CHAPTER **39**

ROLE OF THE SESAMOID APPARATUS IN HALLUX LIMITUS/RIGIDUS

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The majority of attention in both the evaluation and treatment of hallux limitus and hallux rigidus has been historically focused on the metatarsophalangeal joint articulation. However, an equally significant degree of deformity is often present at the metatarsal-sesamoid joint level. Moreover, it is the opinion of this author that dysfunction of the metatarso-sesamoid joint is the causative factor which leads to degeneration of the metatarsophalangeal joint. A discussion will be presented which reveals several interesting clinical and radiographic findings in patients with hallux limitus/rigidus. The significance of these findings, with regard to conservative and surgical management will be addressed.

THE METATARSAL-PHALANGEAL-SESAMOID JOINT

The articulation of the first metatarsal to the proximal phalangeal base and the sesamoid apparatus represents a unique mechanical device by which the foot propels the body forward in ambulation. The first metatarsal-phalangeal-sesamoid joint (MPS joint) has been extensively analyzed, both anatomically and biomechanically (Durrant & Siepert 1993). The approximate percentage of total joint articulation between these three osseous structures is as follows: metatarsophalangeal joint (60%), and metatarsal-sesamoid joint (40%). Together, the metatarsophalangeal and metatarso-sesamoid joints articulate with approximately two-thirds of the total joint surface of the metatarsal head at any one point in time. Although a significant percentage of this joint's articulating surface area involves the metatarsal-sesamoid articulation, little attention has been paid to its role in joint dysfunction. In the majority of patients with hallux limitus/rigidus, there is sagittal plane immobility of the joint. Conversely, there is often little instability in the transverse plane, and the sesamoids are typically well-aligned beneath the metatarsal head. This is in contradistinction to the transverse plane instability of the sesamoid apparatus in hallux abductovalgus deformities.

In normal propulsion, the first metatarsal glides over a fixed sesamoid apparatus, permitting the normal degree of closed-chain kinetic dorsiflexion of the metatarsophalangeal joint. The windlass mechanism provided by the plantar musculature and fascia supports the longitudinal arch of the foot in propulsion, and aids in hallux purchase and digital roll-off. This complex and concerted series of events is essential for normal joint function. Any deviation from the norm in this series of events is potentially catastrophic to the articular surfaces of these joints. Considering the repetitive nature of ambulation, it is not surprising that we frequently treat patients with degenerative arthritis of this three-surface, dual-joint structure.

PREDISPOSING FACTORS IN HALLUX LIMITUS/RIGIDUS

Any disruption in the normal cycle of first metatarsal-phalangeal-sesamoid joint function can potentially "throw a wrench" into the mobility of this joint complex. In patients with spasm of the short flexors of the hallux (flexor hallucis brevis), there is a complete locking of the motion in the entire joint. The result is an immobile sesamoid apparatus and flexor plate which may lead to degeneration of the metatarsophalangeal joint surface. A normal first metatarsal-phalangeal-sesamoid joint has an axis of rotation which is located centrally within the metatarsal head (Fig. 1). With locking of the sesamoid apparatus, this axis of rotation is shifted plantarly to the junction of the metatarsal head and the articular surface of the sesamoids (Fig. 2). In this manner, the fulcrum for motion about the joint is inferior to the central axis of the joint, acting as a hinge at the level of the sesamoid-phalangeal ligament. As a result of this shifted axis of rotation, the proximal phalangeal base is prevented from migrating dorsally on the

