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Anatomy, Bony Pelvis and Lower Limb, Leg Posterior Compartment

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Introduction

The lower leg divides into three fascial compartments:

- Anterior
- Lateral
- Posterior

These compartments are formed and separated via divisions by the anterior and posterior intermuscular septa, and the interosseous membrane.[1] Each compartment contains its distinct set of muscles, vasculature, and innervation:

- The anterior compartment musculature functions to primarily dorsiflex the foot and ankle
- The lateral compartment musculature functions to plantar flex and evert the foot
- The posterior compartment musculature functions to plantarflex and invert the foot

The posterior compartment of the leg (often referred to as the "calf") further divides into distinct superficial and deep compartments by the transverse intermuscular septum. The larger, superficial compartment of the lower leg contains the gastrocnemius, soleus (GS) and plantaris muscles.

The deep layer of the leg's posterior compartment contains the popliteus, flexor digitorum longus, flexor hallucis longus, and tibialis posterior muscles. The various muscles of the posterior compartment primarily originate at the two bones of the leg, the tibia, and the fibula. The tibia is a large weight-bearing bone, often referred to as the "shin bone," and articulates with the femoral condyles superiorly and the talus inferiorly.[2] The fibula articulates with the tibia laterally at proximal and distal ends; however, it has no involvement in weight bearing.[3]

Structure and Function

The divisions of the lower leg are made up by intermuscular septa that are extensions of the overlying fascia. Within the posterior compartment of the leg, an additional septum further separates the compartment into two additional layers; superficial and deep. It was traditionally felt that the fascia overlying specialized organs and tissues within the body are irrelevant and only served to hold in place a particular tissue type.[4][5] However, current thinking realizes that fascia, such as that of the posterior leg compartment, are not only involved in creating osteofascial compartments for muscles but also provide protective tunnels for neovascular bundles irrespective of limb positions, facilitate venous return, act as protective sheaths, dissipate external pressures, and are clinically significant in the spread or

containment of infections.[4]

The three muscles within the superficial posterior compartment include the gastrocnemius, soleus, and plantaris muscles. Together these three muscles form the triceps surae.[1] The gastrocnemius is involved in plantar flexion of the ankle, while the knee is in extension, and also is involved in flexing the leg at the knee joint. The soleus is involved in plantar flexion of the ankle, irrespective of knee position. The plantaris, a muscle that is thought to be absent in 10% of the population, is also involved in plantar flexion of the ankle but plays a limited compared to the other two superficial posterior muscles. The deep posterior compartment muscles include the flexor hallucis longus, flexor digitorum longus, tibialis posterior and popliteus muscles.[1] The flexor hallucis longus is primarily involved with flexing the big toe while also having limited contribution to plantar flexion of the ankle. Additionally, it supports the medial longitudinal arch of the foot. The flexor digitorum longus is involved in flexion of the other four toes of the foot, plantarflexion of the ankle as well, and also maintains the lateral and medial longitudinal arches of the foot. The tibialis posterior is primarily involved in ankle plantar flexion as well as inversion of the foot. The popliteus muscle controls weak flexion of the knee and medial rotation of the tibia. Independent activation, as well as coordination amongst the seven muscles of the posterior leg, is crucial in maintaining balance, facilitating gait, and allowing actions such as stair climbing, jumping, and landing.

Gait is most commonly subdivided into a stance phase and a swing phase (although additional phases can exist depending on clinical scenario).[6] The stance phase of gait can further divides into a heel strike (where initial contact is made with the ground), a loading response (where the weight of the body is accepted), a midstance (where the knee stabilzation occurs), and a terminal stance (where mass is accelerated forward). During midstance and terminal stance, in particular, the plantar flexors of the ankle play a pivotal role as they eccentrically control dorsiflexion, and concentrically allow acceleration of the foot.[7]

Embryology

Limb development in the human embryo originates from mesenchymal tissue, derived from the lateral plate of the mesoderm, encased within ectoderm.[8] Outgrowths of the lateral plate mesoderm, referred to as limb buds, arise in the middle of the 4th week in utero under the influence of signaling from mTORC1.[9] The exact morphogenesis of these limb buds is dependent upon several transcription factors, and each limb bud develops a positional value in respect to the proximo-distal, anteroposterior and dorsoventral axes of the fetus.[10] Dependent on its position, a slew of specific transcription factors may be expressed, including retinoic acid, fibroblast growth factor, sonic hedgehog, and hoxc6, allowing for the development of the particular upper or lower limb.[11]

