Passive Muscular Insufficiency

The Etiology of Gastrocnemius Equinus

Jarrod Shapiro, DPM[a,*], Benjamin Kamel, DPM[b]

KEYWORDS

• Gastrocnemius equinus • Lower extremity biomechanics • Silfverskiöld
• Passive insufficiency

KEY POINTS

- The test for ankle range of motion originally described by Silfversköild has been incorrectly adopted for use on patients without neuromuscular diseases.
- Passive insufficiency, as in the gastrocnemius muscle, occurs in muscle/tendon units that pass across multiple joints, leading to a decrease in available range of motion of the distal joint when the proximal joint is extended.
- The unloaded, unstable foot is acted on by the normal gastrocnemius muscle complex, creating lower extremity dysfunction.

INTRODUCTION

Ankle equinus is a condition often discussed in the foot and ankle literature, and is now a term increasingly applied to various pathologic entities. Originally discussed in the orthopedic literature in relation to diseases involving muscular spasticity, most commonly cerebral palsy,[1] the concept of ankle equinus and its various treatments is now commonly applied to patients with complications related to diabetic peripheral neuropathy, pathologic flatfoot conditions, and other nonspastic lower extremity conditions.

The current opinion within the orthopedic and podiatric literature proposes that ankle equinus arises from a limitation of ankle joint dorsiflexion due to a pathologic shortening of the muscle fibers of the gastrocnemius and/or gastrocnemius-soleus muscle complex.[2] This shortening is thought to create or exacerbate pathology of the lower extremity in various ways, including increasing plantar forefoot pressures

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[a] PMSR/RRA Podiatric Residency, Western University College of Podiatric Medicine, Chino Valley Medical Center, 309 East Second Street, Pomona, CA 91766, USA; [b] PMSR/RRA Podiatric Residency, Chino Valley Medical Center, 5451 Walnut Avenue, Chino, CA 91710, USA
* Corresponding author.
E-mail address: jshapiro@westernu.edu

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leading to diabetic neuropathic ulceration[3,4] and excessive midfoot compensation in various pathologic entities.

However, an alternative evaluation of the medical literature may lead one to a different explanation of muscular ankle equinus and its role in pathology of the lower extremity. The purpose of this article is to present an alternative hypothesis of gastrocnemius equinus: diminished ankle joint dorsiflexion is a physiologic process resulting from normal functional anatomy that applies force to an already pathologic foot.

HISTORICAL BACKGROUND AND A NEW HYPOTHESIS

Early medical literature focused on ankle equinus in relation to spastic muscular disorders, especially in relation to cerebral palsy.[5] In 1923, Nils Silfverskiöld presented what became the foundation for the examination technique currently used to diagnose ankle equinus.[1] In 1971, Root and colleagues[6] defined normal ankle joint range of motion as greater than 10° dorsiflexion of the foot on the leg with the subtalar joint held in neutral position with the knee in an extended position. The investigators explained that when the foot is about to enter the toe-off phase of gait, the leg is positioned 80° to the foot, requiring 10° of dorsiflexion for appropriate gait immediately following propulsion.[5] Since then, multiple investigators proposed various definitions as well as measurement techniques discussed in detail elsewhere.[7–20]

The original literature about spastic calf muscle contracture has been extrapolated to the nonspastic patient population with the assumption that tightness of the posterior calf musculature is abnormal. It is important to note that the Silfverskiöld test, currently used for patients with nonspastic disorders, was not described for this patient population. Rather, Silfverskiöld[1] discussed this test in relation to patients with neurologic conditions and subsequent hyperactive muscle contractures.

Silfverskiöld[1] discussed the concept of "many-joints muscles," describing muscle-tendon units that pass across more than one joint. Many-joint muscles are common in the human, for example, the rectus femorus (crosses the hip and knee). The gastrocnemius muscle is also a many-joint muscle, originating on the medial condyle and lateral epicondyle of the femur and inserting on the posterior calcaneal surface, thus crossing the knee, ankle, and subtalar joints. This anatomic configuration creates the 3 joint-crossing gastrocnemius muscle.

