Dynamic Chiropractic – June 17, 1994, Vol. 12, Issue 13

Chiropractic Management of the Painful Bunion

By James Brantingham, DC, CCF , Thomas Michaud, DC, Alison Deliman, DC, CCFC, Randy Snyder, DC, CCFC and Charles Brantingham, DPM

also by: Jay Silverman, DC, CCFC

Hallux abductovalgus (HAV) or bunion, is a commonly seen deformity of the first metatarsophalangeal joint (MPJ) in which the hallux is abducted and everted, frequently overriding the second toe. Although the terms HAV and bunion are often used synonymously (as is done in this paper), it should be noted that a bunion actually refers to the callus and inflamed adventitious bursa overlying the HAV deformity.

Even though bunions have been described in the medical literature for several hundred years (the word bunion is believed to be derived from the Latin, bunio, meaning turnip), there continues to be much controversy concerning its etiology. This is most likely because the development of HAV is multifactorial, stemming from a variety of structural and functional aberrancies.

For example, structural malformations such as a rounded first metatarsal head, obliquity of the first tarsometatarsal articulation and/or elongated great toe (the Egyptian foot type), may all allow for a lateral migration of the hallux on the metatarsal head. These structural variations are even more likely to produce HAV in the presence of functional abnormalities such as an excessively low medial longitudinal arch, an increased range of dorsiflexion in the first tarsometatarsal articulation and/or excessive subtalar pronation, all of which undermine the mechanical stability of the first MPJ.[1]

Despite the fact that over 80 operative procedures have been described for the treatment of HAV, the rationale for even the most current surgical interventions have not been conclusively demonstrated.[2] Because complications associated with surgery include nonunion osteotomy, avascular necrosis and/or laceration of the medial sensory branch of the superficial peroneal nerve, surgical intervention should never be considered for prophylactic or cosmetic reasons.[3] In fact, O'Connor and Baxter[3] state that surgical intervention, which may consist of a combination of soft tissue procedures (releasing the adductor tendons with medial capsule reinforcement) and/or osteomies to improve alignment of the first metatarsal and/or hallux, should only be considered if pain and deformity continue to increase despite conservative care. Furthermore, Bordelon[4] states that "it is probably best not to perform a surgical procedure on a serious athlete unless absolutely necessary, as he or she may not be able to return to the previous activity level."

To be comprehensive, conservative treatment of HAV should include well-fitting shoe gear (a spacious toe-box is essential), orthotics, when indicated, to improve mechanical stability and redistribute plantar pressure patterns, and various manual therapies such as manipulation and myofascial techniques, to lessen pain patterns that may be arising from soft tissue adhesions associated with the faulty biomechanics.

In regards to the manual techniques, James Mennel[5] reported that manipulation of the bunion joint itself could relieve pain but not change the deformity (which is our common experience). Also, Hiss,[6] having had the experience of manipulating over 100,000 feet, made it clear that although only surgery could change the HAV, approximately 50 percent of patients with a painful bunion could be made more comfortable with manipulation alone. Mennel and Hiss recommend manipulation or mobilization of any stiff "fixated" dysfunctional foot or ankle joints in the manual treatment of HAV. The following case study demonstrates the importance of incorporating manipulative techniques in the conservative management of HAV.

Case Study

A 39-year-old male presented to our office on June 4, 1991 after undergoing bunionectomy on December 21, 1990. The patient’s pain began early in 1980, and consisted of moderate to marked discomfort beneath the 2nd and 3rd metatarsal heads with mild 1st MPJ pain. By 1988 the pain was frequently severe and was particularly exacerbated by even moderate amounts of exercise such as stair climbing, martial arts or racquetball. In fact, the patient eventually had to stop exercising altogether due to persistent foot pain. Because of the increasing severity of forefoot pain, the patient consulted a podiatrist who performed a bunionectomy; a modified Austin procedure with a base wedge osteomy including a screw fixation was used. A rigid cast was applied for one week followed by a fiberglass walking cast for three more weeks. He was temporarily disabled for three weeks and for the entire first week rested with his foot elevated.

Unfortunately, the surgery did not appreciably change the patient’s pain pattern and he was then fitted with functional rigid and semi-rigid orthotics. When he complained to the podiatrist that the pain was essentially the same, another surgery was suggested for an elongated second metatarsal bone (which the podiatrist felt was possibly the cause of the persistent pain). The patient declined the second operation and not only continued to have pain in his first MPJ but also began to experience a great deal of discomfort, burning, and tingling in the ball of the foot, especially beneath the 2nd and 3rd metatarsal heads. At this point, he contacted our office for evaluation and treatment.