Blood Supply and Lymphatics

The posterior compartment of the leg receives vascular supply from the posterior tibial artery.[12] This artery is a continuation of the popliteal artery, with the latter arising from the femoral artery. The posterior tibial artery supplies various muscles of the posterior compartment via its muscular branches and also supplies the tibial bone via the nutrient artery of the tibia. The posterior tibial artery provides a fibular artery as well.[13] The fibular artery not only provides blood supply via muscular branches to specific posterior compartment muscles, but it also provides the nutrient artery of the fibula. The posterior tibial artery continues towards the foot where it divides into the medial and lateral plantar arteries. The posterior tibial artery travels deep to the triceps surae muscle within the intermuscular septum and travels along with the tibial nerve.

Nerves

The tibial nerve provides innervation of the posterior leg compartment.[14] This nerve is the larger branch of the sciatic nerve which divides into the tibial nerve and common fibular (peroneal) nerve at the popliteal fossa. The tibial

nerve continues along the length of the leg and into its terminal branches known as the medial and lateral plantar nerves. Both the common fibular nerve and the tibial nerve innervate various muscles of the posterior leg. The tibial nerve provides the medial sural cutaneous nerve, which unites via the sural communicating branch from the common fibular nerve to create the sural nerve. The sural nerve supplies skin overlying the lateral and posterior leg.

Muscles

Muscles of the posterior compartment are in two subdivisions: a superficial and deep layer.[1]

Superficial Posterior Muscles:

The gastrocnemius muscle is made up of separate lateral and medial heads.[1][15] The medial is the larger of the two. The medial head originates at the femur and the medial femoral condyle whereas the lateral head originates at the femur and lateral femoral condyle. The gastrocnemius directly overlies the soleus muscle, and together these two muscles blend into a calcaneal (Achilles) tendon (AT), which inserts into the tuberosity of the calcaneus. Between the AT and the overlying superficial fascia is a sub-calcaneal bursa that allows for frictionless motion between skin and tendon.

A deep calcaneal bursa between the tendon and bone facilitates frictionless gliding of the tendon over the bone. The soleus muscle originates on the posterior surface of the fibula as well as the soleal line on the tibia. As a unit, the gastrocnemius and soleus muscles form the triceps surae.[1]

Deep to the triceps surae lies the plantaris muscle. This muscle originates on the lateral supracondylar line of the femur and inserts onto the tuberosity of the calcaneus adjacent to the AT. The plantaris muscle is commonly a source for grafting material. The triceps surae muscles provide for plantar flexion of the foot. However, the gastrocnemius muscle itself can also facilitate flexion of the knee. The plantaris muscle allows minor plantar flexion as well.[1]

Deep Posterior Muscle

The most superior muscle of the deep posterior compartment of the lower leg is the popliteus muscle.[16][17] The popliteus has its origin at the lateral condyle of the femur and inserts onto the posterior tibial surface superior to the soleus muscle. The popliteus muscle facilities unlocking of the knee joint via internal rotation.[18][17]

The flexor hallucis longus (FHL) originates from the posterior surface of the fibula and inserts into the base of the great toe's distal phalanx. The FHL tendon travels below the sustentaculum tali and is held in place by the annular ligament. The FHL muscle plantarflexes the talocrural joint and the metatarsophalangeal and interphalangeal joints of the great toe.

The flexor digitorum longus (FDL) muscles originate from the posterior surface of the tibia and inserts onto the distal phalanges of the 2nd to 5ith digits in the foot. The FDL muscle crosses both the talocrural joint and metatarsophalangeal joints, serving as an active plantar flexor of both joints. The FDL also plantarflexes the metatarsophalangeal and interphalangeal joints of the 2nd through 5th toes.

An accessory FDL muscle is said to be present in 15% of the population.[19] Finally, the tibialis posterior muscles originate on the interosseous membrane and inserts onto the navicular tuberosity and cuneiform bones of the foot. Its tendon travels through the tarsal tunnel. The tibialis posterior muscle provides for plantar flexion across the talocrural join and inversion of the subtalar joint.

