Medial column foot systems: an innovative tool for improving posture

Brian A. Rothbart

Introduction

Morton (1935) describes a foot in which the 1st metatarsal is shorter than the 2nd, visually identified as a deep 1st web space (Fig. 1). Clinical studies uncover a foot in which the 1st metatarsal is *structurally inverted and elevated* relative to the 2nd metatarsal, referred to as the Rothbart Foot Structure (RFS) (Rothbart 1988). Morphologically, Morton and Rothbart both foot structures are the same: both arising from an embryological retention of talar supinatus. Rothbart (1988) demonstrates that it is this *elevated position of the 1st metatarsal that hyperpronates the walking foot*. Hyperpronation draws the posture forward (Rothbart McCombs et al. 1992, Rothbart Yerratt 1994, Rothbart Hansen et al. 1995, Schneider 1995, Filner 1996, Liley 1996). The body adapts. Slowly and progressively, strain and deformation patterns develop that lead the patient into chronic pain (Rothbart Esterbrook 1988, Rothbart McCombs et al. 1992, Rothbart Hansen et al. 1995, Petersen 1995, Schneider 1995, Filner 1996, Liley 1996) (Fig. 2).

Travell and Simons (1992) state: 'Morton's foot structure is a major initiator and perpetuator of trigger points'. Since Morton's foot structure and Rothbart's foot structure (RFS) is the same foot structure, it is logical to state 'RFS is a major initiator and perpetuator of trigger points'.

An innovative medial column foot system, *designed to improve posture* in patients with Morton/Rothbart's Foot Structure, is introduced. This foot appliance visually, and at times dramatically, reduces pelvic tilts (unleveled pelvis), shoulder protractions (rounded shoulders) and forward head positions. The drawn forward posture is reversed (Fig. 3). And with improved posture, trigger points/chronic pain syndromes are more easily resolved. Intuitively we know feet affect posture. Engineers use this concept daily: as goes the foundation (foot), so goes the building (posture). This article describes the foot–posture relationship dynamically, e.g. the impact the walking foot has on posture.

Morton (1932) asserts a short 1st metatarsal (relative to the 2nd and 3rd metatarsals) prevents the first metatarsal head from fully

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Box 1

1.0 Normal embryonic development (weeks 1–8 pf).
- Week 3 post fertilization (pf) lower limb bud appears
- Week 6 pf limb bud at right angles to rump: soles and posterior surfaces face cephalad (Fig. 4, Right slide, top)
- Week 8 pf soles and posterior surface face one another (Fig. 4, right slide, middle, Fig. 5)

1.1 Normal foetal development (weeks 9 pf to full term).
- Week 9 pf primordial ankle and knee joints appear (Fig. 4 Left slide, bottom)
- Week 10 pf thigh and leg unwinding around longitudinal axis (Fig. 4, Right slide, bottom)
- Week 11 pf calcaneal supinatus unwinding
- Week 12 pf talar {head} supinatus unwinding
- Week 36 pf heel and sole plantargrade to leg

1.2 Abnormal foetal development
- 1.21 Talar head fails to unwind: Rothbart foot structure
- 1.22 Calcaneus fails to unwind: Clubfoot deformity

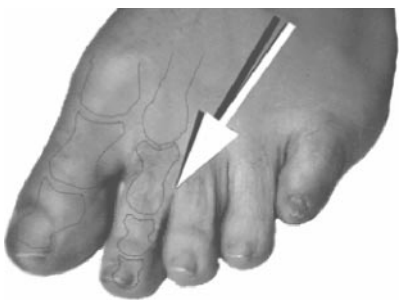

Fig. 1 Deep 1st web space seen in Morton's foot structure. The 1st web space (space between the 1st and 2nd toes) is more proximal (nearer to the heel) than the 2nd web space.

- Plantar fasciitis
- Chondromalacia
- Sacral iliac joint inflammation
- Sciatica
- Low back pain
- Thoracic outlet syndrome
- Headaches
- Temporal mandibular joint dysfunction

Fig. 2 Chronic pain syndromes associated with Rothbart's foot structure.

participating in weight bearing (Fig. 1). While this concept appears correct, it is only a partial explanation of the pathodynamics engaging hyperpronators. Embryologically the foot goes through a series of torsional changes (Streeter 1945, Lash et al. 1997, Aiton et al. 1995, McLachlan et al. 1997, O'Rahilly et al. 1987, Smith 1999, Jirasek & Keith 2001, Gasser 1975, Patten 1946). If these torsions end prematurely (Tabibzadeh & Pettersson 1995), the 1st metatarsal and big toe (proximal phalanx and hallux) remain structurally elevated and inverted (in elevatus) relative to the lesser metatarsals and phalanges (Straus 1927, Olivier 1962). It is this retained elevatus that forces the walking foot into hyperpronation (Rothbart & Esterbrook 1988, Rothbart & Hansen 1995, Filner 1996, Liley 1996). It is hyperpronation that draws the posture forward (Rothbart et al. 1992, Rothbart & Hansen 1995, Schneider et al. 1995, Filner 1996,