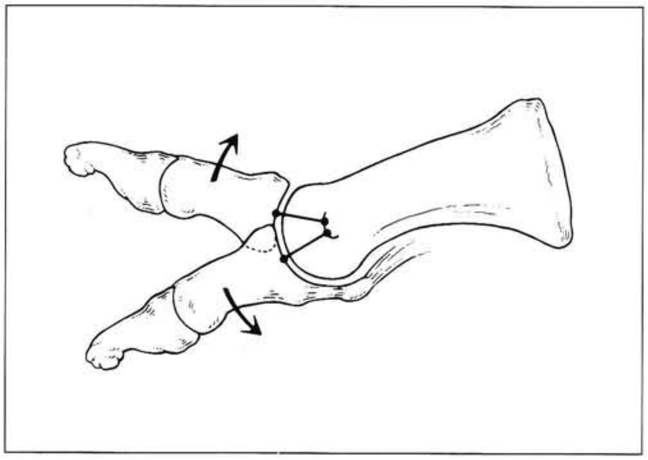

Figure 1. Normal axis of rotation of the first metatarsal-phalangeal-sesamoid joint.

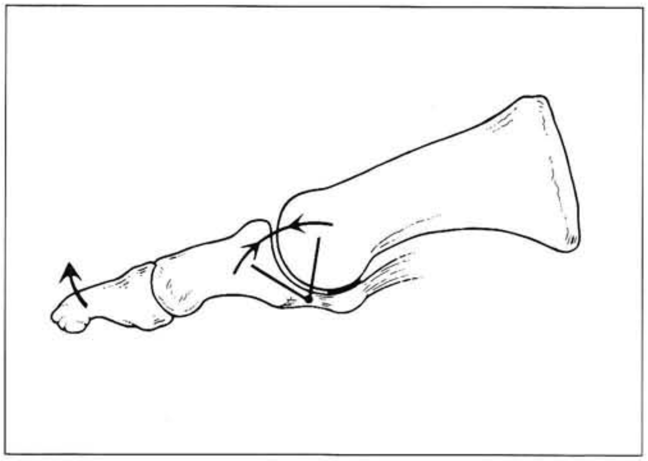

Figure 2. The axis of motion (hinge) occurs at the level of the sesamoido-phalangeal ligament when there is immobility of the sesamoid apparatus, causing impingement of the dorsal metatarsophalangeal joint.

metatarsal head. This sets up an impingement syndrome, by which the dorsal articular surfaces of these two adjacent bones develop articular degeneration, with subsequent arthrosis and joint surface remodeling (flattening, hypertrophy, and dorsal osteophyte formation). As a response to dorsal impingement, the short flexors of the hallux forcefully contract in an attempt to stabilize the joint and prevent motion. This is a cyclic process of joint immobility which further propagates joint immobility resulting in degenerative arthritis. A typical scenario is seen in the patient with an uncompensated forefoot varus. The hallux plantarflexes in an attempt to grip the weight-bearing surface and over time the sesamoid apparatus becomes immobile and dorsal joint impingement occurs. This is followed by spasm of the short flexors causing a "functional" joint fusion. This entire sequence sets the stage for progressive degenerative arthritis, further joint immobility, and ultimately hallux limitus or rigidus.

It is important to realize that arthrosis of the metatarsophalangeal joint represents the end-stage of this disease process. There are, however, earlier clinical and radiographic findings which suggest a precursor-state of this condition. This includes the well-described entity known as functional hallux limitus (Dananberg, 1986). In this early stage condition, the metatarsophalangeal joint is already functioning as an auto-fused joint. Although the joint may have an unrestricted range of motion in open-chain kinetics, as well as no apparent radiographic evidence of arthrosis, examination of the "loaded" or weight-bearing joint reveals total sagittal plane immobility.

Assuming that spasm of the short flexors of the hallux is a precursor to degenerative arthritis, we must then identify what it is that leads to immobility of the flexor-sesamoid apparatus. There are congenital, developmental, acquired, and iatrogenic conditions which predispose to the development of hallux limitus/rigidus. Most of these conditions have the common denominator of an uncompensated forefoot or rearfoot varus deformity. This may present as medial column instability in a collapsing flatfoot (Fig. 3), or secondary to a more proximal locus of uncompensated varus (rearfoot or ankle varus).

