Review Article

Pediatric Foot Notes: A Review of Common Congenital Foot Deformities

Jose F. Jackson, MD; Stephen J. Stricker, MD

Abstract

The primary care physician has the first opportunity to recognize congenital problems such as metatarsus adductus, clubfoot, calcaneovalgus, vertical talus, tarsal coalition, and flexible flat foot. The authors review the clinical features of these conditions, as well as associated conditions, in order to facilitate early diagnosis. While none of these foot deformities delays initial ambulation, rigid deformities may result in painful callosities or difficult shoe wear later in childhood. Some of these foot deformities require early orthopaedic treatment for optimal outcome while others require only observation or reassurance. *Int Pediatr. 2003;18(3):133-140.*

Key words: foot deformity, clubfoot, flexible flat foot, metatarsus adductus, tarsal coalition, vertical talus

Introduction

Congenital and acquired foot deformities are common in children.[1] Flexible foot deformities are defined as those which are passively correctable. Such deformities generally resolve spontaneously and cause few functional problems. Rigid foot deformities, on the other hand, do not correct without treatment and will eventually result in foot pain. An effort is made to complete corrective casting or surgical treatment prior to walking age, so early diagnosis and orthopaedic referral are important. The authors review the important clinical and radiographic features to help the primary care physician identify the more common congenital foot deformities in children.

Metatarsus Adductus (MTA)

Simple metatarsus adductus is a congenital deformity characterized by medial deviation of the forefoot, resulting in convexity of the lateral border of the foot (Fig. 1). It is quite common, occurring in approximately 3% of live births, and is usually bilateral. The etiology is believed to be intrauterine positioning,[2-4] but a familial association has been reported.[5] Anatomically, the medial deviation occurs primarily at the tarso-metatarsal joints. It may cause intoeing after walking age. MTA is also a component of congenital clubfoot deformity, discussed later in this review.

Most congenital foot deformities, including MTA, may be associated with developmental hip dysplasia (DDH). Kumar[6] reported a ten-fold increase in the incidence of DDH in infants with MTA. Although this high association with DDH has been questioned recently, careful examination of the hips is advisable when any type of congenital foot deformity is suspected.

The natural history of MTA is usually benign, with 87% resolving by age 6 years and 95% by age 16 years;[7] however, the prognosis is dependent upon the classification. MTA must be distinguished from dynamic metatarsus primus varus, in which contraction of the abductor hallucis muscle causes medial deviation

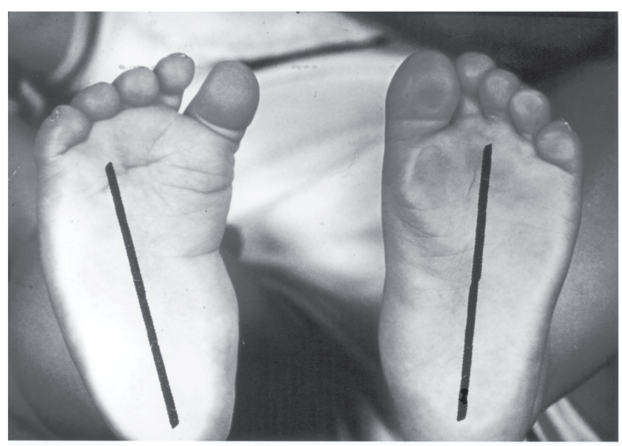

Fig 1 - Right metatarsus adductus is confirmed by the convex lateral border of the foot. The heel-bisector line is normal on the left, passing in the 2nd-3rd toe web space. The line passes through the 4th toe on the right, indicating moderately severe deformity.

From the Jackson Memorial Medical Center, Miami, Florida, Chief Resident in Orthopaedic Surgery (Dr Jackson) and Professor and Chief of Pediatric Orthopaedics at the University of Miami School of Medicine (Dr Stricker).

Address reprint requests to Stephen J. Stricker, MD, Department of Orthopaedic Surgery (D-27), P.O. Box 016960, Miami, FL 33101.

of the great toe and forefoot during ambulation. A rare condition which may simulate MTA is congenital epiphyseal bracket of the first metatarsal, a growth plate anomaly resulting in a short hallux with varus deformity.[8]

Classification of MTA

The most widely accepted clinical classification described by Bleck[7] utilizes the heel-bisector line (Fig. 1) which passes through the longitudinal axis of the heel. When the foot is held in the simulated weight-bearing position, the line should pass through the 2nd toe. MTA is mild if the line passes through the 3rd toe, moderate if through the 4th toe, and severe if through the 5th toe.