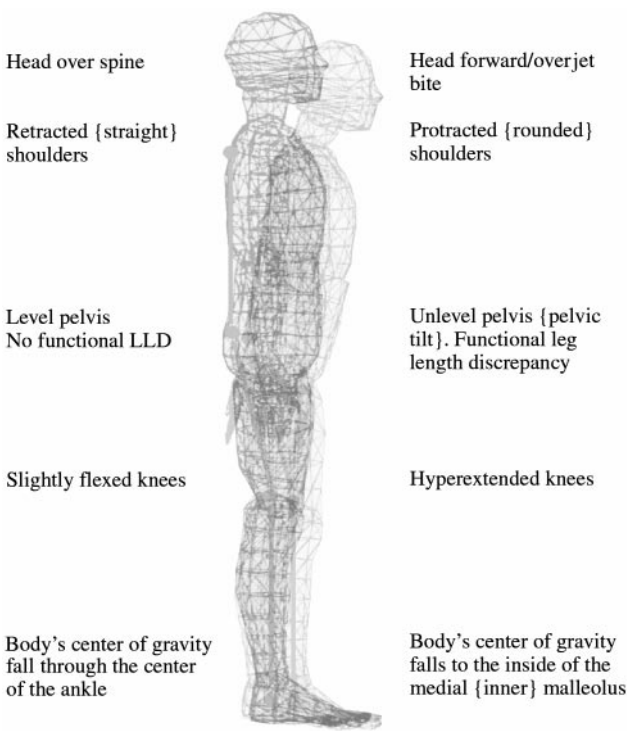

Fig. 3 BioImplosion. Gravity induced skeletal shift. Hyperpronation shifts the body's center of gravity forward. The axial framework responds by moving out of vertical towards a forward, inward and downward position (typical hyperpronation pattern of left foot > right foot produces the following postural pattern: left PSIS is anterior [forward] and superior [higher] relative to right PSIS, pelvis is unleveled, left leg is functionally longer than right leg, thoracic cage is rotated counterclockwise, left shoulder is protracted [forward] and superior [higher] relative to right shoulder). Cranial and connective tissue compensations are commonly initiated and/or perpetuated by this forward postural shift which, in turn, can be responsible for many of the chronic pain conditions seen clinically. Adapted and reproduced with permission from the American Journal of Pain Management, 1995.

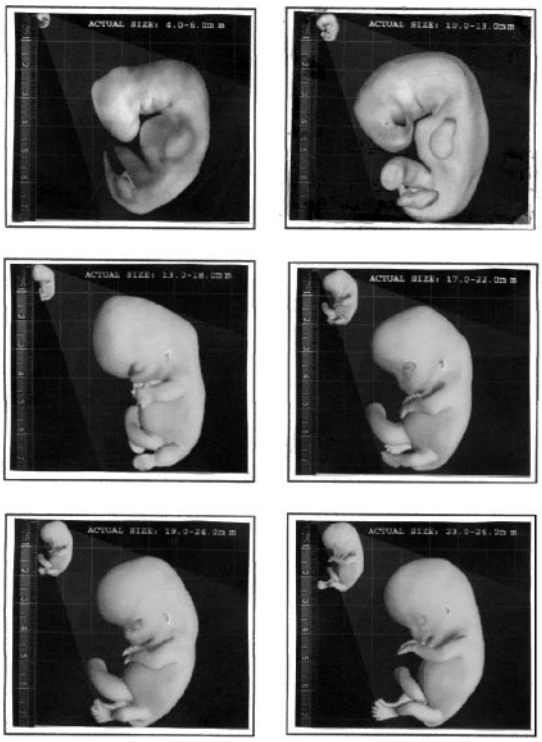

Fig. 4 Left slide, top: Week 3 pf. Limb bud appears as a swelling opposite the lower lumbars. Right slide, top: Week 6 pf. Limb bud sits at right angles to rump of embryo. Soles of feet and posterior compartments of leg and thigh face cephalad. Left slide, middle: Week 8 pf. Limb bud has rotated 90° around its longitudinal axis. Soles and posterior surfaces of thigh and legs now face one another. Right slide, middle: Week 8.5 pf. Primordial toes appear in the developing foetus. Left slide, bottom: Week 9 pf. Primordial ankle and knee joints appear. Right slide, bottom: Week 10 pf. Leg and thigh are unwinding around their longitudinal axes (clockwise left, counterclockwise right) leaving the foot (calcaneus and talus) in supinatus. Adapted and reproduced with permission from Visible Embryos © Mousework Inc.

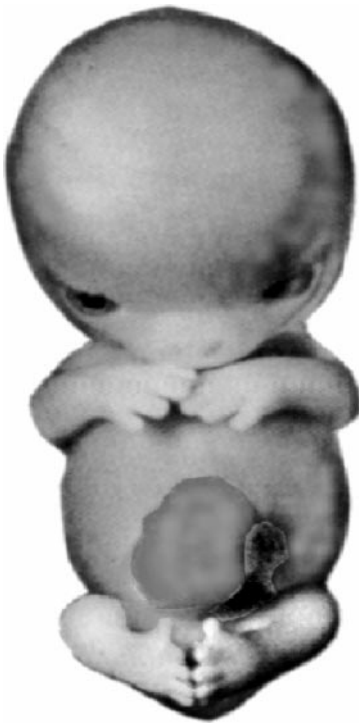

Fig. 5 Frontal view embryo week 8.5 pf. Lower limb bud is rotating 90° around its longitudinal axis (clockwise left, counterclockwise right). Posterior thigh and leg compartments face one another, as do the heels (calcaneal supinatus) and soles (talar supinatus) of the feet. USPTO #6,092,314 Sheet 2, Figure 2. Adapted and reproduced with permission from GRD BioTech Inc., 2000.

Liley 1996). And it is this forward posture and ensuing compensations that lead the patient into chronic pain (Rothbart & Esterbrook 1988, Rothbart et al. 1992, Rothbart & Hansen 1995, Petersen 1995, Schneider 1995, Filner 1996, Liley 1996) (Figs 2 and 3).

Section 1 (Embryology) briefly outlines [1] the normal ontogenetic stages within the lower limb bud and [2] the abnormal ontogenetic events that result in either the Clubfoot deformity (Cfd) or Rothbart foot structure (RFS). (This paper does not deal with the positional or structural deformations that can occur postnatally, e.g. rearfoot varum from massive trauma to the heel bone, leg length discrepancy from sepsis of the femoral growth plate, etc) Section 2 (RFS, clinical significance of PME) discusses RFS impact on posture and gaiting, and provides a methodology for diagnosing RFS in the adult foot. Section 3 (stabilizing RFS) introduces an innovative foot appliance that [1] reduces static *and dynamic* hyperpronation and [2] reverses postural shifts generated from RFS.

