The Natural History and Pathophysiology of Flexible Flatfoot

Edwin J. Harris, DPM, FACFAS

Flat feet in infants, children, and adolescents are so common that the lack of agreement about the natural history and pathophysiology of the condition is surprising. There is great controversy about the role that flat feet play in health, and disagreement on the indications for treatment. The frequent occurrence raises the question of whether many of the mild forms are really a part of normal development and not a sign of disease.

Flat feet are considered by parents and some physicians as diseased, deformed, and in need of treatment simply because they exist. Staheli[1,2] suggested that part of this attitude may be cultural, because high arches have long been considered a sign of aristocracy, virtue, and well-being and therefore, good. Lower arches were traditionally considered a deformity, evidence of poor health, and something that needed to be treated and therefore bad.[1,2] It is the deviation from some ideal foot structure that supposedly makes flatfoot abnormal and presumably will result in long-term morbidity and disability in adulthood. Conversely, high-arched feet are indicators of muscle imbalance and are signs of underlying conditions that include static encephalopathy, myopathy, spinal dysraphisms, and other serious pathologic conditions. Triathletes with supinated foot types are more likely to sustain overuse injuries.[3] Manoli and Graham[4] regarded subtle cavoid feet as underpronators. This raises the question of whether most forms of flat feet constitute a morbid process with the characteristic symptoms and distinct natural history that, if left unmodified, will prove to be disabling. Only then does flatfoot meet the definition of a disease. Flatfoot becomes a medical issue only when symptoms develop. The mere absence of a well-formed medial longitudinal arch does not necessarily imply a pathologic condition.[5]

Flat feet continue to generate parental concern and result in many visits to health care professionals for consultation and treatment. Parents themselves may have been diagnosed with flat feet when they were children. They may have worn special

Department of Orthopaedics and Rehabilitation, Section of Podiatry, Loyola University Medical Center, Loyola University Chicago, Maguire Building Room 1700, 2160 South First Avenue, Maywood, IL 60153, USA
E-mail address: eharrisdpm@AOL.com

shoes, orthoses, or perhaps had surgery. They may be convinced that the treatments they received gave them a better prognosis and wish the same for their children. They may be aware of contributing factors that include genetics, ligamentous laxity, and association with other named syndromes. Their presumption is that available treatment influences the natural history of flatfoot in a positive way and brings about a long-term change in the function and the anatomy of the child's feet. If told by a physician that the child needs no treatment, parents may shop from doctor to doctor until they find someone who will satisfy their perceived needs.

There are huge gaps in our knowledge about flatfoot. Terminology of foot movement is confusing.[6,7] There is no agreement on a name for this entity. It is variously referred to as flatfoot, pes planus, pes valgoplanus, pes planovalgus, talipes valgus, and pronation syndrome. It is an anatomic lesion and not a diagnosis or even a single condition. It is a collection of clinical entities that are grouped together because they share similar features.

It is unfortunate that the term ''flatfoot'' enjoys such universal usage. It is misleading because it concentrates only on the sagittal plane component and the foot surface contact area, to the exclusion of the other planes. A literature review identified 22 articles dealing with height of the navicular from the floor and ''navicular drop.''[8–29] Flatfoot is a triplane deformity. Although the deformity is on 3 planes, 1 plane often dominates. Newer additions to biomechanical theory call this planal dominance.[30,31] As research continues, there is less concentration on the subtalar joint and more on the talocalcaneonavicular joint complex (the acetabulum pedis).[32–35]

The American College of Foot and Ankle Surgeons sponsored a project to develop clinical pathways for clinical diagnosis and treatment recommendations.[36] Published in 2004, the investigators identified several subsets of pediatric flatfoot, including flexible flatfoot, rigid flatfoot, skewfoot deformity, and flatfoot associated with some more specific diagnoses. Talipes calcaneovalgus and oblique talus deformity were not included in this classification, even though talipes calcaneovalgus is often referred to as ''infantile flatfoot.''[37–39]

Flexible flatfoot was divided into physiologic and nonphysiologic flatfoot. Nonphysiologic flatfoot may be asymptomatic or symptomatic. Rigid flatfoot was divided into congenital convex pes valgus, flatfoot associated with tarsal coalition, peroneal spastic flatfoot without tarsal coalition, and iatrogenic flatfoot. Skewfoot combines severe rearfoot pronation and rigid forefoot adductovarus. Flat feet associated with other issues are caused by neurologic disease, muscular disease, syndromes, and collagen vascular disease. There is no progressive relationship between flexible flatfoot and rigid deformities. Simple flatfoot does not become congenital convex pes valgus. Flexible flatfoot does not progress to rigid deformity in most cases.

There is difficulty even in defining flexible flatfoot. There is agreement that there is a normal arch when non–weight-bearing and a flattened arch when the child stands. It is hard to recognize the transition from the physiologic to the pathologic.[40–42] Nonphysiologic flexible flatfoot progresses over time instead of improving or at least stabilizing. It is more severe than physiologic flexible flatfoot and has excessive heel eversion with an unstable talonavicular joint. Tight heel cords and gait disturbance are commonly associated with nonphysiologic flatfoot. Children with equinus secondary to tight heel cords may benefit from stretching exercises, and, occasionally, heel cord lengthening. Orthoses may also be indicated.[43]

Most flexible flat feet are physiologic, asymptomatic, and require no treatment.[44–46] The natural history of physiologic flexible flatfoot is presumed to be one of improvement over time. Children with asymptomatic flexible flatfoot should be monitored clinically for development of symptoms and signs of progression. It is difficult to

identify clinical factors in young children that may lead to a change in classification. Initial evaluation of the child should be thorough. Continued progression requires reassessment to search for underlying disease. Rigid flatfoot is identified by a stiff, flattened arch on and off weight-bearing. There is little argument that rigid flatfoot, skewfoot, and flatfoot associated with neuromuscular abnormalities, congenital syndromes, and collagen diseases are clearly pathologic and require treatment. There are no data available to suggest that these forms of flatfoot have any natural history that may result in clinical improvement over time. The status of flexible flatfoot is much less clear.

FLEXIBLE FLATFOOT

The controversy relates to that member of the flatfoot spectrum referred to as flexible flatfoot. Should all forms of flexible flatfoot be grouped together? Are all forms pathologic? There is little argument about the need to treat those forms of flatfoot that are clearly pathologic. There is a presumption that all flexible flatfoot is disease. Aggressive long-term management of all pronation has been advocated historically.[47] There is little discussion about the morbidity experienced by the child. It seems that it is the anatomic lesion that is considered to be objectionable. This leads to the question: if flexible flatfoot is a common issue, asymptomatic and without long-term morbidity, then when is treatment justified?

Asymptomatic flexible flatfoot is an almost universal finding in toddlers. This high frequency has been attributed to several things. One explanation is that the thickness of the plantar soft tissue is made up of "baby fat." This produces a plantigrade foot that may only appear flat. It is difficult to determine true flatness by physical examination alone. The only way is through evaluation of standardized weight-bearing antero-posterior (AP) and lateral radiographs of the feet. These can be difficult to obtain in small children, and radiographic techniques vary from investigator to investigator. Interpretation is complicated by incomplete ossification of the foot structures. The presumption that the ossific nuclei represent the true shape of the cartilaginous anlagen has been successfully challenged.[48–52] This makes radiologic interpretation difficult because age changes the location and shape of ossification centers until they become truly representative of the bone near skeletal maturity.