MTA may also be classified according to its rigidity.[7] Flexible MTA can be easily corrected by the examiner, or by eliciting contraction of the peroneal musculature. No treatment is required for flexible MTA since spontaneous correction is expected. Rigid MTA cannot be passively corrected, and will usually require orthopaedic referral for serial cast treatment of moderate or severe deformities.

Imaging of MTA

Radiographs are not routinely required, but should be considered for progressive deformities or for those feet with short hallux. A simulated weight-bearing AP view of the foot will show medial deviation of the metatarsal, especially the first. MTA can be quantified radiographically by measuring the talo-first metatarsal angle.[9]

Treatment of MTA

Observation or forefoot stretching by parents are reasonable management options for flexible MTA, since spontaneous correction is predictable. Rigid MTA of moderate or severe degree is best treated with serial manipulation and corrective casting. Optimal results occur when casting is begun between 3-8 months of age, although casting has been shown to be somewhat effective up to 2 years of age.[3,7] Typical treatment involves a short leg cast changed every 1-2 weeks, for a total of 6-12 weeks. Recurrence is uncommon. Use of the Denis-Browne bar or placement of shoes on the wrong feet are of dubious value. Surgical treatment is rarely indicated, but severe persistent MTA can be corrected with early medial soft-tissue releases,[10] or with late midfoot osteotomies.[11]

Idiopathic Clubfoot/Talipes Equinovarus (TEV)

Congenital clubfoot, also called talipes equinovarus, consists of rigid ankle plantarflexion (equinus), heel inversion (varus), and metarsus adductus (Fig. 2). None of these deformities is passively correctable. The foot and calf may be slightly atrophic, and a midfoot transverse crease is usually present. Idiopathic TEV is a common deformity, occurring in 1-2 per 1000 live births. The condition is bilateral in 50%, and 65% of those affected are male. The anatomic deformity is best envisioned as medial rotation and inversion of the entire foot around the talus. Rigid clubfeet do not delay walking, but they will eventually cause painful callosities due to weight-bearing on the lateral borders of the feet.

The etiology is unknown but may be due to an arrested stage of fetal development. Nongenetic factors such as birth order and smoking are also implicated in the etiology of TEV, but their significance is uncertain.[12-15]

Genetic factors may be important since the risk of idiopathic TEV increases to 2-4% if a first-degree relative has clubfoot. The risk is 25% if both the parent and sibling had clubfoot deformity. Concordance is 32.5% in monozygotic twins compared to 3% for dizygotic twins.[14] Clubfeet may

Fig 2 - Bilateral congenital clubfeet show the typical clinical features of heel inversion, ankle equinus, and metatarsus adductus.

be inherited in a strict Mendelian fashion as part of certain syndromes, such as diastrophic dwarfism (autosomal recessive), Pierre-Robin (X-linked recessive), or Freeman-Sheldon (autosomal dominant). Teratologic TEV is commonly seen in association with myelodysplasia, arthrogryposis, Streeter's amniotic band syndrome, or in prune-belly syndrome.

Classification of TEV

Clubfeet may be postural due to intrauterine positioning. Such feet can be passively corrected at birth and will typically correct spontaneously in the first weeks. Most clubfeet are rigid and cannot be passively corrected. Rigid clubfeet are either idiopathic or teratologic, with the latter group occurring in association with syndromes, chromosomal anomalies, or neuromuscular conditions. All rigid clubfeet require orthopaedic referral, preferably within the first 2 weeks of life, so that serial casting may be initiated.

Imaging of TEV

Radiographic examination is usually not required for diagnosis at birth. Initial radiographs are typically obtained at age 2-3 months to check the progress of serial cast treatment. In a rigid clubfoot, the talus and calcaneus will become parallel, causing the talo-calcaneal angles to be subnormal.[16] With the foot in maximum dorsiflexion, the lateral talo-calcaneal angle should normally be 30-50°, but is typically much less in a clubfoot. Rigid equinus may also be verified radiographically by noting plantarflexion of the calcaneus on a radiograph taken in maximum dorsiflexion.