Embryology

Ontogenetic retention of talar supinatus: the Rothbart foot structure

One thousand and six Egyptian feet were evaluated for talar supinatus (Sewell 1906). Angular variances, up to 20°, were reported. If the talar head fails to unwind (remains in supinatus), the navicular remains relatively supinated (torsionally twisted inward around its longitudinal axis), and with it, the internal cuneiform, 1st metatarsal and big toe (Straus 1927, Olivier 1962). Rothbart terms the retained structural elevation of the 1st metatarsal and big toe: *Primus Metatarsus Elevatus* (*PME*; see Fig. 6). PME values are clinically quantified using microwedges (Fig. 7).

In the postnatal foot, the navicular articulates with all three cuneiforms. From a structuralist view point, one might conclude that the navicular impacts all three cuneiforms. From an embryological

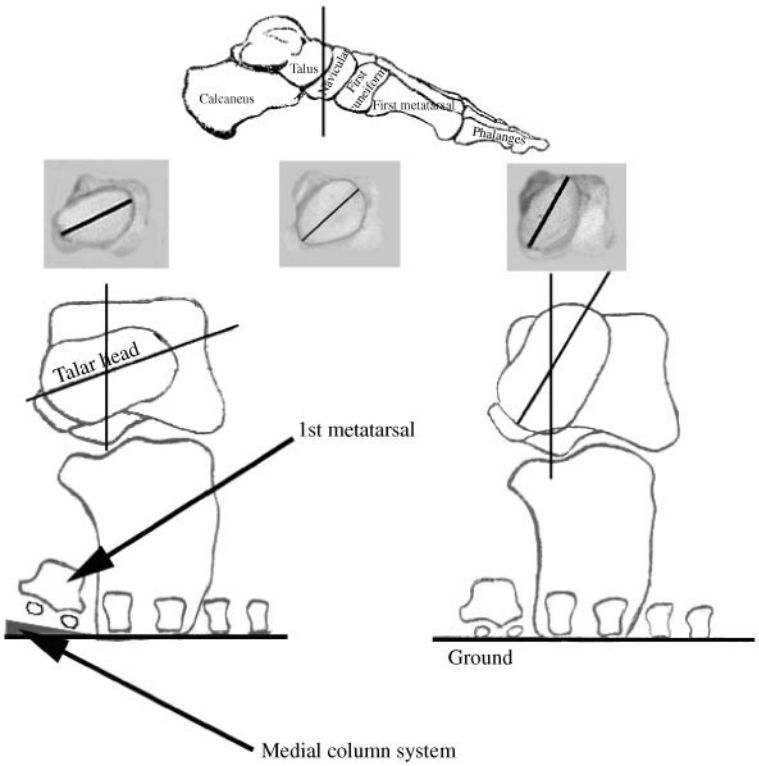

Fig. 6 Talar supinatus, frontal view, left foot. Torsional unwinding of the talar supinatus delineated (see upper diagrams, left to right). As the talar head unwinds, Primus Metatarsus Elevatus is decreased. Lower diagram left illustrates retention of talar supinatus and resulting twist and elevation of the 1st metatarsal (PME) relative to the ground. Lower diagram right illustrated complete unwinding of the talar head and resulting plantargrade position of the 1st metatarsal relative to the ground. USPTO #6,092,314 Sheet 3, Figure 4. Adapted and reproduced with permission from GRD BioTech Inc., 2000.

1st metatarsal/big toe (standing position, subtalar joint neutral).

Ontogenetic retention of calcaneal supinatus: the Clubfoot deformity

If the calcaneus fails to unwind (remains in relative supinatus), the cuboid remains in supinatus, and with it, the lateral two cuneiforms,

perspective, this is not the case. Straus (1927), Streeter (1945) and Olivier (1962) depict that is the unwinding of the talar head, that determines the shape and contour of the navicular, internal cuneiform, 1st metatarsal and big toe. That is, the relative structural position of the 1st metatarsal/big toe is determined by the sculpturing of the talar head. Retention of postnatal talar supinatus results in a visibly elevated