Active and passive tension activates muscles to create forces by pulling equally on their attachments. Active tension refers to the force created in the sarcomere of activated motor units using energy stored in ATP.[21] Muscles have the unique ability to provide active forces, unlike ligaments and tendons. The shape of the active tension potential in muscle is the force-velocity relationship of muscle.[21]

When components of the muscle-tendon unit are stretched, they create a passive tension force. During a passive gastrocnemius wall stretch, the internal resistance of the Achilles tendon that prevents full range of motion is attributed to passive tension. This passive tension can be quite large and may be responsible for the muscular weakness seen in muscles following stretching.[22] Limitation of any joint range of motion is largely attributed to the passive tension created by the tendon that attaches to it. When examining passive tension on a multiarticular level, passive tension is magnified due to passive insufficiency. Elasticity also exists in the production of active muscle tension, which is likely a mixture of actin/myosin filaments, sarcomere nonuniformity, and bridge stiffness.[23–26]

The functional significance of the multijoint-crossing gastrocnemius is profound. Silfverskiöld[1] described a *transmission effect* caused by this muscular formation. When one joint in this complex moves it causes associated movement of the

subsequent distal joint(s). This effect is exemplified by a runner jumping hurdles. While in mid-leap, the runner fully flexes the hip, placing the hamstring muscles on full stretch, reaching the limit of muscular length. This maximally stretched position of the hamstrings thus limits the full extension capability of the knee. Silfverskiöld[1] termed this phenomenon *passive insufficiency*. If the hip was then extended (relaxed) the hamstring muscles would now have a greater potential length, and the knee would be able to fully extend. This effect occurs throughout the body and is described by Silfverskiöld[1] as normal. In fact, it is a beneficial mechanism that increases muscular strength and decreases energy expenditure.[1]

This same transmission effect may also be applied to the gastrocnemius muscle. As noted previously, this muscle passes across multiple joints, including the knee and ankle. If one flexes the knee, the foot may be dorsiflexed on the ankle far beyond the previously stated 10° past neutral, noted as the minimum range of motion necessary for nonpathologic gait.[2,6] However, if one fully extends the knee, then the foot may be dorsiflexed on the ankle to approximately 90° and no farther (**Fig. 1**). Due to the transmission effect and passive insufficiency, the gastrocnemius muscle has reached its maximal stretch, and an apparent lack of dorsiflexion is noted. Again, this is described by Silfverskiöld[1] as being a normal physiologic process. When this same limitation occurs during gait (caused instead by an antagonist muscle actively contracting, thereby limiting joint range of motion) it is termed *active insufficiency*.

Silfverskiöld[1] described his examination technique for determining treatment of patients with spastic forms of equinus. His test is described as follows[1]:

> *I have tested this partly by observing the walk and partly by the following method: Passive and active dorsal flexion of the foot, with bent and with stretched knee. The degree of spasticity and passive insufficiency can be measured by the strength that is needed to produce passiv [sic] dorsal flexion of the foot with the knee bent or stretched respectively. The greater the difference in strength with bent or with stretched knee, the greater the spasticity of the gastrocnemius muscle and the passive insufficiency respectively.*

Silfverskiöld[1] hypothesized that the same passive insufficiency present in the healthy human would be enhanced in patients with spastic neurologic diseases. As such, his test was simply examining the amount of spasticity rather than a pathologic contracture of the triceps surae muscle group. This test also helped him to evaluate the hypertonicity of the soleus muscle (assumed to be present if the contracture was evident with the knee both flexed and extended).