Surgical Considerations

Acute compartment syndrome (ACS)

Compartment syndrome develops when pressures within a closed compartment (i.e., the lower leg) exceeds the perfusion pressure of the vessel, leading to tissue ischemia, nerve compression, and subsequent pain out of proportion on exam.[20][21] The syndrome falls into classifications of acute or chronic, with the former more associated with trauma, and the latter more associated with athletic exertion.[22]

ACS commonly presents in patients sustaining blunt trauma from motor vehicle accidents. Young patients suffering from tibial fractures of the diaphysis are at higher risk for developing compartment syndrome and must be monitored accordingly.[23]

Four compartment fasciotomy

Current management of compartment syndrome involves releasing all four lower leg compartments.[24] Common complications of the procedure include:

- neurovascular injury
- soft tissue injury

Pediatric considerations

Tibial fractures are among the most common fractures sustained by the pediatric population.[25] Delayed diagnosis of tibial fractures, especially secondary to high energy trauma, can include collateral vascular lesions, neurovascular compromise, and the development of ACS.[26] Typical management utilizes closed reduction and casting; however surgical intervention may include external fixation, intramedullary nailing, and open reduction internal fixation (ORIF) with plates/screws.

Indications for lower extremity amputations include the presence of lower extremity ischemia from arterial occlusion (including critical limb ischemia), distal extremity infection, or presence of a malignant tumor.[27][28] Surgical techniques for below the knee amputations (BKA) can be utilized, with the posterior flap being most common. Complications may arise postoperatively, with infections rates varying secondary to various factors such as the use of skin clips over sutures, and use of suction drains.[29]

Achilles tendon rupture

The Achilles tendon is the strongest tendon, and susceptible to injury and rupture.[30][31] Achilles tendon ruptures commonly occur in athletes (e.g., sprinters), but there is a well-recognized predilection for the recreational athlete (the so-called "weekend warrior"). The latter occurs secondary to acute paroxysmal stress sustained by an already weakened tendon after even mild degrees of tendinopathy. Degenerative Achilles tendinopathy can occur over time and presents clinically as recurrent pain and swelling over the Achilles tendon insertion, or at various levels proximal to its insertion.[32]

Treatment

Nonoperative management options include[31]

- Rest/activity modification
- NSAIDs
- Physical therapy (e.g., eccentric exercises)

Though tendinopathy management is generally with nonoperative treatment modalities, acute AT ruptures may require surgical intervention. A common repair technique for tears located near the musculotendinous junction

includes a direct end-to-end repair (open versus percutaneous techniques). Direct repairs are most amenable to healing within six weeks out from the injury date. The data remains controversial regarding the outcome following surgery compared to nonoperative management (i.e., functional bracing/rehab protocols).

Gastrocnemius tears

Tearing of the gastrocnemius muscle is a common injury in patients over the age of 40 years, and is often referred to as tennis leg, because of its prevalence within the sport.[33] Its most common presentation is pain localized to the posterior leg, accompanied by a palpable defect on the belly of the gastrocnemius. This is accompanied by loss of strength on plantar flexion and decreased range of motion. Typically, the medial head of the gastrocnemius is involved, however, an injury to the plantaris tendon or soleus tendon may also present similarly. Diagnosis is typically made clinically; however, ultrasound may also be useful in assessing the injury and differentiating it from other pathology. Conservative treatment includes the limitation of activity, crutch walking, rest, ice, compression, and elevation. However, medical and surgical management may be necessary in cases of a hematoma or acute compartment syndrome.[33]

Clinical Significance

Accessory muscles of the posterior leg compartment may be present in patients and lead to potential sequelae. Reported accessory muscles have included an additional muscular tissue connecting the lower flexor hallucis longus and the tibialis posterior muscle. There are cases where this muscle crosses the posterior tibial artery and may be a source of entrapment. Additional accessory muscles have been said to originate in the interosseous membrane, near the tibia, and insert into connective tissue surrounding the posterior tibial vessels. These situations also serve as a potential source of compression for the vasculature.[34]

