Isolated Gastrocnemius Tightness

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Background: Contracture of the gastrocnemius-soleus complex has well-documented deleterious effects on lower-limb function in spastic or neurologically impaired individuals. There is scarce literature, however, on the existence of isolated gastrocnemius contracture or its impact in otherwise normal patients. We hypothesized that an inability to dorsiflex the ankle due to equinus contracture leads to increased pain in the forefoot and/or midfoot and therefore a population with such pain will have less maximum ankle dorsiflexion than controls. We further postulated that the difference would be present whether the knee was extended or flexed.

Methods: This investigation was a prospective comparison of maximal ankle dorsiflexion, as a proxy for gastrocnemius tension, in response to a load applied to the undersurface of the foot in two healthy age, weight, and sex-matched groups. The patient group comprised thirty-four consecutive patients with a diagnosis of metatarsalgia or related midfoot and/or forefoot symptoms. The control group consisted of thirty-four individuals without foot or ankle symptoms. The participants were clinically examined for gastrocnemius and soleus contracture and were subsequently assessed for tightness with use of a specially designed electrogoniometer. Measurements were made both with the knee extended (the gastrocnemius under tension) and with the knee flexed (the gastrocnemius relaxed).

Results: With the knee fully extended, the average maximal ankle dorsiflexion was 4.5° in the patient group and 13.1° in the control group ($p < 0.001$). With the knee flexed 90°, the average was 17.9° in the patient group and 22.3° in the control population ($p = 0.09$). When gastrocnemius contracture was defined as dorsiflexion of ≤5° during knee extension, it was identified in 65% of the patients compared with 24% of the control population. However, when gastrocnemius contracture was defined as dorsiflexion of ≤10°, it was present in 88% and 44%, respectively. When gastrocnemius-soleus contracture was defined as dorsiflexion of ≤10° with the knee in 90° of flexion, it was identified in 29% of the patient group and 15% of the control group.

Conclusions: On the average, patients with forefoot and/or midfoot symptoms had less maximum ankle dorsiflexion with the knee extended than did a control population without foot or ankle symptoms. When the knee was flexed 90° to relax the gastrocnemius, this difference was no longer present.

Clinical Relevance: These findings support 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.

Tightness of the gastrocnemius-soleus complex has long been documented in spastic and neurologically impaired individuals[1-3]. Since the first description of tendo Achillis lengthening in the early 1800s by Delpech[4], release or attenuation of the superficial posterior compartment of the leg has been performed in many ways to relieve equinus contracture and to improve gait and muscle balance across the foot and ankle[5-16]. Very little attention, however, has been paid to the cumulative pathological effects or even the existence of a more subtle equinus contracture of the gastrocnemius that can be found in the "normal," otherwise unaffected population. This is surprising considering that isolated gastrocnemius tightness and its treatment with surgical recession were first described in the early 1900s by Silfverskiold[17] and by Vulpius and Stoffel[18], respectively, albeit in spastic patients[19-21]. To date, even the definition of and method of examining for equinus contracture remain controversial, with the maximal ankle dorsiflexion values used as a measure of equinus deformity ranging from 0° to 25° and the examination performed with the knee in varying amounts of flexion or extension[22-28]. As a result of such confusion, the reliability of clinical examination for correctly identifying contracture also has never been determined.

Except for a few still controversial examples of plantar fasciitis, forefoot ulceration in diabetics, or progressive hallux valgus or flatfoot, the relationship between tightness of the superficial posterior compartment and progressive pathological changes in the foot in non-spastic individuals has been overlooked entirely by the orthopaedic community[26,29-32]. In contradistinction, more attention has been paid to this phenomenon in the podiatric literature over the past three decades, although most of those reports are observational or anecdotal[10,23,28,33-38]. We know of no paper specifically addressing the prevalence of isolated gastrocnemius contracture or its long-term effects in otherwise normal, healthy people.

Our experience has suggested that the gastrocnemius muscle in particular is the predominant deforming force in people with structural breakdown or chronic pathological changes related to the foot and ankle. We suspect that contracture of this muscle is not only common but often partially responsible for many of the chronic forefoot and midfoot symptoms identified in non-neurologically impaired patients. The purpose of our study was to identify the prevalence of equinus contracture in a group of individuals with and without foot symptoms, with a specific focus on the presence of isolated gastrocnemius tightness and our ability to accurately diagnose it. We hypothesized that an inability to dorsiflex the ankle as a result of equinus contracture leads to increased pain in the forefoot and/or midfoot and therefore a population with such pain will have less maximal ankle dorsiflexion than controls. We further postulated that the difference would be present whether the knee was extended or flexed.

Materials and Methods

Patient Enrollment

Subsequent to obtaining Institutional Review Board approval, we prospectively identified sixty-eight subjects for this investigation between April 1, 1999, and July 31, 1999. The patient population comprised thirty-four consecutive patients who presented to our University and Veteran's Administration foot and ankle clinics because of isolated forefoot or midfoot pain of any duration. The exclusion criteria for the approximately 1000 patients screened for this group included any history of neurological disease including neuroma, any systemic disease potentially affecting the foot or ankle such as the vasculitides or rheumatoid arthritis, any confounding preexisting foot or ankle surgery or trauma, any osseous block to ankle dorsiflexion, or any irreducible foot deformity (i.e., one making it impossible to obtain a neutrally aligned foot by reducing the talonavicular joint) precluding proper testing and evaluation of ankle equinus. Individuals with symptoms related solely to the hindfoot or ankle, those with severe hindfoot deformity, and those with midfoot pain suspected to be the result of ankle or hindfoot pathology were also excluded. The control population consisted of thirty-four randomly identified spouses of patients or hospital personnel from multiple services who had no foot or ankle symptoms. Approximately seventy-five people were interviewed for inclusion in the control group before a sufficient number who met the exclusion criteria described above could be enrolled. All people fulfilling these criteria were then approached by an independent provider to ask them if they were willing to participate in the study and, if they were, to sign an informed-consent form.

