The Gastrocnemius
A New Paradigm for the Human Foot and Ankle

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INTRODUCTION

Until recently, attention to the gastrocnemius as a primary etiology in the foot and ankle has been sparse and not well understood. Understanding in this matter, however, is coming of age. As addressed in this issue of *Foot and Ankle Clinics of North America*, there is more to the gastrocnemius than meets the eye.

In order to move forward, 2 vital questions must be answered: Why would the calves tighten? and, more importantly, How does a tight calf actually cause problems remotely in the foot and ankle? In this overview I proffer a concept that explains why the human calf might tighten in otherwise normal people. Javier Pascual Huerta explains in detail elsewhere in this issue how an isolated gastrocnemius contracture mechanically damages the foot and ankle, with a particular emphasis on plantar fasciitis.

Once it is accepted that the gastrocnemius contracture, which is usually silent, is an integral problem, then 2 final challenges can be considered. The first is to identify all
the problems that result, in part or principally, from this seemingly benign process. If the calf can be accurately linked to a particular pathologic entity, such as plantar fasciitis, then treatment in turn becomes more accurate. Then and only then can treating the calf be definitely chosen rather than just treating the result of this contracture (ie, symptomatic treatment of the foot). The second challenge is to refine the treatment of the gastrocnemius contracture itself, whether conservative stretching regimens or surgical lengthening. The ultimate challenge is the realization that if the gastrocnemius contracts with age, then this pathology can be avoided by appropriate preventative treatments.

A LIMITED REVIEW OF LITERATURE

The literature is now replete with evidence to support the association of the gastrocnemius and several foot and ankle conditions, including plantar fasciitis, insertional Achilles tendinosis, posterior tibialis tendon rupture, metatarsalgia, neuropathic ulcers, and others. The expense of treating just plantar fasciitis every year in the United States is staggering. Tong and Furia projected that in 2007 the cost of treatment to third-party payers ranged from $192 to $376 million.” They went on to state, “Our estimates do not account for all diagnostic tests and treatments common for PF [plantar fasciitis]. As a result, it is likely that this study understates the true costs of care for the disease.”

A consensus on a standard nonoperative treatment protocol has not been established, in part because a majority of these studies have addressed only the apparent problem in the foot and not the cause (ie, the tight calf). The other problem with these protocols is that the treatment options are lumped together in a variety of combinations, including calf stretches, immobilization, rest, orthotics, injections, platelet-rich plasma and cortisone injections, physical therapy, ice, stretching, extracorporeal shock wave therapy, surgery, and so forth. Although each of these treatments alone might help symptoms to some extent, the variety of treatments available only serves to reinforce that the cause of the problem has not been well understood. Although calf stretching has been shown effective, it has been mostly discounted because of a general feeling of lack of compliance in study groups. Yet, a prospective, randomized, double-blinded study on definitive Achilles stretching by Porter and colleagues was a seminal yet largely overlooked work. Porter and colleagues standardized conservative treatment by examining “one of the more effective nonsurgical modalities for treatment of painful heel syndrome” comparing 2 calf stretching regimens and confirmed that Achilles stretching alone was an effective treatment. Other studies have shown the benefits of calf stretching also. This was subsequently confirmed by DiGiovanni and colleagues, who concluded the “existence of isolated gastrocnemius contracture in the development of forefoot and/or midfoot pathology in otherwise healthy people. These data may have implications for preventative and therapeutic care of patients with chronic foot problems.” They stated that it is the gastrocnemius muscle that is contracted, not the Achilles: “such equinus positioning of the foot has frequently been called an Achilles contracture. However, this is a misnomer because the majority of the perceived stiffness or stretch occurs within the muscle bellies themselves, not in the tendon; the tendon can be responsible for only about 3% to 5% of this change in position”; they further noted, “We suspect that this pathologic entity plays a vital role in chronic mechanical breakdown or inflammation of both the foot and ankle.”