Treatment of TEV

Idiopathic and teratologic clubfeet are usually treated shortly after birth with serial manipulation and casting. While casting often fails, it may facilitate subsequent surgery by decreasing soft-tissue contractures. The duration of cast treatment varies widely, but is usually for at least 2 months, with cast changes every 1-2 weeks. The reported success of cast treatment also varies, ranging from 20-80%.[17-19] Many orthopaedic surgeons now perform percutaneous Achilles tenotomy to facilitate cast correction.[18] A potential complication of casting is skin pressure sore, especially if the foot slides up into the cast. If the child exhibits marked irritability in the cast and the parents are unable to contact the orthopaedic surgeon, it may be prudent for the parents to quickly remove the cast by soaking off the plaster in warm water. This may prevent a skin sore which can cause infection or scarring.

Surgical correction consists of posteromedial soft-tissue release, usually performed between the ages of 6-12 months. The goal of surgery, as with casting, is to achieve a painless, plantigrade foot which permits normal gait and shoe wear. This is accomplished 75-95% of the time with the initial surgery, but some patients require additional procedures to achieve or to maintain correction.[20,21] Even with well-executed surgery, clubfeet can never be made completely normal - all congenital clubfeet will have some degree of permanent stiffness, weakness, and hypoplasia of the foot and ipsilateral leg. Rigidity and recurrent deformity are more likely in teratologic clubfeet.

Congenital Vertical Talus (CVT)

Congenital vertical talus (CVT) is also called talipes convex pes valgus or "congenital rocker-bottom foot". It is a rare deformity (one-tenth the incidence of clubfeet) which results in reversal of the normal longitudinal arch at birth. The etiology is unknown, but may be related to muscle imbalance in some cases.[22,23] Both sexes are affected equally, and CVT is bilateral in 50% of cases. Anatomically, the pathognomonic feature of CVT is a fixed dislocation of the tarsal navicular dorsal to the talar head. The dislocation is difficult to palpate clinically but the resulting prominence of the talar head in the plantar-medial arch of the foot is apparent. The down-pointing talus results in equinus and eversion (valgus) of the heel. It is the equinus hindfoot and the upward pointing forefoot which results in the rocker-bottom sole.

Approximately 50% of vertical tali are idiopathic, but the remainder are associated with various syndromes and neuromuscular conditions such as myelomeningocele, arthrogryposis, chromosomal anomalies, and syndromes (nail-patella, Larsen's, Streeter's dysplasia).[23-25] Therefore, any patient with CVT deserves a careful evaluation for an underlying cause.

CVT is sometimes confused with congenital calcaneovalgus foot deformity (CCV). In CCV, the newborn ankle is hyper-dorsiflexed due to intrauterine positioning. CCV differs from vertical talus because CCV has hindfoot dorsiflexion and no prominence or dislocation of the talar head. As with other congenital foot deformities, CVT and CCV may occur in association with DDH.[26] CCV resolves spontaneously prior to walking age.

Classification of CVT

Like clubfoot, CVT may be classified into 2 groups - idiopathic and teratologic. The former group has a better prognosis because there is no associated muscle imbalance. CVT will not delay walking, and painful callosities will not develop until late childhood. Nevertheless, early treatment is much easier and offers superior results compared to late treatment of symptomatic feet.

Imaging of CVT

The typical radiographic evaluation of CVT includes an AP view of the foot as well as lateral views of the foot in dorsiflexion and maximum plantarflexion. The dorsiflexion view will confirm the fixed equinus of the hindfoot. The plantarflexion view is necessary to confirm the pathognomonic irreducible dislocation of the talonavicular joint. Because the navicular does not ossify until 3-5 years of age, one cannot directly visualize the talonavicular dislocation on plain radiographs. One may assume that the talonavicular joint remains dislocated in plantarflexion if the axis of the first metatarsal does not line up with the long axis of the talus (Fig. 3). It is not necessary to obtain an MRI of the foot to confirm the presence of talonavicular dislocation.

Treatment of CVT

The goal of treatment is to obtain a painless, plantigrade foot with correction of the talonavicular dislocation. Initial serial casting soon after birth may improve the soft tissue contractures anterior to the ankle, but casting is rarely effective as the definitive treatment.[5,27]

Surgical correction is almost always required, and good results are achieved in 60-85%.[25,28,29] Two decades ago, extensive correction of the talonavicular joint and the hindfoot contractures were performed separately as staged procedure. More recently, one-stage correction has been shown to give equally good results. Best results are obtained if surgery is performed before 2 years of age. An attempt is made to complete correction prior to walking age for children with idiopathic CVT. As with clubfeet, feet with CVT cannot be restored to complete normality following surgery. Mild stiffness and flatfoot deformity are common long-term sequelae; however, functional results are very good.