A discussion of metatarsal length pattern aberrations as a cause of hallux limitus/rigidus must also be included. An excessively long first metatarsal obligates the medial column to contact the ground

Figure 3. Severe pediatric collapsing flatfoot, with medial column instability, hallux equinus, and an apparent metatarsus primus elevatus.

with excessive pressure beneath the first metatarsal, causing two effects: the first is to prematurely load the joint, thereby causing flexion of the proximal phalanx on the metatarsal head (i.e. premature windlass effect); the second effect is that the long first metatarsal causes a retrograde inversion of the forefoot on the rearfoot, forcing the patient to roll-off on the lateral side of the foot in propulsion. This precludes the normal progression of forces from passing through the first ray, and restricts dorsal displacement of the proximal phalanx on the metatarsal head.

In a similar but opposing manner, a short first metatarsal fails to contact the ground. This leads to flexion of the hallux on the metatarsal, in an attempt to provide medial support for weight-bearing forces. This is often the case in iatrogenic first metatarsal shortening, or with iatrogenic metatarsus primus elevatus. In either instance, the first metatarsal fails to contact the weight-bearing surface, and the hallux compensates by assuming a fixed position of flexion (hallux equinus). Repetitive contraction of the short flexor tendons, in an attempt to stabilize the hallux on the ground, leads to eventual spasm and a fixed contracture of the sesamoid apparatus. A similar process occurs in any form of uncompensated varus, where the medial column fails to contact the ground, and the hallux plantarflexes in an attempt to stabilize the foot.

HALLUX EQUINUS – CLINICAL AND RADIOGRAPHIC FINDINGS

Although there may be only limited or minimal evidence of arthrosis on x-ray, the first metatarsal-phalangeal-sesamoid joint may already be functioning as a fused joint. In addition to an immobile "loaded" joint, there are interesting radiographic findings which reflect this process. A hallux flexus or hallux equinus deformity is the most common of these findings. This has previously been described by Rzonca, Levitz, and Lue (1984) as a stage in the progression of hallux limitus.

Hallux equinus can be readily observed on a lateral weight-bearing radiograph in patients with hallux limitus/rigidus. This is a subtle but consistent finding which often precedes evidence of degenerative arthritis. The hallux assumes a position which places it in direct line with the first metatarsal shaft. The most obvious evidence of this finding is that the dorsal cortical surfaces of the first metatarsal and proximal phalanx assume a straight line position with one another. The proximal phalanx is thus in a position of declination and assumes the same degree of declination as the first metatarsal. With the proximal phalanx "gripping" the ground, the two bones act as one long medial segment, with the interposed metatarsophalangeal joint functioning as a fused entity. This appears to be the body's attempt to fuse this joint on its own. As the joint continues to function in this manner, and as the sesamoid apparatus remains immobile through the chronic and constant spasm of the short flexor tendons, the joint progresses to total destruction.

Also of interest is the effect that this "functional fusion" of the first metatarsal-phalangeal-sesamoid joint has on the appearance of the rest of the foot. As the hallux assumes a fixed position of plantarflexion, the medial column acts much like a very long first metatarsal, causing the forefoot to assume a relatively inverted position. This is observed as an "apparent" metatarsus primus elevatus, where the first metatarsal appears to be dorsally deviated as compared to the second (Figs. 4, 5). There is an angular deviation between the first and second metatarsals (separation on lateral radiograph), which has been previously thought to be a primary metatarsus primus elevatus.

The effect of this "functional fusion" of the first metatarsal-phalangeal-sesamoid joint can involve adjacent joints, either proximally or distally. Degeneration of either the hallux interphalangeal joint or the first metatarsocuneiform joint is not an uncommon finding. With respect to the interphalangeal joint of the hallux, it is common to observe a "cock-up" hyperextension deformity (Fig. 4). The next, more proximal level of compensation occurs as degenerative arthritis of the first metatarsocuneiform joint. It is interesting to note that more significant degeneration often occurs proximal or distal to the first metatarsal-phalangeal-sesamoid joint.

Figure 4. Severe hallux limitus, with functional fusion of the metatarsal-phalangeal-sesamoid joint, hallux equinus, retrograde metatarsus primus elevatus, and degeneration of the hallux interphalangeal joint (cock-up hallux).