In many of these works, the measurement method is the Silfversköld test. The amount of or lack of dorsiflexion an individual exhibits with this test may be supportive,
but a negative Silfverskiöld test does not rule out the calf as the source of the pathology. In other words, a negative Silfverskiöld test does not confirm that there is not a clinically significant calf contracture present. More recently, Barouk and Toulec and Abbassian and colleagues described what I term the modern method to surgically lengthen the isolated gastrocnemius contracture, a proximal medial gastrocnemius release. Abbassian and colleagues commented, “Despite this, the exact etiology of the condition is still subject to debate...more recent study, however, reduced ankle dorsiflexion was associated with a much greater risk of developing the condition [plantar fasciitis] than either BMI [body mass index] or activity type.” Although it is somewhat a leap of faith to surgically lengthen the gastrocnemius behind the knee to fix plantar fasciitis, this procedure has been demonstrated effective. It is possible in the future that this will be the gastrocnemius lengthening procedure of choice for the isolated gastrocnemius contracture after failed conservative treatment because it directly addresses the contracted gastrocnemius, the degree of release is controlled, and it allows for immediate weight bearing.

None of these concepts is new, as John Joseph Nutt published his book Diseases and Deformities of the Foot in 1913, in which he discusses similar principles described by Dr Huerta in this issue. Nutt goes on to state, “Treatment consists in lengthening the gastrocnemius. As a very slight lengthening is all that is necessary, the tendo-Achilles being attached to so short a lever arm, an operation is scarcely demanded.” The work of Barouk and Toulec and Abbassian and colleagues, follows this statement; however, I would contend that calf stretching would be equally as effective in a high percentage of cases. Nutt stated, “The chief characteristic of this condition is a shortening of the gastrocnemius and the soleus. This shortening is not enough to produce the deformity of equinous but limits dorsal flexion.” What he essentially described in 1913 is the isolated gastrocnemius contracture. The biggest takeaway from Nutt’s work is the understanding of the ubiquitous source of calf contractures in otherwise normal humans, and he alludes to the mechanism of injury placed on the foot as a result (Figs. 1 and 2).

THE ORIGINS OF THE CALF CONTRACTURE

Where there may be many reasons why this progressive gastrocnemius tightness occurs, there are 4 common categories described in this article: activity changes, physiologic changes in muscles and tendons, genetics, and reverse evolution. Three of these principles generally apply to all muscles, whereas the fourth, reverse evolution, applies particularly to the calf, hamstring, and hip flexors.

The current thoughts on epidemiology of many acquired foot and ankle problems are addressed first. The prevailing thought on the epidemiology of these resulting problems may not be correct. The factors cited—obesity, sedentary life style, medical comorbidities, shoe wear, concrete floors, overuse, and so forth—although commonly associated with various foot and ankle problems, all may have a common pathway as the cause of these problems via calf contractures. In other words, although they are indeed present, these factors may not directly cause the resulting foot or ankle problems or pathology. Each of these factors creates an avenue to an increase in the human calf contracture, which in turn causes the foot and ankle pathology. If a tightened gastrocnemius is implicated as the cause of foot and ankle problems, then how and why would this happen in otherwise normal people? My premise is based on the notion that a gastrocnemius contracture is a contracture of the connective tissue surrounding and within the muscle, and less the tendon, and may have little to do with the muscle fibers themselves.
Fig. 1. Nutt depicts the effect gastrocnemius has as the passenger passes over the planted foot. (From Nutt J. Diseases and deformities of the foot. New York: E.B.Treat & Co; 1913. Available at: books.google.com/books?id=UWgQAAAAYAAJ.)

Fig. 2. Nutt depicts the strain of the plantar supportive tissue placed under additional tension and “lowering of the dome” or additional dorsal compressive forces placed on the longitudinal arch. (From Nutt J. Diseases and deformities of the foot. New York: E.B.Treat & Co; 1913. Available at: books.google.com/books?id=UWgQAAAAYAAJ.)
ACTIVITY CHANGES: LIFESTYLE INFLUENCES

General Decreased Activities as People Age

As people age, they are simply not as active as they may have been when they were children. Because of this more limited exposure to range of motion, muscle-tendon units fail to reach their ultimate length with regularity. A society of an increasingly sedentary lifestyle does not help matters either. If these stationary habits occur consistently and over a long enough period of time, the muscle-tendon units will shorten. The law of Davis⁵³ says that over time soft tissue contracts to the shortest position possible, given the opportunity. The isolated gastrocnemius contracture is a slower process, taking years to develop, but is the most common form of contraction of the lifestyle changes. This concept occurs every day when people experience the start-up stiffness or pain that settles as they get going. When sitting or sleeping, the ankles are in relaxed equinus and the calves tighten, only to stretch out just enough when becoming more mobile again.