Tarsal Coalitions (TC)

A tarsal coalition (TC) is an abnormal fusion of two tarsal bones. Ninety percent of coalitions involve

Fig 3 - The pathognomonic radiographic feature of congenital vertical talus is demonstrated on the lateral foot radiograph taken in maximum plantarflexion. Persistent dorsal displacement of the first metatarsal relative to the axis of the talus signifies a fixed dorsal talonavicular dislocation. The soft-tissue shadows show a “rocker-bottom” sole.

either the calcaneonavicular gap or the talocalcaneal joint.[30] The tissue bridging the tarsal joint at birth may be fibrous, cartilaginous, or osseous. Coalitions are rarely diagnosed at birth because they do not cause signs or symptoms until later in childhood. The incidence is <1% without sex predilection, and involvement is bilateral in 50-80%.[30,31] The natural history is variable, but many coalitions remain asymptomatic. Some coalitions cause pain in late childhood and may result in rigid, progressive flatfoot deformity.

Coalitions are due to failure of segmentation of embryonic mesenchyme.[31] The inheritance of TC is autosomal dominant with nearly full penetrance.[30] Coalitions are usually isolated, but may occur in association with clubfeet, fibular hemimelia, symphalangism, and Apert's syndrome.[31-33]

Coalitions are typically fibrous or cartilaginous at birth, but many begin to ossify in late childhood. As the coalitions turn into rigid bone, subtalar motion becomes limited, and peroneal muscle spasm may occur. This occasionally results in progressive hindfoot eversion, called "spastic peroneal flatfoot". Most patients present at age 8-16 years complaining of pain in the region of the subtalar or talonavicular joint. The diagnosis may be strongly suspected simply by the presence of limited or painful subtalar motion. Differential diagnosis includes tarsal rheumatoid arthritis or peroneal tendinitis.

Imaging of TC

Initial plain radiographic assessment should include AP, lateral, and oblique views of the foot as well as Harris axial views of the hindfoot. Because of the high incidence of bilaterality, it is reasonable to image both feet even if symptoms are unilateral. The lateral view and Harris view are sometimes useful for visualizing talocalcaneal coalitions. The oblique view shows the calcaneonavicular coalition if it is beginning to ossify (Fig. 4). Dorsal talar "beaking" may be seen on the lateral view, possibly indicative of excessive compensatory motion at the talonavicular joint. Subtalar coalitions are poorly localized or quantified on plain radiographs, so a CT scan is usually the best tool for imaging.[34] The CT scan may confirm narrowing and irregularity of the middle or posterior subtalar facets. Occasionally, an MRI scan or bone scan is required to help confirm the presence of a fibrous coalition. The orthopaedic surgeon will typically obtain a CT scan of both feet to confirm the coalition, to rule out multiple coalitions, and to check for bilateral coalitions prior to recommending treatment such as surgical excision.

Treatment of TC

Asymptomatic TC's do not require treatment, even if subtalar motion is limited. Symptomatic TC's are typically treated nonsurgically with short leg casting for 3-6 weeks. This may be followed by supportive shoes (hiking boots) or with orthoses such as the University of California Biomechanical Laboratory (UCBL) arch support with the goal of preventing recurrent symptoms.[5] Conservative treatment successfully reduces pain symptoms at least temporarily in 30-68% of cases.[35]

When conservative treatment fails, surgical treatment may become necessary. Young patients with small coalitions may undergo excision of the coalition, providing relief in 60-85% of patients.[35-41] In older patients with joint degeneration, with large coalitions, or with severe flat feet, fusion of the subtalar and/or midfoot joints may be required to achieve a painless plantigrade foot.