Clinical evidence of compensation for an immobile metatarsal-phalangeal-sesamoid joint appears as a distinct pattern of shoe-wear and callus formation. In patients with hallux limitus or

Figure 5. Hallux rigidus with hallux equinus, retrograde metatarsus primus elevatus, and a functionally fused metatarsophalangeal joint, as evidenced by the straight-line relationship between the first metatarsal and proximal phalanx.

hallux rigidus, there is a distinct pattern of increased shoe-wear beneath the hallux interphalangeal joint, as well as the lateral heel region. The patient compensates for lateral roll-off by striking the ground with the foot in an inverted position; and as the patient's gait enters midstance and propulsion, the force of progression fails to pass through the metatarsal-phalangeal-sesamoid joint. Instead, the force of progression passes through the hallux interphalangeal joint, and is commonly observed as a plantar-medial callus beneath this joint (Figs. 6A-6D).

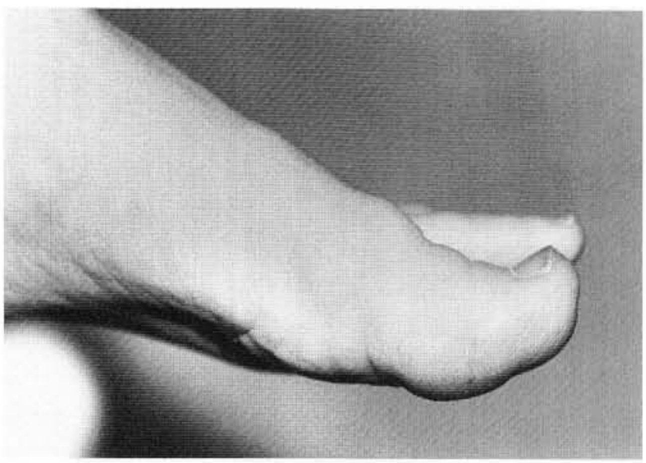

Figure 6A. Medial view of a patient with hallux rigidus and hallux equinus. Note the compensation at the hallux interphalagneal joint, which presents as a cock-up hallux.

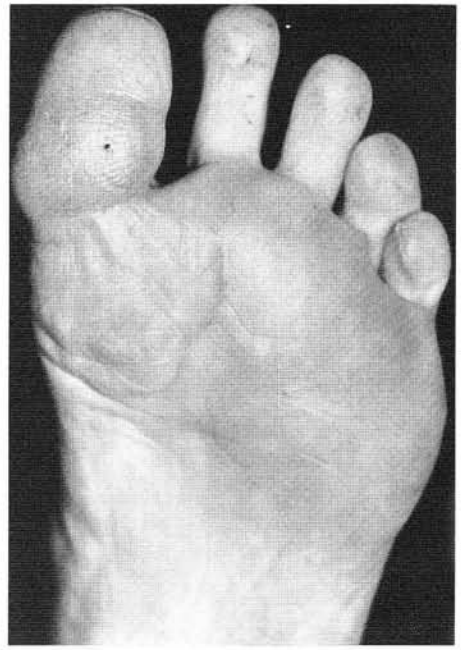

Figure 6B. Plantar view demonstrating distinct callus formation beneath the hallux interphalangeal joint. Also present is a severely painful and inflamed bursa.

Figure 6C. Lateral radiograph. Note the equinus position of the proximal phalanx of the hallux.

Figure 6D. Dorsoplantar radiograph demonstrating the retracted position of the sesamoid bones.

SESAMOID HYPERTROPHY

Another peculiar finding in patients with long-standing hallux limitus or hallux rigidus is hypertrophy of the sesamoid bones (Figs. 7-10). This is most readily observed on dorsoplantar radiographs of the foot. Constant and repetitive traction on the sesamoid bones produces a reactive proliferation of bone (enthesiopathy), both proximally and distally. In advanced cases, the plantar-proximal aspect of the proximal phalanx also demonstrates osseous hypertrophy. In severe cases, the sesamoido-phalangeal ligament can appear as an osseous bridge of bone.

