Four Peer-Reviewed Articles That Every Podiatrist Should Read

These seminal works help define modern pedal biomechanics.

BY PAUL SCHERER, DPM

Misconceptions that shoes or genetics cause common deformities such as HAV (hallux abducto valgus) or HR (hallux rigidus) have been disproved. Pronation of the subtalar joint is not the cause of plantar fasciitis. Advancements have been made in the theories that explain these pathologies, and are clearly revealed in the peer-reviewed literature.

This article will focus on four undervalued published articles that explore these concepts, and should further the understanding of the mechanical origins of foot pathology for every podiatrist. The four papers that will be reviewed and discussed were written by Thomas Roukis, Kevin Kirby, Geza Kogler and Douglas Richie. The articles relate to these topics: hallux abducto valgus, plantar fasciitis, sinus tarsi syndrome, and adult-acquired flat foot. These papers have a much greater implication to our collective understanding of pathology than what is presently common knowledge in our profession.

1. Hallux Abducto Valgus, a New Theorem

Roukis wrote a rarely-referenced article in *JAPMA* which provides a new hypothesis for the mechanical origin of the deformities of hallux abducto valgus (HAV) and hallux rigidus (HR) and provides a better understanding of the pathology of functional hallux limitus. The simple yet significant experiment hypothesized that the sagittal plane motion of the first ray had a demonstrative effect on hallux dorsiflexion; the greater the first ray dorsiflexion in stance and gait, the less the hallux could dorsiflex. The premise of the experiment was that if this effect were true, we might have the answer to why only some people with poor foot mechanics and structure develop hallux valgus and hallux rigidus.

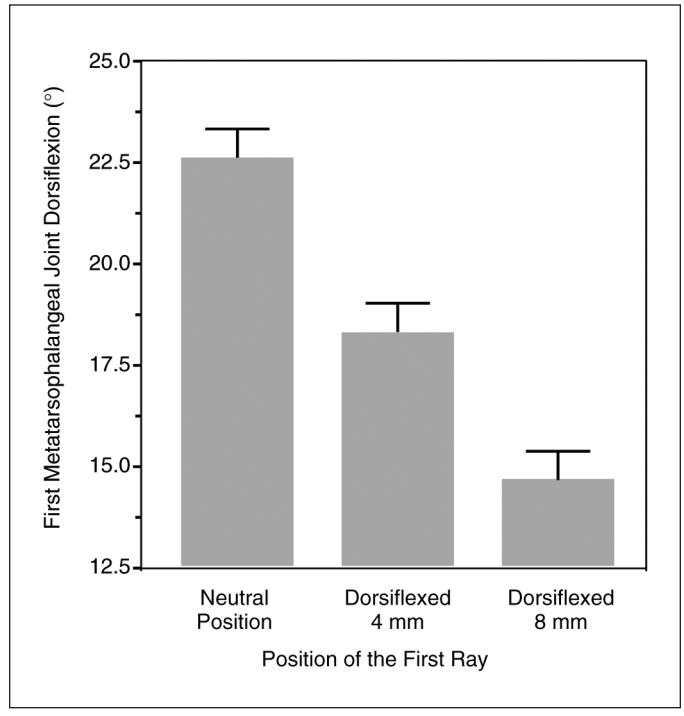

Figure 1: Reprinted with permission of the APMA.

Previous to this experiment, the majority of the literature blamed heredity, osteoarthritis, osteochondritis, avascular necrosis, congenital fragmentation of the epiphysis at the base of the proximal phalanx, or a short flexor hallucis brevis muscle for the lack of dorsiflexion that ultimately led to HAV dislocation or hallux rigidus. Although some literature did suggest that the position of the first ray had an effect on motion of the first metatarso-phalangeal (MP) joint, there was no known quantitative documentation. His experiment involved ten subjects (20 feet).

Each subject had an extensive examination of mechanical characteristics and motion of the foot joints, as

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well as forefoot to rearfoot position and the heel position to the ground. An apparatus was designed to accurately measure hallux dorsiflexion while the subjects were weight-bearing. Dorsiflexion of the hallux was measured without elevating the first ray, and when elevating it by 4mm and 8mm. The data were analyzed to compare the mean degree of dorsiflexion in each elevated position of the first ray.