Other orthopedic variables also operate in this age group. Tibia varum is physiologic up to 2 years of age. Because this produces a varus or inverted tibial relationship to the support surface, the only way that the infant's medial column of the foot can reach the ground is for the rearfoot to pronate. By 2 years of age, most children have parallel tibias or genu valgum. At this time, the presence or absence of abnormal pronation becomes more evident. Toddlers have an abducted externally rotated gait pattern. This gait places the long axis of the foot external to the line of progression and allows propulsion off the medial side of the foot. There is also the mistaken notion that all abduction of the foot is a sign of pronation. In reality, abduction is likely to be supra-malleolar (Fig. 1).[44]

To summarize: factors that can modify the natural history of pediatric flexible flatfoot include ligamentous laxity, obesity, proximal rotational abnormalities, tibial influence, pathologic tibia varum, equinus, presence of an os tibiale externum, and presence of tarsal coalition.[53]

THE NATURAL HISTORY OF FLEXIBLE FLATFOOT

There are few studies on the natural history of flexible flatfoot if left untreated and on the subsequent natural history of the condition when it is treated. The available

Fig. 1. A 12-year-old girl has severe external rotation of the lower extremities secondary to external tibial torsion and externally rotated hips. Although there is obvious pronated foot structure, the supramalleolar component produces the greatest component of the abducted gait.

literature is of questionable merit in the light of today's insistence on evidence-based medicine, levels of clinical evidence, and study construction. There are no data to conclusively prove that flexible flatfoot in infants and children leads to long-term morbidity in adults. The lack of agreement on the need to treat flexible flatfoot has resulted in the development of 2 polarized, dogmatic, and opposite philosophies regarding treatment. Today's physicians are forced to make decisions based on their personal training and experience and on conclusions drawn from the literature. It is difficult to evaluate the validity of these so-called authoritative conclusions. Some decisions are based on data, but many are "expert opinion." Even those based on data arrive at conclusions supported by statistical analyses that seem counterintuitive to common clinical experience.

This lack of data affects the evaluation of long-term benefits from the use of exercises, physical therapy, special shoes, and orthoses. One study dealing with the question of shoes and orthoses as modifiers of the natural history of flexible flatfoot shows how difficult it is to construct a valid scientific prospective study.[54] Interested readers should study the article by Wenger and colleagues[54] and the Editorial and the 2 Letters to the Editor that appeared in the same issue. Operative therapy is not immune to criticism and generates its own share of controversy. Surgical intervention certainly changes the anatomy, but it is permanent and not without risk. When potential morbidity and cost are factored in, it becomes even more imperative to demonstrate that such intervention is medically necessary and that therapy is likely to achieve the proposed goals.

There is a natural history to the development of the child's arch that cannot be denied. Staheli and colleagues[55] studied 441 normal subjects from 1 to 80 years of

age and concluded that flat feet are usual in infants, common in children, and within the normal range of observations made in adult feet. Their recommendation for management was documentation and observation.

Gould and colleagues[56] studied 225 beginning walkers and followed them for 4 years. All of the apparently normal toddlers had pes planus determined by radiographic and photographic parameters. Arches developed regardless of the footwear worn. Children who had arch-support footwear developed arches faster. Hyperpronation was evident in 77.9% and genu valgum in 92.3% of the 5-year-olds.

Garcia-Rodriguez and colleagues[57] studied the prevalence of flexible flatfoot in a population of 4- to 13-year-old schoolchildren in Malaga, Spain. They graded by severity a sample of 1181 children from a total population of 198,858 primary school children. They made 3 age groups (4–5, 8–9, and 12–13 years old) and classified their footprints into 3 grades of flat feet. They found the prevalence of flat feet to be 2.7% of the 1181 children sampled. Of the patient sample, 168 (14.2%) were receiving orthopedic treatment, but only 2.7% met the diagnostic criteria for flat feet. Of the group that met the criteria for flat feet, only 28.1% were being treated. Overweight children in the 4- to 5-year-old age group had increased prevalence for flat feet. Their data suggested that an excessive number of children within the study group were being treated.

Lin and colleagues[58] studied flexible flatfoot in preschool children in Taiwan using gait analysis. Two hundred and seventy-seven preschool children (201 boys and 176 girls), from 2 to 6 years of age, were enrolled in the study. The results showed that age, height, weight, foot progression angle, occurrence of physiologic knock knees, and joint laxity scores correlated with flat feet. Children with flat feet, compared with children without, performed physical tasks poorly and walked slowly, as determined by gait parameters. They concluded that flatfoot should not simply be regarded as a problem of static alignment of the ankle and foot complex but may be a consequence of dynamic functional change in the whole lower extremity.

El and colleagues[59] studied longitudinal arch morphology in 579 schoolchildren and evaluated generalized joint laxity, foot progression angle, frontal-hindfoot alignment, and longitudinal arch. They evaluated 82.8% as normal and mild flatfoot, and 17.2% were evaluated as moderate and severe flexible flatfoot. There was a significant negative correlation between arch index and age and between hypermobility score and age.

Pfeiffer and colleagues[60] studied 835 children between the ages of 3 and 6 years, basing their diagnosis of flatfoot on a valgus position of the heel and poor formation of the arch. Prevalence of flexible flatfoot in the 3- to 6-year-old age group was 44%. Incidence of pathologic flatfoot was less than 1%. Ten percent of the children were wearing arch supports. The prevalence of flatfoot decreased by age from 54% in the 3-year-old age group to 24% in the 6-year-old age group. There were more boys than girls with flat feet (52% vs 36%). Obesity was a complication in 13%. They concluded that likelihood of flatfoot was influenced by age, gender, and weight. They concluded that more than 90% of the treatments instituted in their patient population were unnecessary.

Other investigators have studied specific populations to determine the relevance of flatfoot in adults and carry this information over to the pediatric population. The classic article by Harris and Beath[43] from 1948 not only described hypermobile flatfoot with short tendo-Achilles but also discussed some data from the 1944 to 1945 Royal Canadian Army Medical Corps study on Army foot problems. Among 3619 Canadian soldiers, there were 25 cases of severe hypermobile flatfoot with short tendo-Achilles, 192 cases of mild hypermobile flatfoot with short tendo-Achilles, 74 cases of peroneal

spastic flatfoot, and 524 cases characterized by low arch. They further concluded that the natural history of hypermobile flatfoot with short tendo-Achilles is for it to become more severe, and more incapacitating, with increasing age. The disability is mild in childhood and may not be expressed until adolescence. It worsens in young adult life, and by middle life it may have reached severe proportions.[43]

Forriol and Pascual[61] studied the footprints of 1676 schoolchildren (1013 girls and 663 boys) between the ages of 3 and 17 years. They noted a high percentage of lowered medial longitudinal arches in the young age groups and a lower percentage in the older age groups. Their conclusion was that the medial longitudinal arch is a physiologic development in the earlier years of growth.

Cowan and colleagues[62] studied 246 US Army infantry trainees and their conclusions did not support the hypothesis that low-arched individuals are at increased risk of injury.

Rudzki[63] studied 350 men in the Australian Army and concluded that pes planus was not a significant factor in the development of injury during recruit training.

Hogan and Staheli[13] investigated the concept that treatment of flexible flatfoot in children will prevent disability in adult life. Proponents of not treating flexible flatfoot cite military studies that show that flexible flat feet are not a source of disability in soldiers. They studied 91 physically active civilian adult men and women and found no relationship between arch configuration and pain, suggesting that in the civilian population flexible flat feet are not a source of disability. They concluded that their study was consistent with previous studies and that it provided additional evidence against the practice of treating flexible flat feet in children.