Flexible Flat Foot (FFF)

While an accepted definition of flexible flat foot (FFF) is lacking, definitions always include flattening

Fig 4 - The oblique radiographic view of the foot readily shows narrowing of the calcaneonavicular space (arrow), confirming subtotal ossification of a calcaneonavicular tarsal coalition.

of the medial longitudinal arch of the foot with weight-bearing (Fig. 5). "Flexible" implies that the flat arch is correctable, and this can be demonstrated by observing restoration of the arch when the patient sits or stands on tiptoes. In more severe FFF there may be excessive eversion of the heel (hindfoot valgus) as well as forefoot abduction. Subtalar motion should be normal. The etiology of FFF is usually benign familial laxity which allows the ligament support of the arch of the foot to stretch under weight-bearing loads.[5,42]

It is not entirely clear whether FFF should be considered congenital or acquired. The appearance of flat (pronated) feet is often present at birth and during toddler years due to the presence of fatty tissue in the arch. The arch gradually develops to its mature form by age 4-7 years.[27,43,44] The prevalence of flat foot decreases with age, but may remain 10-23% by adulthood.[45,46]

The natural history for most FFF is benign, rarely causing disability or pain even in adulthood. Parents may be reassured that the United States Army no longer prohibits individuals from military service because of FFF. Reports regarding the natural history of FFF show variable results due to difficulties quantifying flatness clinically and radiographically; however, gradual development of the arch of the foot has been reported to improve by age 3 years and into adulthood. Therefore, there is generally no need to refer a child for orthopaedic evaluation of flexible flatfeet prior to age 3 years, unless the deformity is unusually severe, painful, or progressive. Previous generations were often treated with orthopaedic "corrective" shoes and arches did improve, consistent with the natural history.

Fig 5 - The clinical diagnosis of flexible flat feet always includes flattening of the medial longitudinal arches during weight-bearing.

Therefore, one of the more difficult contemporary orthopaedic challenges is to convince parents "whose childhood braces helped them outgrow their flat feet" that orthotics are of no proven benefit in curing their children's FFF!

There are several associated conditions which may worsen the prognosis of FFF, resulting in progressive flat foot deformity or pain. These include tight Achilles tendon, accessory navicular, or ligament hyperlaxity. If passive ankle dorsiflexion above neutral is not possible when the knee is extended, the Achilles contracture will exaggerate hindfoot eversion and increase the tension on the plantar fascia, sometimes resulting in painful plantar fasciitis or progressive flatfoot deformity. An accessory or prominent tarsal navicular may rub against the shoe when the foot is pronated. The presence of an ossicle in the accessory navicular may result in an incompetent insertion of the posterior tibial tendon, the tendon most important for dymamic support for the arch of the foot.[42] Severe ligament laxity, such as seen in trisomy-21, Marfan's syndrome, or Ehlers-Danlos, may also result in severe or progressive flatfoot deformity. Patients with these risk factors should be referred for orthopaedic evaluation of flat feet. Orthopaedic referral is also suggested for rigid flatfeet or for flexible flat feet which appear to be painful or progressive.

Classification of FFF

Flat feet may be categorized as flexible or rigid. Congenital vertical talus and tarsal coalition are examples of rigid flatfeet. Flexible flat feet have flattened arches which are correctable. The degree of FFF deformity is rather subjective, clinically and radiographically; therefore, treatment decisions are usually based upon the presence or absence of pain, Achilles contracture, or accessory navicular.

Imaging of FFF

Radiographs are rarely indicated in the routine evaluation of FFF. Standing AP, lateral, and oblique views may be taken to estimate deformity and to rule out causes of pain, such as tarsal coalition, accessory navicular, stress fracture, or tumor. On the standing lateral view of the foot, the talo-first metatarsal angle may be measured.[9] The normal value is > 0°, but the apex-plantar midfoot sag seen in FFF may result in a negative value.

Treatment of FFF

A prospective study has shown that the use of custom orthopaedic shoes and orthotic inserts does not influence the ultimate arch development compared to untreated controls.[47] Therefore, asymptomatic FFF are treated with reassurance and supportive regular shoes. Frequent stretching exercises may be reasonable if Achilles contracture prevents ankle dorsiflexion above 0°.

Orthotics such as the UCBL arch support may successfully alleviate activity-related midfoot pain and premature breakdown of the medial shoe.[5,48] Parents should not be advised that the UCBL will result in permanent restoration of the arch. Rigid arch supports may increase pain if there is medial prominence of the navicular or talar head, or if there is significant Achilles contracture.

Surgical treatment for idiopathic FFF is rarely indicated, and should virtually never be performed in asymptomatic feet. Surgical options include judicious Achilles lengthening, excision of the prominent accessory navicular, distal calcaneal lengthening osteotomy,[49] calcaneal medial translation osteotomy, subtalar fusion, and triple arthrodesis.