Figure 7A. Dorsoplantar radiograph of the left foot of a patient with bilateral hallux limitus and mild degenerative arthritis of the metatarsophalangeal joint.

Figure 7B. Dorsoplantar radiograph of the right foot of the patient in 7A. Note the degree of sesamoid hypertrophy and advanced degenerative arthritis of the first MTP joint.

Figure 7C. Lateral radiograph of the left foot. Note the position of the hallux (equinus), retrograde metatarsus primus elevatus, and straight medial column of the first metatarsal and proximal phalanx.

Figure 7D. Lateral radiograph of the right foot demonstrating findings similar to 7C. However, note the narrowed joint space subchondral sclerosis, and dorsal spur formation of the first metatarsal.

Figure 8A. DP radiograph of a patient with hallux limitus and early degenerative arthritis. Note the degree of sesamoid hypertrophy, as well as a bipartite tibial sesamoid.

Figure 8B. Oblique view of the patient in 8A. The position of the proximal phalanx on this view demonstrates hallux equinus in a non-weight-bearing attitude.

Figure 9A. DP radiograph of another patient with hallux limitus, and severe degenerative arthritis of the metatarsophalangeal joint. Note the pronounced sesamoid hypertrophy, corresponding to the degree of arthritis.

Figure 9B. Oblique radiograph of the patient in 9A.

Figure 10. Radiograph of end-stage hallux rigidus, with severe hypertrophy and degeneration of the sesamoids.

RATIONALE FOR CONSERVATIVE TREATMENT

Current therapy for the treatment of hallux limitus and hallux rigidus includes a variety of conservative and surgical methods. Some of these methods aim to accommodate the immobile joint, while others attempt to restore joint mobility.

Steinberg (1971) advocated the use of serial peri-articular local anesthetic injections (often with hyaluronidase) in an attempt to break the cycle of muscular spasm. He reported a high rate of success (89%) through this semi-invasive technique, although frequent and repeated treatments were required. The rationale for this method of treatment is consistent with the previously described cyclic nature of the deformity.

The use of a Morton's extension pad or metatarsal bar pad effectively reduces motion about the metatarsal-phalangeal-sesamoid joint. An arthritic joint is only symptomatic when the joint is allowed to move. Hence, the common finding that a totally immobile (or fused) joint is not a painful joint. As in any arthritic joint, an attempt to provide immobilization will provide some degree of symptomatic relief. The findings of joint enlargement and osteophyte formation represent the body's attempt to auto-fuse the joint. Since the body is rarely able to fully accomplish auto-fusion, the arthritic joint normally becomes chronically symptomatic. The supportive effect of a Morton's extension augments the body's attempt at auto-fusion, however, it will not aid in restoration of motion or arrest the spasm of the short flexors of the hallux. When used in a joint with some degree of mobility or only mild arthrosis (functional hallux limitus), the Morton's extension can theoretically accelerate the process of joint degradation by further restriction of motion.

The use of an orthotic device which allows the first metatarsal to plantarflex, by supporting the lateral forefoot, is another sound method of addressing the etiology of hallux limitus. As previously mentioned, there is a high correlation between uncompensated varus and hallux equinus. The benefit of permitting the medial column to bear weight, through a medial relief cut-out in an orthotic device, is reasonable in theory. However, in most patients with an uncompensated varus deformity, there is little adaptability which will permit the first metatarsal to assume a plantigrade alignment with the rest of the foot. In patients with sufficient mobility and a voluntarily reducible forefoot supinatus deformity, this method is of particular value. However, this is typically not the case in instances of iatrogenic first metatarsal shortening, iatrogenic metatarsus primus elevatus, or a fixed varus deformity, regardless of location (forefoot, midfoot, rearfoot, or ankle).