Abdel-Fattah and colleagues[64] studied 2100 military recruits between the ages of 18 and 21 years in the Saudi Arabian Army. The incidence of flatfoot was 5%. Their conclusion was that family history, wearing shoes during childhood, obesity, and urban residency were significant issues associated with flatfoot. Because no flatfoot-related complaints were reported among the cases, their conclusion was that flexible flatfoot did not seem to be the cause of any disability.

DATA FROM FORMS OF IMAGING
Footprint Analysis

To diagnose and follow the natural history of flexible flatfoot, some sort of imaging must be used. Footprint recordings are inexpensive and easy to obtain, but more difficult to standardize and interpret. In addition, they merely represent the contact area of the plantar surface of the foot without giving any information on bony interrelationships. Radiographs are more frequently used but suffer the same problem with standardized positioning and angle-drawing techniques. Footprint and radiographic interpretations have issues with interobserver and intraobserver reliability.

El and colleagues[59] studied 579 primary school children and evaluated generalized joint laxity, foot progression angle, frontal-hindfoot alignment, and longitudinal arch height in dynamic positions. They used footprints obtained from the Harris-Beath mat and concluded that there is a negative correlation between arch height and age and between hypermobility score and age and that flexible flatfoot and hypermobility are developmental profiles.

Garcia-Rodriguez and colleagues[57] studied the incidence of flexible flatfoot in a population of 4- to 13-year-old schoolchildren and grouped their footprints into 3 flatfoot grades. They determined that the prevalence of flat feet was 2.7% of the 1181 children sampled.

Kanatli and colleagues[65] studied the relationship of radiologically measured angles and the arch index obtained from footprint analysis of 38 children with flexible flatfoot. They concluded that footprint analysis could be used effectively for screening studies and in individual office examinations.

Mickle and colleagues[66] used plantar footprints to study Australian preschool children to determine whether flatfoot was influenced by gender. They concluded that more boys had flat feet than girls. This finding was due to a thicker plantar fat pad in boys.

Radiology Analysis

Radiology has a historical role in the diagnosis and management of flat feet. Some studies relied heavily on this form of imaging. In the 1944 to 1945 Royal Canadian Army Medical Corps survey, all 3619 subjects had radiographs.[43]

Akcali and colleagues[8] studied 20 children with flexible flatfoot and external tibial torsion and a control group of 10 children with flexible flatfoot without rotational problems. Talar declination angle, talo–first metatarsal angle, and dorsoplantar talocalcaneal angles were measured on standing radiographs. Tibial torsion was measured by computed tomography (CT). They identified increased plantar talar declination and increased AP talocalcaneal angle. There was also prominent naviculocuneiform sag. Their conclusion was that abnormal external tibial torsion may affect the foot deformity and can change the benign nature of flexible flatfoot.

Harty[67] identified imaging pediatric foot disorders as a challenging task. He concluded that optimally exposed and well-positioned radiographs can answer many questions. CT and magnetic resonance imaging (MRI) are often needed to provide additional information to assist in the management of congenital and acquired foot lesions.[67] Positioning infants and young children can be difficult because of poor patient cooperation. Unless present during the exposure, the interpreter of the study is at the mercy of the technician.

Pehlivan and colleagues[68] evaluated the value of radiology to distinguish symptomatic and asymptomatic flexible flatfoot in young men. They concluded that an increased lateral talo–first metatarsal angle might be an important risk factor for development of symptoms in otherwise normal flexible flat feet.

Kuhn and colleagues[69] radiographically evaluated flexible pes planus patients with and without orthoses and concluded that there were statistically significant improvements in weight-bearing foot alignment with orthosis use. They concluded that their study supports the use of custom flexible orthotics for the improvement of pedal structural alignments.

Vanderwilde and colleagues[70] performed a radiographic measurement study of the feet in normal infants and children to establish standard radiographic values by evaluating weight-bearing radiographs of 74 normal infants and children admitted to a hospital for issues other than orthopedic disease. They ranged in age from 6 to 127 months and were grouped into 10 age groups. They examined AP, true lateral, and maximum dorsiflexion lateral radiographs. On the AP, the knees were flexed with the central ray directed at the talus. They measured the talocalcaneal angle, the calcaneus–fifth metatarsal angle, and the talus–first metatarsal angle. On the lateral radiograph, they measured the talocalcaneal angle, tibiocalcaneal angle, tibiotalar angle, talus–first metatarsal angle, and talo-horizontal angles. They also measured the talocalcaneal and tibiocalcaneal angles on stress dorsiflexion radiographs. The talocalcaneal index is calculated by adding the values of the AP and lateral talocalcaneal angles. Their results were that girls and boys and right and left feet had similar findings. AP talocalcaneal and fifth metatarsal–calcaneus angles

decreased with age. Lateral talocalcaneal and talo–first metatarsal angles decreased less with age. Lateral tibiotalar, talo-horizontal, and maximum dorsiflexion talocalcaneal angles showed the least decrease with age.

Bleck and Berzins[71] studied flatfoot in children using the Helfet heel seats or the University of California Biomechanics Laboratory (UCBL) orthoses. They called this deformity pes valgus with plantarflexed talus, flexible. Follow-up examination of 71 cases revealed that 79% of the patients treated for more than a year had clinical and roentgenographic improvement. They recommended the Helfet heel seat if the plantarflexion angle of the talus is 35° to 45° and the UCBL shoe insert if plantarflexion of the talus is greater than 45°.

LEVELS OF EVIDENCE
Case-control Studies, Case Series, and Articles Primarily Expressing Expert Opinion

Based on levels of evidence for primary research, case-control studies (level III), case series (level IV), and articles relating primarily expert opinions (level V) are the least reliable. However, there are many such articles in the literature.

Bahler[44] discussed management of the more pronounced form of flexible flatfoot with the use of various types of insoles and also differentiated 5 components in the development of flexible flat feet. He concluded that a slight form of flatfoot is physiologic in children and that more pronounced forms require treatment.

Wenger and Leach[72] stated that flexible flatfoot is a manifestation of a constitutional laxity of ligaments and joints and seems abnormal because of weight-bearing stresses. They concluded that most children with flatfoot achieve a partial correction spontaneously and that current research at the time of their writing did not document that treatment with corrective shoes or inserts produced a result better than the partial correction that occurs naturally.

Jani[40] identified the difficulty in recognizing the transition of flexible flatfoot from a physiologic condition to a pathologic condition that makes assessment of therapy difficult. He questioned the usefulness of arch supports. However, he felt that the therapy was indicated for severe flatfoot deformities recognized by heel valgus of more than 20° and lack of a medial arch. Results of follow-up examinations of treated and untreated cases of flexible flat feet suggest that the value of the arch support insoles that are used widely is more than questionable.[40]

Zollinger and Fellmann[41] also noted the difficulty in separating normal variations of children's feet from pathologic conditions. It was their contention that flexible flatfoot disappears during growth. There was little pathologic significance if it persisted in adults. Differentiation is made between a benign pain-free course of development with no functional restrictions and pathologic deformities that require conservative or surgical therapy. Zollinger and Exner[42] stated that the spectrum of normal variations of children's feet is extremely broad and often difficult to separate from pathologic conditions. They concluded that flexible flatfoot normally disappears during growth. Even if it persists into adult life, it has no real pathologic significance. The natural course, even of severe flexible flatfoot in children, leads to good results that are often better than the surgical results, and more discretion with surgical treatment was advocated.