Recent Changes in Activities

Bed rest due to illness or recovery from recent injury or surgery and even after pregnancy often causes a dramatic change in activities, such that the muscles, especially the calf, tighten up because of the abrupt reduction of otherwise normal mobility. As the gastrocnemius relaxes, it is under little, if any, tension, and according to the law of Davis, the calves get tighter. Then a return to normal activities creates problems solely because of the resulting calf contracture. This is a relatively quick process, taking just weeks to a few months.

Athletes and Increased Activity Situations

On the other end of the spectrum, many athletes, in particular distance runners, seem more at risk for contracture. One would think that athletes should be immune to this phenomenon; however, the repetitive action of running, especially for joggers, raises frequent issues. While running, the gastrocnemius and hamstrings of these athletes are not extended to their full length. In fact, slow motion video shows that the knee only comes to full extension in distance running after toe off and the ankle is plantar-flexed as opposed to walking. During a high-mileage run, these muscle groups are over-active, and the opposing muscles, the quadriceps, and the anterior tibialis might get relatively weaker. Over time, the muscles eventually adapt to this shortened position.

PHYSIOLOGIC CHANGES TO MUSCLES AND TENDONS: INTERNAL INFLUENCE

There are definable physiologic changes that take place with aging when it comes to collagen and connective tissue. Cross-linking of collagen increases with age, and connective soft tissues get less compliant and lock the structure in the shortened position if allowed to do so. No doubt this is part of the law of Davis. In addition, the percentage of elastin in connective tissues reduces, which lowers the flexibility of muscles and tendons. This is a fact of aging and is mostly inescapable.

GENETICS

There is no doubt that genetics play, if nothing else, a subtle role in the progressive contracture of connective tissue. This would account for a possible familial proclivity to calf tightness or other muscle groups.

Genetics might also account as to why an individual person might be more prone to muscle contractures that lead to tendon and joint problems in multiple areas of the...
body or recurrent issues in one location. Some people experience muscle tightness more than others and genetics could be a reason this might occur.

**REVERSE EVOLUTION: THE HUMAN INFLUENCE AND THE PREDILECTION PATTERN**

Evolution suggests that humans evolved from quadrupeds to a bipedal species, but bipedal gait, as a primary method of locomotion, evolved approximately 2.2 to 3 million years ago.\(^{22,51}\) Before that time, bipedal gait was used by human ancestors, but only part time. In order for the human foot to adapt to bipedal gait, 2 basic structural changes were required. In the leg, certain muscle groups had to lengthen whereas their opposing muscle groups had to shorten. Simultaneously, the ankle had to unwind and dorsiflex approximately 70°. This dorsiflexion brings the heel down to contact the ground, thus making the foot plantigrade. Additionally, the knee and the hip had to extend to achieve the positioning to allow for a bipedal gait.

There are 2 key lower extremity muscle groups that certainly adapted late in this evolutionary process. In the quadruped, the hamstrings and the triceps surae (gastrocnemius and soleus) are all shortened as the knee is continuously flexed and the ankle joint is in a highly plantar flexed position.