RATIONALE FOR SURGICAL TREATMENT

A large body of literature exists regarding the surgical management of hallux limitus and hallux rigidus. Although each type of procedure claims to address a different component of the deformity, or aims to gain a certain effect, the procedures which have been somewhat successful provide symptomatic relief in one of three ways. These three basic effects are: elimination of motion through joint fusion (McKeever, 1952), radical and aggressive joint resection arthroplasty (Keller, 1904), and increasing joint motion in the direction of dorsiflexion (Watermann; Bonney & Macnab 1952, Kessel & Bonney 1958; Youngswick, 1984).

Fusion of the first metatarsophalangeal joint effectively reduces pain in that joint. This is the surgeon's means of accelerating nature's attempt at auto-fusion. However, fusion of the metatarsophalangeal joint does not address the metatarso-sesamoid joint, which can be a source of prolonged pain. As was previously described, the next proximal or distal joint (metararsocuneiform or hallux interphalangeal joint) is often subject to accelerated degeneration through compensation for loss of this motion. The potential for adjacent joint degeneration can be reduced by accurate positioning of the arthrodesis site based on the patient's activity level and style of shoes typically worn.

Resection arthroplasty of the base of the proximal phalanx of the hallux provides motion about the metatarsophalangeal joint by releasing the attachments of the sesamoido-phalangeal ligament, and increasing the space within the joint. This does not restore mobility to the sesamoid apparatus, and the sesamoids will assume a passive role in joint function. Regarding the cyclic etiology of hallux limitus/rigidus, release of the short flexor insertion also releases the plantar fulcrum of rotation, which is causing dorsal impingement of the joint. Adequate osseous resection from the base of the proximal phalanx is critical in that the chronicity of the hallux equinus leads to circumferential contracture about the joint, and a further tendency for dorsal impingement. Conversely, over-aggressive joint resection leads to a loss of extrinsic stability of the hallux, seen as the common flail-hallux or cock-up hallux deformity.

The third category of surgical procedures is more varied in approach, but common in effect. These procedures include both metatarsal and proximal phalangeal osteotomies. The Watermann osteotomy (Fig. 11) is proported to dorsally relocate the effective articular cartilage, while the osteotomy by Bonney, Macnab, and Kessel (Fig. 12) attempts to increase dorsiflexion within the joint.

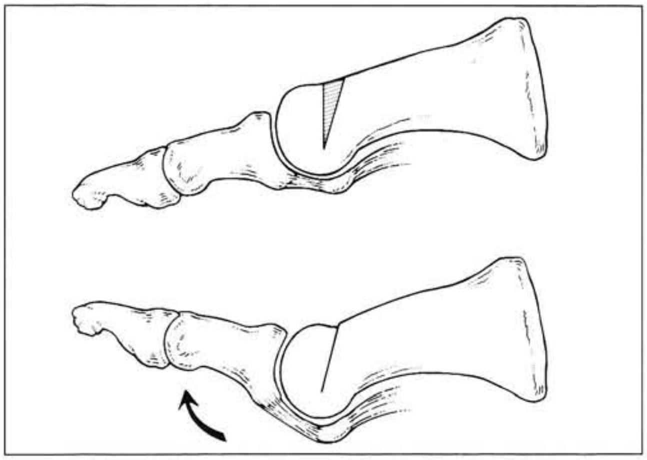

Figure 11. Watermann osteotomy of the first metatarsal for hallux limitus. The effect on joint mobility is attributed to shortening of the dorsal aspect of the joint. However, there is little effect on sesamoid mobility with this procedure.