Cappello and Song[45] stated that infants are born with flexible flatfoot and that a normal arch develops in the first decade of life. They concluded that flexible flatfoot rarely causes disability, and asymptomatic children should not be burdened with orthotics or corrective shoes. Flexible flatfoot with tight heel cords may become symptomatic and can be addressed with a stretching program. Surgical intervention for

flexible flatfoot is reserved for patients who have persistent localized symptoms despite conservative care. Rigid or pathologic flat feet have multiple causes, and many will require treatment to alleviate symptoms or improve function.

Hefti and Brunner[73] noted that many parents have anxiety about insufficient foot arches of their children. They stated that the arch is physiologically flattened by a hypervalgus of the hindfoot, and these feet do not need treatment.

Li and Leong[74] grouped intoeing gait, flexible flatfoot, bow legs, and knock knees in 1 category of physiologic problems that occur in normal children.

Sullivan[46] noted that the exact incidence of flatfoot in children is unknown but that it is a common finding. He further stated that all children have only a minimal arch at birth, and more than 30% of neonates have calcaneovalgus deformity of both feet. He concluded that calcaneovalgus is not painful and generally resolves without treatment. The same thing is true of flexible flatfoot. His recommendation was that the examining physician must rule out the existence of those conditions that do require treatment.

Attempts at Higher Level Studies

Whitford and Esterman[29] performed a randomized controlled trial of 2 types of in-shoe orthoses in children with flexible excessive pronation of the feet between the ages of 7 and 11 years. They made the diagnosis by observing calcaneal eversion and navicular drop. They found no evidence to justify the use of in-shoe orthoses in the management of flexible excessive pronation in children.

Evans studied the relationship between "growing pains" and foot posture in children, investigating the complaint of aching legs and its relation to pronated foot posture using 8 single-case experimental designs. The foot posture is believed to be deleterious and is often treated with in-shoe devices. This intervention proved helpful for children with pronated foot posture and aching legs.[75] Four years later, Evans and Scutter[76] compared foot posture with functional health between children aged 4 to 6 years with and without leg pain and reached the conclusion that navicular height was not predictive for growing pains. They also concluded that there was no support for the anatomic theory for growing pains and did not find a meaningful relationship between foot posture or functional health measures and leg pain in young children.[76]

PATHOPHYSIOLOGY

Understanding of the pathology of flatfoot is based on anatomic experimental data, theoretical biomechanics, and clinical observations in patient care. Anatomic studies dating back to the 1930s and 1940s led to the Root biomechanical theory of foot function. This approach to foot abnormality relies heavily on subtalar joint biomechanics and coronal plane forefoot-to-rearfoot interrelationship. As a natural consequence of gaining knowledge, the Root approach to foot biomechanics is not so much being challenged as being added to.

Much of Root biomechanical theory is based on the subtalar joint neutral position. However, it is impossible to anatomically define the subtalar joint neutral position with any degree of precision. At least 4 techniques have been described. First, it is traditionally defined as the position the calcaneus occupies when it is placed at one-third of the total subtalar range of motion moving from the position of full eversion. A second technique involves lining up the lateral calcaneus with the fibula. A third technique is palpating for full coverage of the talar head by the navicular. Fourth, it has been described as that position in which the foot is neither pronated nor supinated.

Kirby[77] introduced the concept of foot function based on the spatial location of the subtalar joint axis in relation to the weight-bearing structures in the plantar foot, using the concept of subtalar joint rotational equilibrium to explain how externally generated forces, such as ground reaction forces, and internally generated forces, such as ligamentous and tendon tensile forces, and joint compression forces affect the mechanical behavior of the foot and lower extremity.

McPoil and Cornwall[78] found that, contrary to previously published theory, the "neutral" position of the rearfoot for the typical pattern of rearfoot motion during the walking cycle was found to be the resting rather than subtalar joint neutral position. It is clear that equating flatfoot pathology with subtalar joint function is a gross oversimplification of an extremely complicated anatomic area.

Ball and Afheldt[79] challenged the Root orthotic theory and stated that the casting and evaluation techniques have poor reliability and unproven validity and that the principles are rarely followed. They also challenged the concept that excessive foot eversion leads to excessive pronation and that orthotics provided beneficial effects by controlling rearfoot inversion and eversion. It was their contention that control of internal/external tibial rotation is the most significant factor in maintaining proper supination and pronation mechanics. They also suggested that proprioceptive influences play a large role.

Detailed discussion of the pathophysiology of flexible flatfoot is outside of the scope of this article. However, the highlights can be stressed. Too much of the literature concentrates on abnormal foot-to-surface contact and failure of foot structure on the sagittal plane (loss of the medial arch). Abnormal pronation is triplanar. It is usual to find deformity on all 3 planes, but it is more pronounced on 1 plane. This tendency has led to the concept of planal dominance.[30] By recognizing the plane of the greatest component of the deformity, treatment options can be more accurately selected. Coronal (frontal) plane deformity is recognized by marked increase in subtalar eversion motion. Transverse plane pronation is recognized by transverse talonavicular instability without excessive heel eversion and without failure of the medial column in the sagittal plane. Sagittal plane pronation can be identified by breech along the medial column. This condition can be seen on clinical inspection but is more apparent on weight-bearing lateral radiographs. More emphasis is currently being placed on evaluating the rearfoot as if it were a complex functional talocalcaneonavicular joint unit (the acetabulum pedis).[32–35]

There are certain aspects of the pathophysiology of flatfoot that are not controversial. Painful pronated foot structures with rearfoot rigidity are often caused by tarsal coalitions. Barroso and colleagues[80] placed the incidence of congenital tarsal coalition at about 1% and recognized it as the main cause of painful rigid flatfoot in the pediatric population. Blakemore and colleagues[81] also identified tarsal coalitions as a major cause of painful rigid flat feet in children and adolescents. They identified the most common types as talocalcaneal and calcaneonavicular coalitions. Lowy[82] discussed pediatric peroneal spastic flatfoot in the absence of coalition.

The cause of flexible flatfoot is unknown. There is evidence that there are genetic tendencies toward excessive pronation. It is not unusual to see flexible flatfoot in multiple siblings and to trace it back through several generations. Additional diagnoses, such as ligamentous laxity and hypotonia, are often combined with flexible flatfoot. This combination is often the point at which flexible flatfoot ceases to be physiologic and becomes pathologic.

Flexible flatfoot can be influenced by tibia varum, genu valgum, gastrosoleus contracture, and primary ankle joint valgus. Primary ankle joint valgus is often overlooked in the assessment of pronated feet. If it is present, foot eversion becomes

the sum of subtalar eversion plus supramalleolar valgus (**Fig. 2**). Obesity also adversely modifies the course of flexible flatfoot and may be a major cause for foot and leg discomfort.

Planal Dominance

From the pathophysiologic point of view, flexible flatfoot is identified by abnormal subtalar joint pronation, some degree of transverse plane uncovering of the talonavicular joint, and flatness of the medial longitudinal arch. There are 4 types of flexible flatfoot based on the concept of planal dominance. The first is coronal or frontal plane pronation, characterized by abnormal eversion of the calcaneus in the coronal plane. It is difficult to attach specific numbers to calcaneal eversion, but more than 15° is considered excessive (**Fig. 3**).

Transverse plane pronation is characterized by uncovering of the talar head medially at the talonavicular joint in the absence of excessive heel eversion. This condition increases the AP talocalcaneal angle and results in some degree of abduction of the lateral forefoot. Calcaneal eversion rarely exceeds 10°. Lateral radiographs are surprisingly normal. There is little or no failure of the medial column in the sagittal plane. It gives the impression that the lateral column is short (**Fig. 4**).