Clinical and Radiographic Examination

A brief medical and surgical history was obtained from each individual to be studied, and a directed physical examination of both feet and ankles was performed. The patient's height and weight, any previous treatments, and all medications were recorded. Any history of smoking, diabetes, or ligamentous laxity (defined as elbow hyperextension) was also noted. Examination was performed to identify any physical or structural foot or ankle abnormalities, with attention paid to the chief symptom. A subjective clinical assessment of the presence or absence of equinus was then made by the examiner. Gastrocnemius contracture was simply recorded as "present" or "absent" by the examiner on the basis of the Silfverskiold test[17]. Weight-bearing anteroposterior, lateral, and oblique radiographs of the foot and/or ankle were not routinely made for the purposes of this study, but any that had been made incidentally during a patient's clinic visit were evaluated. No

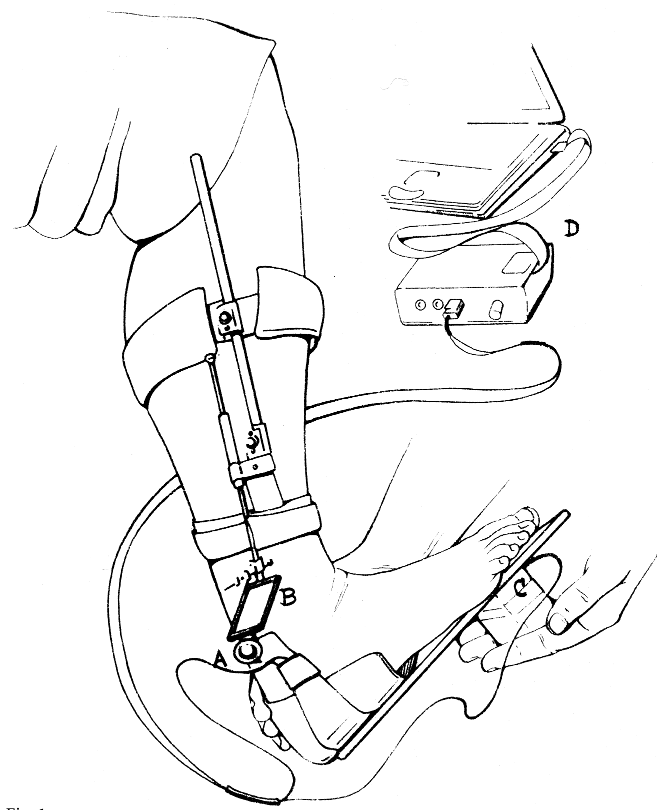

Fig. 1

The equinometer comprises an electrogoniometer (A) attached to the lateral aspect of the leg by means of a four-bar linkage (B), which is connected to a rigid foot-plate attached underneath the foot with a force transducer (C) positioned beneath the second metatarsal head. The ankle acts as the center of rotation for an upwardly applied force to the plantar surface of the foot. Resultant maximal ankle dorsiflexion is measured by the computer (D), which accrues data only when the upward force on the foot is appropriate for a predetermined torque of 10 N-m.

radiographs had been made for any person in the control group.

Equinometer Testing

Maximal ankle dorsiflexion was measured with an equinometer affixed to the subject's leg (Fig. 1)[39,40]. Data from this electrogoniometric device were processed by a Macintosh G3 series computer software package (Apple Computer, Cupertino, California), which depicts ankle position in the sagittal plane as a result of an upwardly directed load applied to the undersurface of the foot. The site of torque application is directly under the second metatarsal head, the approximate center of force transmission in the human foot. The instrument shows a reading of 0° when attached to a neutral, plantigrade foot, with subsequent positive change indicating relative dorsiflexion and negative change indicating plantar flexion. A predetermined, constant torque of 10 N-m was arbitrarily selected for use in this experiment. This value represents the average pressure placed on the foot by two of our orthopaedists and one rehabilitation medicine specialist while testing for equinus contracture in an office setting. We had previously validated the equinometer in our laboratory for such use. The instrument is accurate to within 1.31°, with a documented average daily variation in individual subjects of <0.5° (0.45° ± 0.43° [standard deviation])[41]. Both a right-sided and a left-sided equinometer were used, with recalibration to a plastic foot and ankle model every few weeks to ensure reproducibility and accuracy.