Finally, the primary care physician may be asked by parents whether it is more beneficial for infants and toddlers to wear shoes or to go barefooted. While our modern society promotes shoe wear from early age, there is little evidence to support the notion that toddlers' feet require support. Investigators studying unshod populations in Africa and India found fewer flat foot deformities or functional problems compared to children who wore shoes.[50,51] However, the same studies showed that shoes do serve a protective function since most unshod foot problems were related to infection or to trauma. Standard shoes appear innocuous as long as they fit properly, but high-fashion shoes for young women (high-heels with narrow pointed toe-box) may promote ankle sprains, lesser toes deformities, and bunion formation.

References

1. Widhe T. Foot deformities at birth: a longitudinal prospective study over a 16-year period. *J Pediatr Orthop* 1997;17:20-24.
2. Berg EE. A reappraisal of metatarsus adductus and skewfoot. *J Bone Joint Surg* 1986;68-A:1185-1196.
3. Rushforth GF. The natural history of hooked forefoot. *J Bone Joint Surg* 1978;60-B:530-532.
4. Staheli LT. Rotational problems of the lower extremities. *Orthop Clin North Am* 1987;18:503-512.
5. Crawford AH, Gabriel KR. Foot and ankle problems. *Orthop Clin North Am* 1987;18:649-666.
6. Kumar SJ, MacEwen GD. The incidence of hip dysplasia with metatarsus adductus. *Clin Orthop* 1982;164:234-235.
7. Bleck EE. Metatarsus adductus: classification and relationship to outcomes of treatment. *J Pediatr Orthop* 1983;3:2-9.
8. Mubarak SJ, O'Brien TJ, Davids JR. Metatarsal epiphyseal bracket: treatment by cenral physiolysis. *J Pediatr Orthop* 1993;13:5-8.
9. Vanderwilde R, Staheli LT, Chew DE, Malagon V. Measurement on radiographs of the foot in normal infants and children. *J Bone Joint Surg* 1988; 70-A:407-415.
10. Asirvatham R, Stevens PM. Idiopathic forefoot-adduction deformity: medial capsulotomy and abductor hallucis lengthening for resistant and severe deformities. *J Pediatr Orthop* 1997;17:496-500.
11. Gvildys P, Clark B Jr, Prigoff MM. Surgical treatment of metatarsus adductus using a combined soft tissue and osseous approach. *J Foot Surg* 1989;28:454-458.
12. Honein MA, Paulozzi LJ, Moore CA. Family history, maternal smoking, and clubfoot: an indication of a gene-environment interaction. *Am J Epidemiol* 2000;152:658-665.
13. Chesney D, Barkeer S, Miedzybrodzka Z, et al. Epidemiology and genetic theories in the etiology of congenital tallipes equinovarus. *Bull Hosp Joint Diseases* 1999;58:59-64.
14. Wynne-Davies R. Genetic and environmental factors in the etiology of talipes equinovarus. *Clin Orthop* 1972;84:9-13.
15. Cowell HR, Wein BK. Current concepts review. Genetic aspects of club foot. *J Bone Joint Surg* 1980;62-A:1381-1384.
16. Simons GW. A standardized method for the radiographic evaluation of clubfeet. *Clin Orthop* 1978;135:107-118.
17. Ikeda K. Conservative treatment of idiopathic clubfoot. *J Pediatr Orthop* 1992;12:217-223.
18. Ponseti IV. Current concepts review. Treatment of congenital club foot. *J Bone Joint Surg* 1992;74-A:448-454.
19. McKay DW. New concept of and approach to clubfoot treatment: Section II–correction of the clubfoot. *J Pediatr Orthop* 1993;3:10-21.
20. McKay DW. New concept of and approach to clubfoot treatment: Section III—evaluation and results. *J Pediatr Orthop* 1993;3:141-148.
21. Simons GW. Complete subtalar release in club feet. Part II – comparison with less extensive procedures. *J Bone Joint Surg* 1985;67-A:1056-1065.
22. Jacobsen ST, Crawford AH. Congenital vertical talus. *J Pediatr Orthop* 1983;3:306-310.
23. Seimon LP. Surgical correction of congenital vertical talus under the age of 2 years. *J Pediatr Orthop* 1987;7:405-411.
24. Ogata K, Schoenecker PL, Sheridan J. Congenital vertical talus and its familial occurrence: an analysis of 36 patients. *Clin Orthop* 1979;139:128-132.
25. Stricker SJ, Rosen E. Early one-stage reconstruction of congenital vertical talus. *Foot Ankle Int* 1997;18:535-543.