Aside from anatomical compressions of neurovascular bundles, injury may also take place iatrogenically. For example, fibular graft harvests, tibial osteotomies, and fasciotomy are all procedures in which there is an increased risk of tibial nerve injury. Knowledge of the branching points of the tibial nerve into the specific muscles of the posterior leg is essential to avoid such injuries and subsequent motor loss.[35]

Focal myositis is a rare inflammatory pseudotumor that may present in the posterior leg compartment. Diagnosis is difficult as clinicians may often misinterpret the tumor for other neoplastic processes and inflammatory myopathies. However specific histopathological changes that should be noted include B cell and dendritic plasmacytoid cell lesions, without any viral or molecular infectious source. Additionally, the presence of IgG4 and fibrosis has also been associated with focal myositis.[36][37]

Physiologically, the pressure of surface veins in the leg decreases when a person is ambulatory.[38] This pressure decrease occurs because the muscles of the posterior leg compartment can compress deeper veins via contraction. When said muscles relax, these deep veins will increase the amount of blood they accept from the surface veins, thus decreasing pressure in the surface veins. However, patients may have venous abnormalities in which the surface vein pressures do not adequately decrease, causing ambulatory venous hypertension. Such insufficiencies arise from destruction, incompetence, or embolization of the veins, which in turn leads to reflux that leads to inadequate reduction in venous pressure. This venous hypertension, in turn, leads to hyperpigmentation, fibrosis, and ulceration in the leg. It is hypothesized that there is a deficiency in the nutrition and oxygenation sent to the cells which affect their functionality. Treatment of chronic venous insufficiency varies from compression therapy to sclerotherapy or radiofrequency ablation.[38]

Restless leg syndrome is characterized by spontaneous, nocturnal leg movements and has a prevalence of about 2-9% in elderly patients.[39] The sensorimotor activation of the leg occurs at rest and most commonly at night, leading to severe sleep disturbance.[40] The condition most commonly affects the calf muscles. However, the genetic basis and

pathophysiology are not well known. Patients with iron deficiency may be at increased risk for restless legs syndrome.[41] Typical management includes the use of dopamine agonists.

Leg cramps are characterized by involuntarily, episodic, skeletal muscle contractions of the calf muscles, associated with pain, and typically occurring at night.[42] Though the etiology of leg cramps is not well understood, associations exist with electrolyte imbalances, renal disease, polyneuropathy's, myopathies, and administration of certain drugs. Additionally, trauma and deep venous thrombosis also have associations.[43] A third of the general population over the age of 50 is affected by nocturnal leg cramps, with the incidence increasing with age. Like restless leg syndrome, leg cramps may lead to sleep disturbances and subsequent sequelae. Quinine was historically prescribed for control of nocturnal leg cramps, but it has fallen out of favor after an FDA-issued warning regarding potentially dangerous side effects.[44]