Each subject to be tested was seated on an examination table with the bare foot and leg exposed. Care was taken to ensure that the subjects were relaxed. The fibula and the second metatarsal head were then identified by the examiner. A measurement from the tip of the fibula to the center of the second metatarsal head (i.e., the moment arm) for each person was recorded in the computer (Fig. 2). The device was carefully positioned alongside the lateral aspect of the leg in line with the fibula. This reference serves as a very reproducible anatomic landmark and has an axis that closely approximates the center of rotation of the ankle joint. The foot-plate and accompanying force transducer were then attached to the undersurface of the neutrally aligned foot (Fig. 3). The talonavicular joint is held reduced by the examiner, thus reducing the hindfoot and locking the midfoot. Midtarsal motion is prevented because the heel cup is attached to a rigid steel bar running the full course of the foot. With the foot held in neutral in the device, ankle dorsiflexion could be measured after the 10-N-m load was applied to the undersurface of the foot. Because various data suggest that the gastrocnemius muscle is under no tension with the knee flexed ≥25°, ankle dorsiflexion was recorded with the knee in full extension and in 90° of flexion[42]. Three recordings of maximal resultant ankle dorsiflexion were obtained for each knee position. Subjects were randomized (alternated) with regard to whether testing was started with the knee in extension or flexion. These latter two steps were taken to isolate independent contributions of both the gastrocnemius and the soleus muscle to any existent contracture and to negate any stretching effect that repetitive testing might have on the tissues. Twenty-eight people in the patient group and thirty-three people in the control group were tested with the equinometer on the left side, and twelve and fifteen, respectively, were tested with it on the right side. There were six bilateral examinations in the patient group and fourteen in the control population. All examinations and equinus testing were performed by a single examiner.

The data accrued from this testing were used to compare the subjective determinations (by the clinician) and the objective determinations (with the equinometer) of the presence or absence of equinus. Since absolute criteria for identifying gastrocnemius contracture either subjectively or objectively have yet to be established and we are still in the early phase of studying this problem, we used two cutoff determinants, ≤5° and ≤10° of maximal ankle dorsiflexion after force application, as potential definitions of gastrocnemius contracture. The four

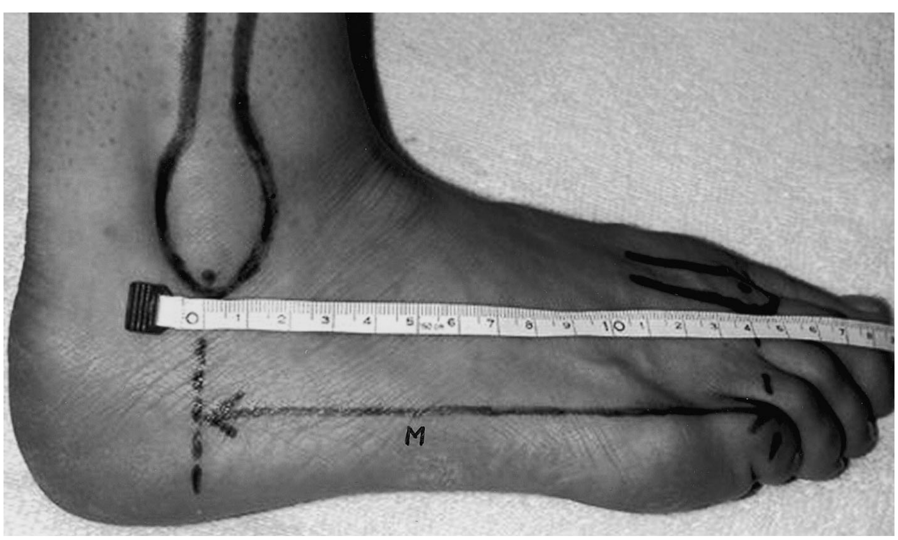

Fig. 2

Moment-arm measurement of the distance (M) between the distal tip of the lateral malleolus and the center of the second metatarsal head (as extrapolated along the lateral column of the foot) standardized a constant torque of 10 N-m for each individual. This measurement (M), in the equation torque = force × moment (M), allows the computer to provide feedback on appropriate upward pressure (force) to keep torque constant.

analyses included (1) clinical determination versus equinometer measurement of ≤5° with the knee flexed, (2) clinical determination versus equinometer measurement of ≤5° with the knee extended, (3) clinical determination versus equinometer measurement of ≤10° with the knee flexed, and (4) clinical determination versus equinometer measurement of ≤10° with the knee extended. Thus, for each individual, the presence or absence of gastrocnemius contracture was decided subjectively on the basis of clinical opinion and objectively on the basis of two different criteria: maximal ankle dorsiflexion of ≤5° or of ≤10° with the knee fully extended. The examiner was considered to have made a correct diagnosis when the clinical decision (present or absent) correlated with the objective determination as defined by the above criteria in each scenario. Gastrocnemius-soleus contracture was arbitrarily defined as present when the individual exhibited ≤10° of maximal ankle dorsiflexion in 90° of knee flexion as determined objectively with the equinometer.

Statistical analysis was performed between matched groups with the use of the chi-square test and the Student paired t test. The p value was set at 0.05 for determination of significance. Study power was calculated for the resultant maximal ankle dorsiflexion values.

Results

Sixty-eight people were included in this investigation. The average age of the patient group was forty-seven years (range, twenty-one to seventy-six years) compared with forty-five years (range, twenty-eight to sixty-three years) for the controls. There were eighteen men and sixteen women in the patient population compared with sixteen men and eighteen women in the control group. The average weight of the individuals was 183 lb [83 kg] (range, 104 to 345 lb [47.2 to 156.5 kg]) in the patient group and 171 lb [77.6 kg] (range, 120 to 260 lb [54.4 to 117.9 kg]) in the control group. The primary diagnoses, determined on the basis of the patient's history, the results of physical examination, and radiographs, in the patient group were classified into six categories (Table I). Although only seven of the thirty-four patients were noted to have isolated metatarsalgia, nineteen (56%) had some component of metatarsalgia as part of the chief symptom. We defined a Morton foot as a constellation of symptoms commonly identified in our clinics: hallux valgus, extensor recruitment, medial column hypermobility, and a long second metatarsal with overload. Patients with insufficiency of the posterior tibial tendon were found to have either stage-one or stage-two disease[43]. We describe this deformity as dorsolateral peritalar subluxation, and we excluded any patient with stage-three disease because feet with irreducible deformity cannot be properly tested for equinus contracture. We noted one stress fracture, at the base of the fifth metatarsal without antecedent trauma. In the control group, all subjects were found clinically to have structurally sound feet.