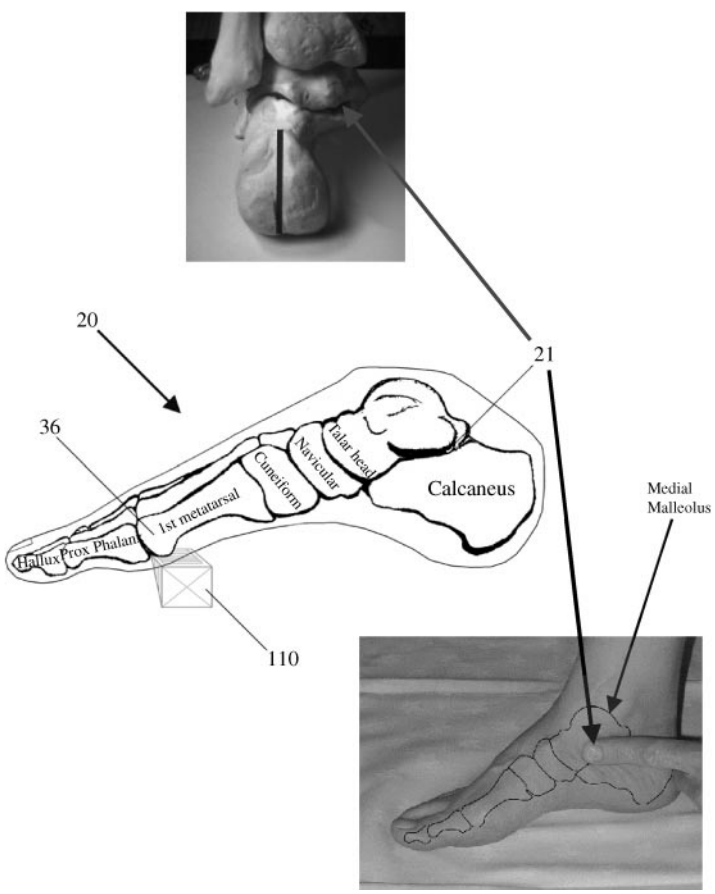

Fig. 7 Measuring PME. With the client standing, locate the subtalar (talocalcaneal) joint (21) as follows: one finger width below and anterior to the medial malleolus (see bottom photograph). Keeping your finger over the subtalar joint space, have your client transfer 90% of their weight to the other foot. With your other free hand, slowly guide the foot through pronation (inversion) – supination (eversion) until the margins of the subtalar joint space feel parallel to one another (see top photograph). If the subtalar joint is pronated, the joint space disappears. If the subtalar joint is supinated, the joint space gaps. Slide the microwedge (110) under the ball of the foot (1st metatarsal) until slight resistance is met. Record the PME value off the microwedge.

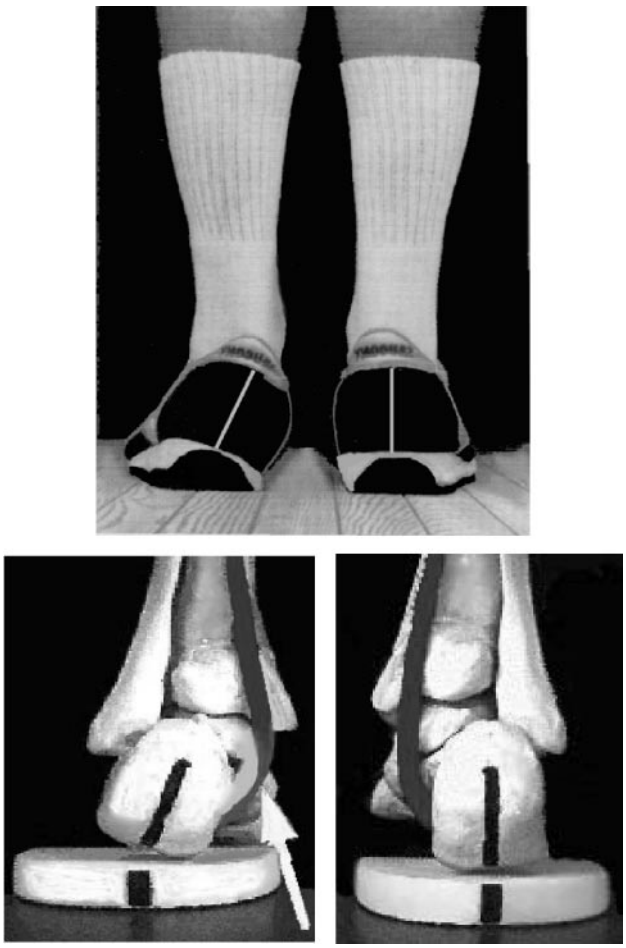

Fig. 8 The neurovascular network sits posteriorly and inferiorly to the medial malleolus. Hyperpronation associated with PME >20 mm (see left foot) can compress this network, significantly reducing blood flow and innervation to the bottom of the foot (see bottom, left diagram).

lesser four metatarsals and accompanying phalanges (Bohm 1929). The footplate unwinds heel to toe (Streeter 1945, 1948, 1951), thus the maxim: *as goes the calcaneus, so goes the talus*. Retention of calcaneal supinatus is always accompanied by talar supinatus, the Clubfoot deformity (Cfs) (Bohm 1929).

In the postnatal foot, the calcaneus articulates with the cuboid, which in turn articulates with the navicular, lateral cuneiform and lateral two metatarsals. From a structuralist viewpoint, one might conclude that the calcaneus only impacts these structures. From an embryological prospective, this is not the case. Bohm (1929) describes how the ontogenetic unwinding of the calcaneus affects the lateral column of the embryonic foot. That is, the relative structural position of the cuboid, the lateral two cuneiforms and four lateral metatarsals/phalanges are determined by the sculpturing of the calcaneus. Retention of calcaneal supinatus in the prenatal foot is manifested as the Cfd in the postnatal foot.

RFS: clinical significance of PME

RFS is subtle in the newborn's foot due to the bulging longitudinal fat pad and incomplete ossification of the tarsal bones. At birth, the primary ossification site of the talus has already appeared (Sewell, 1906). However, the onset of ossification of the navicular is variable (2.7–4.0 years of age) and late compared to the other tarsal bones (Caffey 1972, Lang 1972, Bardeen 1905, Hoeer et al. 1962). The topographical contouring of the inner longitudinal arch (ILA) starts to develop between 12 and 16 months of age. A definite ILA is present by age 2. By age 30 months, maximal inner longitudinal arching is attained, the apex corresponding to the navicular tuberosity (Blais & Green 1956). As the ILA takes on its adult contour and the navicular bone ossifies, PME becomes more visible. However, measuring PME in a child less than 4 years of age is prone to error because the navicular bone has not yet ossified.

In the adult foot (aged 4 and over), PME values between 10 mm and 25 mm are pathognomic of RFS. Flexible arches (higher arch sitting than standing) and flexible flatfeet (arch sitting, no arch standing) are functional compensations commonly associated with RFS. The supinatory 'strike', in which the body's weight is carried on the outside margins of the heel, is another common compensation associated with RFS. Visual inspection of the shoes reveals an excessive wear pattern on the outside margins of the heels. When dealing with RFS, measuring forefoot varum across the entire sole (metatarsals 1–5) is a misdirected clinical assessment, since talar supinatus only affects the 1st metatarsal.

A preliminary investigation correlating PME values to compensatory patterns in 37 infants, ages 2–4, was undertaken at the Bellevue Foot and Ankle Center in Bellevue Washington (1988–1994).