In the course of evolution, these muscle groups (hip flexors, hamstrings, and gastrocnemius) had to lengthen while the opposing muscle groups (quadriceps and anterior tibialis) had to shorten and gain power. Because these muscle groups adapted later in the evolutionary process, they are the first to move backward and tighten as a person ages, reverting back to their former positions. This is called a predilection pattern, leading to many problems with the calves and the hamstrings. This relative motor imbalance is due, in part, to this evolutionary process and also as a function of the calf, which is required to be stronger for normal human bipedal locomotion. Simultaneously with these muscle changes, to provide a larger weight-bearing base for balance and a lever arm that could adequately propel humans forward in bipedal mode, it was necessary for the foot to drop out of its equinus position, moving the heel to the ground. This rotation of approximately 70° mostly occurs at the ankle joint. This concept can be examined further by looking at the perfect foot.

**The Perfect Foot**

The perfect foot, from a mechanical standpoint, would be one that evolution left behind long ago, as humans moved away from being quadrupeds. Structurally, a horse or dog has a better foot for wear and abuse than humans. It would be correct to argue that this might be the case because quadrupeds weight bear on 4 legs and, therefore, there is less force exerted on the hind foot. When considering the foot as a biomechanical structure unto itself and the forces humans put on each of theirs through every day, however, it is apparent that the construction of the foot for a quadruped is more mechanically sound; however, the weight-bearing platform of an equinus foot is too small for prolonged maintenance of a bipedal gait. So, the human foot evolved and rotated to place the heel on the ground (described previously). This adaptation provided a much larger plantar surface area for improved force distribution, balance, and necessary leverage for bipedal locomotion. This same new foot position placed the human foot in a biomechanically disadvantaged position, however, due to new leveraged forces it had to withstand.

With weight bearing, the human foot is oriented such that forces shift from a vertical, pure compressive load (perfect foot) to leveraged/bending or vector forces, resulting in higher compressive (dorsal foot and anterior ankle) and tensile forces (plantar foot and posterior ankle). Vector or bending forces clearly produce more asymmetric force...
on the parts of the system compared with vertical axial loading. Evolution robbed people of stable axial forces, creating bending forces.

**The Gastrocnemius: Cause and Effect**

If it is believed and accepted that the isolated gastrocnemius contracture is associated with problems in the foot and ankle, there must be a valid reason how this can happen. Nutt described this mechanical linkage in 1913 in the form of Schaeffer’s foot. Today, Huerta describes in more detail how a tight gastrocnemius causes plantar fasciitis.

When younger, a flexible, compliant gastrocnemius allows the necessary smoother deceleration of ankle dorsiflexion as the ankle progresses forward late in the ankle rocker phase just as the forefoot rocker begins. The forces are gradual and damped producing a softer end point, thus a more cushioned force transfer to the foot and ankle.

The damaging forces occur with the isolated gastrocnemius contracture when a tremendous, leveraged force is transmitted to the foot and ankle as the tibia and body pass over the planted or stance foot. Instead of a gradual force transfer, which occurs when younger, there is an abrupt end point in forward progression of necessary ankle dorsiflexion. This effect occurs during the late part of midstance or the ankle rocker phase, just before the heel lifts off the ground. If the calf becomes contracted, this indirect leveraged force is exponentially magnified, creating abnormal, pathologic bending or vector stress to the foot and ankle, thus resulting in stress and strain to the joints and supporting ligaments and tendons. Dorsal compressive and plantar tension forces are magnified. It is also at this point in the gait cycle that individuals who have symptomatic plantar fasciitis demonstrate a characteristic shortened type of antalgic gait as they begin to walk with a characteristic limp. This is the gait of effective pain avoidance until the rest induced by the shortened gastrocnemius can lengthen just enough to allow the forces foot or ankle to reduce, followed by improvement of the gait. Over time, and as a result of taking thousands of steps per day, the foot and ankle succumb to an occult, unrecognized overuse, which ultimately leads to damage.

**DISCUSSION**

I believe that there is a simple, singular, silent, and remote cause of the many foot and ankle problems, which are mechanically created, leading to incremental damage to the foot and ankle through leveraged forces: the human calf that becomes too tight with age. In short, the isolated gastrocnemius contracture is the common denominator that leads to the many of the human nontraumatic foot and ankle problems. Calves tighten with age: activity changes, physiologic changes in muscles and tendons, genetics, and reverse evolution.