A significant difference was found between the patient

TABLE I Primary Diagnoses in the Patient Group

Diagnosis	No. of cases
Metatarsalgia	7
Morton foot	13
Idiopathic Lisfranc arthrosis	1
Posterior tibial tendon insufficiency	9
Plantar fasciitis	3
Stress fracture (5th metatarsal)	1

Fig. 3
Achievement of a neutrally aligned foot in the jig was an extremely important step in testing tightness of only the gastrocnemius-soleus complex.

TABLE II Comparison of Average Maximal Ankle Dorsiflexion Between the Two Groups with the Knee in Either Extension or Flexion

	Maximal Ankle Dorsiflexion*	
	Knee in 0° of Flexion	Knee in 90° of Flexion
Patient group	4.5° ± 4.5° (–1.1° to 14.3°)	17.9 ± 9.0° (6.1° to 37.0°)
Control group	13.1° ± 8.2° (1.1° to 30.7°)	22.3° ± 10.9° (7.6° to 51.1°)
P value	<0.001	0.09
Power ratio	0.99	0.40

*The values are given as the average and the standard deviation, with the range in parentheses.

TABLE III Comparison, Between Groups, of Number of Subjects in Whom Gastrocnemius Contracture, Identified with the Equinometer, Was Diagnosed Clinically

	Gastrocnemius Contracture*		Correct Diagnosis*†	
Group	≤5°	≤10°	≤5°	≤10°
Patient	65% (22/34)	88% (30/34)	76% (26/34)	88% (30/34)
Control	24% (8/34)	44% (15/34)	94% (32/34)	79% (27/34)

*≤5° and ≤10°, which refer to the maximal ankle dorsiflexion, indicate the two possible definitions of contracture. †The correct diagnosis refers to the correct identification of gastrocnemius contracture as either present or absent.

and control populations with regard to the average maximum ankle dorsiflexion with the knee in the extended position (the gastrocnemius under tension) as opposed to the flexed position (the gastrocnemius relaxed) (Table II). The difference between the ankle dorsiflexion range with the knee flexed and that with the knee extended was 11.5° ± 6.0° in the patient group compared with 8.4 ± 4.8° in the control group ($p = 0.02$).

No significant difference was identified, during knee extension ($p = 0.68$) or flexion ($p = 0.67$), between the results for the right and left sides of patients who had a bilateral examination in either group. There was also no difference in either group between the readings for the first and last (third) trials ($p = 0.6$ to 0.9) or between male and female subjects ($p > 0.05$). The average maximal ankle dorsiflexion in knee extension was found to be independent of the initial starting position of the knee (either flexion or extension) ($p > 0.05$). Similarly, there was no significant difference between the results of the whole patient group and those in the subpopulation of patients who presented with only metatarsalgia ($p > 0.05$). When this subpopulation was compared with the control group, however, the difference in average maximal ankle dorsiflexion was found to be significant ($p = 0.001$).

Despite the small numbers of individuals, there were a few evident differences between groups with regard to some of the medical parameters that we measured. Five individuals in the patient group were found to have diabetes mellitus as opposed to none in the control group. Seven individuals in the patient group had a history of smoking compared with only three in the control group. Only one individual in the patient group had ligamentous laxity compared with five in the control group.

Regardless of whether we defined gastrocnemius contracture as ≤5° or ≤10° of ankle dorsiflexion during full knee extension, there seemed to be at least a twofold increase in the rate of equinus in the patient group compared with that in the control population and an approximately 85% rate of correct clinical diagnosis of this entity (Table III). Similarly, the number of individuals in the patient group exhibiting a so-called Achilles tendon contracture (defined as combined equinus of the gastrocnemius and soleus muscles) was nearly double that in the control group (Table IV).

Discussion

For two centuries, much attention has been paid to equinus contracture of the gastrocnemius-soleus complex in patients with neurologic or spastic imbalance[3,5-16]. Associated long-term sequelae of ankle equinus, such as gait disturbance and structural breakdown of the foot and ankle, have also been well documented in these patients[2,17,20,22,25,26,28,31,35,44,45]. 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. Hence, although we often speak of "Achilles" tightness and address the Achilles tendon directly to treat this disorder,

TABLE IV Comparison, Between Groups, of Number of Subjects with Either No Evidence of Gastrocnemius Contracture or Evidence of Gastrocnemius-Soleus Contracture

Group	No Gastrocnemius Contracture*		Gastrocnemius-Soleus Contracture†
	≤5°	≤10°	
Patient	35% (12/34)	12% (4/34)	29% (10/34)
Control	76% (26/34)	56% (19/34)	15% (5/34)

*≤5° and ≤10°, which refer to the maximal ankle dorsiflexion, indicate the two possible definitions of contracture. †Gastrocnemius-soleus contracture was defined as ≤10° of maximal ankle dorsiflexion in 90° of knee flexion.

the root of the problem is actually at the level of the gastrocnemius and soleus muscles. Since 1816, when the first tendo Achillis lengthening was described in France, multiple methods of managing this problem have been reported[4,10-16,18,20]. Improved gait, decreased muscular imbalance, and slowed progression of foot deformity have often been noted after these therapies in affected patients[2,6,7,9,16,19,27,28,39].

