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Understanding The Biomechanical Effects Of Hallux Limitus

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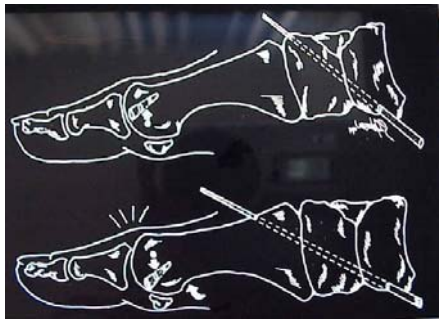

Given the multifaceted nature of hallux limitus, having a strong understanding of the subject is vital for anyone who treats the feet. Indeed, hallux limitus is part of the discussion for a variety of conditions including hallux rigidus, hallux valgus, first MPJ arthritis, osteochondritis and first ray hypermobility. There are countless treatment solutions for hallux limitus depending on the nature of the pathology.

Can one make any sense of the variety of topics related to hallux limitus? Is there a common ancestor to this pathology? If we know more about hallux limitus, is there a way to improve clinical outcomes? Can we at least agree on a common terminology and definition?

In order to address some of these questions, let us take a closer look at the terminology and the pathomechanical algorithm for the progression of pathology to deformity, and review related research that has an impact on the clinical treatment of hallux limitus. I will also try to explain the dysfunction or pathomechanics of the first metatarsophalangeal (MPJ) joint. This will help one better understand the deformities of hallux rigidus (HR) and hallux abducto valgus (HAV), and the related problems of sub-hallux ulcers, recurrent bunions and pinch callus.

Having a better understanding of this pathology and deformity may lead to a better interpretation of the literature and the treatment options, regardless of whether one is utilizing a surgical or a conservative approach. Direction from the literature always helps but understanding the data is essential.

Defining Hallux Limitus

Unfortunately, clinicians commonly use the term hallux limitus to define a pathology, a deformity and a clinical sign when one should only use the term to define a pathology. In regard to hallux limitus, there are actually two different pathologies: structural hallux limitus and functional hallux limitus.

The term structural hallux limitus (SHL) refers to a pathology involving the lack of hallux dorsiflexion with non-weightbearing and weightbearing. Structural hallux limitus usually originates with trauma that disrupts the joint, consistently limits motion and usually progresses rapidly to the deformity of hallux rigidus.

The other hallux limitus pathology is functional hallux limitus (FHL). This pathology, first described in 1972, exhibits a normal range of hallux dorsiflexion during non-weightbearing but there is a dramatic decrease in hallux dorsiflexion during weightbearing.1

One can consider both SHL and FHL as pathologies that progress to deformities. Structural hallux limitus always progresses to the deformity of hallux rigidus and FHL progresses to either hallux rigidus or hallux valgus. The common characteristic of both pathologies is the decreased ability to dorsiflex the big toe joint when the heel comes off the ground in gait, forcing the joint to move when there is limited motion. This causes the deterioration of the joint surfaces through increased periarticular pressure and tension, leading to subsequent subchondral destruction, inflammation and osseous proliferation.

It is easy to imagine the progression of structural hallux limitus from a fractured proximal phalangeal base, sesamoid fracture or prolonged exposure to an arthropathy but how does the progression occur from functional hallux limitus to hallux valgus? To understand this, we have to keep the pathology/deformity terminology straight and follow a simple pathomechanical algorithm.

Hallux valgus and hallux rigidus are deformities that originate from the effects of the pathology of functional hallux limitus. It is important to understand this distinction because a good clinician directs his or her attention to the deformity and the pathology.

Focusing only on deformity without consideration for the original pathology often results in a recurrence of the original problem after treatment.

What The Research Reveals About The Biomechanical Impact

Many have investigated the mechanics of FHL for the past decade and a half, and have expressed theories about the exact normal and abnormal mechanism of the first MPJ. However, researchers have only recently published experiments that clearly explain the process.

It is simple to understand the mechanics of how trauma can limit hallux dorsiflexion in SHL. Cartilage degeneration, subchondral atrophy and osteophytic lipping are all logical extensions of trauma. How can a joint have limited range of motion when a patient is in stance but have a full range of motion when the foot is non-weightbearing?

This phenomenon of FHL as a pathology was the topic of an experiment published in 1996.3 The hypothesis of the research was that the axis of motion in the joint, the hinge pin, moved lower in the metatarsal head when the first metatarsal was dorsiflexed, thus jamming the joint. The more the first metatarsal dorsiflexed, the less the joint was allowed to dorsiflex. Actually, this did not happen in just some of the patients in the experiment but in every one of them. Every normal foot can demonstrate FHL by dorsiflexing the first ray. Eight mm of elevation of the first ray produced an average 35 percent decrease of hallux dorsiflexion.

What could be causing the first ray to dorsiflex in patients with FHL? For many years, researchers believed it was “pronation” and first ray “hypermobility.” If it were pronation, why doesn’t everyone who is maximally pronated demonstrate FHL? Why do some patients who are not maximally pronated demonstrate FHL? Further, if FHL is the precursor to HAV and HR, why don’t all pronated feet develop some degree of hallux abducto valgus or hallux rigidus?