This prior research generates the foundation of a novel hypothesis: what is measured as gastrocnemius equinus is actually anatomically normal passive gastrocnemius insufficiency. Presence of “pathologic” equinus in the non-neurologically impaired patient is

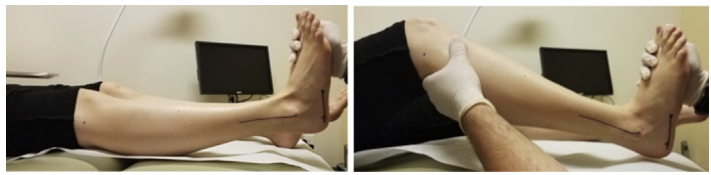

Fig. 1. The Silfverskiöld test as is commonly performed in contemporary clinical practice on this asymptomatic, healthy patient. Left image demonstrates 90° of ankle dorsiflexion with the knee extended. Right image shows the greatly improved ankle dorsiflexion with the knee flexed. Flexing the knee eliminates the transmission effect, creating greater available muscle/tendon unit length.

an erroneous diagnosis, and the Silfverskiöld[1] maneuver that is commonly performed to test for the presence of gastrocnemius versus gastrocnemius-soleus equinus is testing normal lower extremity anatomy rather than a pathologic state.

This test has been inappropriately applied to the nonspastic population in which normal passive gastrocnemius insufficiency is present. As a result, passive gastrocnemius insufficiency is incorrectly considered to be pathologic contracture. The Silfverskiöld test may be, under this new paradigm, a less essential biomechanical examination technique with decreased utility in the neurologically normal population.

RELATIONSHIP OF PASSIVE INSUFFICIENCY WITH FOOT STRUCTURE

Under optimal conditions, the foot acts through a specific timed firing of musculature, with supportive passive structures (ligaments and aponeuroses), to create a relatively "locked" foot position in late midstance.[2,27–30] The nonpathologic foot assumes this locked position, providing a mechanically stable lever arm over which the ankle dorsiflexes during midstance. The triceps surae complex fires at terminal stance, thereby plantarflexing the foot on the ankle as one unit with pedal stability maintained.

To summarize, the normal action of the triceps surae is predicated on a "preloaded" foot, which has an appropriately supinated subtalar position, creating a stable foot position in which to plantarflex during the push-off period of the stance phase of gait.

The foot may become preloaded through a variety of proposed mechanisms, including, but likely not limited to the following:

- Subtalar supination leading to a "locked" midtarsal position.[2,31,32]
- Close packed position of the calcaneocuboid joint leading to an advantageous position of the peroneus longus and plantarflexion of the first metatarsal with subsequent retrograde plantarflexion of the medial column (close packed medial column position) and stable medial longitudinal arch.[28,33]
- Tibialis posterior muscle concentric action on the midtarsal and subtalar joints.[2]
- The windlass mechanism provides passive stability at terminal stance and toe off.[34]
- Bony architecture, with the medial arch keystone effect, provides passive medial longitudinal arch stability.[34,35]

If these preloading mechanisms fail, then the foot remains in a loose "unloaded" position with loss of mechanical stability. When normal ankle dorsiflexion occurs at midstance (in the presence of normal anatomic passive gastrocnemius insufficiency) the foot is unprepared to accept the forces of the triceps surae proximally and ground reactive forces plantarly. The subtalar joint will increasingly pronate as compensation leading to the typical flatfoot appearance. At toe off, when the ankle is plantarflexing and triceps surae actively firing, the unstable condition is further worsened with midfoot dorsiflexion as compensation for the lack of ankle dorsiflexion.

Various mechanisms have been proposed to explain the causes of an unstable foot, such as the subtalar joint neutral theory proposed by Root and colleagues[2] and rotational equilibrium theory of Kirby.[36] Passive gastrocnemius insufficiency may be incorporated into this system as well. In the pathologic foot, if the tibialis posterior and peroneus longus contract too late or inefficiently due to pathologic events described previously, then unstable structures are overpowered by an otherwise anatomically normal triceps surae complex.