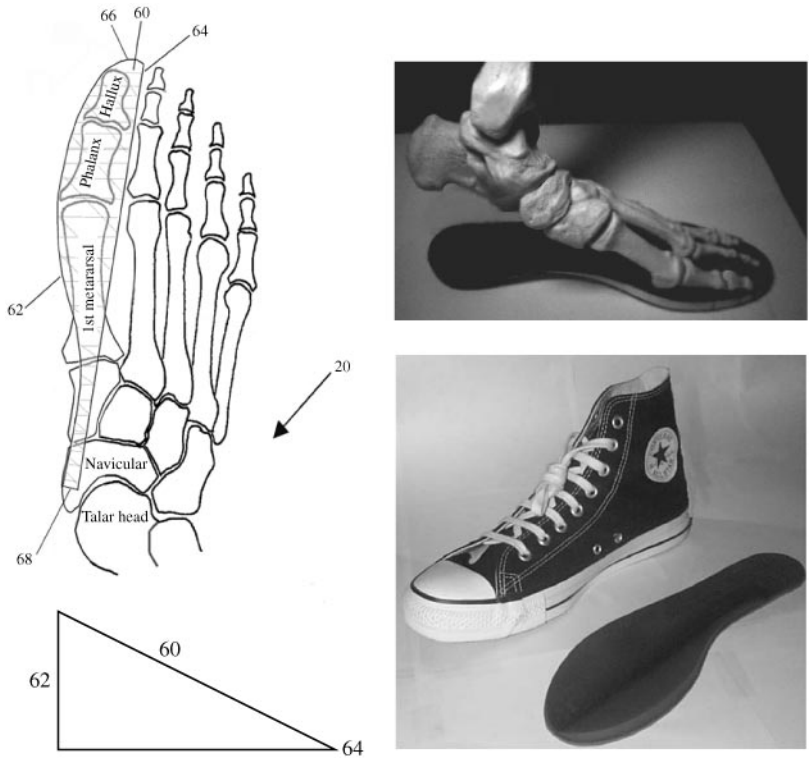

Fig. 9 Medial column system. Tactile feedback loop extending from the navicular to the hallux. The system is sloped (60) medial to lateral. The vertical dimensioning discussed in the paper is represented by line (62), which extends along the medial border of the foot. Typically arch supports are not incorporated into the medial column system. Visual gait studies on 317 clients delineate: 30% tactile feedback results in approximately 70% decrease in dynamic hyperpronation (the 30–70 rule). USPTO #6,092,314 Sheet 4, Figure 5. Adapted and reproduced with permission from GRD BioTech Inc., 2000.

The following factors were measured: foot type (flexible arches or flexible flatfeet), position of feet (pigeon-toed, straight or out-toed), heel strike (inside or outside), and PME values. The results suggest that infants (under age 4) with PME values >20 mm tend to adduct (pigeon-toe). As children they are typically inside heel strikers and flat-footed (flexible flat-feet). Infants with 11–19 mm PME tend to abduct (out toe). As children they are outside heel strikers and have flexible arches.

The above findings provide a possible pattern of compensations associated with various PME ranges. Infants with higher PME values (>20 mm) tend to instinctively reduce (adapt to) their dynamic hyperpronation by turning their feet inward. Infants exhibiting lower PME values (11–19 mm) tend to either 'fall into' a mild to moderate hyperpronation pattern (feet turned outward), or adapt by walking on the outsides of their feet (feet straight forward). In both cases, as children they appear to have flexible arches. The above observations are insightful but not definitive (26 of the 37 children follow these patterns). This study needs to be repeated using larger sample sizes over a longer period of time.

Clinical data compiled at GRD BioTech Inc in Seattle Washington (2001) collaborates earlier studies linking higher PME values to RFS (Rothbart et al. 1992, Cummings 1994, Schneider et al. 1995, Rothbart & Hansen 1995). Three hundred and seventeen patients were measured with microwedges (Fig. 7). Three hundred and six (96.5%) demonstrated an RFS with a mean PME of 16 mm (distance [freeway space] between the 1st metatarsal and ground, subtalar joint in neutral [joint congruity] position). Of these, 271 (88.5%) had flexible arches (mean PME of 14 mm), and 35 (11.5%) had flexible flat-feet (mean PME of 24 mm). Ten (3.1%) demonstrated a non-RFS (mean PME of 6 mm). 1 (0.3%) had a preclinical Cfd (PME of 37 mm).

The typical pronated posture

PME (>10 mm) significantly forces the walking foot to roll inward, forward and downward (typically left foot $>$ right foot) until the 1st metatarsal reaches the ground (Rothbart & Esterbrook 1988, Rothbart et al. 1995, Schneider 1995, Filner 1996, Liley 1996). This shifts the body's center of gravity forward and downward, which in turn, pulls the innominates forward and downward (typically left $>$ right). The pelvis is unleveled, resulting in a functional leg length discrepancy (left longer than right). As these displacements cascade up the axial framework, scoliotic and kyphotic curves are exaggerated. The thoracic cage twists (usually counterclockwise). The shoulders. protract (left $>$ right) The right shoulder drops. Cranially, the maxilla moves anteriorly relative to the mandible resulting in an overjet bite. This gravity-induced skeletal 'collapse' is termed BioImplosion (Rothbart et al. 1992, Rothbart & Yerratt 1994, Schneider 1995, Petersen et al. 1995, Filner 1996, Liley 1996), which over time can initiate strain and trigger point patterns, foot to jaw (Rothbart et al. 1992, Rothbart & Hansen 1995, Schneider 1995,