Many foot deformities, such as hallux valgus, plantar ulceration, stress fracture, peritalar subluxation (acquired flatfoot), and metatarsalgia, seem to have a multifactorial origin in neurologically "normal" people. Few orthopaedic papers, however, have discussed the role of a tight superficial posterior compartment in these people; furthermore, to our knowledge, none have really addressed the existence or role of an isolated gastrocnemius contracture[26,29,30,32,46]. Most of these few reports deal with the relationship between a tight heel cord and flatfoot. Lin et al.[47] also reiterated the importance of a tight heel cord in diabetics, noting a markedly improved rate of healing of plantar ulcers treated with a combination of a total-contact cast and heel-cord lengthening as opposed to a cast alone; presumably the improvement was due to decreased foot pressures. Brodersen et al.[22] followed more than 750 children over the course of their education in the Danish school system and found that, in the two oldest classes, students with a high arch had a 39% prevalence of a tight superficial posterior compartment compared with prevalences of 56% and 77%, respectively, in those with a normal arch and those with a depressed arch (flatfoot). They recommended stretching of the gastrocnemius-soleus complex in all school-age children. Some papers, such as that by Sinacore[48], have described an exhaustive list of risk factors associated with foot problems but have never mentioned a tight superficial posterior compartment.

While equinus contracture is well recognized as the inability to dorsiflex through the tibiotalar joint, to our knowledge there is still no good study providing a numerical definition of gastrocnemius tightness or describing its prevalence, natural history, or treatment or the clinical ability to diagnose the condition in humans. This is probably due to the subtleties of midfoot motion and hence the clinician's difficulty in accurately performing an evaluation for equinus contracture. Correct examination requires locking of the talonavicular joint, and hence neutralization of the hindfoot, and subsequent evaluation of the change between the ankle dorsiflexion possible with the knee flexed 0° and that possible with the knee flexed 90°. The modicum of information on this seemingly underappreciated phenomenon is anecdotal and is mostly in the podiatric literature[23,25,28,35]. Since the early 1970s, podiatrists have sought to define and diagnose gastrocnemius and combined equinus deformity, with some disagreement[10,24,26-28]. The maximal ankle dorsiflexion values used as a measure of equinus deformity have ranged from 0° to 25°, and various knee positions have been used to calculate these values[22-28]. A number of recent reports have corroborated evidence linking equinus release with decreased foot symptoms and breakdown as a result of improved ankle dorsiflexion, particularly in diabetics[44,49-51]. Subotnick[28], Downey and Banks[23], and Hill[35] independently emphasized improvement in gait mechanics and foot function after the effects of gastrocnemius or soleus tightness had been alleviated in non-spastic individuals; Subotnick stated that "gastrocnemius or soleus equinus is the greatest symptom producer in the human foot."[28] In fact, two podiatrists described the first (to our knowledge) isolated gastrocnemius lengthening for treatment of a non-neurological deformity in 1974[52]. Although these papers would be heavily criticized on a scientific basis by today's standards, they linked equinus deformity with such foot conditions as Achilles tendinitis, plantar fasciitis, metatarsalgia, peroneal tendinitis, Morton neuroma, and heloma durum and provided some of our only insight into the role of the gastrocnemius under normal conditions. Furthermore, Grady and Saxena[24] challenged our ability to stretch a tight gastrocnemius-soleus complex, noting only a few degrees of change in muscle flexibility after twenty-six weeks of a commonly prescribed home stretching regimen. This technique levers the foot between a dominant muscle group on one side and the unyielding ground on the other. If the muscle does not actually stretch, such a combination could lead eventually to foot breakdown.

We believe that we were the first to attempt to prospectively identify (1) the prevalence of isolated gastrocnemius contracture in neurologically normal people, (2) its potential relationship with forefoot and/or midfoot pain, and (3) our ability to correctly diagnose it during physical examination. On the basis of the data obtained from our research, we believe that the inability to dorsiflex the ankle due to gastrocnemius tightness represents a real entity in "normal" patients, and, although we have not proved causality, we demonstrated its significant association with increased pain in the forefoot and/or midfoot. Patients with such pain had an almost threefold higher prevalence of isolated gastrocnemius contracture (when defined as ≤5° of dorsiflexion during knee extension) in comparison with a control population without foot symptoms. They also had twice the prevalence of combined gastrocnemius-soleus contracture compared with controls. When evaluated with the knee in extension, these patients had only one-third (4.5°) of the average maximal ankle dorsiflexion demonstrated by the asymptomatic group (13.1°). However, no significant difference in dorsiflexion was identified between the two groups when the knee was flexed and the gastrocnemius was relaxed. These data support the concept that chronic mechanical overload of the foot and thus inflammation or wear and tear can result from equinus contracture (Fig. 4). Because it crosses the knee, ankle, and subtalar joint, the gastrocnemius muscle seems the most prone to being tight in the leg and would therefore have the greatest effect during upright alignment.