No doubt many more studies will and should emerge that qualify and quantify the role of the tight gastrocnemius on the human foot and ankle. It is likely that study methods, such as high resolution and ultra–high-speed gait laboratory motion analysis, will show this concept to be correct, which, in turn will lead to improved prevention and treatment efforts.

The Silfverskiöld test, in my experience, is only an approximate estimate as a clinical test and is generally only useful for following the results of a particular treatment method. Historically, the Silfverskiöld test, relied on in clinical studies for the subtle or isolated gastrocnemius contracture, will likely be found to have been a hindrance to understanding. Even though Abbassian
and colleagues used the Silfverskiöld test, they were suspicious of its validity. A negative Silfverskiöld test does not exclude the gastrocnemius as a possible cause of the underlying foot or ankle problem. The symptoms and even the diagnosis itself may be the best link to the tight gastrocnemius as the culprit. The classic symptoms, such as start-up pain or stiffness, or even the diagnosis itself, such as posterior tibialis dysfunction or insertional Achilles tendinosis, determine the cause, not this test. In other words, if patients have plantar fasciitis, then by default they have calves that are too tight regardless of what the Silfverskiöld test indicates. Think of it as sort of reverse logic: where there is smoke there is fire, even if the fire cannot be seen.

It has been postulated that epidemiologic factors, such as obesity, sedentary lifestyle, medical comorbidities, shoe wear, concrete floors, advanced age, female gender, and overuse issues, to name a few, are responsible for a variety of foot and ankle pathology. Although these factors might consistently coexist with a variety of foot and ankle problems and seem to have a causal relationship, it is my assertion that they have little if any direct relationship.

The singular and real association of each of these epidemiologic factors is a contracture of the gastrocnemius muscle, which is camouflaged in this list. Most every other cause of these foot and ankle problems is likely mediated by contributing to the degree and/or rate of an already contracting gastrocnemius. These problems promote gastrocnemius tightness, which in time causes incremental damage to the foot and/or ankle. To put it frankly, the obvious evidence of this is that overweight people with plantar fasciitis stretch and get over their problem and they are still overweight. Women have a calf-lengthening procedure for insertional Achilles tendinosis with resolution of their problem and they are still women. Older people stretch and move on pain-free, yet they do not get magically younger. Gradual, silent calf contracture happens in most people, some less and some more, depending on life choices and circumstances.

**SUMMARY**

It seems that merely being human places a risk for developing acquired foot and ankle problems. This damage is mediated through the gastrocnemius that tightens for several reasons with age and these same contracted calves do incremental harm to the human foot and ankle. Drs Nutt and Huerta have eloquently described how a silent gastrocnemius contracture, that seemingly has little to do with the foot and ankle, can gradually do so much harm when left undetected and unattended.

Considering this knowledge, I assert, if not challenge, that the calf is a common source of a majority of acquired, nontraumatic adult foot and ankle problems, such as plantar fasciitis, nontraumatic midfoot osteoarthritis, insertional Achilles tendinosis, posterior tibialis tendon dysfunction, and Achilles tendinitis, to name a few. Further investigation should sort out these associations and issues.

As the realization and understanding of the ubiquitous role of the contracted gastrocnemius in many foot and ankle problems move forward, so will the search for optimal treatment: nonoperative and operative.

Controversy, varying opinions, and even urban myth abound when it comes to the best method of calf stretching. No doubt much work remains to sort out the optimal stretch for the calf, whether static, dynamic, eccentric, or other. When it comes to surgical lengthening procedures, whether at the Achilles, at the musculotendinous junction, or more proximal, the search must move on to find the safest, most accurate, and quickest recovery method possible. Of course, prevention, in the form of calf stretching, should be ultimate goal.
Addressing the calf contracture as definitive treatment and, better yet, as prevention, will no doubt become a mainstay of the treatment of many foot and ankle problems. Regardless of whether a preference of treatment is calf stretching or performing lengthening by surgical means, the mission must be to bring awareness of the detrimental role of the gastrocnemius contracture to everyone as soon as possible. It has already been a century since Nutt\(^{23}\) described much of this the first time; let us not wait too much longer.

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