas drained by different river systems. Indeed, the Achilles tendon has been shown to have multiple sources of blood supply originating distal, proximal, and from the peritendinous structures[7,8] The question is, however, does this anatomic distribution of vessels within the tendon tissues cause a true hemodynamic compromise or ischemia. As early as 1958, Hastad[9] tested the hemodynamic flow in the tendon using a sodium washout technique and noted uniform blood flow throughout the tendon, which challenges the watershed-based ischemia theory. Astrom and colleagues[10] in 1994 used laser Doppler flow analysis to assess real-time tendon circulation in 28 healthy volunteers. They tested the subjects with the Doppler probe inserted into the tendon and assessed hemodynamic flow at rest, during calf muscle contracture and after vascular occlusion. Their findings showed pulsatile flow synchronous with the heart rate evenly distributed throughout the tendon with only a slight decrease at the tendon insertion at the calcaneus.[11] This research challenges the anatomic-based ischemic theory put forth by Lagergren and colleagues.[7] Further hemodynamic research in live subjects has further strengthened the notion that blood flow is uniform throughout the tendon, including the watershed zone.[10,12] Other research has shown an increase in blood flow in the tendon with exercise. Langberg and colleagues[12] used Xe flow measurements to identify a fourfold increase in tendon blood flow 5 cm from the Achilles insertion during exercise. Boushel and colleagues[13] noted that both muscle tendon blood flow increased concurrently with exercise without abnormal shunting of blood.[14] Kubo and colleagues[15] reported both blood volume and oxygen saturation after repetitive muscle contracture did not change.[16] In 2015, Kubo[14] then found that although oxygen consumption did not change when comparing eccentric to concentric muscle contraction, inflow of blood to the tendon was significantly greater during eccentric compared with concentric contractions.

It has been suggested based on work by Cummins and Anson[11] in 1946 that the twisting of the Achilles tendon fibers distally is a possible cause of the purported ischemia of the midsubstance of the tendon. This so called "wringing out" of the tendon, as it has been described by many, is based on these anatomic observations. Although this an attractive circumstantial piece of evidence, the effect of this fiber twisting on real-time blood flow in living tissue has not been confirmed through experimentation. The studies that have shown uniform blood flow throughout the tendon at rest, with contracture and exercise noted previously, would argue against this effect. Further research is needed to make the causal connection between the tendon spatial anatomy (twisting) and the blood flow within the tendon at all levels. Many observations in medicine lead to theories regarding the role that an observation has for

function. When these observations are connected with theories that have a strong component of "common sense" or appear on the surface to fit nicely with what we already have learned, the idea begins to be accepted as true through a process of repeated quoting and testing despite lack of true proof. As clinicians and researchers, we must take care not to perpetuate ideas that have not been tested to prove a connection between anatomic observations and theoretic application to physiologic function.

BIOMECHANICS

The gastrocnemius and the soleus act together as the main plantar flexors of the foot at the ankle joint during gait. They are both active during the latter 80% of the stance phase of gait and have a major role in propulsion and balance. The posterior muscles, especially the soleus, act as stabilizers and play a critical role in balance by resisting the ground reactive force on the stance leg as the swing phase extremity moves forward, creating a dorsiflexory moment at the ankle joint. Stabilization forces are converted to push-off force in the stance-phase foot beginning at the point in the gait cycle just after maximal ankle dorsiflexion is achieved with the tibia moving forward producing ankle plantarflexion. The posterior muscle group is inactive just after toe-off and throughout swing. Conditions that result in weakness of the gastrocsoleus unit result in significant imbalance and alterations of the normal gait. Lack of the stabilizing function of the superficial posterior muscles prevents the person from resisting the forces generated from the center of gravity anterior to the knee, which is present in normal gait and results in the person having a tendency to fall forward. In addition, weakness results in compensation with increased activity of the deep posterior muscle group (flexor substitution), which may lead to a variety of foot and digital deformations. Equinus or lack of adequate ankle dorsiflexion in gait can lead to compensation through pronation of the foot. It has been recognized that this effect of compensation for a lack of ankle dorsiflexion can lead to repetitive stress issues in the foot, such as plantar faciosis and forefoot pain. This will be discussed in subsequent articles. Depending on the position of the Achilles tendon relative to the subtalar joint axis, the triceps surae may be a relative supinator or pronator of the subtalar joint. This function will vary depending on foot position and on segmental leg alignment. A more lateral relative position contributes to pronation and more medial position contributes to supination. The position of the tendon insertion can be altered during surgical procedures and thereby change the relative contribution of the gastrocsoleus unit on pronation or supination of the foot.

Because it crosses the knee joint, the gastrocnemius is also a contributor to knee flexion. The contribution of each muscle to plantarflexion power at the ankle has been studied. Silver and colleagues[17] suggested that the soleus is the main plantarflexor at the ankle during normal gait. They showed the soleus to exert nearly double the plantarflexion force at the ankle compared with the gastrocnemius.[17] The medial and lateral heads of the gastrocnemius have the same overall plantarflexion function but have different degrees of contribution, with the medial gastrocnemius head providing more than 70% of the muscle force. Force through the Achilles tendon during exercise can approach 12 times body weight, making the Achilles vulnerable to repetitive stress injury.[18] Late in the stance phase just before heel lift (60%–88%), the knee is in maximal extension and the ankle is in dorsiflexion.[19] This is the point at which the gastrocsoleus is subjected to the maximal stretching force, and therefore there is increased incidence of foot and ankle compensations causing potential symptoms and pathology.[20]

The size and unique structure of the Achilles tendon allow it to function under high loads. In the case of many of the acute and chronic clinical conditions that are discussed in this issue, it is these high forces coupled with structural abnormalities of the foot, ankle, and lower leg that are contributory factors in problems such as midsubstance tendinosis and degeneration. The stress-strain resistance properties of the Achilles are similar to all tendons, with physiologic collagen fiber stretching occurring from 2% to 4% stretch length, microscopic fibril failure at 6% to 8% stretch, and macroscopic failure beyond 8% stretch. Below the failure points of the stress-strain curve, the tendon fibers have the elastic capability to rebound and release energy that is valuable in function.[21] A variety of abnormalities, deformities, and activities can place loads on the tendon that are beyond its capacity to rebound and result in internal tendon fiber damage and degeneration. In contrast to injury, subrupture force application through the tendon is necessary for biochemical signaling of fibroblasts to produce collagen for normal tendon health and to heal injury.[22] This mechanical stress is a necessary component of prevention and recovery from injury and has been shown to increase recovered tendon strength. These principles highlight the importance of continued activity during recovery, with both tendon movement and controlled stress being necessary to promote proper healing. These principles will be applied in other sections in which the detriments of prolonged immobilization and the benefits of movement and controlled force are highlighted as necessary components of tendon recovery.

ACKNOWLEDGMENTS

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