The phenomenon of "early heel off" in the pathologically pronated foot also may be explained through the lens of passive insufficiency. Early heel off is seen clinically during the midstance phase of gait and may occur due to a normal passive gastrocnemius

insufficiency acting on a pathologically non-preloaded foot that dorsiflexes at the midfoot (Fig. 2, right). The same is true for early heel off in the pes cavus type foot except the foot is appropriately preloaded and the entire foot elevates off the ground early as a result of passive gastrocnemius insufficiency (see Fig. 2, left).

RELATIONSHIP OF CURRENT RESEARCH TO PASSIVE INSUFFICIENCY

All current research literature has assumed pathologic gastrocnemius equinus, rather than normal passive insufficiency. This factor may be part of the explanation for the controversy behind the variable measuring techniques as well as the definition of ankle equinus itself.[14–19]

Hill[37] prospectively examined 176 of 209 consecutive new patients presenting to a foot and ankle clinic over a 6-week period. Of the 176 patients with a biomechanically related complaint, 96.5% also had equinus defined as less than 3° ankle joint dorsiflexion using a tractograph. Hill[37] concluded the high incidence of equinus reflected an "acquired deformity." It is difficult to extrapolate these numbers to the healthy population because gathering data from a clinic leads to selection bias in favor of symptomatic patients. However, despite this, the high incidence may be explained by the normal presence of passive gastrocnemius insufficiency rather than an acquired deformity.

Saxena and Kim[38] in 2003 looked at the prevalence of ankle equinus in a healthy adolescent athlete population. Using a hand-held goniometer with patients supine and foot in neutral position, they examined 40 healthy high school athletes, measuring ankle joint dorsiflexion with the knee both extended and flexed. Mean ankle dorsiflexion in this asymptomatic young, athletic population was found to be zero degrees with the knee extended and 5° with the knee flexed. The investigators explained their method of measurement and small cohort as limiting factors with increased potential for variability. They hypothesized, "Adolescent athletes have a component of gastrocnemius equinus." These findings in a young, healthy, asymptomatic population may be more adequately explained by the presence of anatomic passive gastrocnemius insufficiency rather than a pathologic process.

DiGiovanni and colleagues[32] compared ankle joint range of motion in 2 groups of patients. Group 1 consisted of 34 patients with metatarsalgia and related forefoot

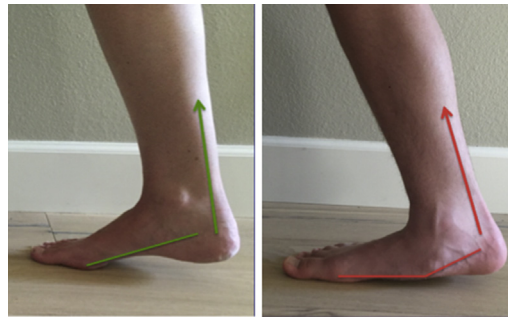

Fig. 2. In the left image, the Achilles transmits its force to an intact medial longitudinal arch via a long lever arm with normal heel elevation. In the right image, a pathologic medial longitudinal arch dorsiflexes during push off, creating a short lever arm on which the Achilles tendon may act. In both cases, the gastrocnemius-soleus complex is normal but acting on a pathologic foot in the right image.

and midfoot symptoms, whereas the control group consisted of 34 patients without foot or ankle pain. These investigators measured ankle range of motion using an equinometer (goniometer linked to a pressure sensor to standardize the amount of pressure when dorsiflexing the foot with computerized angle determination). They found a significant difference in the amount of ankle dorsiflexion between groups. However, in both groups, a difference was noted in the amount of ankle dorsiflexion with the knee extended versus flexed. These results may, again, be explained by gastrocnemius passive insufficiency. However, potential confounding issues are demonstrated with this study due to methodological limitations. The experimental and control groups were heterogeneous. Five patients in the experimental group had diabetes, whereas none in the control group did. This may have led to a selection bias toward decreased ankle range of motion because diabetic individuals have been shown to have glycosylation of tendons.[39,40] Seven patients in the experimental group were smokers versus 3 in the control group. It is unknown what specific effect smoking has on tendon length and function, although it is considered detrimental to appropriate tendon function.[41] In addition, 5 members of the control group had ligamentous laxity whereas only 1 in the experimental group did, pointing again to heterogeneity of sample groups. Based on the study's limitations, the investigators' conclusions may be inaccurate.