26. Hoffinger SA. Evaluation and management of pediatric foot deformities. *Pediatr Clin North Am* 1996;43:1091-1111.
27. Sullivan JA. Pediatric flatfoot: evaluation and management. *J Am Acad Orthop Surg* 1999;7:44-53.
28. Kodros SA, Dias LS. Single-stage surgical correction of congenital vertical talus. *J Pediatr Orthop* 1999;19:42-48.
29. Dodge LD, Ashley RK, Gilbert RJ. Treatment of the congenital vertical talus: a retrospective review of 36 feet with long-term follow-up. *Foot Ankle* 1987;7:326-332.
30. Mosier KM, Asher M. Tarsal coalitions and peroneal spastic flat foot: a review. *J Bone Joint Surg* 1984;66-A:976-984.
31. Vincent KA. Tarsal coalition and painful flatfoot. *J Am Acad Orthop Surg* 1998;6:274-281.
32. Spero CR, Simon GS, Tornetta P III. Clubfeet and tarsal coalition. *J Pediatr Orthop* 1994;14:372-376.
33. Grogan DP, Holt GR, Ogden JA. Talocalcaneal coalition in patients who have fibular hemimelia or proximal femoral focal deficiency: a comparison of the radiographic and pathological findings. *J Bone Joint Surg* 1994;76-A:1363-1370.
34. Herzenberg JE, Goldner JL, Martinez S, Silverman PM. Computerized tomography of talocalcaneal tarsal coalition: a clinical and anatomic study. *Foot Ankle* 1986;6:273-288.
35. Takakura Y, Sugimoto K, Tanaka Y, Tamai S. Symptomatic talocalcaneal coalition: its clinical significance and treatment. *Clin Orthop* 1991;269:249-256.
36. Luhmann SJ, Schoenecker PL. Symptomatic talocalcaneal coalition resection: indications and results. *J Pediatr Orthop* 1998;18:748-754.
37. Pachuda NM, Lasday SD, Jay RM. Tarsal coalition: etiology, diagnosis, and treatment. *J Foot Surg* 1990;29:474-488.
38. Gonzalez P, Kumar SJ. Calcaneonavicular coalition treated by resection and interposition of the extensor digitorum brevis muscle. *J Bone Joint Surg* 1990;72-A:71-77.
39. Scranton PE Jr. Treatment of symptomatic talocalcaneal coalition. *J Bone Joint Surg* 1987;69-A:533-538.
40. Wilde PH, Torode IP, Dickens DR, Cole WG. Resection for symptomatic talocalcaneal coalition. *J Bone Joint Surg* 1994;76-B:797-801.
41. McCormack TJ, Olney B, Asher M. Talocalcaneal coalition resection: a 10-year follow-up. *J Pediatr Orthop* 1997;17:13-15.
42. Basmajian JV, Stecko G. The role of muscles in arch support of the foot: an electromyographic study. *J Bone Joint Surg* 1963;45-A:1184-1190.
43. Staheli LT, 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* 1987;69-A:426-428.
44. Barry RJ, Scranton PE Jr. Flat feet in children. *Clin Orthop* 1983;181:68-75.
45. Gould N, Moreland M, Alvarez R, Trevino S, Fenwick J. Development of the child's arch. *Foot Ankle* 1989;9:241-245.
46. Rao UB, Joseph B. The influence of footwear on the prevalence of flat foot: a survey of 2300 children. *J Bone Joint Surg* 1992;74-B:525-527.
47. Wenger DR, Mauldin D, Speck G, Morgan D, Lieber RL. Corrective shoes and inserts as treatment for flexible flatfoot in infants and children. *J Bone Joint Surg* 1989;71-A:800-811.
48. Staheli LT, Giffin L. Corrective shoes for children: a survey of current practice. *Pediatrics* 1980;65:13-17.
49. Mosca VS. Calcaneal lengthening for valgus deformity of the hindfoot: results in children who had severe, symptomatic flatfoot and skewfoot. *J Bone Joint Surg* 1995;77-A:500-512.
50. Engle ET, Morton DJ. Notes on foot disorders among natives of the Belgian Congo. *J Bone Joint Surg* 1931;13:311-318.
51. Sim-Fook L, Hodgson AR. A comparison of foot forms among the non-shoe and shoe-wearing Chinese population. *J Bone Joint Surg* 1958;40-A:1058-1062.