CASE STUDY:
Gastrocnemius Equinus
By: Dr. Greg Laakmann, DPM

One of the more challenging problems I see in my office are patients with symptomatic pathomechanics related to an equinus deformity. Regardless of the related diagnosis, the biomechanics in these individuals are complex, particularly when contemplating foot orthoses in the treatment plan. With musculotendinous equinus, I generally tend to use a more forgiving or softer device rather than the more traditional semi-rigid, controlling orthosis. As with any treatment plan, prescription foot orthotic therapy is merely one component of a comprehensive approach to addressing the patient’s complaints; additional interventions may include strengthening exercises, stretching, physical therapy, medications, activity modifications, and appropriate shoe gear selection (home, work and play).

Case Presentation

A male runner (39 years old) presented to the office who had been suffering shin splints for the three weeks leading up to the visit. The patient had been gradually increasing his mileage over a six month period, including an increase in the frequency and intensity of speed and hill workouts. Progressive pain was noted at the anterior aspect of each leg during running sessions, with the onset of pain beginning earlier with each successive run. The patient had been a runner for the past four years and had been running in a motion control running shoe. Typically he replaced his shoes every 800 km. Patient also relates a remote history of “heel pain” that he experienced twenty years earlier.

Lower Extremity Physical Exam

Physical exam revealed:

Non-weightbearing:
- Neurovascular status intact
- Dermatological exam unremarkable
- 4° flexible forefoot valgus
- Stable 1st ray range of motion (ROM)
- 1st MTPJ ROM: 70° dorsiflexion (DF) and 0° plantar flexion (PF)
- Point tenderness along the anterolateral border of the tibial crest
- No pain to palpation of the plantar fascia, calcaneus or Achilles tendon
- Midtarsal and subtalar joint ROM - WNL
- Positive Silfverskiold test (ankle joint DF 0° with knee extended and 10° with knee flexed)
- Manual muscle testing of the anterior muscle groups (AT, EHL, EDL) reveals grade 5/5 and no pain B/L

Weightbearing:
- RCSP: 2° eversion
- NCSP: 2° inversion
- Negative Coleman block test
- WNL 1st MTPJ DF with 1st ray loading
- Gait evaluation revealed: partial arch collapse; medial rearfoot translocation; adductory twist; early heel off; and extensor substitution
- Shoe gear reveals: adequate integrity with mild excessive wear throughout the sole (FF > RF); and scuffing noted at anterior toe box.
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Assessment

1. Anterior lower extremity myofasciitis secondary to gastrocnemius equinus
2. Compensated forefoot valgus
3. Rearfoot varus with abnormal pronation
4. Query history of plantar fasciitis

Plan

As with any biomechanical imbalance, it is particularly important to address the main etiological contributing factor, which in this case was gastrocnemius equinus. The patient was instructed on daily gastrocnemius stretches performed following light activities. A neutral shoe with single density midsole and a higher heel height was recommended. Training regimen was temporarily modified to avoid speed and hill workouts with regular flat-terrain training allowed at slightly slower and shorter distances than normal and to tolerance and pain avoidance. Localized icing to the anterior lower extremity was also instituted. Night splints could have also been considered but were not used in this instance. Note for recalcitrant and severe gastrocnemius equinus deformities with associated compensatory symptoms, one may also need to consider the risks and benefits of a gastrocnemius recession.

Alternatively, one could also utilize another device with a semi-flexible shell or a reverse Morton’s extension in lieu of the extrinsic forefoot valgus post extended to sulcus.

As both midtarsal and subtalar pronation have components of dorsiflexion within the foot, both these motions will assist in compensating for a gastrocnemius equinus imbalance. Having the orthoses maximize midtarsal pronation will decrease the negative effects of the equinus deformity (anterior lower extremity muscle overuse) without creating any additional secondary issues. Posting the calcaneus to neutral but using a more forgiving device will also allow some (but not full) STJ pronation and thus some assistive pseudodorsiflexion. Keep in mind that this patient may have had a remote history of plantar fasciitis so I tend to lean away from allowing full maximum STJ pronation.

Differential diagnoses would include anterior tibial stress syndrome or anterior compartment syndrome, although I would tend to see these in a more rigid, pes cavus individual with an equinus deformity and an associated negative Silfverskiold test.

Proper training and stretching along with appropriate running shoe selection is imperative in minimizing complications in runners of all distances and levels. Understanding the complex biomechanics and interactions of the feet and legs can allow for the successful use of suitable prescription foot orthoses which can be a valuable adjunct in resolving symptoms and preventing future compensatory problems (and keeping your running patients running).

Figure 1: Standard Mold

From a functional orthoses standpoint, the author prefers a more forgiving, softer device in runners unless there is considerable and excessive abnormal pronation without an equinus deformity or coalition. In this instance, a Standard Mold device was prescribed. The calcaneus was posted to vertical and the FF posted to 4° of valgus (2° intrinsically and 2° extrinsically extended to sulcus). Minimal arch fill and a loose 3 mm heel lift were also used with the intent of removing the heel lifts once the equinus deformity resolved.
CLINICAL BIOMECHANICS: Isolated Gastrocnemius Equinus
By: Christopher MacLean, Ph.D.

INTRODUCTION

It is interesting when you review the literature that there is very little information on Gastrocnemius Equinus. The majority of the research has focused on gastrocnemius-soleus complex inflexibility in spastic and neurologically impaired individuals. Very little attention has been paid to this condition in otherwise healthy individuals who present with foot related complications. In healthy individuals, gastrocnemius equinus has been associated with a number of foot-related pathologies (Table 1) (Aronow et al., 2006; DiGiovanni et al., 2002 & 2007; Lavery et al., 2002; Richie, 2007).

Table 1: Foot-related pathology/deformity associated with Gastrocnemius Equinus in otherwise healthy individuals:

- Plantar fasciitis
- Achilles tendinopathy
- Forefoot ulceration in diabetes
- Metatarsalgia
- Adult acquired flatfoot
- Hallux valgus
- Ankle instability

Increased pressure under the forefoot as a result of ankle equinus has been studied to a great extent due to its potential role in the development of ulcers in the diabetic foot (Lavery et al., 2002). Additionally, reduced stride length and an abducted angle of gait may be strategies employed to the dorsiflexion requirements (Kirby, 1997).

Aronow et al. (2006) have also described that the muscles of the anterior compartment of the leg may be recruited to increase active dorsiflexion and to overcome the inflexibility of the muscles of the posterior compartment.

Probably the most important compensatory mechanism as it relates to pathology is that of increased dorsiflexion in the joints of the hindfoot and midfoot. This would typically occur as the subtalar joint pronates unlocking the talonavicular and calcaneocuboid joints (Aronow et al., 2006). Over time, this may lead to a loss of ligamentous integrity in the spring ligament, long and short plantar ligaments and plantar aponeurosis (Richie, 2007).

It has been suggested that there a number of compensatory mechanisms that occur secondary to an isolated gastrocnemius equinus. Clinically, it is likely that the predominant deforming force in individuals with structural breakdown and chronic pathological conditions of the foot and ankle is produced by the gastrocnemius muscle.

Early heel off is perhaps the most commonly cited mechanism and involves prolonged loading of the metatarsal heads and potentially, the development of metatarsalgia and/or plantar tyromas (Aronow et al., 2006; Kirby, 1997).