Lavery and colleagues[3] examined 1666 diabetic patients in a prospective observational cohort study. They found an ankle equinus (defined as $<0^{\circ}$ dorsiflexion at the ankle) prevalence of 10.3% in this cohort, which is significantly lower than prior studies. This may be because of their definition of equinus, which would have underestimated limitation of ankle range of motion. In addition, the method of examination was not stated, making this study difficult to compare with prior research.

Similarly, Frykberg and colleagues[42] in 2012, while prospectively comparing equinus in 43 diabetic versus 59 nondiabetic patients found a 24.5% prevalence (odds ratio 3.3). These researchers used a biplane goniometer to measure ankle joint range of motion with a definition identical to Lavery and colleagues.[3] Similarly, the definition of equinus may have affected the observed prevalence.

Surgical biomechanical research additionally lends support to this concept. In 2009, Arangio and Salathe[43] compared the force of a 10-mm medial displacement calcaneal osteotomy on the medial column ligaments of an experimental cadaver flatfoot with intact foot as a control. The calcaneal osteotomy decreased first metatarsal force, decreased the talonavicular joint moment, and increased fifth metatarsal and calcaneocuboid joint loads.[43] The gastrocnemius-soleus complex was not modified during the study and had no effect on the outcome. Creating an abnormally functioning medial column was the primary factor in this experimental model. Similar outcomes were found with the use of subtalar arthroeiresis[44] and Evans osteotomy.[45] Nyska and colleagues[46] further found that increasing the pull of the Achilles tendon in a cadaver model increased the flatfoot deformity, but only in the feet that were made unstable by sectioning the ligaments of the medial column. An artificially tightened Achilles tendon only had an effect on the experimentally unstable foot.

FUTURE DIRECTIONS

This model calls into question the modern concept of muscular ankle equinus. Understanding correct physiologic function is a prerequisite to advancing medical science and patient care, and future research may demonstrate modifications to nonsurgical and surgical methodology.

Contemporary applications of this new hypothesis may be considered in both the nonsurgical and surgical realms. For example, during reconstructive surgery for pathologic pronatory conditions, it is common to perform either a tendoachilles lengthening or gastrocnemius recession with the intent to reduce what was previously considered a pathologic equinus. However, with this alternative model, the goal of flatfoot surgery would be to first create a stable foot with an improved ability to preload, allowing the normal gastrocnemius muscle to function appropriately. One purpose of the medial displacement calcaneal osteotomy is to medialize the pull of the tendoachilles (in addition to placing the calcaneal bisection in line with the tibia). If the triceps surae insertion were placed medial to the subtalar joint axis (rather than lateral to it) the gastrocnemius muscle would be altered from a subtalar joint pronator to supinator. In this case, performing a gastrocnemius recession or tendoachilles lengthening procedure would be contraindicated in flatfoot surgery. A normal triceps surae redirected to an improved anatomic location would be a more effective lever than an artificially lengthened one.

Similarly, foot orthosis therapy may benefit from a more accurate anatomic model. A current application of this concept may be to incorporate a heel lift into the orthosis shell to limit passive insufficiency. This method is supported by a study by Johanson and colleagues,[16] who found increased ankle dorsiflexion and increased time to heel off in patients with less than 5° ankle dorsiflexion after placement of a 6-mm or 9-mm heel lift.

A review of the literature reveals that it has never been asked how a pathologic contracture of the gastrocnemius muscle can be so prevalent in the human population. Despite the apparent high prevalence of gastrocnemius equinus, most researchers have assumed this is pathologic. In reality it is not the “pathologic” contracture of this muscle that creates foot dysfunction but rather the normal passive insufficiency created by the gastrocnemius being a multijoint muscle. Further research is necessary to confirm this hypothesis before it is accepted into common clinical practice.

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