FOREFOOT DERANGEMENT: ETIOLOGY OF HAMMERTOES AND BUNIONS

Thomas J. Merrill, DPM

Ibn A. Imani, DPM

The most common cause of bunion deformity and associated digital deformities is the long term progressively destabilizing effects of excessive pronation. Abnormal pronation produces a muscle tendon imbalance across the joints of the forefoot. Over time, these asymmetrical forces, if not controlled, can eventually alter normal alignments of the metatarsals and digits. Inappropriate surgical intervention can produce this derangement much more rapidly.

Long-term excess subtalar joint pronation changes the normal function of the joints and muscles of the foot during the gait cycle. During the contact phase of gait, the normal subtalar joint goes through the motion of pronation to aid in shock absorption. At the end of the contact phase the subtalar joint stops pronating at a few degrees past neutral and then begins the motion of supination. This motion of resupination during midstance produces a rigid stable forefoot to accept the weight of the body and to allow propulsion.

During forefoot loading, both axes of motion at the midtarsal joint pronate to a locked position. The locked position of the midtarsal joint is dependent on the position of the subtalar joint. Every degree of subtalar joint pronation produces an exponential increase in midtarsal joint pronation. Therefore, in abnormal subtalar joint pronation, the midtarsal joint cannot reach the locked position and the forefoot continues to evert and abduct during midstance. The longitudinal axis of the midtarsal joint and the range of motion of each metatarsal attempt to compensate for the everted position of the forefoot in relation to the weightbearing surface. The first metatarsal has the largest dorsiflexory range of motion, followed by the fifth and fourth metatarsals. The second and third metatarsals have a limited dorsiflexory range of motion. Palpation of the neutral subtalar joint position and locking of the midtarsal joint allows evaluation of each of the metatarsal's range of motion.

The peroneus longus muscle is a major factor in the stabilization of the first metatarsal against the ground, and abnormal pronation alters its function. The fulcrum of the tendon, the cuboid, loses stability and the muscle pull decreases as its functional length increases. Because of the everted position of the forefoot, the insertion of the peroneus longus into the base of the first metatarsal is lower in relationship to the fulcrum of the cuboid. The plantarflexory force vector of the tendon on the first metatarsal is decreased or lost. The first metatarsal becomes hypermobile and develops an increase in dorsiflexion, adduction, and inversion around its normal axis of motion.

Abnormal subtalar joint pronation with calcaneal eversion and talar plantarflexion will cause the forefoot to abduct on the rearfoot. Abduction of the forefoot contributes to digital deformities by changing the normal relationship between the intrinsic muscles, the long flexors and extensors, and their digital insertions. Although abnormal subtalar joint pronation may occur for many reasons,

the derangement of the forefoot that results develops from this same pattern of joint instability.

IATROGENIC ETIOLOGY OF ABNORMAL PRONATION

Surgical intervention can produce dramatic realignment of muscle tendon function, but can also produce devastating muscle tendon imbalance. There are several reasons why the forefoot derangement that is iatrogenically produced is much more progressive than the forefoot derangement secondary to abnormal pronation. The attempted surgical procedure did not realign the muscle tendon imbalance and that imbalance continues. Normal anatomy in the area that had previously provided stability may have been destroyed. The underlying etiology of abnormal pronation is probably still present and these forces will continue to influence the postoperative result.

The approach to an iatrogenic deformity is the same as to other biomechanical deformities. A current inventory of the functional anatomic structures is the first step. Identification of the individual deforming forces influencing the muscle tendon imbalance is the second step. The selection of surgical procedures to redirect or neutralize these forces is the third step. The immediate and long term postoperative management is the fourth step.

Hallux Abducto Valgus

Patients often develop a hallux abducto valgus deformity secondary to the hypermobility resulting from excess pronation. With the hypermobile motion of the first metatarsal, the first metatarsal head dorsiflexes, inverts, and adducts. As pronation increases forefoot abduction and first metatarsal motion, the tension on the transverse head of the adductor hallucis tendon increases. The anatomy of the first metatarsophalangeal joint effects the development of a hallux abducto valgus deformity because no muscles attach into the first metatarsal head. The intrinsic muscles insert into the capsule, the sesamoids and the base of the proximal phalanx. The adductor hallucis tendon and the deep transverse intermetatarsal ligament have strong attachments into the lateral aspect of the joint capsule and the fibular sesamoid. These structures are held stable as the first metatarsal head moves away from the second metatarsal head. The lateral collateral, fibular sesamoidal and intersesamoidal ligaments remain intact. The first metatarsal head effectively leaves the fibular sesamoid behind as it moves along its increased axis of motion. The flexor hallucis longus and brevis tendons are rotated from under the metatarsal head gradually into the interspace. All of the periarticular structures that cross the first metatarsophalangeal joint become rotated or displaced. The abductor hallucis tendon is displaced plantarly and the extensor hallucis longus and brevis tendons are displaced lateral to the joint.