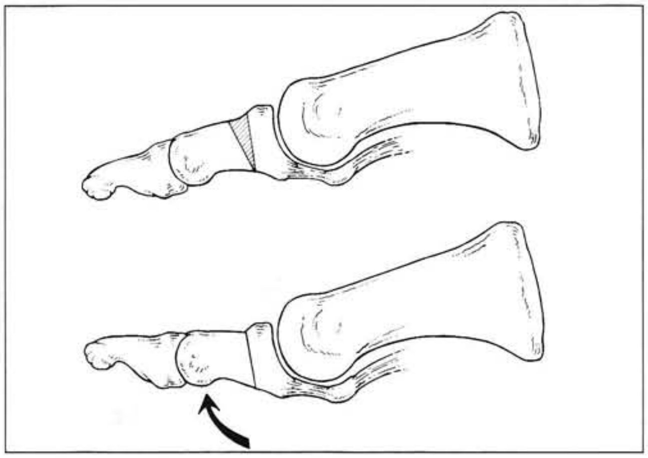

Figure 12. Proximal phalangeal osteotomy for hallux limitus. The primary effect on the first ray is a "relative" increase in dorsiflexion of the hallux. However, the 1st MTP joint is not altered. it is only the position of the distal hallux relative to the base of the proximal phalanx that has been changed. Again, there is little effect on sesamoid mobility.

Youngswick's modification of the Austin bunionectomy attempts to plantarflex and shorten the distal first metatarsal, thereby allowing the proximal phalanx to dorsiflex without restriction (Fig. 13). In each of these procedures, the functional

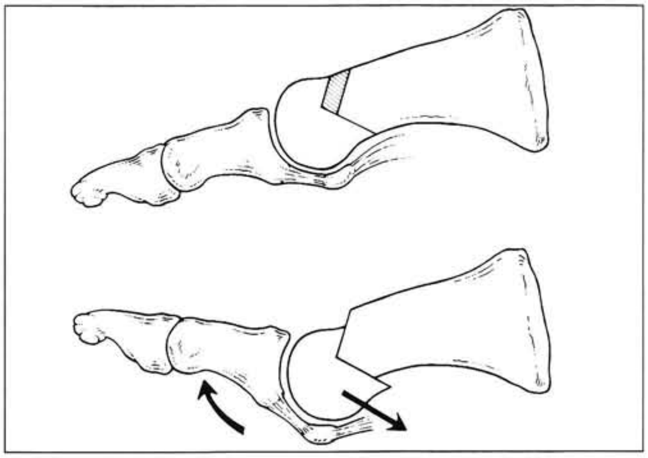

Figure 13. Youngswick's modification of the Austin osteotomy provides increased dorsiflexion due to shortening of bone within the joint, as well as by reducing the effect of contracted plantar structures. In a joint with a mobile sesamoid apparatus, there is a potential to increase the mobility of the sesamoid apparatus.

effect is similar in that the plantarly-displaced fulcrum of rotation at the metatarsal-sesamoid level is accommodated by the removal of bone from the dorsal aspect of the joint (actually, just proximal or distal to the joint). However, in none of these procedures is there actually a relocation of available or functional cartilage. There is simply a removal of bone from the side of the joint which is under restrictive compression. These procedures, in and of themselves, do not restore motion of the metatarsal-sesamoid articulation. Adjunctive soft tissue releases are required to restore mobility to the flexor plate and sesamoid apparatus. In a joint in which the sesamoid apparatus has not ankylosed to the metatarsal head, the Youngswick modification could potentially provide for restoration of motion by effectively releasing tension from the plantar soft tissue structures.

SUMMARY

The natural history of hallux limitus and hallux rigidus is based on a complex cascade of events which propagate joint destruction. The sequence of uncompensated varus, hallux equinus (gripping), compensatory short flexor spasm, sesamoid apparatus immobility, dorsal impingement, degenerative arthritis, and chronic joint spasm was detailed. Secondary radiographic findings, such as functional joint fusion with flexion contracture of the metatarsal-phalangeal-sesamoid joint, and apparent metatarsus primus elevatus, were also presented. The change in position of the axis of rotation of the metatarsophalangeal joint, from centrally within the metatarsal head to the metatarsal-sesamoid articulation region, was implicated as the cause of joint destruction. Likewise, sesamoid hypertrophy was identified as a sequale of excessive plantar soft tissue traction. Finally, the rationale for both conservative and surgical management of hallux limitus/rigidus, was expounded upon and justified based on the pathogenesis of the deformity.

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