This study also indicates that isolated gastrocnemius tightness in otherwise normal, healthy patients seems to be subtle and that our ability to diagnose it clinically is, at best, only good. Our ability to make this diagnosis correctly in the patient group was only 76% when we used a definition of ≤5°

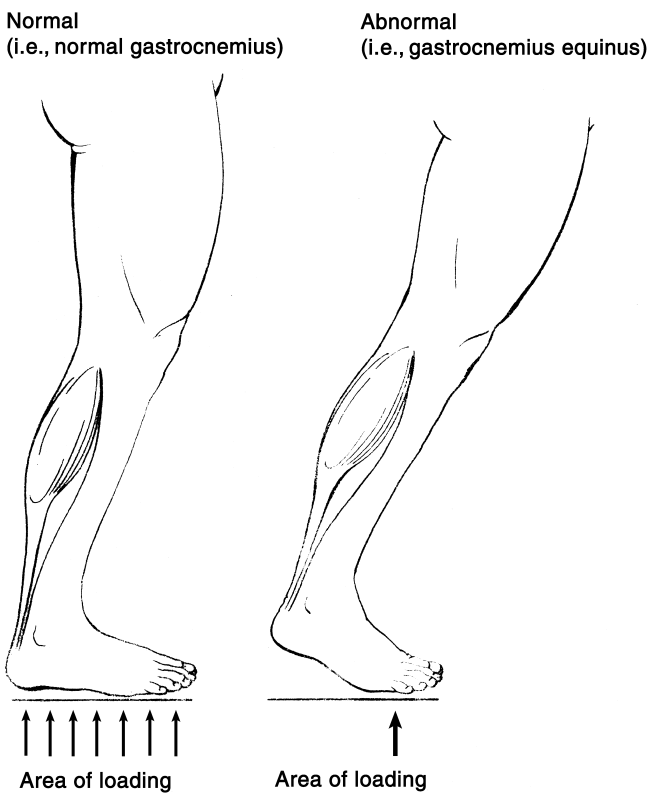

Fig. 4

Graphically depicted load patterns during gait. This illustration demonstrates the anticipated variation in forefoot and midfoot loading during late-stance-phase gait between "normal" subjects (i.e., those with normal gastrocnemius length) and patients with an abnormal (i.e., contracted) gastrocnemius length.

during knee extension; it increased to 88% when the definition was ≤10°. Although gait analysis data suggest that the average individual relies maximally on about 10° of ankle dorsiflexion during the late-stance phase of normal walking[53], the gastrocnemius equinus remains ill-defined. We have selected ≤5° of maximal ankle dorsiflexion with the knee in full extension as our definition because it allowed us to diagnose the problem in those who were at risk (symptomatic patients) with fairly good reproducibility (76%) and, more importantly, we were able to reliably avoid (in 94% of the cases) unnecessary treatment of those who were not at risk (asymptomatic people). This definition is complemented by our chosen definition of combined gastrocnemius and soleus contracture as ≤10° of maximal dorsiflexion with the knee in 90° of flexion (i.e., regardless of knee position).

Interpretation of these findings is limited by the shortcomings confounding our study. Generalization of our data would have been more accurate with a larger number of patients in both groups and with the inclusion of multiple blinded examiners. Also, our measurements relied on anatomic landmarks and thus were not exact, and weight-bearing radiographs were not compared between the two groups. In addition, electromyographic data may have permitted an assessment of the impact on patients of dorsiflexion end points, and application of 10 N-m of force on our equinometer was considered the so-called gold standard against which the clinical diagnosis was compared. We also included diabetic patients, who could be considered to have a form of neurological impairment, and, while we extrapolated data from ankle excursion with varying knee positions as an approximation of gastrocnemius tightness, it is not an exact measurement of actual muscle tension.

In summary, we believe that this report provides the first evidence of isolated gastrocnemius contracture and its potential relationship with foot symptoms in otherwise healthy, nonneurologically impaired individuals. It demonstrated that (1) patients with metatarsalgia or related forefoot and/or midfoot symptoms had less average maximal ankle dorsiflexion with the knee in extension than did a control population without foot symptoms, and (2) when the knee was flexed to 90° to relax the gastrocnemius, this difference was no longer present.

The literature remains confusing regarding the definition of isolated gastrocnemius or combined gastrocnemius-soleus tightness. On the basis of the data obtained in the present investigation, we offer the following standardization of terms to describe the inability to dorsiflex through the tibiotalar joint: gastrocnemius equinus represents maximal ankle dorsiflexion of ≤5° with the knee in full extension, and Achilles tightness represents maximal ankle dorsiflexion of ≤10° with the knee in 90° of flexion. To our knowledge, this paper is also the first to define an approximate prevalence of both gastrocnemius and gastrocnemius-soleus contracture in the general population. Depending on the chosen definition of contracture, roughly three-quarters of the patients with a symptomatic foot and ankle in this study had isolated gastrocnemius contracture and roughly one-third had a combined contracture, whereas approximately one-quarter of the asymptomatic individuals had gastrocnemius contracture and one-sixth demonstrated a combined contracture. Clinical examination was demonstrated to be fairly reliable in identifying muscle tightness.