The results clearly documented the proportionate decrease of first MP joint dorsiflexion (Figure 1) that occurred with increased dorsiflexion of the first ray. Although this study showed a consistent proven effect and inter-relationship of one foot part on the other, the real value of this paper is actually the introduction of the theorem of the pathomechanics of hallux abducto valgus and hallux rigidus, which to date, no other author has yet to question or disprove.

The theory proposed is that the final deformity (HAV or HR) depends on the actual amount of first ray dorsiflexion that occurs during gait, rather than anatomical variances or inherent properties of the first MP joint. The theory also proposed that certain foot types increased ground reactive force (GRF) under the first metatarsal head which leads to the dorsiflexed first ray. These foot types included an everted calcaneus, a flexible forefoot valgus, a plantarflexed first ray, or any combination of these, which ultimately dorsiflex the first ray and decrease motion at the first MP joint, unless compensated by midtarsal joint supination.

An individual with one of these foot types and the resultant decreased ability to dorsiflex the hallux, creates increased compression between the articular surface of the base of the proximal phalanx and the head of the first metatarsal when lifting the heel in gait. The compression creates sufficient force to sublux, or dislocate the joint in HAV, or even traumatize the joint sufficiently to produce hallux rigidus.

Accordingly, the author proposed that the HAV deformities are the end result of a large amount of first ray dorsiflexion, and that hallux rigidus deformities are the end result of a smaller amount of first ray dorsiflexion. The concomitant inversion of the first ray with dorsiflexion plays a major role here.

The significance of this article eluded some of the early makers of the first MP joint implants who didn't include in their design the interrelationship between first ray motion and hallux dorsiflexion. Also, clinicians who use custom orthoses to treat functional hallux limitus could use this concept to affirm the effectiveness of plantarflexing the first ray during negative casting, or incorporating a reverse Morton's extension in the orthotic prescription to increase hallux dorsiflexion.

This concept may also stimulate new treatment options, or at least prophylaxis for sub-hallux ulcerations, since virtually all of these wounds are associated with limited dorsiflexion of the hallux, creating increased shear under the hallux during gait.

The majority of readers probably believe that the etiologic cause of plantar fasciitis is pronation of the foot, because pronation increases the tension on the plantar fascia.

2. Plantar Fasciitis: What Causes the Pain?

The majority of readers probably believe that the etiologic cause of plantar fasciitis is pronation of the foot, because pronation increases the tension on the plantar fascia. A simple demonstration on your next ten patients will show that pronation does not stretch the plantar fascia. Test the tension of a patient's plantar fascia in the neutral position, and then in a pronated position, and you will find the tension to be the same. So when the arch lowers to stretch the plantar fascia, which joint is really moving?

The *Journal of Bone and Joint Surgery (JBJS)* published an infrequently-cited article by Geza Kogler that attempted to explain what foot positions stretch the plantar fascia. Kogler suggested that plantar fasciitis also had a mechanical origin and investigated the foot mechanics that stretch the plantar fascia. His experiment and article led to the theorem that it is the supination of the midtarsal joint as a compensation in patients with forefoot valgus and everted heels that places a stretch on the plantar fascia, which creates a periostitis at its proximal attachment.

His experiment was an in vitro test that simulated static stance in nine fresh cadaver lower limbs. Each specimen was mounted in a testing machine which was previously proven to simulate axial load. A transducer was implanted in the plantar fascia (aponeurosis) to measure and record the amount of strain in each segment of the experiment. The foot was tested under nine different situations to determine which situation produced the most tension on the plantar fascia using varus and valgus wedges placed under the forefoot, the rearfoot, or any combination of the two. The leg was loaded and the strain on the plantar fascia was recorded during each condition for each specimen. This data was compared to the control measures of the same specimen with no wedges underneath.

Most clinicians consider the rearfoot position as an influence in stretching the plantar fascia. Many clinicians also use a varus wedge under the rearfoot to remove tension from the fascia. The data showed no significant difference between the tension on the plantar fascia with the wedge under either the medial side (varus wedge) or the lateral side (valgus wedge) of the rearfoot. These findings were consistent from specimen to specimen. In his paper, Kogler cited five published references where resolution of symptoms was achieved with varus wedges under rearfoot, and questioned why this technique is

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used if it did not reduce tension on the plantar fascia.

