Hyperpronation and Foot Pain

Steps Toward Pain-Free Feet

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In Brief: Primary care physicians often see patients who have foot pain. Although foot disorders may have many diagnostic possibilities, the majority can be explained via the pathologic biomechanics of hyperpronation and the resulting changes in the kinetic chain. Four common problems often associated with hyperpronation are plantar fasciitis, posterior tibial tendon dysfunction, metatarsalgia, and hallux valgus. Interventions that seek to reduce hyperpronation and strengthen foot muscles are often recommended for treating foot pain.

Foot pain is an extremely common problem. Exact prevalence rates in the general population are unknown, but various small studies in women report rates between 32% and 80%. Evaluating and diagnosing foot pain can be daunting for physicians because of the wide array of conditions that can cause discomfort. One article listed 49 different possible diagnoses for subcalcaneal heel pain alone. Although diagnoses may differ, hyperpronation (ie, pronation too early in the gait cycle) is likely an associated event. An understanding of the causes and effects of hyperpronation will greatly assist the evaluation and treatment of patients who have foot pain.

Defining the Problem

The discussion of foot disorders begins with an understanding of terminology and a review of the gait cycle. Foot pronation and supination are active processes that must be distinguished from pes planus and pes cavus, which are terms describing a static foot. Pronation entails calcaneal eversion, a downward migration of the midfoot, then forefoot abduction and dorsiflexion. With supination, the calcaneus inverts, and the forefoot adducts and plantar flexes.
Pes planus signifies a flatfoot and pes cavus denotes a hollow foot. While pes planus is typically described from visual observation alone, the actual definition depends on the metatarsal bones losing their normal longitudinal arch. Thus, people with hypertrophied muscles on the plantar surfaces of their feet, such as lifelong barefoot walkers, can be mistakenly viewed as having flatfeet, when, in fact, their bones maintain a normal longitudinal arch. The term "flexible flatfoot" describes an arch that is high when unloaded but flattens with standing if weight bearing does not cause calcaneal eversion. Hyperpronation occurs if weight bearing causes calcaneal eversion, in which case the static property of the foot cannot be clinically specified. The bottom of a "fixed" flatfoot remains flat whether the patient is sitting or standing.

**The Gait Cycle**

The normal gait cycle begins with a heel strike, and then very brief supination with force moving forward. This action is followed by pronation of the foot, whereby the weight becomes distributed over the midfoot, and finally a toe-off (figure 1). Toe-off is associated with a brief supination (ie, calcaneal inversion) caused by the windlass mechanism of the medial longitudinal arch. In normal ambulation, the force of the body is transmitted over the toes with approximately one third of body weight going over the first toe, and one sixth of body weight going over each of the lateral four toes.

Individuals who lack physiologic pronation are said to be supinators, and their feet have difficulty absorbing the shock of weight bearing. The weight does not disseminate over the middle part of the foot, but rather remains on the bony lateral side. Much more commonly, people who have foot pain lack the initial supination, and thus pronate too early in the gait cycle (ie, hyperpronate).
What Causes Hyperpronation?

With too-early pronation, the force is transmitted medially while the weight is still on the hindfoot and proximal midfoot. The medial longitudinal arch loses height, and that may set up a cascade of biomechanical problems related to the causes and effects of hyperpronation (figure 2).

Biomechanically, the precipitating events in hyperpronation can be viewed in relation to the position of the talus. Although kinetic chain reactions occur from the hip down to the foot, the interdependent relationships of the talus, calcaneus, and navicular are especially important. A key point is that the talus does not simply sit atop the calcaneus; rather, it is positioned anteriorly and medially on the calcaneus (figure 3). The talus contacts the anterolateral edge of the proximal navicular bone, the most superior bone of the medial longitudinal arch. The talus has no tendinous attachments and thus depends on the static support of surrounding ligaments and bones. Malposition of one bone affects the adjacent proximal or distal bone.
The position of the calcaneus is greatly determined by the Achilles tendon. The Achilles tendon inserts onto the calcaneus slightly lateral to midline. A tight Achilles provides not only plantar flexion, but also eversion to the calcaneus. Both of these actions translate force medially on the talus and downward and medially on the navicular, possibly causing subsequent loss of height of the medial longitudinal arch.

The position of the talus is supported distally by the navicular bone. In standing, the navicular bone maintains its position high on the medial longitudinal arch through the static support of surrounding bones and ligaments. A natural alignment between the talus and the navicular and a spring ligament (ie, the calcaneonavicular ligament) adjoining these bones locks the foot in place (figure 4). With ambulation, dynamic support from the posterior tibial tendon (PTT) is needed to maintain the superior position of the navicular. A weak PTT is unable to support the position of the navicular, and, once again, a loss of the medial longitudinal arch may occur.
Collapse of the medial longitudinal arch everts the calcaneus in relation to the talus; that is, the foot pronates. This may stretch the soft-tissue structures located posterior to the medial malleolus (namely, the PTT and the posterior tibial nerve), manifesting as posterior tibial tendinopathy or posterior tibial nerve entrapment. A collapsed arch can also stretch the spring ligament and plantar fascia, producing plantar fasciitis. With calcaneal eversion (pronation), the forefoot abducts and increases force through the medial rays, which can result in problems over the first ray, such as hallux valgus, and over the second ray, such as metatarsalgia.