As the lateral joint structures rotate into the interspace, the proximal phalanx of the hallux assumes a valgus position. The dorsomedial aspect of the joint capsule stretches and the medial aspect of the first metatarsal head becomes prominent. The soft tissue overlying the dorsiflexed, adducted metatarsal head becomes inflamed from shoegear and a bursa may form because of the irritation.

The abducted hallux position and the periarticular structural displacement combine to produce the retrograde buckling force that increases the metatarsus primus adductus and progressively accentuates the deformity. The adaptive soft tissue contracture of the flexor hallucis brevis and the adductor tendon acting in conjunction with the displaced fibular sesamoid can produce a tension-band like effect that will resist relocation of the proximal phalanx on the metatarsal head. The adductor tendon may also be responsible for lateral tracking of the proximal phalanx on the metatarsal head during dorsiflexion. As the joint gradually dislocates, the abnormal lateral forces on the articular cartilage surfaces can produce adaptive or degenerative changes.

The lateral collateral ligament, fibular sesamoidal ligament, adductor hallucis tendon and flexor hallucis brevis undergo shortening and contracture over time. Each of these anatomic structures must be identified and addressed individually in the surgical repair of the hallux abducto valgus deformity.

A pinch callus at the medial plantar aspect of the medial condyle of the head of the proximal phalanx may develop because of the changes in hallux function with excess pronation. As the

hypermobile first metatarsal dorsiflexes and the hallux moves into a valgus position, the hallux deviates from the line of progression. The body weight falls more medially than in a normal foot and the patient subsequently rolls over the medial side of the abducted hallux.

Iatrogenic Hallux Abducto Valgus

An unsuccessful surgical attempt can produce a more progressive deformity. A hyperkeratotic lesion plantar to the second metatarsal head would be expected with a hypermobile first metatarsal and a hammertoe of the second digit. The plantar tyloma is not due to a structural deformity of the second metatarsal, but rather the muscle tendon imbalance of the first ray and the digits. Realizing the etiology of the plantar lesion, a procedure that realigns the intrinsic muscle balance across the first metatarsophalangeal joint is indicated. Procedures that appear to address the immediate symptoms, in reality can actually increase the forefoot derangement. The bunionectomy procedure alone will reduce the medial capsular stability and allow for a more rapid development of the hallux abducto valgus deformity. The elevating osteotomy of the second metatarsal will dramatically increase the abnormal pronation of the subtalar joint when associated with a neglected hypermobile first metatarsal.

Contracted Toes

One of the most common deformities caused by muscle tendon imbalance is the contracted toe. Digital deformities usually occur in conjunction with other forefoot changes such as hallux abducto valgus, lesser metatarsalgia and plantar tylomas. These deformities are closely related and the interaction between them is the key to the evaluation and treatment. Digital contractures affect the function of the metatarsophalangeal joint and metatarsals. As the toes contract, the metatarsophalangeal joints are dorsiflexed and a retrograde plantarflexory force is placed on the metatarsals. A decision must be made as to the etiology of a plantar hyperkeratotic lesion: structural metatarsal etiology or functional digital contracture etiology.

A normal foot will resupinate as the leg begins to rotate externally and the tibialis posterior contracts. As the subtalar joint resupinates the dorsiflexory force on the forefoot locks the midtarsal joint and allows the peroneus longus to act over a stable cuboid to plantarflex the first ray and stabilize the forefoot. This allows the intrinsic muscles to stabilize the toes against the ground without buckling. Both the interossei and lumbricales help to stabilize the toes, although the interossei may be the primary stabilizers during stance, whereas the lumbricales function primarily during the swing phase. A partial resection of the base of the proximal phalanx also has no biomechanical basis. The resultant muscle tendon imbalance will deviate and eventually dislocate the metatarsophalangeal joint. The total resection of the base of the proximal phalanx has no biomechanical basis. The loss of the intrinsic muscle stabilization reproduces the effects of abnormal pronation immediately. The resultant floating digit has no sagittal plane stability and does not purchase the ground.