The plantar fascia's reaction to forefoot wedging was even more of a curiosity. A valgus wedge under the forefoot decreased strain in the plantar fascia compared to either no wedge or a varus wedge. In fact, the three scenarios where a valgus forefoot wedge was used reduced the tension the most; and conversely the varus wedge used under the forefoot increased the tension the most (Figure 2).

Is there an explanation for this finding? Perhaps the increased tension in the plantar fascia (that causes proximal periostitis at the calcaneal tuberosity) has nearly nothing to do with pronation of the subtalar joint, but rather supination of the midtarsal joint (think inversion of the forefoot on the rearfoot). This would explain the increased tension with a forefoot varus wedge, because it supinates the midtarsal joint. The decreased tension with a forefoot valgus wedge is a result of pronation of the midtarsal joint. This may also explain why we see plantar fasciitis heel pain in non-subtalar joint pronated patients, and even in patients with cavus feet. For these patients, it is their forefoot valgus deformity which has to compensate by inverting at the midtarsal joint when the foot hits the ground that stretches the plantar fascia.

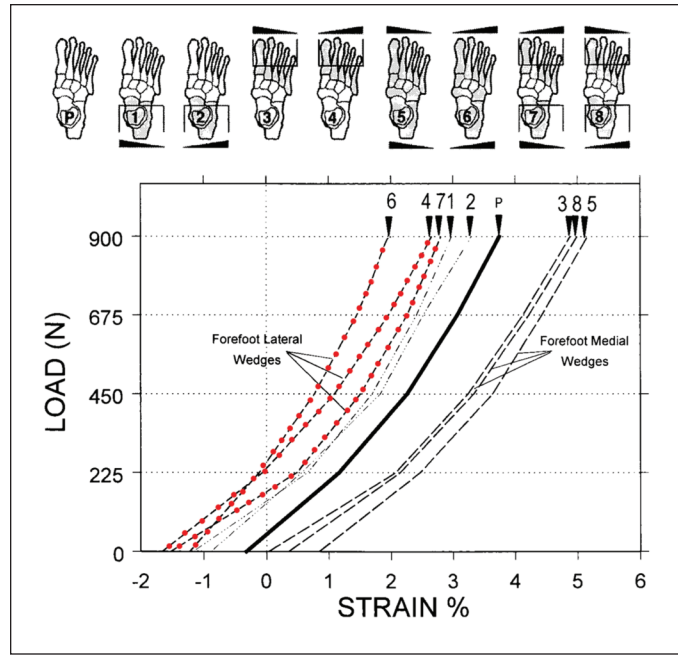

Figure 2

Most importantly, this experiment sheds light on the etiology of mechanically-induced heel pain and possibly, if used properly, can improve treatment outcomes. While Kogler did show decreased strain with forefoot valgus wedging, it is not known how much of a reduction may result in clinical relief of pain. This important piece of information is very valuable when prescribing an orthotic for a patient with plantar fasciitis. Should a clinician add a forefoot valgus wedge if you want to reduce tension on the fascia? Probably, yes! Is it still important to control rearfoot motion? Also yes, because less control of the rearfoot produces supination of the midtarsal joint. This data may also stimulate research to investigate new methods of treating heel pain, since we seem to be much closer to understanding the mechanical origins of this pathology.

3. Rotational Equilibrium, Explaining Foot Pronation

The concept that foot biomechanics is related to foot pathology is relatively recent, starting in the 1950's and gaining traction in the late 1960's. During that time most of the research and educational information focused on the subtalar joint and its motion. Regardless of the correct and incorrect assumptions at that time, most investigators still regard that proper function of the subtalar joint as critical for normal function of the foot. Today, most researchers and some clinicians describe rotational forces called moments that cause motion at the subtalar joint. These moments are primarily supination moments and pronation moments acting across the subtalar joint axis. As more researchers use these terms, clinicians must understand their meaning if new understanding of treatments are to be possible.