Sagittal plane pronation involves the other 2 planes, but the defining feature is failure of the medial column at the talonavicular joint, the cuneonavicular joint, the first metatarsocuneiform joint, or at several of these locations (**Fig. 5**).

Triplane pronation shows excessive heel eversion, transverse plane talar head uncovering, and collapse of the medial column without any real dominance on any of the planes (**Fig. 6**).

Fig. 2. In the workup for pronated feet, the possibility that the ankle joint may not be horizontal must be taken into consideration. If there is primary ankle valgus deformity, the calcaneus is everted with reference to the weight-bearing surface independent of any subtalar joint position. This condition cannot be modified by any in-shoe orthosis.

Fig. 3. Coronal plane dominant pronation without change on the remaining planes. There is marked heel eversion noted clinically. The radiographs show a normal AP talocalcaneal angle and normal lateral talocalcaneal relationship with preservation of the medial column.

Fig. 4. Transverse plane dominant pronation. Clinically, the calcaneus is everted to the weight-bearing plane and the forefoot is abducted on the rearfoot. The radiographs show much of the medial talar head uncovered on the AP radiograph, with minimal failure of the medial column in the sagittal plane on the lateral radiograph.
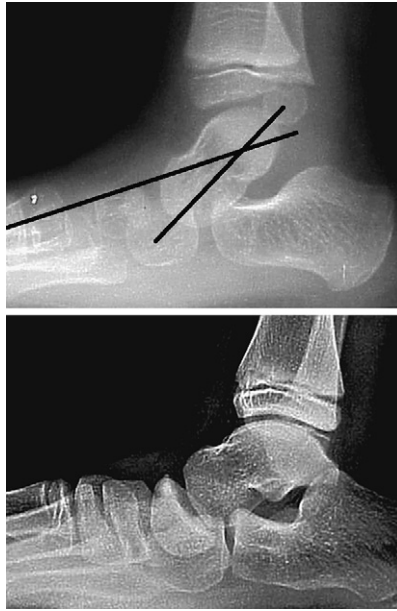
Fig. 5. Sagittal plane dominant pronation. Medial column collapse may be at the talonavicular joint or at some point distal.

Forefoot varus is often a manifestation of sagittal plane dominant deformity. In pediatric practice, the incidence of forefoot varus before the age of 6 years is almost nonexistent, which suggests that much of the adult forefoot varus is acquired.

The importance of equinus deformity as a complication of pronation cannot be overemphasized. Like forefoot varus, congenital equinus in the pediatric age group is uncommon. If present, the congenital form is almost always associated with neuromuscular disease. Acquired equinus is first seen toward the end of the first decade

Fig. 6. Triplane pronation. The AP and lateral radiographs show equal signs of pronation. The transverse and sagittal planes are affected.

of life. If there is inadequate ankle joint dorsiflexion, the lack of ankle movement has to be compensated for by pronating the rearfoot, or the heel cannot reach the ground. This method may require maximal or supramaximal pronation to the point at which joint surfaces are actually subluxed (**Fig. 7**).

The angle made by the tibia and the weight-bearing plane is also important. In children younger than 2 years, physiologic tibia varum is seen. To get the medial forefoot down to the ground, the rearfoot must pronate. Physiologic tibia varum persists until 2 years of age, at which time it slowly changes to become genu valgum. This condition, too, encourages pronation. The proposed mechanism is movement of the center of gravity to the medial side of the weight-bearing foot, but there may be other explanations for it.

Primary ankle valgus is frequently overlooked in the workup of flatfoot. The exact incidence is unknown but, if overlooked, will result in error in control of the rearfoot because the pathology is proximal to the talocalcaneonavicular joint. In addition, orthoses cannot change this rigid and fixed eversion of the entire foot and ankle.

Talipes Calcaneovalgus

The position of talipes calcaneovalgus in the flatfoot spectrum has been largely ignored. The American College of Foot and Ankle Surgeons study failed to mention it.[36] Because of the use of the word talipes, it is grouped with the congenital foot and ankle deformities. It is included in several articles on congenital lower extremity deformities in infancy.[83–89] Some investigators believe that it spontaneously corrects.[46,72,90–92] Several investigators have studied its incidence in the population.[46,93] Nunes and Dutra[94] estimated the incidence at 4.2 per 10,000 live births.

Fig. 7. As a consequence of attempting to compensate for severe ankle equinus, the talonavicular joint has transversely subluxed.

Its clinical appearance is distinctive. The foot is maximally dorsiflexed at the ankle so that the dorsum of the foot may make contact with the anterior tibia. The talocalcaneonavicular joint complex is maximally pronated. The forefoot is abducted. The anterior compartment muscles may be contracted. Although it is semiflexible, there is some resistance to full passive supination on manipulation. It is often associated with breech deliveries. Developmental hip dislocation and knee extension deformities are common.[95]

The differential diagnosis includes posteromedial bow deformity[96,97] and congenital convex pes valgus.[98] If there is a pure calcaneus deformity, it is necessary to verify that there is S1 function to exclude a paralytic deformity caused by myelomeningocele and other neurologic issues.[99] Examination of the contour of the tibia will help exclude posteromedial bow, but radiographs are often necessary (**Fig. 8**). Congenital convex pes valgus is similar to talipes calcaneovalgus. One key difference is the extreme rigidity in congenital convex pes valgus. The radiographic distinction is easily made. On lateral studies of calcaneovalgus deformity, the ankle is in a calcaneus position, whereas in congenital convex pes valgus the rearfoot is in equinus (**Fig. 9**).

The calcaneus ankle position spontaneously corrects or is treated by serial casting. The hyperpronation of the talocalcaneonavicular joint complex persists in untreated infants.

Oblique talus deformity was described by Kumar and colleagues.[37,38] Two types were described, depending on the position of the calcaneus. In 1 form, the calcaneal inclination angle is preserved and the talus is deviated significantly downward. In the second type, the talus is angled downward and the calcaneal inclination angle is reversed (**Fig. 10**). Oblique talus deformity may be persistence of the talocalcaneonavicular hyperpronation of talipes calcaneovalgus deformity.

Fig. 8. An infant with posteromedial bow deformity could be mistaken for talipes calcaneovalgus or congenital convex pes valgus. The radiograph clearly demonstrates a posteromedial bow.

Fig. 9. Calcaneovalgus deformity is recognized radiographically on lateral view. (*A*) The ankle is in a calcaneus position with the talus dorsiflexed in the ankle mortise. (*B*) In congenital convex pes valgus, the talus is in a maximally plantarflexed position, and the ankle is in equinus.

DISCUSSION

Pediatric flatfoot is more than just a low arch. It is a complex condition of the rearfoot that may or may not be pathologic. Several subsets can be identified. Types such as congenital convex pes valgus (congenital vertical talus), flatfoot associated with tarsal coalitions, skewfoot deformity, flatfoot complicated by traumatic or iatrogenic

Fig. 10. Oblique talus deformity. The midfoot is flexible and can be manually reduced. The talus if plantarflexed at the talonavicular joint and the calcaneal inclination angle is reversed.

arthrosis, and flatfoot associated with systemic disease are clearly not physiologic. There is little argument that these will require some form of treatment. Their natural history tends more toward worsening, development of symptoms, and secondary joint changes over time. There are no data to suggest that they improve over time.