Examination

The patient's feet revealed bilateral HAV deformities. There was a healed surgical curvilinear scar over the distal half and dorsal proximal half of the left first metatarsal and first phalangeal shafts. Palpation revealed only a mildly tender first MPJ but there was marked tenderness beneath the plantar aspects of the 2nd and 3rd metatarsal heads. Also, medial/lateral compression of the metatarsal heads (the Morton's squeeze test) produced pain between the 2nd and 3rd metatarsal heads. Range of motion testing revealed a dorsiflexed first ray (i.e., the first metatarsal-cuneiform joint acting as a single long metatarsal shaft) which was hypermobile in dorsiflexion and hypomobile in plantar flexion. The left second and third metatarsals possessed a decreased range of superior-inferior intermetatarsals glide and the hallux possessed 55 degrees dorsiflexion (normal is 65 degrees). There was only five degrees of ankle dorsiflexion (10 degrees is required for noncompensated ambulation) and subtalar range of motion was within normal limits.

Motion palpation of the left foot revealed a loss of inferior glide of the first metatarsal upon the medial cuneiform (a very common fixation with HAV), long axis extension of the talocrural joint and inferior glide of the cuboid on the calcaneus. The "Hiss test" (i.e., fixing the heel with one hand while circumducting the forefront with the opposite hand) revealed a normal range of midtarsal and central tarsometatarsal mobility.

Gait analysis revealed a bilateral 20 degree toe-out gait pattern (resulting from retroverted hips) with excessive subtalar pronation noted throughout all periods of stance phase. Static stance evaluation revealed bilateral eversion of the calcanei (10 degrees on the left and five degrees on the right) with excessive abduction of the forefoot at the midtarsal joint (indicative of a vertically displaced oblique midtarsal joint axis).

Treatment

Treatment consisted of warm hydrotherapy baths (with a metallic screw fixation, diathermy and like modalities were contraindicated) to relieve pain and facilitate less forceful manipulations and pulsed ultrasound beneath the plantar 2nd and 3rd metatarsal heads to lessen inflammation of the intermetatarsal bursae and the involved interdigital nerve. A grade 4 mobilization was performed on the left foot to restore intermetatarsal glide between the 2nd and 3rd metatarsals (Figure 1) and a grade 5 manipulation was performed to restore inferior glide of the first metatarsal upon the medial cuneiform (Figure 2). Also, a grade 5 manipulation was used to restore a long axis extension at the talocrural joint and inferior glide of the cuboid upon the calcaneus (Figures 3 and 4). Finally, the hallux was very gently mobilized in all six planes of motion.

ARNETTA: SCAN IN FIGURES 1,2,3, and 4

Figure 1 -- Grade 4 mobilization used to restore intermetatarsal glide between the 2nd and 3rd metatarsals. Reproduced with permission from reference.[1]

Figure 2 -- Grade 5 manipulation used to restore inferior glide of the 1st metatarsal upon the medial cuneiform. Reproduced with permission from reference.[1]

Figure 3 -- Grade 5 manipulation used to restore long axis extension at the talocrural joint. Reproduced with permission from reference.[1]

Figure 4 -- Grade 5 manipulation used to restore inferior glide of the cuboid upon the calcaneus. Reproduced with permission from reference.[1]

In order to prevent these fixation patterns from returning, the patient was encouraged to wear his previously prescribed semi-rigid orthotics in order to lessen the range of calcaneal eversion present at the end of the midstance period. After only five treatments the patient reported nearly 100 percent relief of his original symptoms. He was contacted more than one year after his final treatment and he continued to have 90 or greater relief of pain.

Discussion

Root et al.[7] describe four stages in the development of HAV. Initially, there is a pure lateral shift of the entire hallux upon the first metatarsal head (stage 1). This is quickly followed with abduction of the hallux (stage 2). Because abduction of the hallux displaces the long flexor and extensor tendons laterally, contraction of these muscles during the propulsive period produces a retrograde medially directed component of force as the proximal phalanx pushed the first metatarsal in an adducted position (stage 3). The authors note that the fourth stage of deformity, which involves complete dislocation of the first MPJ, rarely occurs without underlying rheumatic inflammatory disease or neuromuscular disorder.