Modern society, with our rising obesity and dependence on footwear, may contribute to more people having hyperpronation and subsequent foot pain. Obese individuals have an altered gait with more extensive rearfoot eversion. Heavier body weight results in higher plantar pressures, with the largest effect under the longitudinal arch and metatarsal heads. Greater foot pronation occurs when wearing shoes than when walking barefoot. Shoes elevate the calcaneus, shorten the Achilles tendon, and effectively splint the foot, thereby limiting muscle contraction during ambulation. Extensive observational data suggest that wearing shoes in childhood is detrimental to the development of a normal longitudinal arch and that shoeless populations have less chronic foot pain.

**Examining the Feet**

When evaluating patients who have foot pain, it is essential to view their bare legs and feet from at least the midcalf down. The feet are examined when patients are seated with their feet off the ground, standing, and with ambulation. The "too many toes" sign (when the examiner can see the lateral four toes as the patient walks away) is often attributed to hyperpronation and forefoot abduction (figure 5). While this is generally the case, visualization of the lateral four toes may also result from more proximal causes, such as external rotation of the hip (eg, in the gait of classic dancers) without hyperpronation of the feet. More emphasis should be placed on viewing the Achilles tendon and noting if calcaneal eversion occurs. With the patient walking toward them, clinicians should observe if the medial longitudinal arch is maintained or whether the navicular bone seems to drop toward the floor with midstance.
It is also essential to note the strength of the PTT and the flexibility of the Achilles tendon. The PTT can be assessed by observing the patient while he or she does heel raises. Normally, the calcaneus inverts and the foot supinates with heel raises. Calcaneal eversion is often a sign of a weak PTT. Achilles tendon flexibility testing should be done with both a bent and a straight knee to differentiate the soleus (evident when knee is bent) from the gastrocnemius. The opposite foot can be used for comparison. It is important to place the subtalar joint in neutral alignment, and then apply a laterally directed force to the talar neck while pushing the forefoot medially to lock the foot (figure 6). Otherwise, calcaneal eversion or forefoot dorsiflexion may give a false impression of Achilles flexibility.

**Common Foot Disorders**

The deleterious effects of hyperpronation have been implicated in four common disorders of the feet. It is important to note that, while we discuss the physiologic plausibility whereby hyperpronation may lead to foot pain, prospective data are lacking.
**Plantar fasciitis.** The most common cause of hindfoot pain, plantar fasciitis results from a degeneration of the fibrous aponeurosis that courses the medial longitudinal arch. Patients report pain, generally near the distal medial border of the calcaneus, that is most prominent with the first step after a long rest. Plantar fasciitis is seen in patients who have a rigid cavus foot and in those who hyperpronate, and either deformity may increase the stress on the plantar fascia. Excessive body weight, genu valgus, and gastrocnemius-soleus contracture are all associated with increased pronation of the feet and are known precipitants of plantar fasciitis. 3,8

Treatment options have historically included rest, physical therapy, ice, heat, heel cups, pads, splints, shoe modification, orthoses, nonsteroidal anti-inflammatory medications, injections, and surgery. Of all the treatment options, the most consistently positive results have come from Achilles stretching programs, both active and passive, using night splints. 9,10 Exercises to stretch and strengthen the Achilles tendon are an effective means of decreasing hyperpronation, thus reducing pain. A large study 8 found that the use of heel cups, which raise the calcaneus (the opposite of Achilles tendon stretching), was the least effective of the various treatments evaluated.

**Posterior tibial tendinopathy.** The main insertion for the PTT is on the medial navicular bone. The PTT is essential for initiating inversion and thus counteracting the forces of too-early pronation. Proper functioning of the PTT is necessary for dynamic stabilization of the medial longitudinal arch. A weak PTT cannot maintain the usual talonavicular alignment. The talonavicular joint capsule and plantar spring ligament will stretch out with time. With the resulting dorsolateral subluxation around the talar head, the midfoot is "unlocked," taking away the ability to push off. PTT dysfunction can cause compensatory forefoot supination, leading to problems such as hallux valgus and metatarsalgia. Thus, a strong PTT is necessary to protect against hyperpronation. Additionally, hyperpronation from other causes may result in PTT weakness, because the tendon becomes overstretched.

PTT dysfunction, once considered a rare entity, is now recognized with increasing frequency, perhaps because of increasing obesity and improvements in diagnostic techniques. 11 Patients who have PTT dysfunction may report pain along the tendon's course, or pain more distally caused by changes in forefoot biomechanics. In the later stages, the lateral ankle may sustain compressive forces.