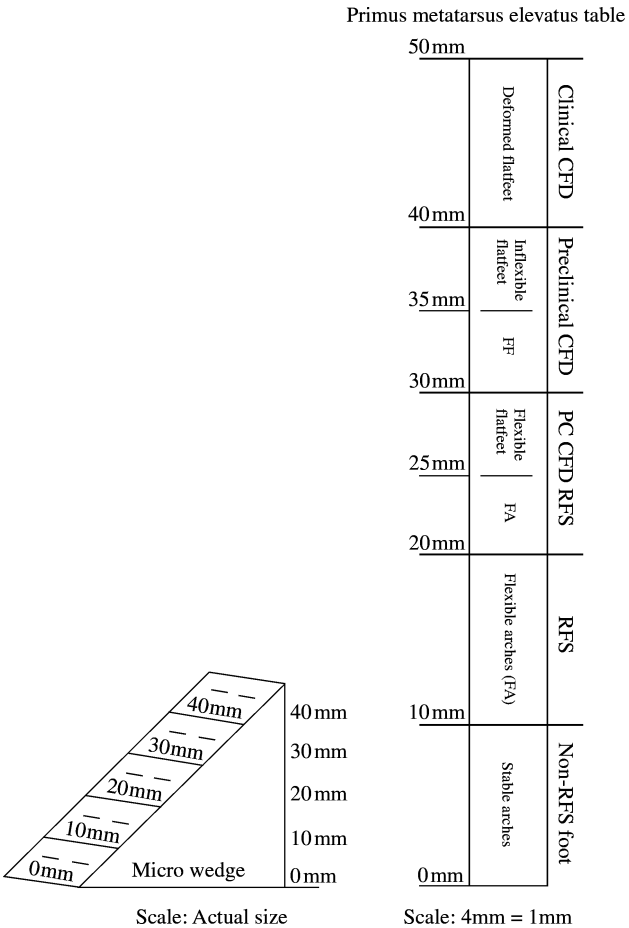

Fig. 10 Primus metatarsus elevatus table. A linear correlation is seen between PME values and foot structure. Low PME values (<10 mm ± 1–2 mm) are associated with a non-RFS. In the absence of significant soft tissue or cranial adaptations: midrange PME values (10–25 mm ± 1–2 mm) are associated with RFS, ranging from flexible arches (10–19 mm ± 1–2 mm) to flexible flatfeet (25–35 mm ± 1–2 mm). High PME values (>40 mm) are associated with Clubfoot deformities with overt arch deformation.

Petersen 1995, Filner 1996, Liley 1996).* (See Fig. 2).

Johnson and Cross (1990) describe a common compensatory pattern (CCP) that is very similar to the BioImploded posture outlined above. Johnson and Cross link posture to torsions within the pelvis. Rothbart et al. (1992) link posture to torsions (hyperpronation) within the feet. Zink & Lawson (1979) describe a disparent (atypical) CCP. Interesting enough, this disparent CCP closely parallels the Bio-Imploded posture resulting from the less common hyperpronation pattern of right > left.

Hyperpronation associated with PME > 20 mm can mechanically compress the posterior tibial vessels (nerve, artery and vein) as they enter the foot behind the medial malleolus. This is quickly determined by taking standing PT pulses: foot neutral and then pronated (Fig. 8).

From clinical observations, RFS appears to be a dominant, single autosomal Mendelian characteristic. This would explain the familial

*For example, chronic shoulder protraction can lead to a functional thoracic outlet syndrome.

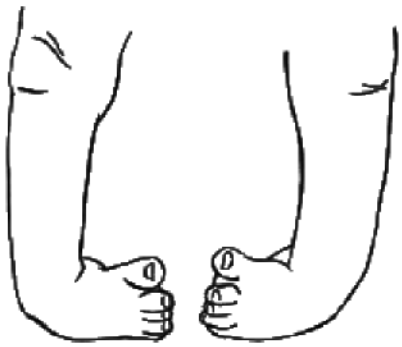

Fig. 11 The Clubfoot deformity.

diathesis and the inordinate rate of expansion in the genetic pool (Garbalosa et al. 1994). Harris and Beath (1949) evaluated the foot structure of 3619 Canadian men. Their study suggests a 40% incidence of Morton's foot structure (short 1st metatarsal). A more recent investigation, presented at the Annual Conference of the American Academy of Pain Management (Rothbart 1995), suggests a 95% incidence of Morton/Rothbart's foot structure (short and elevated 1st metatarsal).

Stabilizing RFS

Heel wedges and arch supports

Calcaneal wedging decreases standing hyper-pronation. Calcaneal wedging *does not* decrease walking hyperpronation generated from the elevated 1st metatarsal. In fact, inverting the whole foot in this fashion can increase the relative elevation of the 1st metatarsal to the ground, which in turn can increase the hyperpronation generated at heel-lift. In a similar fashion, arch supports decrease midstance hyperpronation, but are ineffective as the ball of the foot engages in weight bearing. Paradoxically, arch supports affect feet like immobilization casts affect muscles: function is improved at the price

of muscle strength. In time, these same feet become weaker/more pronated (when barefooted) than they were prior to arch support therapy. For this reason, the author rarely uses heel wedges in RFF, and judiciously only uses arch supports in feet that are functionally flat.

Medial column systems

Medial column systems effectively reduce dynamic hyperpronation associated with RFS. The effect of these systems extend from the navicular, medial cuneiform, 1st metatarsal bone, to the proximal phalanx and hallux (Fig. 9). With each step, a tactile feedback loop is triggered that auto corrects the hyperpronation being initiated by the PME. The suggested rule of thumb is: 30% tactile = 70% improvement (the 30–70 rule is observational, based on empirical data derived through gait evaluation of 317 patients at the GRD BioTech facility, 1996–1998, and hence not meant to be definitive or all inclusive). For example, a 6 mm medial column system (Fig. 9[62]) under a foot measuring 20 mm PME (assuming no significant hypertonicity patterns or cranial deformations) tends to decrease the observable hyperpronation by approximately 70%. Via proprioception, this system provides feedback to the big toe (proximal phalanx and hallux) and 1st metatarsal. All the other weight bearing structures proximal to the big toe and 1st metatarsal (ankle, knee, pelvis, spine, neck, head and jaw) tend to spontaneously correct themselves around this change. The body's center of gravity shifts posteriorly. The posture is *visually* more vertical. The inner longitudinal arch is not supported in feet with flexible arches. (Arch supports are used in flatfeet to facilitate the transfer of vector forces across the ILA). The heel is neither cupped nor wedged.