Questions

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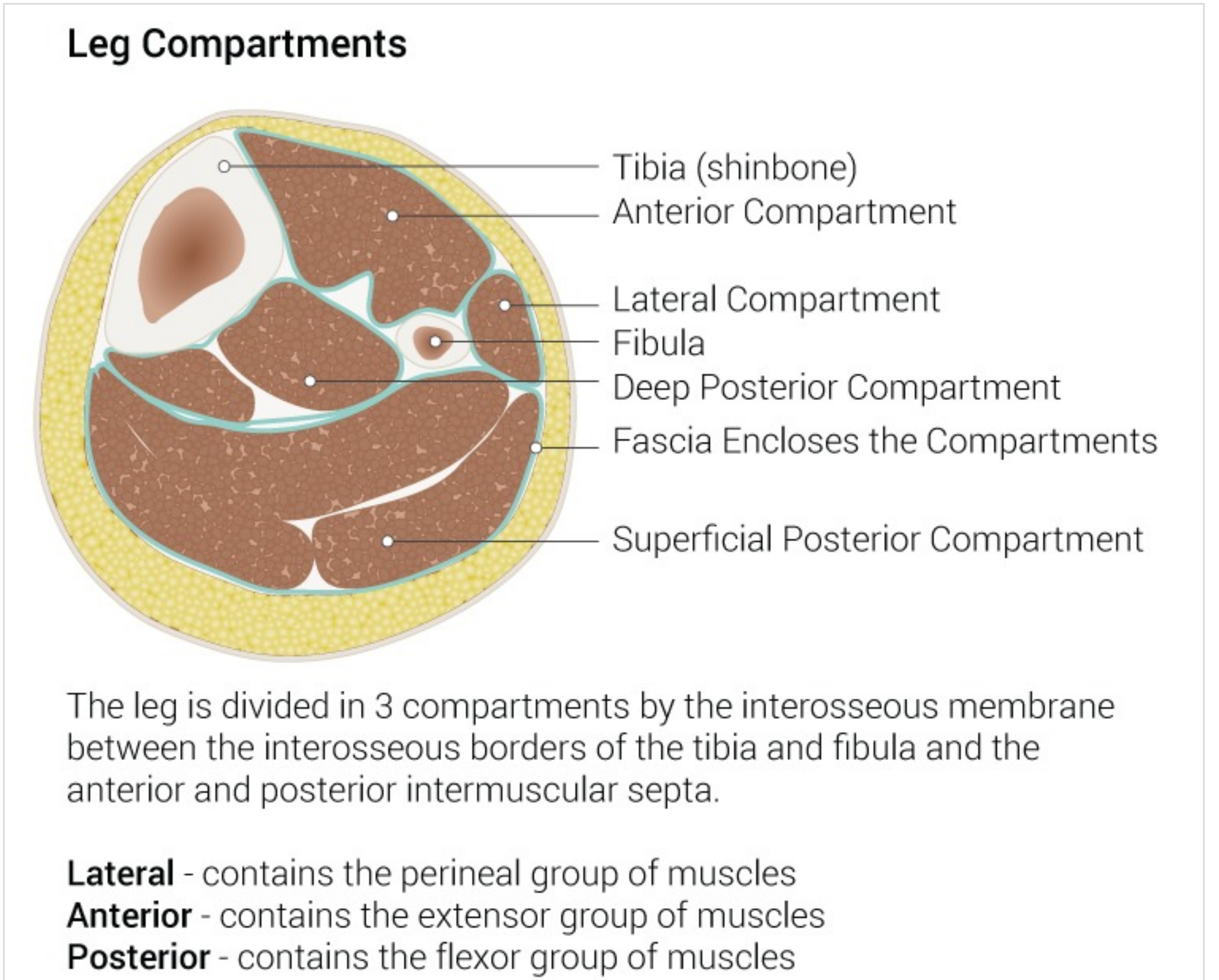
References

1. Binstead JT, Varacallo M. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Nov 13, 2018. Anatomy, Bony Pelvis and Lower Limb, Calf. [PubMed: 29083629]
2. Bourne M, Murphy PB. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Dec 9, 2018. Anatomy, Bony Pelvis and Lower Limb, Tibia. [PubMed: 30252309]
3. Gupton M, Kang M. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Dec 6, 2018. Anatomy, Bony Pelvis and Lower Limb, Fibula. [PubMed: 29261984]
4. Benjamin M. The fascia of the limbs and back--a review. J. Anat. 2009 Jan;214(1):1-18. [PMC free article: PMC2667913] [PubMed: 19166469]
5. Taborri J, Palermo E, Rossi S, Cappa P. Gait Partitioning Methods: A Systematic Review. Sensors (Basel). 2016 Jan 06;16(1) [PMC free article: PMC4732099] [PubMed: 26751449]
6. Tao W, Liu T, Zheng R, Feng H. Gait analysis using wearable sensors. Sensors (Basel). 2012;12(2):2255-83. [PMC free article: PMC3304165] [PubMed: 22438763]
7. Tickle C. How the embryo makes a limb: determination, polarity and identity. J. Anat. 2015 Oct;227(4):418-30. [PMC free article: PMC4580101] [PubMed: 26249743]
8. Jiang M, Fu X, Yang H, Long F, Chen J. mTORC1 Signaling Promotes Limb Bud Cell Growth and Chondrogenesis. J. Cell. Biochem. 2017 Apr;118(4):748-753. [PMC free article: PMC5296292] [PubMed: 27606668]
9. Wolpert L. Vertebrate limb development and malformations. Pediatr. Res. 1999 Sep;46(3):247-54. [PubMed: 10473037]
10. Cunningham TJ, Duester G. Mechanisms of retinoic acid signalling and its roles in organ and limb development. Nat. Rev. Mol. Cell Biol. 2015 Feb;16(2):110-23. [PMC free article: PMC4636111] [PubMed: 25560970]
11. Chmielewski P, Warchoł Ł, Gala-Błądzińska A, Mróz I, Walocha J, Malczak M, Jaworek J, Mizia E, Walocha E, Depukat P, Bachul P, Bereza T, Kurzydło W, Gach-Kuniewicz B, Mazur M, Tomaszewski K. Blood vessels of the shin - posterior tibial artery - anatomy - own studies and review of the literature. Folia Med Cracov. 2016;56(3):5-9. [PubMed: 28275266]
12. Tanaka K, Matsumura H, Miyaki T, Watanabe K. An anatomic study of the intermuscular septum of the lower leg; branches from the posterior tibial artery and potential for reconstruction of the lower leg and the heel. J Plast Reconstr Aesthet Surg. 2006;59(8):835-8. [PubMed: 16876081]
13. Vloka JD, Hadzić A, April E, Thys DM. The division of the sciatic nerve in the popliteal fossa: anatomical implications for popliteal nerve blockade. Anesth. Analg. 2001 Jan;92(1):215-7. [PubMed: 11133630]