Although many factors may be responsible for the development of HAV, the most commonly described causes are genetic (Johnston[8] claims that HAV is transmitted as an autosomal dominant with incomplete penetrance), inflammatory disease affecting the MPJ, muscle imbalances where the abductor forces exceed adductor forces, and excessive subtalar joint pronation during the propulsive period, which undermines the mechanical stability of the entire foot.[1]

More recently, Klaue et al.[2] claimed that an increased range of sagittal plane motion at the first tarsometatarsal articulation is a common cause of HAV that is frequently overlooked. By measuring the distance that the first metatarsal head could be dorsiflexed after stabilizing the heel and forefoot, the researchers noted that individuals with painful bunions possessed larger ranges of first metatarsal cuneiform dorsiflexion compared to an asymptomatic population; i.e., the symptomatic group averaged 9.3 mm of vertical displacement versus 5.3 mm in the asymptomatic group. This exaggerated range of the first metatarsal dorsiflexion may be responsible for producing a painful bunion because the first metatarsal, unlike the second metatarsal, possesses an axis of motion that allows for relatively equal amounts of frontal and sagittal plane motion. In other words, for every 10 degrees the first metatarsal dorsiflexes, it will simultaneously invert 10 degrees. These coupled movements, when excessive, may greatly exacerbate bunion pain because the adventitious bursa overlying the dorsomedial metatarsal head becomes sheared between the skin (which is held in a fixed position by shoe-gear) and the rotating metatarsal head. A functional orthotic in this situation is often invaluable because it supports the medial longitudinal arch, thereby lessening the range of first metatarsal dorsiflexion. Typically, the more rigid orthotic shells are prescribed to treat a painful bunion because they more effectively limit the overall range of pronation.[9]

In regards to manipulation, clinical experience dictates that although the degree of HAV typically remains unchanged, the individual is often made more comfortable by improving the flexibility of soft tissue contracture so often associated with this deformity. This is particularly true in post-surgical cases where prolonged immobilization results in widespread joint dysfunction, particularly in the first metatarsocuneiform and calcaneo-cuboid joints. It is also of clinical interest that D.D. Palmer[10] repeatedly refers to the benefits associated with manipulating the joints of the foot and ankle, particularly in regards to lessening the pain associated with an inflamed bunion. It should be emphasized, however, that manipulation of a painful bunion should always be gentle and should only be performed once other causes of first MPJ pain have been ruled out; e.g., gout, psoriatic and rheumatoid arthritis and severe degenerative joint disease. As a safety precaution it is recommended that this protocol be initially followed in mobilization and manipulation of the HAV or bunion patient:

1. Rule out metabolic diseases such as gout, rheumatoid arthritis, and severe or advanced degenerative joint disease by radiographs.

2. Perform light traction of the hallux and gentle lateral glide joint play mobilization, then ice the bunion. Very mild soreness is acceptable therefore continue treatment. Severe pain is unacceptable. Stop bunion mobilization and only treat other foot fixations.

3. Next, traction and with moderate force, mobilize the hallux in adduction (Figure 5) (adduction toward the midline of the body). Apply ice and assess as in # 2.
   ARNETTA: SCAN IN Figure 5

   Figure 5 -- Moderate force mobilization used to mobilize the hallux in adduction.

4. Next, use strong mobilization, ice, reassess, and at the next visit use high velocity, low amplitude manipulation of the bunion joint (Figure 6). Constant patient assessment is necessary.

   ARNETTA: SCAN IN Figure 6

   Figure 6 -- High velocity, low amplitude manipulation of the bunion joint.

References

1. Michaud TC. Foot Orthoses and Other Forms of Conservative Care. Baltimore: Williams and Wilkins, 1993.

2. Klaue K et al. Clinical, quantitative assessment of first tarsometatarsal mobility in the sagittal plane and its relationship to hallux valgus deformity. Foot Ankle 1994;15(1): 9-13.

3. O’Connor PL, Baxter DE. Developmental disorders: adult foot. In: Gould JS. The Foot Book. Baltimore: Williams and Wilkins, 1988: 216.

4. Bordelon RL. Surgical and Conservative Foot Care. Thorofare, NJ: Slack Inc., 1988:48.

5. Mennell J. The Science and Art of Joint Manipulation. Philadelphia: The Blakiston Co., 1949.

6. Hiss JM. Functional Foot Disorders. Los Angeles: Oxford Press, 1949:307-21.

7. Root MC, Orien WP, Weed JH. Normal and abnormal function of the toot. Los Angeles: Clinical Biomechanics, 1977.

8. Johnson O. Further studies on the inheritance of hand and foot anomalies. Clinical Orthopedics 8:146, 1956.

9. Smith LS et al. The effects of soft and semi-rigid orthoses upon rearfoot movement in running. J Am Podiatr Med Assoc 1986; 76(4):227.

10. Palmer DD. The Chiropractors Adjustor. Portland OR: Portland Publishing Co., 1910.

James Brantingham, DC, CCFC
Thomas Michaud, DC
Alison Deliman, DC, CCFC

Jay Silverman, DC, CCFC

Randy Snyder, DC, CCFC

Charles Brantingham, DPM

■