When the subtalar joint fails to resupinate, the distal joints cannot be stabilized. The intrinsic muscles lack the strength to stabilize the foot and they fatigue in the attempt to do so. An additional attempt to regain digital stability is initiated with excessive firing of the flexor digitorum longus muscle. The pull of the long flexor tendons produces a hammer toe deformity because the proximal and middle phalanges are no longer stabilized. This grasping of the toes, called flexor stabilization, occurs during late midstance and propulsion. Flexor stabilization can also cause an adducto varus deformity of the digits to develop. As the foot pronates and the forefoot abducts, the pull of the long flexor shifts medially and increases the amount of adduction that occurs with flexion. The fifth toe contracts most frequently, with the fourth and third toes also being affected in more serious cases. The digital effects of abnormal pronation can be observed as a separate entity.

Flexor Cap Dislocation

One of the problems that occurs after long term pronation with digital contracture is dislocation of the metatarsophalangeal joint flexor cap. This occurs most commonly at the second metatarsophalangeal joint and is aggravated by an abducted hallux that forces the second toe dorsal-

ly and medially. As the toe displaces, the long flexor and extensor tendons are pulled to the medial side of the joint, and the flexor cap may ride up around the medial side of the metatarsal head. As this occurs, contraction of the long flexor no longer just plantarflexes the toe, but also adducts it.

A hallux abducto valgus deformity often contributes to toe deformities, especially those of the second toe. As the hallux drifts laterally it tends to push the lesser toes laterally, then it may under-ride or override them. As the hallux moves under the second toe, a hammer toe develops and the entire second metatarsophalangeal joint will eventually dislocate. A patient with this problem may present only with a chief complaint of a painful second toe, but the underlying hallux abducto valgus deformity needs to be addressed.

Metatarsal Disorders

Metatarsal problems are intimately related to contracted digits in excessively pronated feet, although isolated metatarsal problems are sometimes seen. Patients may complain of metatarsalgia, painful plantar hyperkeratoses, tailor's bunions, stress fractures, or neuromas. As the foot abnormally pronates, the lesser metatarsals are forced to bear excess weight. The forefoot becomes hypermobile and the first ray dorsiflexes. This pressure falls primarily on the second metatarsal and a diffuse shear callus will form just distal to the metatarsal head. If the second toe becomes contracted also, the retrograde force that plantarflexes the second metatarsal may lead to the development of a nucleated hyperkeratotic lesion. The lesion may also occur if the metatarsal is anatomically plantarflexed. However, a hypermobile first metatarsal is much more common and a true structurally plantarflexed second metatarsal is rare. Failure to differentiate between the two is a common problem and can result in a very difficult iatrogenic postoperative situation.

The resection of the second metatarsal head has no biomechanical basis and exaggerates the hammertoe deformity and the hallux abduction deformity. The weightbearing distribution of the remaining metatarsals is destroyed and stress fractures or plantar lesions result. The tension distribution between the division of the flexor digitorum longus and flexor digitorum longus is also disrupted. A loading phenomenon muscle tendon imbalance develops and creates progressive hammertoe deformities of the remaining digits.

Bibliography

Basmajian JV, Stecko P: Role of muscles in arch support of the foot. *J Bone Joint Surg* 45A:1184-1190, 1963.

Green DR, Ruch JA, McGlamry ED: Correction of equinus related forefoot deformities, a case report. *J Am Podiatry Assoc* 66:768-780, 1976.

Grode SE, McCarthy DJ: The anatomical implications of hallux abducto valgus, a cryomicrotomy study. *J Am Podiatry Assoc* 70:539-551, 1980.

Haines RW: The anatomy of hallux valgus. *J Bone Joint Surg* 36B:272-293, 1954.

Jarrett BA, Manzi JA, Green DR: Interossei and lumbricales muscles of the foot, an anatomical and functional study. *J Am Podiatry Assoc* 70:1-13, 1980.

Long C: Intrinsic-extrinsic muscle control of the fingers, electromyographic studies. *J Bone Joint Surg* 50A:973-984, 1968.

Root ML, Orien WP, Weed JH: *Normal and Abnormal Function of the Foot*, Vol 2. Los Angeles, Clinical Biomechanics Corp, pp. 28-41, 1977.

Sage RA, Hugar DW: Hallux pinch callouses: some etiologic considerations, *J Foot Surg* 19:148-151, 1980.

Schlefman BS, Fenton CF, McGlamry ED: Peg in hole arthrodesis. *J Am Podiatry Assoc* 73:187-195, 1983.

Sgarlato JE: *A Compendium of Podiatric Biomechanics* San Francisco, California College of Podiatric Medicine, p 94-104, 1971.

Shaw AH: The biomechanics of hallux valgus in pronated feet. *J Am Podiatry Assoc* 64:193-201, 1974.