In 1989, Kevin Kirby thought that developing a better understanding of the mechanical origins of foot pathology would be achieved by describing these moments. He explained in his *JAPMA* article how the balance of pronation and supination moments determine where the subtalar joint position will fall in stance, by a concept called rotational equilibrium. This concept is important for any clinician who contemplates a mechanical alteration of the foot by calcaneal osteotomies or by prescribing orthoses for sinus tarsi syndrome.

The article contains a brief and understandable explanation of how the forces of a particular muscle or the forces of the ground are converted into moments, and how a surgical procedure could intentionally alter these moments. The anterior advancement of the Achilles tendon done in a Murphy procedure is a good example of reducing the moment of the triceps surae muscle (by moving the attachment forward a specific percentage) in order to decrease pressure under the forefoot during propulsion.

This concept can also be used for understanding why some people have pronated subtalar joints, others have supinated subtalar joints, and why still others are neutral. Consider that the ground exerts a force "up" under the foot on both sides of the subtalar joint axis. The force on the lateral side produces pronation, and the force on the medial side supination. If both moments (the force and the distance from the axis) are equal, the joint achieves rotational equilibrium and the foot stands neutral.

What if the ground forces are the same but the patient is born with a medially deviated subtalar joint axis (Figure 3)? This increases the lateral force area and changes the moments, and now the foot must pronate because the ground is pushing up with a greater advantage on the lateral side

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of the axis. Think just the opposite with a cavus foot, which has a laterally deviated subtalar joint axis or greater force on the medial side of the axis. Clinicians can use this concept to their advantage by attempting to alter the moment on the medial side of the subtalar axis in pronated feet, or by moving the calcaneus more medially with an osteotomy to increase the supination moment.

The concept of a less than ideal location of the subtalar joint axis is not the only way a joint becomes unstable or "not in rotational equilibrium." The moments can be changed by muscle pathology. Consider adult-acquired flatfoot, in which the tibialis posterior cannot exert its necessary force, which results in a decreased supinatory moment, or an overpowering of the pronatory moment, and the foot badly pronates. Based on the rotational equilibrium concept, clinicians can now consider ways to change this by increasing the supinatory moment or decreasing the pronatory moment. Can this be achieved by surgically altering the axis position more laterally? There are a lot of treatment options to think about when gaining an appreciation for this concept.

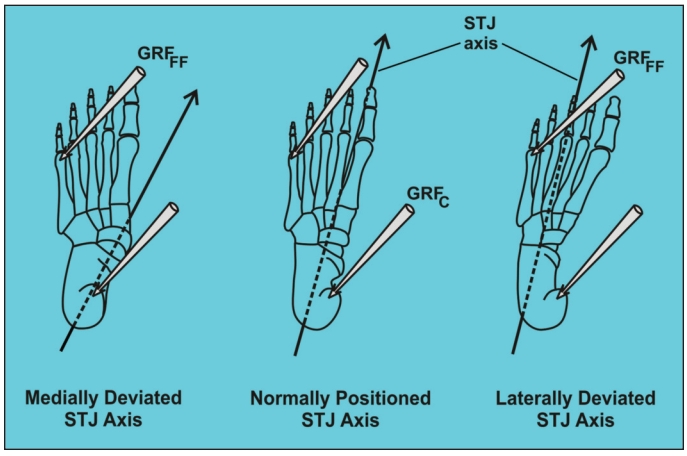

Figure 3

When the subtalar joint is acted upon by a strong pronation moment (or a weak supinatory moment), the joint pronates until the lateral process of the talus comes in contact with the floor of the sinus tarsi. Pronation only stops when the floor of the sinus tarsi absorbs sufficient abnormal compression. Could this excessive compression on bone in some patients, especially when the compression exceeds osseous tolerances, produce the inflammation and pain of sinus tarsi syndrome? The author suggests that this is

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not only true, but that foot orthoses work by creating a supinatory moment which decreases interosseous compression, and reduces symptoms without creating a change in joint position.

This idea may also help explain why some patients benefit more from orthotic therapy than others. If the pronation moment of the foot is greater than the supination moment of the orthoses, little change will occur in the joint or the symptoms. Would thickening the medial side of the heel cup of an orthosis increase the ground force on the medial side of the axis and increase the supination moment of orthoses, therefore increasing their effectiveness? The author thought so and this was the premise for a later article on the medial skive technique.