Tactile feedback systems are based on the observation that small repetitive stimuli bring about significant changes. Interesting enough, in terms of foot mechanics, this occurs through kinesthetic reposturing. The foot-brain connection recognizes this proprioceptive input as innately correct. The foot is reminded where it should be (not here, but over there) and automatically makes the adjustment. With each step, the foot becomes stronger, the posture straighter. Hyperpronation is reduced. And not surprisingly, as the soft tissue and cranial adaptations are addressed, the tactile input in the medial column system may require recalibration (adjustment). An unexpected outcome using foot tactile systems is the observation that hypertonic muscles can become *disassociated* from Morton/Rothbart's foot structure. That is, these short and tight muscles evolve into self-perpetuating loops. Their associated pain referral patterns prove intractable to foot therapy alone. This underscores the importance of concurrent foot and soft tissue therapy when dealing with chronic pain issues.

The medial column foot system is used as a proprioceptive stimulator. It is dimensioned to partially fill-in the 'freeway space' that exists between the 1st metatarsal/big toe and ground (Fig. 6). If this system is dimensioned so that it completely fills in the 'freeway space', it becomes a supportive device. Such devices over a period of time weaken structure and should not be used under the RFS. Using a medial column foot system in a *non*-RFS places a disruptive upward load under the 1st metatarsal head. This can dramatically limit the range of dorsiflexion within the 1st metatarsal-phalangeal articulation and lead to a potential functional hallux limitus.

Summation

The foetal development of the lower limb bud, and specifically the footplate, is reviewed. Clinical studies describe the impact talar and calcaneal supinatus have on the medial column of the foot: incomplete unwinding of the talar head results in the Morton/Rothbart foot structure; incomplete unwinding of the calcaneus results in the Clubfoot deformity (Fig. 11).

Zitzlesperger (1960) and Elftman's (1960) foot models demonstrate an inverse relationship between pronation and arch stability: as pronation increases, arches flatten. Clinical data from GRD BioTech (2001) demonstrates a similar inverse relationship between PME (elevation of the 1st metatarsal and big toe) and arch stability: as PME increases, arch stability decreases. PME values less than 10 mm correlate to stable arches (same arch height sitting or standing). PME values between 10 mm and 20 mm correlate to flexible arches (higher arch sitting than standing), 25 mm–30 mm to flexible flatfeet (arch sitting, no arch standing), 35 mm–40 mm to inflexible flatfeet (no arch sitting or standing), and >40 mm to rigid and structurally deformed feet (Fig. 11 – the Clubfoot deformity).

PME between 10 and 25 mm are associated with RFS, a foot in which the 1st metatarsal is short and structurally elevated relative to the lesser metatarsals. Rearfoot posts destabilize the RFS. Arch supports weaken the RFS. And forefoot varum posts (Root

et al. 1971) structurally strain the RFS.**

Published studies link PME to hyperpronation and hyperpronation to BioImplosion. Dimensioning medial column systems at 30% of the measured PME tend to decrease dynamic hyperpronation by 70% ± 10%. This in turn tends to reduce pelvic tilts by 50% ± 20%, shoulder protractions by 40% ± 20% and forward head positions by 30% ± 20%. In the absence of any significant psychological or nutritional imbalances, hypertonic muscles become significantly more amenable to long-term resolution as posture becomes more vertical.

REFERENCES

Aiton JF, McDonough A, McLachlan JC, Whiten SC, Smart SD 1995 World Wide Web Access to the British Universities Human Embryo Database. Journal of Anatomy 190: 149–154

Cummins H 1929 The topographic history of the volar pads in human embryo. Contributions Embryology 20: 105

Bardeen CR 1905 Studies of the development of the human skeleton. American Journal Anatomy 4: 265

Blais MM, Green WT et al. 1956 Lengths of the growing foot. Bone Joint Surgery, 38[A]: 998

Bohm M 1929 The embryologic origin of clubfoot. Journal Bone Joint Surgery, 11: 2, 229

Caffey JP 1972 Pediatric X-Ray Diagnosis. Vol 2: 884. 6th Edition, YearBook Medical Publishers, Chicago

Cichoke AJ 1999 The Back Pain Bible. Ch. IV, pp 35. Keats Publishing, Chicago

Cummings G 1994–95 Personal communications with Author and on campus visit: Blind study-using microwedges to measure vertical dimensions under patients seen by students. Clinical Director, Department of Physical Therapy, Georgia State University, Atlanta GA

Elftman H 1960 The transverse tarsal joint and its control. Clinical Orthopedics 16: 41

Filner B et al. 1995 The relationship of anterior upper torso muscular dysfunction with structural hyperpronation in chronic pain patients. Annual Meeting, American Academy of Pain Management, Dallas, September

Filner B 1996 Postural Analysis: Differential Diagnosis. Annual Conference, American Academy of Pain Management, Washington DC

Gasser RF 1946 Atlas of Human Embryos. Harper and Row Publishing, Hagerstown

Garbalosa JC et al. 1994 The frontal plane relationship of the forefoot to the rearfoot in an asymptomatic population. JOSPT 24: 4, 200–206

GRD BioTech Inc 2001 317 clients evaluated between 1996–1998 using microwedges and visual gait analysis, with and without postural insoles. Seattle, WA

GRD BioTech Inc 2000 Foot Support (Medial Column) System and Use in Shoe Lasts. United States Patent Number 6,092,314. Seattle, WA, July