Although our data are clearly preliminary, such findings may have implications for preventative care and should heighten awareness of the existence and potential long-term effects of tightness of the calf muscles, particularly the gastrocnemius muscle, in patients without neurological impairment. We suspect that this pathological entity plays a vital role in chronic mechanical breakdown or inflammation of both the foot and ankle. ■

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References

1. **Banks HH, Green WT.** The correction of equinus deformity in cerebral palsy. *J Bone Joint Surg Am.* 1958;40:1359-79.
2. **Damron TA, Greenwald TA, Breed AL.** Chronologic outcome of surgical tendoachilles lengthening and natural history of gastroc-soleus contracture in cerebral palsy. A two-part study. *Clin Orthop.* 1994;301:249-55.
3. **Nicoladoni C.** The classic. On the treatment of pes equinus paralyticus. *Clin Orthop.* 1978;135:2-3.
4. **Delpech J.** *Chirurgie clinique de Montpellier; où, observations et reflexions tirées des travaux de chirurgie clinique de cette école*. Paris: Gabon; 1823-8. p 147-231.
5. **Baker LD.** A rational approach to the surgical needs of the cerebral palsy patient. *J Bone Joint Surg Am.* 1956;38:313-23.
6. **Beckerman H, Becher J, Lankhorst GJ, Verbeek AL.** Walking ability of stroke patients: efficacy of tibial nerve blocking and a polypropylene ankle-foot orthosis. *Arch Phys Med Rehabil.* 1996;77:1144-51.
7. **Camacho FJ, Isunza A, Coutino B.** Comparison of tendo-Achilles lengthening alone and combined with neurectomy of the gastrocnemius muscle in the treatment of equinus deformity of the foot associated with clonus in children with cerebral palsy. *Orthopedics.* 1996;19:319-22.
8. **Conine TA, Sullivan T, Mackie T, Goodman M.** Effect of serial casting for the prevention of equinus in patients with acute head injury. *Arch Phys Med Rehabil.* 1990;71:310-2.
9. **Corry IS, Cosgrove AP, Duffy CM, McNeill S, Taylor TC, Graham HK.** Botulinum toxin A compared with stretching casts in the treatment of spastic equinus: a randomised prospective trial. *J Pediatr Orthop.* 1998;18:304-11.
10. **Feehery RV Jr.** Surgery of the achilles tendon and posterior muscle group. *Clin Podiatr Med Surg.* 1992;9:781-811.
11. **Graham HK, Fixsen JA.** Lengthening of the calcaneal tendon in spastic hemiplegia by the White slide technique. A long-term review. *J Bone Joint Surg Br.* 1988;70:472-5.
12. **Javors JR, Klaaren HE.** The Vulpius procedure for correction of equinus deformity in cerebral palsy. *J Pediatr Orthop.* 1987;7:191-3.
13. **McCall RE, Frederick HA, McCluskey GM, Riordan DC.** The Bridle procedure: a new treatment for equinus and equinovarus deformities in children. *J Pediatr Orthop.* 1991;11:83-9.
14. **Olney BW, Williams PF, Menelaus MB.** Treatment of spastic equinus by aponeurosis lengthening. *J Pediatr Orthop.* 1988;8:422-5.
15. **Sindou M, Mertens P.** Selective neurotomy of the tibial nerve for treatment of the spastic foot. *Neurosurgery.* 1988;23:738-44.
16. **Strayer LM Jr.** Recession of the gastrocnemius. An operation to relieve spastic contracture of the calf muscles. *J Bone Joint Surg Am.* 1950;32:671-6.
17. **Silfverskiold N.** Reduction of the uncrossed two-joints muscles of the leg to one-joint muscles in spastic conditions. *Acta Chir Scand.* 1924;56:315-30.
18. **Vulpius O, Stoffel A.** *Orthopadische Operationslehre*. Stuttgart: Ferdinand Enke; 1913. Tenotomie der end schnen der mm. gastrocnemius el soleus mittels rutschenlassens nach vulpius; p 29-31.
19. **Craig JJ, van Vuren J.** The importance of gastrocnemius recession in the correction of equinus deformity in cerebral palsy. *J Bone Joint Surg Br.* 1976;58:84-7.
20. **Sharrard WJ, Bernstein S.** Equinus deformity in cerebral palsy. A comparison between elongation of the tendo calcaneus and gastrocnemius recession. *J Bone Joint Surg Br.* 1972;54:272-6.
21. **Smetana V, Schejbalova A.** [The Strayer surgical technic as the basic operation for treatment of pes equinus in cerebral palsy]. *Acta Chir Orthop Traumatol Cech.* 1993;60:218-24. Czech.
22. **Brodersen A, Pedersen B, Reimers J.** [Foot deformities and relation to the length of leg muscles in Danish children aged 3-17 years]. *Ugeskr Laeger.* 1993;155:3914-6. Danish.
23. **Downey MS, Banks AS.** Gastrocnemius recession in the treatment of nonspastic ankle equinus. A retrospective study. *J Am Podiatr Med Assoc.* 1989;79:159-74.
24. **Grady JF, Saxena A.** Effects of stretching the gastrocnemius muscle. *J Foot Surg.* 1991;30:465-9.
25. **Hillstrom HJ, Perlberg G, Siegler S, Sanner WH, Hice GA, Downey M, Stienstra J, Acello A, Neary MT, Kugler F.** Objective identification of ankle equinus deformity and resulting contracture. *J Am Podiatr Med Assoc.* 1991;81:519-24.
26. **Root ML, Orien WP, Weed JH.** *Normal and abnormal function of the foot: clinical biomechanics.* Vol 2. Los Angeles: Clinical Biomechanics Corp., 1977. p 174.
27. **Sgarlato TE, Morgan J, Shane HS, Frenkenberg A.** Tendo achillis lengthening and its effect on foot disorders. *J Am Podiatry Assoc.* 1975;65:849-71.
28. **Subotnick SI.** Equinus deformity as it affects the forefoot. *J Am Podiatry Assoc.* 1971;61:423-7.
29. **Harris RI, Beath T.** Hypermobile flat-foot with short tendo Achillis. *J Bone Joint Surg Am.* 1948;30:116-38.
30. **Hibbs RA.** Muscle bound feet. *New York Med J.* 1914;100:797.
31. **Mann RA, Coughlin MJ.** Hallux valgus—etiology, anatomy, treatment and sur-

gical considerations. *Clin Orthop.* 1981;157:31-41.