Treatment in the early stages focuses on correcting the root cause of the hyperpronation, with an emphasis on mitigating gastrocnemius-soleus tightness and strengthening the PTT. Correcting PTT dysfunction early in its course may help avoid surgical intervention later. 11,12 If attenuation occurs, it is exceedingly difficult to treat conservatively.

**Hallux valgus.** Lateral deviation of the proximal phalanx on the first metatarsal head often leads to a painful medial eminence, or bunion. Most patients who have hallux valgus have a genetic predisposition that combines with developmental changes to cause hyperpronation and excessive force on the first ray. Hallux valgus is almost exclusively found in shoe-wearing societies. 13 With rare exception, shoes elevate the heel in relation to the midfoot, producing a downward and medial force on the talus. A tight Achilles tendon has also been implicated. 13

Conservative treatment options include digital splinting, wearing wide-toed shoes, and
stretches the Achilles tendon.

**Metatarsalgia.** Pain over the metatarsal heads without any other obvious diagnosis, such as a fracture, corn, or infection, can be termed metatarsalgia. The second metatarsal head is most frequently involved. Normally, weight is distributed over the toes in a fanlike pattern, with the first toe taking one third of the weight and the rest of the toes equally dividing the remaining two thirds. With hyperpronation, the forces move medially, and the second metatarsal may assume an excessively large percentage of the force. Additionally, weak flexor tendons place extra force on the metatarsal heads. General treatment recommendations include strengthening the plantar muscles, wearing shoes with low heels and wide toe boxes, and, occasionally, using orthoses.

**Treating Foot Pain**

Each patient needs to be assessed individually, but some general recommendations seem to apply to many patients who have foot pain. Low-heeled shoes, Achilles tendon stretching, wide-toed shoes, foot strengthening exercises, and weight loss (if needed) are common suggestions for foot pain associated with hyperpronation.

**Altering calcaneal position.** High-heeled shoes increase force on the forefoot. Low-heeled shoes and Achilles tendon stretching decrease the equinus force from an elevated calcaneus. The talus sits both anteriorly and medially atop the calcaneus, and thus any forward tilt to the calcaneus produces a medially directed downward force on the navicular, causing excessive pressure on the medial longitudinal arch. This changes the forces through the midfoot and forefoot, putting extra stress on the medial digits. A tight Achilles tendon enhances the medial force, because it inserts on the lateral side of the calcaneus. For years, Achilles tendon stretching has been a nearly ubiquitous recommendation in the treatment of foot pain, but a recent study by DiGiovanni et al was the first to document that those with midfoot and forefoot pain had a tighter gastrocnemius tendon than controls who did not have foot pain.

**Increasing muscle strength.** Weak foot muscles may contribute to foot pain. Wide-toed shoes and foot-strengthening exercises are prescribed. Axial loading applied to cadaveric feet without simulating the activity of the plantar flexors causes foot pronation and medial longitudinal arch collapse. Wide-toed shoes allow the flexors to contract, and thereby strengthen, the surrounding musculature. Foot-strengthening exercises, such as toe curls and heel raises, strengthen the PTT, peroneus longus muscles, and the flexor tendons. Walking barefoot, if acceptable to the patient, is a functional method to strengthen foot musculature. In fact, less foot pronation occurs when running barefoot than when wearing shoes.

Foot strengthening and weight loss (for overweight and obese patients) may help prevent chronic overload of the metatarsals that can lead to stress fractures and stress reactions. Foot musculature decreases with normal aging, and this may add to the effects of obesity to cause foot pain. Cadaveric studies have demonstrated that contraction of the flexor hallucis longus and the flexor digitorum longus pedis each decrease force on the metatarsals and, theoretically, prevent bony stress reactions. Strengthening exercises help to preserve the strength of the contractions. Given that excessive body weight results in extra pressure and hyperpronation, weight loss should be encouraged for overweight patients who have foot pain.
A Note of Caution

Not all foot pain stems from hyperpronation. We believe that most cases of foot pain in the general population can be attributed to hyperpronation, but a lack of normal pronation has its own set of problems, namely, difficulty in absorbing the shock of weight bearing. The concept of hyperpronation may be novel to some medical providers, but it is frequently cited in running magazines as the major cause of foot pain. It is not uncommon for us to see runners who lack physiologic pronation and yet believe that their lower-leg or foot pain is caused by hyperpronation. They often are wearing custom rigid orthoses meant for hyperpronators. We recommend gently informing them that their rigid orthoses do not seem to be working and encouraging a trial of increased foot flexibility and strength.

Linking the Kinetic Chain

Foot pain is an extremely common problem, and the incidence will likely increase as our population ages and grows more obese. Although diagnoses may differ, hyperpronation is implicated as a common cause, and treatment recommendations are generally geared toward reducing hyperpronation. Helping patients understand the causes and effects of hyperpronation will increase their compliance with treatment recommendations, such as Achilles tendon stretching, foot strengthening, weight loss, and appropriate footwear.

References

12. Coetzee JC, Hansen ST: Surgical management of severe deformity resulting from
posterior tibial tendon dysfunction. Foot Ankle Int 2001;22(12):944-949

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