Harris RI, Beath T 1949 The short first metatarsal: Its incidence and clinical significance. Journal Bone Joint Surgery [Am] 31: 553–565

Hoerr LN et al. 1962 Radiographic Atlas of Skeletal Development of the Foot and Ankle – A Standard Reference. Charles C Thomas, Springfield

Jirasek JE, Keith LG 2001 An Atlas of the Human Embryo and Fetus: A photographic review of human prenatal development. CRS Press, Parthenon Publishers, 1st edition

Johnson K, Cross N 1990 Common compensatory pattern and its relation to lumbar facet angles. Journal American Osteopathic Association 90: 942

Lang J et al. 1972 Praktische Anatomic Erster Band Vierter Teil – Bein und Statik, p. 31. Berlin, Springer Verlag

Lash J, Alonso L, Benke L, Chang A, Dietrich A, Nathan D 1997 Basic Embryology Review Program: Embryo images. University of Pennsylvania, School of Medicine. Web Home Page: http://www.med.upenn.edu.meded/

Liley P 1996 Postural analysis: Head guidance and ground support. Annual Conference American Academy Pain Management, Washington DC

McLachlan JC, Aiton JF, Whiten SC, Smart SD 1997 3-D Modeling of Human Embryo Morphology using QuickTime VR. In "Molecular Genetics of Human Development". pp 227–237, eds. Strachan T, Lindsay S & Wilson D Bios Scientific Publishers Ltd, Oxford

Morton DJ 1935 The Human Foot. Its evolution, physiology and functional disorders. Columbia University Press, New York

Morton DJ 1932 Means for compensating for foot abnormalities. US Patent Number 1,847,973, USPTO, March

O'Rahilly R, Gardner E et al. 1956 The ectodermal thickening and ridge in the limbs of staged human embryos. Journal Embryology and Experimental Morphology 4: 256

O'Rahilly R, Gardner E 1975 The timing and sequence of events in the development of the limbs in the human embryo. Anatomical Embryology, 1–23

Olivier G 1962 Formation du Squelette des members. pp 145–189. Paris, vigot Freres

Patten BM 1946 Human Embryology. The Blakiston Company, Philadelphia

Petersen M et al. 1995 The relationship of lower leg muscular dysfunction with structural hyperpronation in chronic pain syndrome patients. Posterboard Presentation, Annual Conference, American Academy Pain Management, Dallas

Root ML, Orien WP, Weed JH, Hughes RJ 1971 Biomechanical examination of the foot. 1st ed. Los Angeles: Clinical Biomechanical Corporation

Rothbart BA, McCombs A, Riniker L 1992 BioImplosion. The treatment of chronic pain syndrome. Posterboard Presentation, Annual Conference, American Academy of Pain Management, Albuquerque

Rothbart BA 1995 Postural Kinetics. Two studies presented: [1] Gait compensations observed in 37 infants between 1988–1994. [2] PME taken on 3417 patients between 1984–1995. Annual Conference, American Academy of Pain Management, Dallas

Rothbart BA, Yerratt M 1994 An innovative mechanical approach to treating chronic knee pain: A BioImplosion Model. American Journal Pain Management 4: 3, 123–128

Rothbart BA, Esterbrook L 1988 Excessive Pronation: a major biomechanical determinant in the development of chondromalacia and pelvic lists. Journal Manipulative Physiologic Therapeutics 11: 5, 373–379

Rothbart BA, Liley P, Hansen K, Yerratt K 1995 Resolving chronic low back pain:

**Only the 1st metatarsal is in elevatus in the RFS. Forefoot varum posts elevates metatarsals I–IV. The terms supinatus and varum have been used interchangeably in the literature. By convention, supinatus is used to describe a torsionally inverted relationship in the prenatal foot. Varum is used to describe the same torsional (structural) relationship in the postnatal foot.

The foot connection. American Journal Pain Management, 5: 3, 84–89
Rothbart BA, Hansen K 1995 Postural Analysis: Definition of terms. Posterboard Presentation. Annual Conference, American Academy Pain Management, Dallas
Svae B 2000 Personal Communication with Author, Postural Dynamics, Seattle, WA
Schneider M et al. 1995 The importance of controlling hyperpronation with a postural control device in resolving musculoskeletal pain in chronic syndrome patients. Posterboard Presentation, Annual Meeting, American Academy Pain Management, Dallas
Sewell RS 1906 A study of the astragalus (talus). Part IV. Journal Anatomy Physiology, Vol 40: 152
Smith BR 1999 Visualizing human embryos. Scientific American 280: 76–81
Smith BR, Huff DS, Johnson GA 1999 Magnetic resonance imaging of embryos: An Internet resource for the study of embryonic development. Computerized Medical Imaging and Graphics 23: 33–40
Straus WL 1927 Growth of the human foot and its evolutionary significance. Contributions in Embryology 19: 95
Streeter GL 1945, 1948, 1951 Developmental horizons in human embryos. In Contributions to Embryology, Vols. 21, 32, 34. Washington DC. Carnegie Institution of Washington
Tabibzadeh S, Pettersson TV 1995 An Atlas of Human Embryogenesis. Frontiers in BioScience. Website http://bioscience.igh,cnrs.fr/atlases/fert/embrper.htm
Travell JG, Simons DG 1992 Myofascial Pain and Dysfunction. The Trigger Point Manual. Vol. 2: 20, pp 379–392, Williams and Wilkins, Baltimore
Visible Embryos 2001 Embryos Slides. Mouseworks Inc. Website: WebMaster@visembryo.com
Zink JG, Lawson W 1979 An osteopathic structural examination and function interpretation of the soma. Osteopathic Annals 7: 12
Zitzlesperger S 1960 the mechanics of the foot based on the concept of the skeleton as a statically indetermined space framework. Clinical Orthopedics 16: 47–63