The issue of flexible flatfoot is another matter. There is little supportive evidence that it improves or worsens over time, therefore it is difficult to explain why there is so much polarization and contradiction when groups discuss biomechanical theory, what constitutes normal and abnormal, what is and is not deformity, and, especially, the pros and cons of treatment.[2,100–103]

As in the management of any other disease, treatment of flexible flatfoot should be goal-oriented. To be successful, there has to be a reasonable expectation that the goals can, and will, be met. Relief of clinical symptoms, positive modification of the natural history, and prevention of future complications are all laudable goals. However, their achievement remains scientifically unverifiable. There is room in evidence-based medicine to consider medicine-based evidence. Most people who attempt to manage flexible flatfoot in childhood will affirm that the clinical symptoms of plantar arch pain, leg fatigue, and even possibly the nocturnal pain syndrome respond to the use of orthotics. The real question is whether anything short of surgical reconstruction truly modifies the natural history. As an offshoot of that question, can extensive surgical intervention be justified for asymptomatic, or marginally symptomatic, flexible flatfoot?

Well-designed valid studies of the natural history of flexible flatfoot and the effects of modification of the natural history of flexible flatfoot are needed. There are some impediments to the design and execution of such studies. Assessment is made by clinical examination of ranges of motion, imaging, gait analysis, and subjective survey instruments. Interobserver and intraobserver reliability of clinical measurement of range of motion must be addressed. At present, imaging seems to be limited to the study of footprints and radiographic imaging. Formal gait-laboratory studies can be incorporated, but they are time consuming and expensive.

Radiographic imaging needs to be considered more carefully than it has been in the past. Several studies have used radiographic parameters for their conclusions on the diagnosis, natural history, and effects of treatment.[8,43,56,65,67–71] Measurements of the various angles assign a numerical value to the positional relationship of individual bones. For the skeletally mature person, this is straightforward. For the skeletally immature, these measurements assign a numerical value to the positional relationships of the ossific nuclei embedded in the cartilage anlagen. The ossification center for the talus begins in the neck, and the body ossifies last.[48,49,51] The ossification center for the calcaneus is eccentrically located and is along its inferior surface in the distal two-thirds of the developing bone.[49,51] It is also located lateral to the midline.[49] Consequently, many of the so-called changes in angular measurements with age that have been used to document a corrective natural history may merely represent the progression of normal ossification while the bony interrelationships remains the same. A photomicrograph of a fetal specimen shows the interrelationship of the developing bones appearing normal (**Fig. 11**). The perspective changes when the ossification centers are inserted are shown in **Fig. 12**.

It may be almost impossible to design and implement a study of the natural history of flexible flatfoot and the effects of treatment. It would require a prospective study of a large controlled patient population. There would have to be strict guidelines for enrollment in the study. The subjects would have to be studied for at least 10 to 15 years. The biggest drawback to the study would be repetitive x-ray exposure to the children for the sole purpose of gathering data. As an example, in the 1944 to 1945 Royal Canadian Army Medical Corps study, all 3619 subjects had radiographs. It is

Fig. 11. Sagittal section of a fetal specimen (developmental age unknown) before the appearance of primary ossification centers. The overall alignment is anatomically correct.

unlikely that that could be done today. If treatment is included, there are only 2 possible hypotheses. The first is that treatment will not modify the natural history. Therefore, the study group will receive unnecessary treatment. The second is that treatment will modify the natural history, in which case the control group would not receive treatment that might prove beneficial. These issues raise serious moral and ethical concerns. It is unlikely that such a study would obtain Institutional Review Board approval.
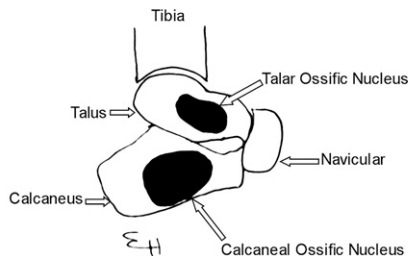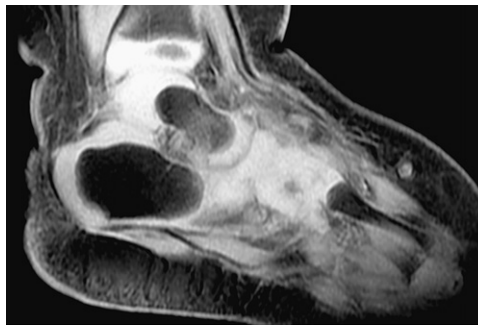

Fig. 12. Sagittal plane MRI of the foot of an 18-month-old girl shows the appearance of the primary ossification centers of the talus and the calcaneus as they relate to the cartilage anlagen. The ossification centers are drawn in relation to the primary cartilage anlagen.

SUMMARY

Those forms of flatfoot that are clearly pathologic are not controversial. The real issue is what to do with asymptomatic flexible flatfoot. It is hard to justify treating all forms of flexible flatfoot on the presumption that it will prevent pathologic conditions in adulthood, because there are no data to support that this actually happens. It is also hard to justify withholding treatment on the presumption that the condition will spontaneously correct, because the presence of flexible flatfoot in adolescence and adulthood proves that they do not all correct. Although not proven, one might be more confident in treating some of these conditions on the presumption that holding the foot in better alignment during rapid growth may prevent progression that may result from change in the developing bones secondary to remodeling during the endochondral ossification process. The same can be said for treating symptomatic flexible flatfoot, although the placebo effect of "doing something" remains unexplored.

Until supportive data are available, physicians must make judgments based on the situation at hand and their own personal experience. It would be wise to avoid the 2 extremes. The nihilistic approach of treating none of them is no better or worse than the approach that all flexible flatfoot is disease and needs forceful management. This statement is particularly true of aggressive surgical management involving ablation of motion segments. A course of action somewhere between the 2 extremes is more appropriate.

This overview of the history, causes, and pathophysiology of pediatric flatfoot can only be accomplished through literature search. A major drawback to this approach is the inability to guarantee recovery of all pertinent articles dealing with the topic. Such a search depends on identifying appropriate keywords. Therefore a possible limitation to this study is that certain important articles may have been omitted.

REFERENCES

1. Staheli LT. Evaluation of planovalgus foot deformities with special reference to the natural history. J Am Podiatr Med Assoc 1987;77(1):2–6.
2. Staheli LT. Planovalgus foot deformity. Current status. J Am Podiatr Med Assoc 1999;89(2):94–9.
3. Burns J, Keenan AM, Redmond A. Foot type and overuse injury in triathletes. J Am Podiatr Med Assoc 2005;95(3):235–41.
4. Manoli A 2nd, Graham B. The subtle cavus foot, "the underpronator". Foot Ankle Int 2005;26(3):256–63.
5. McCarthy DJ. The developmental anatomy of pes valgo planus. Clin Podiatr Med Surg 1989;6(3):491–509.
6. Greiner TM. The jargon of pedal movements. Foot Ankle Int 2007;28(1):109–25.
7. Huson A. Joints and movements of the foot: terminology and concepts. Acta Morphol Neerl Scand 1987;25(3):117–30.
8. Akcali O, Tiner M, Ozaksoy D. Effects of lower extremity rotation on prognosis of flexible flatfoot in children. Foot Ankle Int 2000;21(9):772–4.
9. Allen MK, Glasoe WM. Metrecom measurements of navicular drop in subjects with the anterior cruciate ligament injury. J Athl Train 2000;35(4):403–6.
10. Buchanan KR, Davis I. The relationship between forefoot, midfoot, and rearfoot static alignment in pain-free individuals. J Orthop Sports Phys Ther 2005;35(9): 559–66.
11. Hargrave MD, Carcia CR, Gansneder BM, et al. Subtalar pronation does not influence impact forces or rate of loading during a single-leg landing. J Athl Train 2003;38(1):18–23.