32. **Toolan BC, Sangeorzan BJ, Hansen ST Jr.** Complex reconstruction for the treatment of dorsolateral peritalar subluxation of the foot. Early results after distraction arthrodesis of the calcaneocuboid joint in conjunction with stabilization of, and transfer of the flexor digitorum longus tendon to, the midfoot to treat acquired pes planovalgus in adults. *J Bone Joint Surg Am.* 1999;81:1545-60.
33. **Arkkila PE, Kantola IM, Viikari JS.** Limited joint mobility in type I diabetic patients: correlation to other diabetic complications. *J Intern Med.* 1994; 236:215-23.
34. **Green DR, Ruch JA, McGlamry ED.** Correction of equinus-related forefoot deformities: a case report. *J Am Podiatry Assoc.* 1976;66:768-80.
35. **Hill RS.** Ankle equinus. Prevalence and linkage to common foot pathology. *J Am Podiatr Med Assoc.* 1995;85:295-300.
36. **Katoulis EC, Boulton AJ, Raptis SA.** The role of diabetic neuropathy and high plantar pressures in the pathogenesis of foot ulceration. *Horm Metab Res.* 1996;28:159-64.
37. **Mueller MJ, Diamond JE, Delitto A, Sinacore DR.** Insensitivity, limited joint mobility, and plantar ulcers in patients with diabetes mellitus. *Phys Ther.* 1989;60:453-62.
38. **Simmons RW, Richardson C, Deutsch K.** Limited joint mobility of the ankle in diabetic patients with cutaneous sensory deficit. *Diabetes Res Clin Pract.* 1997;37:137-43.
39. **Lehmann JF, Price R, deLateur BJ, Hinderer S, Traynor C.** Spasticity: quantitative measurements as a basis for assessing effectiveness of therapeutic intervention. *Arch Phys Med Rehabil.* 1989;70:6-15.
40. **Price R, Bjornson KF, Lehmann JF, McLaughlin JF, Hays RM.** Quantitative measurement of spasticity in children with cerebral palsy. *Dev Med Child Neurol.* 1991;33:585-95.
41. **Weaver K, Price R, Cziernecki J, Sangeorzan B.** Design and validation of an instrument package designed to increase the reliability of ankle range of motion measurements. *J Rehabil Res Dev.* 2001;38:471-5.
42. **Davis WL Jr, Singerman R, Labropoulos PA, Victoroff B.** Effect of ankle and knee position on tension in the Achilles tendon. *Foot Ankle Int.* 1999;20:126-31.
43. **Johnson KA, Strom DE.** Tibialis posterior tendon dysfunction. *Clin Orthop.* 1989;239:196-206.
44. **Armstrong DG, Stacpoole-Shea S, Nguyen H, Harkless LB.** Lengthening of the Achilles tendon in diabetic patients who are at high risk for ulceration of the foot. *J Bone Joint Surg Am.* 1999;81:535-8.
45. **Morton DJ.** *The human foot: its evolution, physiology, and functional disorders*. New York: Columbia University; 1935.
46. **Fraser RK, Menelaus MB, Williams PF, Cole WG.** The Miller procedure for mobile flat feet. *J Bone Joint Surg Br.* 1995;77:396-9.
47. **Lin SS, Lee TH, Wapner KL.** Plantar forefoot ulceration with equinus deformity of the ankle in diabetic patients: the effect of tendo-Achilles lengthening and total contact casting. *Orthopedics.* 1996;19:465-75.
48. **Sinacore DR.** Healing times of diabetic ulcers in the presence of fixed deformities of the foot using total contact casting. *Foot Ankle Int.* 1998;19:613-8.
49. **Barry DC, Sabacinski KA, Habershaw GM, Giurini JM, Chrzan JS.** Tendo Achillis procedures for chronic ulcerations in diabetic patients with transmetatarsal amputations. *J Am Podiatr Med Assoc.* 1993;83:96-100.
50. **Frykberg RG, Lavery LA, Pham H, Harvey C, Harkless L, Veves A.** Role of neuropathy and high foot pressures in diabetic foot ulceration. *Diabetes Care.* 1998;21:1714-9.
51. **Ward ED, Phillips RD, Patterson PE, Werkhoven GJ.** 1998 William J. Stickel Gold Award. The effects of extrinsic muscle forces on the forefoot-to-rearfoot loading relationship in vitro. Tibia and Achilles tendon. *J Am Podiatr Med Assoc.* 1998;88:471-82.
52. **Fulp MJ, McGlamry ED.** Gastrocnemius tendon recession. Tongue in groove procedure to lengthen gastrocnemius tendon. *J Am Podiatry Assoc.* 1974; 64:163-71.
53. **Whittle M.** *Gait analysis: an introduction.* 2nd ed. Oxford: Butterworth-Heinemann; 1996. p 58.