14. Nyland J, Lachman N, Kocabey Y, Brosky J, Altun R, Caborn D. Anatomy, function, and rehabilitation of the popliteus musculotendinous complex. J Orthop Sports Phys Ther. 2005 Mar;35(3):165-79. [PubMed: 15839310]
15. Holzmann M, Almudallal N, Rohlck K, Singh R, Lee S, Fredieu J. Identification of a flexor digitorum accessorius longus muscle with unique distal attachments. Foot (Edinb). 2009 Dec;19(4):224-6. [PubMed: 20307482]
16. Barnes M. Diagnosis and management of chronic compartment syndromes: a review of the literature. Br J Sports Med. 1997 Mar;31(1):21-7. [PMC free article: PMC1332468] [PubMed: 9132204]
17. Patel RV, Haddad FS. Compartment syndromes. Br J Hosp Med (Lond). 2005 Oct;66(10):583-6. [PubMed: 16255266]
18. Park S, Ahn J, Gee AO, Kuntz AF, Esterhai JL. Compartment syndrome in tibial fractures. J Orthop Trauma. 2009 Aug;23(7):514-8. [PubMed: 19633461]
19. Apaydin N, Basarir K, Loukas M, Tubbs RS, Uz A, Kinik H. Compartmental anatomy of the superficial fibular nerve with an emphasis on fascial release operations of the leg. Surg Radiol Anat. 2008 Feb;30(1):47-52. [PubMed: 18071623]
20. Mashru RP, Herman MJ, Pizzutillo PD. Tibial shaft fractures in children and adolescents. J Am Acad Orthop Surg. 2005 Sep;13(5):345-52. [PubMed: 16148360]
21. Wozasek GE, Moser KD, Haller H, Capousek M. Trauma involving the proximal tibial epiphysis. Arch Orthop Trauma Surg. 1991;110(6):301-6. [PubMed: 1747311]
22. Desai Y, Robbs JV, Keenan JP. Staged below-knee amputations for septic peripheral lesions due to ischaemia. Br J Surg. 1986 May;73(5):392-4. [PubMed: 3708295]
23. McIntyre KE, Bailey SA, Malone JM, Goldstone J. Guillotine amputation in the treatment of nonsalvageable lower-extremity infections. Arch Surg. 1984 Apr;119(4):450-3. [PubMed: 6703902]
24. Coulston JE, Tuff V, Twine CP, Chester JF, Eyers PS, Stewart AH. Surgical factors in the prevention of infection following major lower limb amputation. Eur J Vasc Endovasc Surg. 2012 May;43(5):556-60. [PubMed: 22342693]
25. Järvinen TA, Kannus P, Paavola M, Järvinen TL, Józsa L, Järvinen M. Achilles tendon injuries. Curr Opin Rheumatol. 2001 Mar;13(2):150-5. [PubMed: 11224740]
26. Alfredson H, Lorentzon R. Chronic Achilles tendinosis: recommendations for treatment and prevention. Sports Med. 2000 Feb;29(2):135-46. [PubMed: 10701715]
27. Nsitem V. Diagnosis and rehabilitation of gastrocnemius muscle tear: a case report. J Can Chiropr Assoc. 2013 Dec;57(4):327-33. [PMC free article: PMC3845475] [PubMed: 24302780]
28. Nayak SB, Shetty SD. Two accessory muscles of leg: potential source of entrapment of posterior tibial vessels. Surg Radiol Anat. 2019 Jan;41(1):97-99. [PubMed: 30353417]
29. Apaydin N, Loukas M, Kendir S, Tubbs RS, Jordan R, Tekdemir I, Elhan A. The precise localization of distal motor branches of the tibial nerve in the deep posterior compartment of the leg. Surg Radiol Anat. 2008 Jun;30(4):291-5. [PubMed: 18283390]
30. Auerbach A, Fanburg-Smith JC, Wang G, Rushing EJ. Focal myositis: a clinicopathologic study of 115 cases of an intramuscular mass-like reactive process. Am. J. Surg. Pathol. 2009 Jul;33(7):1016-24. [PubMed: 19363438]
31. Stacey M. Chronic Venous Insufficiency and Leg Ulceration: Principles and Vascular Biology. In: Fitridge R, Thompson M, editors. Mechanisms of Vascular Disease: A Reference Book for Vascular Specialists [Internet]. University of Adelaide Press; Adelaide (AU): 2011. [PubMed: 30485035]
32. Trenkwalder C, Wetter TC, Stiasny K, Clarenbach P. [Restless legs syndrome and periodic limb movements in sleep]. Nervenarzt. 2001 Jun;72(6):425-36. [PubMed: 11433701]
33. Stiasny K, Oertel WH, Trenkwalder C. Clinical symptomatology and treatment of restless legs syndrome and periodic limb movement disorder. Sleep Med Rev. 2002 Aug;6(4):253-65. [PubMed: 12531131]
34. Yeh P, Walters AS, Tsuang JW. Restless legs syndrome: a comprehensive overview on its epidemiology, risk