12. Headlee DL, Leonard JL, Hart JM, et al. Fatigue of the plantar intrinsic foot muscles increases navicular drop. J Electromyogr Kinesiol 2008;18(3):420–5.

13. Hogan MT, Staheli LT. Arch height and lower limb pain: an adult civilian study. Foot Ankle Int 2002;23(1):43–7.

14. Holmes CF, Wilcox D, Fletcher JP. Effect of a modified, low-dye medial longitudinal arch taping procedure on the subtalar joint neutral position before and after light exercise. J Orthop Sports Phys Ther 2002;32(5):194–201.

15. Lange B, Chipchase L, Evans A. The effect of low-Dye taping on plantar pressures, during gait, in subjects with navicular drop exceeding 10 mm. J Orthop Sports Phys Ther 2004;34(4):201–9.

16. Leung AK, Cheng JC, Mak AF. Orthotic design and foot impression procedures to control foot alignment. Prosthet Orthot Int 2004;28(3):254–62.

17. Menz HB. Alternative techniques for the clinical assessment of foot pronation. J Am Podiatr Med Assoc 1998;88(5):253–5.

18. Mueller MJ, Host JV, Norton BJ. Navicular drop is a composite measure of excessive pronation. J Am Podiatr Med Assoc 1993;83(4):198–202.

19. Paton JS. The relationship between navicular drop and first metatarsophalangeal joint motion. J Am Podiatr Med Assoc 2006;96(4):313–7.

20. Radford JA, Burns J, Buchbinder R, et al. The effect of low-Dye taping on kinematic, kinetic and electromyographic variables: a systematic review. J Orthop Sports Phys Ther 2006;36(4):232–41.

21. Reinking MF. Exercise-related leg pain in female collegiate athletes: the influence of intrinsic and extrinsic factors. Am J Sports Med 2006;34(9):1500–7.

22. Reinking MF, Hayes AM. Intrinsic factors associated with exercise-related leg pain in collegiate cross-country runners. Clin J Sport Med 2006;16(1):10–4.

23. Schuh A, Honle W. [Pathogenesis of hallux valgus]. MMW Fortschr Med 2006;148(48):31–2 [in German].

24. Snook AG. The relationship between excessive pronation as measured by navicular drop and isokinetic strength of the anterior musculature. Foot Ankle Int 2001;22(3):234–40.

25. Trimble MH, Bishop MD, Buckley BD, et al. The relationship between clinical measurements of lower extremity posture and tibial translation. Clin Biomech (Bristol, Avon) 2002;17(4):286–90.

26. Vicenzino B, Griffiths SR, Griffiths LA, et al. Effects of anti-pronation tape and temporary orthotic on vertical navicular height before and after exercise. J Orthop Sports Phys Ther 2000;30(6):333–9.

27. Vicenzino B, Franettovich M, McPoil T, et al. Initial effects of the anti-pronation tape on the medial longitudinal arch during walking and running. Br J Sports Med 2005;39(12):939–43.

28. Vinicombe A, Raspovic A, Menz HB. Reliability of navicular displacement measurement as a clinical indicator of foot posture. J Am Podiatr Med Assoc 2001;91(5):262–8.

29. Whitford D, Esterman A. A randomized controlled trial of two types of in-shoe orthoses in children with flexible excess pronation of the feet. Foot Ankle Int 2007;28(6):715–23.

30. Green DR, Carol A. Planal dominance. J Am Podiatry Assoc 1984;74(2):98–103.

31. Labovitz JM. The algorithmic approach to pediatric flexible pes planovalgus. Clin Podiatr Med Surg 2006;23(1):57–76, viii.

32. Serrafian SK. Biomechanics of the subtalar joint complex. Clin Orthop Relat Res 1993;290:17–26.

33. Epeldegui T, Delgado E. Acetabulum pedis. Part I: talocalcaneonavicular joint socket in normal foot. J Pediatr Orthop B 1995;4(1):1–10.
34. Epeldegui T, Delgado E. Acetabulum pedis. Part II: talocalcaneonavicular joint socket in clubfoot. J Pediatr Orthop B 1995;4(1):11–6.
35. Golano P, Farinas O, Saenz I. The anatomy of the navicular and periarticular structures. Foot Ankle Clin 2004;9(1):1–23.
36. Harris EJ, Vanore JV, Thomas JL, et al. Diagnosis and treatment of pediatric flatfoot. J Foot Ankle Surg 2004;43(6):341–73.
37. Jayakumar S, Ramsey P. Vertical in the oblique talus: a diagnostic dilemma. Orthop Trans 1977;1:108.
38. Kumar SJ, Cowell HR, Ramsey PL. Vertical and oblique talus. Instr Course Lect 1982;31:235–51.
39. Harris EJ. The oblique talus deformity. What is it, and what is its clinical significance in the scheme of pronatory deformities? Clin Podiatr Med Surg 2000;17(3):419–42.
40. Jani L. [Pediatric flatfoot]. Orthopade 1986;15(3):199–204 [in German].
41. Zollinger H, Fellmann J. [Natural course of juvenile foot deformities]. Orthopade 1994;23(3):206–10 [in German].
42. Zollinger H, Exner GU. [The lax juvenile flexible flatfoot–disease or normal variant?] Ther Umsch 1995;52(7):449–53 [in German].
43. Harris RI, Beath T. Hypermobile flatfoot with short tendo Achilles. J Bone Joint Surg Am 1948;30:116–40.
44. Bahler A. [Insole management of pediatric flatfoot]. Orthopade 1986;15(3):205–11 [in German].
45. Cappello T, Song KM. Determining treatment of flatfeet in children. Curr Opin Pediatr 1998;10(1):77–81.
46. Sullivan JA. Pediatric flatfoot: evaluation and management. J Am Acad Orthop Surg 1999;7(1):44–53.
47. Tax H. Conservative treatment of flatfoot in the newborn. Clin Podiatr Med Surg 1989;6(3):521–36.
48. Frisch H, Schmitt O, Eggers R. The ossification center of the talus. Ann Anat 1996;78(5):455–9.
49. Harris EJ. The relationship of the ossification centers of the talus and calcaneus to the developing bone. J Am Podiatry Assoc 1976;66(2):76–81.
50. Howard CB, Benson MK. The ossific nuclei and the cartilage anlage of the talus and calcaneum. J Bone Joint Surg Br 1992;74(4):620–3.
51. Hubbard AM, Meyer JS, Davidson RS, et al. Relationship between the ossification center and cartilaginous anlage in the normal hindfoot in children: study with MR imaging. AJR Am J Roentgenol 1993;161(4):849–53.
52. Meyer DB, O'Rahilly R. The onset of ossification in the human calcaneus. Anat Embryol 1976;150(1):19–33.
53. Napolitano C, Walsh S, Mahoney L, et al. Risk factors that may adversely modify the natural history of the pediatric pronated foot. Clin Podiatr Med Surg 2000;17(3):397–417.
54. Wenger DR, Mauldin D, Speck G, et al. Corrective shoes and inserts as treatment for flexible flatfoot in infants and children. J Bone Joint Surg Am 1989;71(6):800–10.
55. Staheli L, Chew DE, Corbett M. The longitudinal arch. A survey of eight hundred and eighty-two feet in normal children and adults. J Bone Joint Surg Am 1987;69(2):426–8.