The present theory of the pathomechanics or origins of HAV and HR discounts pronation as the simple cause of the deformities and proposes three foot types that increase ground reactive force under the first metatarsal head and subsequently dorsiflex the first ray.

These foot types include everted calcaneus, flexible forefoot valgus and plantarflexed first ray. These three foot types cause greater pressure under the first metatarsal head and do so sooner in the gait cycle with greater force and for a longer period of time.

If this theory is correct, why doesn’t everyone with these three foot types develop FHL and subsequently HAV and HR? This is where the flexibility of the foot at the midtarsal joint plays a role. When ground reactive force from the three foot types attempts to dorsiflex the first ray, sometimes the midfoot collapses instead of the first ray dorsiflexing. Yes, you can have a flatfoot without HAV or HR. This is the theory of proximal stability at work. Only feet with the three foot types and a stable midtarsal joint have a dorsiflexed first ray and all of them have FHL to some degree.

Why do some people who have the three foot types, a stable midtarsal joint and a dorsiflexed first ray get HAV from forcing a limited joint to move at heel lift and others get HR? Remember, we need just as much dorsiflexion of the big toe joint as we do the amount of heel lift.

The work on this answer occurred in 1982.4 This study proved that when the first ray dorsiflexes, it also inverts the axis of the big

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First MPJ Pathology: What You Should Know

How can we prove that this theory is true or valid, and how can we apply it to the treatment of the big toe joint deformities? This was the subject of an experiment that was recently published.1 The experiment started with the premise that there are two essentials for pathology of the first MPJ joint. These essentials are as follows:

- a deforming force that drives the first ray into a dorsiflexed position, limiting hallux dorsiflexion at heel lift; and
- a limited range of motion of the midtarsal joint or, in other words, not enough motion to compensate for the deforming force.

If these two essentials were present, then FHL would always occur and always progress to either HAV or HR.

Researchers designed a protocol to test this concept in stance on humans with FHL and also in gait. Researchers wanted to find out if they could reverse the process of FHL by repositioning the first ray plantarly and increasing the range of hallux dorsiflexion in patients.

In the study, researchers evaluated 27 patients (48 feet) with greater than 50 degrees of dorsiflexion with non-weightbearing but 12 degrees of dorsiflexion with weightbearing. The study authors cast the feet for orthoses but had the patients' first ray plantarflexed to its end range of motion. The patients subsequently underwent testing and standing on a weightbearing goniometer in order for researchers to determine their hallux dorsiflexion with and without the orthoses device under their feet.

Amazingly, 100 percent of the patients increased their range of motion. The average increase in hallux dorsiflexion was 8.5 degrees or, considering the difference between with and without orthoses, an average of 93 percent. Without orthoses, patients had an average dorsiflexion of 9.7 degrees of hallux dorsiflexion while those with orthoses had an average of 18.7 degrees of hallux dorsiflexion. This is a substantial and significant increase in hallux dorsiflexion for clinical purposes.

Not many patients with FHL have discomfort in stance only. Accordingly, study authors designed a second test to evaluate the increase in hallux dorsiflexion by plantarflexing the first ray in gait. The test included the same patients, all in the same shoe, with and without their orthoses.

Researchers measured the determinant of sub-hallux pressure at heel lift. They assumed that in the presence of hallux limitus, heel lift increases sub-hallux pressure. If the orthoses increased hallux dorsiflexion, then one would see less pressure under the hallux at heel lift. The study authors used the Tekscan® insole sensor to measure the hallux pressure at heel lift with and without orthoses. The results of this experiment showed that orthoses designed to plantarflex the first ray in gait decreased the hallux limitus in gait an average of 14.7 percent. The interesting part of this result was that 100 percent of the patients had an increase in big toe joint motion with the orthoses. The greatest increase of dorsiflexion was 49 percent and the least was 2 percent.

Other authors have demonstrated similar results using orthoses or strapping methods but none have related a 100 percent response in their patients.5,6 Those experiments seem to confirm that the position of the first ray produces functional hallux limitus and this is the pathology producing hallux valgus and hallux rigidus. This recent experiment suggests this process may be reversible, or at least attenuated, by plantarflexing the first ray and increasing hallux dorsiflexion.

Applying The Research Findings To Clinical Practice

How can we effectively use this information? Virtually all sub-hallux ulcers are related to hallux limitus.7 Would increasing the range of dorsiflexion in these patients help prevent recurrence or possibly speed healing of the ulcer by removing 14 percent of the ground reactive forces? Perhaps this technique of holding the first ray plantarflexed and increasing hallux dorsiflexion could improve the prognosis of treating sub-hallux ulcers? Might this technique also be a clinical solution to the prevalent problem of hallux pinch callus?

How about bunion surgery? We rearrange the bones and soft tissue of the big toe joint complex, and then put the patient in a postoperative shoe that tends to dorsiflex the first ray. When one reviews the evidence, this does not seem to be the right thing to do postoperatively. Since FHL is a precursor to HAV, perhaps functional orthoses could provide improved clinical outcomes postoperatively in patients who have had bunion surgery. Could this simple change in postoperative protocol help avoid postoperative hallux limitus in some cases?

Our understanding of the mechanical causes of hallux limitus will help guide the decision making process concerning the

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