factors, and treatment. Sleep Breath. 2012 Dec;16(4):987-1007. [PubMed: 22038683]
35. Hallegraeff J, de Greef M, Krijnen W, van der Schans C. Criteria in diagnosing nocturnal leg cramps: a systematic review. BMC Fam Pract. 2017 Feb 28;18(1):29. [PMC free article: PMC5330021] [PubMed: 28241802]
36. Young G. Leg cramps. BMJ Clin Evid. 2009 Mar 26;2009 [PMC free article: PMC2907778] [PubMed: 19445755]
37. Diener HC, Dethlefsen U, Dethlefsen-Gruber S, Verbeek P. Effectiveness of quinine in treating muscle cramps: a double-blind, placebo-controlled, parallel-group, multicentre trial. Int. J. Clin. Pract. 2002 May;56(4):243-6. [PubMed: 12074203]
38. Bordoni B, Varacallo M. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Dec 13, 2018. Anatomy, Fascia. [PubMed: 29630284]
39. Bordoni B, Varacallo M. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Nov 14, 2018. Anatomy, Bony Pelvis and Lower Limb, Gastrocnemius Muscle. [PubMed: 30422541]
40. Hyland S, Varacallo M. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Nov 14, 2018. Anatomy, Bony Pelvis and Lower Limb, Popliteal Region. [PubMed: 30422486]
41. Hyland S, Varacallo M. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Nov 14, 2018. Anatomy, Bony Pelvis and Lower Limb, Popliteus Muscle. [PubMed: 30252340]
42. Varacallo M, Shirey L, Kavuri V, Harding S. Acute compartment syndrome of the hand secondary to propofol extravasation. J Clin Anesth. 2018 Jun;47:1-2. [PubMed: 29476968]
43. Gossman WG, Varacallo M. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Nov 14, 2018. Achilles Tendon Rupture. [PubMed: 28613594]
44. Bordoni B, Varacallo M. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Dec 13, 2018. Myofascial Pain. [PubMed: 30570965]

Figures



Leg Compartments, Tibia (shinbone), Anterior Compartment, Lateral Compartment, Fibula, Deep Posterior Compartment, Fascia Encloses the Compartments, Superficial Posterior Compartment. Contributed Illustration by Beckie Palmer

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