56. Gould N, Moreland M, Alvarez R, et al. Development of the child's arch. Foot Ankle 1989;9(5):241–5.
57. Garcia-Rodriguez A, Martin-Jimenez F, Carnero-Varo M, et al. Flexible flat feet in children: a real problem? Pediatrics 1999;103(6):e84.
58. Lin CJ, Lai KA, Kuan TS, et al. Correlating factors and clinical significance of flexible flatfoot in preschool children. J Pediatr Orthop 2001;21(3):378–82.
59. El O, Akcali O, Kosay C, et al. Flexible flatfoot and related factors in primary school children: a report of a screening study. Rheumatol Int 2006;26(11):1050–3.
60. Pfeiffer M, Kotz R, Ledl T, et al. Prevalence of flat foot in preschool-aged children. Pediatrics 2006;118(2):634–9.
61. Forriol F, Pascual J. Footprint analysis between three and 17 years of age. Foot Ankle 1990;11(2):101–4.
62. Cowan DN, Jones BH, Robinson JR. Foot morphologic characteristics and risk of exercise-related injury. Arch Fam Med 1993;2(7):223–4.
63. Rudzki SJ. Injuries in Australian Army recruits. Part III: the accuracy of a pretraining orthopedics screen in predicting ultimate injury outcome. Mil Med 1997; 162(7):41–3.
64. Abdel-Fattah MM, Hassanin MM, Felembane FA, et al. Flat foot among Saudi Arabian army recruits: prevalence and risk factors. East Mediterr Health J 2006;12(1–2):211–7.
65. Kanatli U, Yetkin H, Cila E. Footprint and radiographic analysis of the feet. J Pediatr Orthop 2001;21(2):225–8.
66. Mickle KJ, Steele JR, Munro BJ. Is the foot structure of preschool children moderated by gender? J Pediatr Orthop 2008;28(5):593–6.
67. Harty MP. Imaging of pediatric foot disorders. Radiol Clin North Am 2001;39(4): 733–48.
68. Pehlivan O, Cilli F, Mahirogullari M, et al. Radiographic correlation of symptomatic and asymptomatic flexible flatfoot in young male adults. Int Orthop 2009; 33(2):447–50.
69. Kuhn DR, Shibley NJ, Austin WM, et al. Radiographic evaluation of weight-bearing orthotics and their effect on flexible pes planus. J Manipulative Physiol Ther 1999;22(4):221–6.
70. Vanderwilde R, Staheli LT, Chew DE, et al. Measurements on radiographs of the foot in normal infants and children. J Bone Joint Surg Am 1988;70(3):407–15.
71. Bleck EE, Berzins UJ. Conservative management of pes valgus with plantar flexed talus, flexible. Clin Orthop Relat Res 1977;Jan–Feb(122):85–94.
72. Wenger DR, Leach J. Foot deformities in infants and children. Pediatr Clin North Am 1986;33(6):1411–27.
73. Hefti F, Brunner R. [Flatfoot]. Orthopade 1999;28(2):159–72 [in German].
74. Li YH, Leong JC. Intoeing gait in children. Hong Kong Med J 1999;5(4):360–6.
75. Evans A. Relationship between "growing pains" and foot posture in children: single-case experimental designs in clinical practice. J Am Podiatr Med Assoc 2003;93(2):111–7.
76. Evans AM, Scutter SD. Are foot posture and functional health different in children with growing pains? Pediatr Int 2007;49(6):991–6.
77. Kirby KA. Subtalar joint axis location and rotational equilibrium theory of foot function. J Am Podiatr Med Assoc 2001;91(9):465–87.
78. McPoil T, Cornwall MW. Relationship between neutral subtalar joint position and pattern of rearfoot motion during walking. Foot Ankle Int 1994;15(3):141–5.
79. Ball KA, Afheldt MJ. Evolution of foot orthotics—part 2: research reshapes long-standing theory. J Manipulative Physiol Ther 2002;25(2):125–34.

80. Barroso JL, Barriga A, Barrecheguren EG, et al. [Congenital synostoses of the tarsus. Concept, classification, diagnosis and therapeutic approach]. Rev Med Univ Navarra 2001;45(1):43–52 [in Spanish].
81. Blakemore LC, Cooperman DR, Thompson GH. The rigid flatfoot. Tarsal coalitions. Clin Podiatr Med Surg 2000;17(3):531–55.
82. Lowy LJ. Pediatric peroneal spastic flatfoot in the absence of coalition. A suggested protocol. J Am Podiatr Med Assoc 1998;88(4):181–91.
83. Correll J, Berger N. [Diagnosis and treatment of disorders of the foot in children]. Orthopade 2005;34(10):1061–72 [quiz 1073–4] [in German].
84. Furdon SA, Donlon CR. Examination of the newborn foot: positional and structural abnormalities. Adv Neonatal Care 2002;2(5):248–58.
85. Gore AI, Spencer JP. The newborn foot. Am Fam Physician 2004;69(4):865–72.
86. Hart ES, Grottkau BE, Rebello GN, et al. The newborn foot: diagnosis and management of common conditions. Orthop Nurs 2005;24(5):313–21 [quiz 322–3].
87. Omololu B, Ogunlade SO, Alonge TO. Pattern of congenital orthopaedic malformations in an African teaching hospital. West Afr J Med 2005;24(2):92–5.
88. Sankar WN, Weiss J, Skaggs DL. Orthopaedic conditions in the newborn. J Am Acad Orthop Surg 2009;17(2):112–22.
89. Wynne-Davies R, Littlejohn A, Gormley J. Aetiology and interrelationship of some common skeletal deformities. (Talipes equinovarus and calcaneovalgus, metatarsus varus, congenital dislocation of the hip, and infantile idiopathic scoliosis). J Med Genet 1982;19(5):321–8.
90. Churgay CA. Diagnosis and treatment of pediatric foot deformities. Am Fam Physician 1993;47(4):883–9.
91. Hensinger RN. Rotational problems of the lower extremity. Postgrad Med 1976; 60(4):161–7.
92. Widhe T, Aaro S, Elmstedt E. Foot deformities in the newborn–incidence and prognosis. Acta Orthop Scand 1988;59(2):176–9.
93. Yu GV, Hladik J. Residual calcaneovalgus deformity: review of the literature and case study. J Foot Ankle Surg 1994;33(3):228–38.
94. Nunes D, Dutra MG. Epidemiological study of congenital talipes calcaneovalgus. Braz J Med Biol Res 1986;19(1):59–62.
95. Paton RW, Choudry Q. Neonatal foot deformities and their relationship to developmental dysplasia of the hip: an 11-year prospective longitudinal observational study. J Bone Joint Surg Br 2009;91(5):655–8.
96. Grimes JB, Blair VP 3rd, Gilula LA. Roentgen rounds #81. Posteromedial bowing of the tibia. Orthop Rev 1986;15(4):249–55.
97. Pappas AM. Congenital posteromedial bowing of the tibia and fibula. J Pediatr Orthop 1984;4(5):525–31.
98. Greenberg AJ. Congenital vertical talus and congenital calcaneovalgus deformity: a comparison. J Foot Surg 1981;20(4):189–93.
99. Rodrigues RC, Dias LS. Calcaneus deformity in spina bifida: results of anterolateral release. J Pediatr Orthop 1992;12(4):461–4.
100. Phillips RD. Planovalgus foot deformity revisited. J Am Podiatr Med Assoc 1999; 89(5):265–8 [author reply 269].
101. Root ML. Planovalgus foot deformity revisited. J Am Podiatr Med Assoc 1999; 89(5):268–9.
102. Evans AM. The flat-footed child—to treat or not to treat: what is the clinician to do? J Am Podiatr Med Assoc 2008;98(5):386–93.
103. Bresnahan P. Letter to the editor: the flat-footed child—to treat or not to treat. What is a clinician to do? J Am Podiatr Med Assoc 2009;99(2):178.