4. Adult-Acquired Flatfoot, a More Accurate Description

Continuing with the thought that a better understanding of the mechanical origins of a particular pathology will lead to more effective and successful treatment pathways, Douglas Richie wrote an article in *Clinics in Podiatric Medicine and Surgery (2007)* delineating an interrelated sequence of biomechanical events that lead to the deformity called adult-acquired flatfoot. The significance of his theory of event sequence helps us look at the pathology that created the foot problem, rather than only look at the deformity of the foot for treatment options. Considering this approach, we have a greater opportunity to not only provide possibly better outcomes, but also to prevent or slow progression of the deformity.

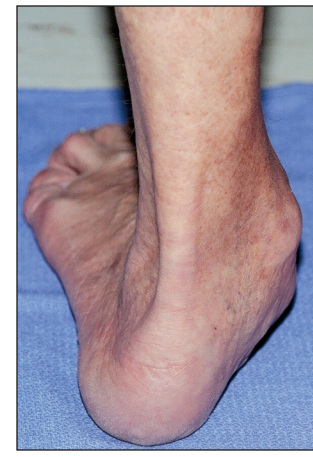

Figure 4

The paper presents significant new discoveries about the pathology and the mechanics that created it, and also provides a concise, interesting, and complete hypothesis of the scenarios that created the deformity. Richie builds a comprehensive review of the pertinent medical literature from Cozen (1965) who first describes the relationship between pronation and the internal mechanical irritation of the tibialis posterior tendon, as well as Goldner's description (1974) of the rupture of the medial plantar calcaneonavicular ligament to explain why surgical failure occurred with simple repair or tenodesis of the tendon.

Few researchers and clinicians, previous to this paper, considered the

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role the peroneus brevis plays as the antagonist to the tibialis posterior in the creation of the progressive adult flatfoot deformity. In addition, Richie was one of the first to consider how the flexor digitorium longus compensates for the loss of the tibialis posterior and could play a role in the future types of treatment plans.

The strongest evidence for building a hypothesis stating that flatfoot deformity may be the etiologic factor, or at least a co-morbidity to this pathology, is Richie's description of Uchiyma's work showing that any flatfoot deformity caused a 30% increase in the gliding resistance of the posterior tibial tendon in its sheath. Another major contribution of this paper is the logical presentation that spring ligament failure is the key event in the pathomechanics of adult acquired flatfoot. Even more significant is his description of the spring ligament complex and its function to control the multi-planar motion of the talocalcaneonavicular joint.

The explanation of the biomechanics of flatfoot deformity alone in this paper should be required reading in schools and the profession for its historical and descriptive nature. Anyone can understand the real, complicated nature of flatfoot deformity from this description in simple, biomechanical terms and how it is applicable to numerous other pathologies; for example, the role of the talonavicular joint as appreciated by studies of arthrodesis of the individual rearfoot joints. Did you know that with fusion of the subtalar joint, much of the remaining rearfoot triarthrodial motion is preserved? The result is the same with fusion of the calcaneocuboid joint, but when the talonavicular joint is fused, virtually all rearfoot motion ceases.

The latter part of the paper builds on the sequence of events in the pathomechanics of adult-acquired flatfoot and uses this scenario, as well as an effective and quantitative examination, to propose joint stabilization procedures, braces, or arthrodesis. The paper is complete, understandable, logical, informative, and immensely relevant to clinical practice.

These four summaries of papers should encourage you to obtain a copy of each and spend time with them. We all see hallux valgus, adult-acquired flat foot, plantar fasciitis, and sinus tarsi syndrome every day. We should be completely knowledgeable in all aspects of the causes of these problems, and not just their treatments. The information is in the literature which explains how the different pathologies develop. You will find a new and stimulating appreciation for the actual concept of hypothesis development, and of the different mechanical origins of foot pathology they present. Through this appreciation more effective therapies may evolve in your office. **PM**

Dr. Scherer is immediate past chair and professor of the Department of Applied Biomechanics at Samuel Merritt College. He is board certified both by American Board of Podiatric Surgery and American Board of Podiatric Orthopedics and Primary Podiatric Medicine. Dr. Scherer has received both public and private grants for his academic work and has published on a wide variety of topics. He is CEO of Pro-Lab Orthotics located in Napa, California.