CHAPTER 22 Disorders of the Foot

Introduction

The human foot is a complex structure capable of supporting body weight, accelerating the body in running, changing position for uneven terrain, and even assuming prehensile function in a child missing upper extremities.[3,5–7,9,11] The major articulation of the hindfoot is the joint between the talus and the complex of the navicular, calcaneus, and cuboid. The calcaneocuboid-navicular complex has been called the acetabulum of the foot, with the talus being the figurative femoral head. The "acetabular" configuration allows motion in several planes—a more complex conceptualization than the older description of an oblique hinge.

The movements of the foot are often described in confusing terms. The hindfoot *inverts* and *everts* into *varus* and *valgus* positions. When the hindfoot *inverts,* the rest of the foot rolls onto the outer border of the foot as it *supinates.* When the heel *everts* into valgus, the forefoot *pronates,* increasing weightbearing on the first ray. Smaller arc movements occur in the midfoot between cuneiforms, navicular, cuboid, and metatarsals.[4,8]

The medial longitudinal arch of the foot is maintained by a combination of ligamentous and muscle attachments. Relative overactivity of the plantar muscles produces a high, cavus arch, whereas underactivity, as in the case of posterior tibial tendon rupture, will cause a flattening of the arch.

The ossific development of the foot begins in utero.[2,10] At birth the talus, calcaneus, and cuboid are ossified whereas the navicular and cuneiforms remain cartilaginous. The metatarsals and phalanges are ossified at birth. The lateral cuneiform ossifies between 4 and 20 months, the medial cuneiform at 2 years, and the intermedial cuneiform at 3 years. The navicular ossifies between the second and fifth years of life (Figs. 22–1 to 22–3).

The foot has its own growth pattern, which differs from the growth rate of the rest of the body. The early work of Blais, Green, and Anderson showed that the foot grows rapidly between infancy and age 5 years, slowing to a rate of 0.9 cm per year between 5 and 12 years in girls and 5 to 14 years in boys, when growth usually ceases.[1] The foot of a 1-year-old girl or an 18-month-old boy has achieved half its adult length. These growth charts have been used for timing procedures such as triple arthrodesis that affect future foot growth (Figs. 22–4 to 22–6).[1–6,8–11]

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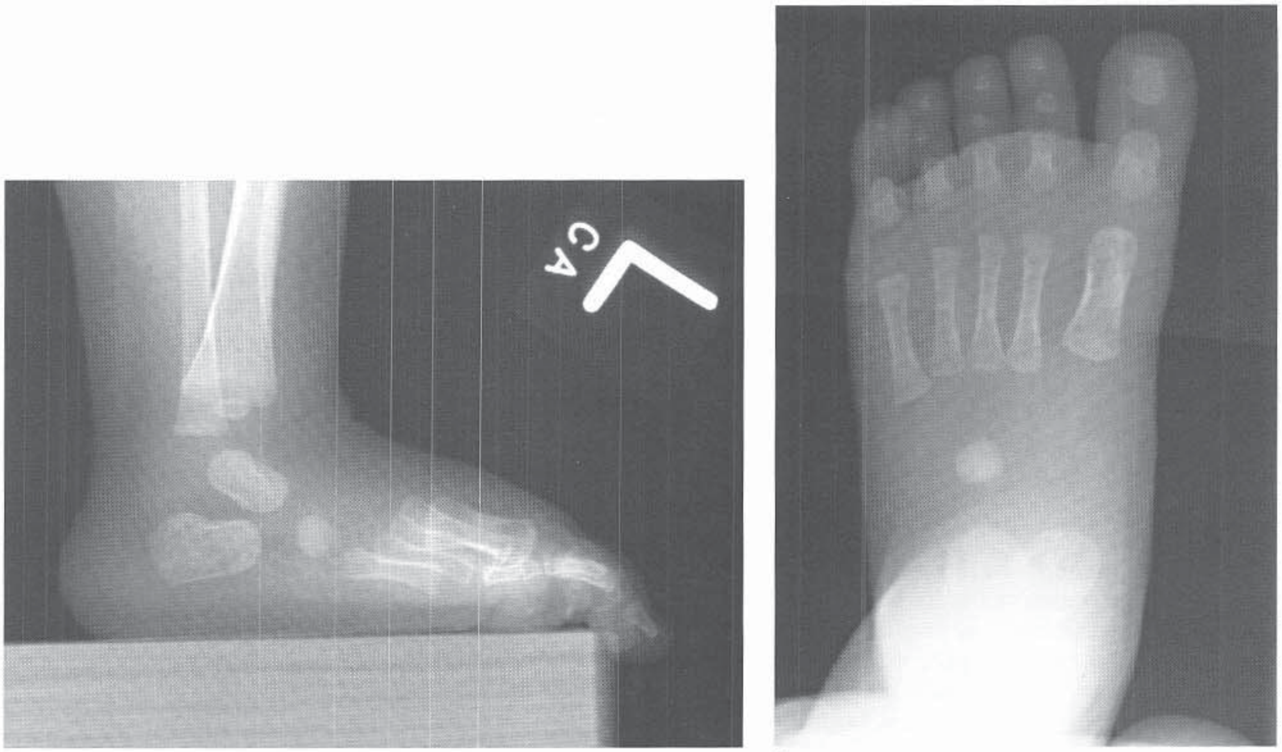
Normal Variations

Many variations of the "normal" foot are seen, especially in the newborn. Intrauterine crowding is blamed for metatarsus adductus and for calcaneovalgus deformities, both of which usually resolve spontaneously. The normal range of the configuration of the arch of the foot varies from high to flat and is not much influenced by shoe or orthotic wear, much to the consternation of many grandmothers.

Many common radiographic variations have been described in the foot, some resembling pathologic conditions. In more than 20 percent of children, one or more accessory bones are seen on radiographs.[20] Figure 22–7 illustrates the commonly recognized accessory bones of the foot. The accessory navicular and os trigonum will be described in detail because they are of clinical importance.

OS TRIGONUM

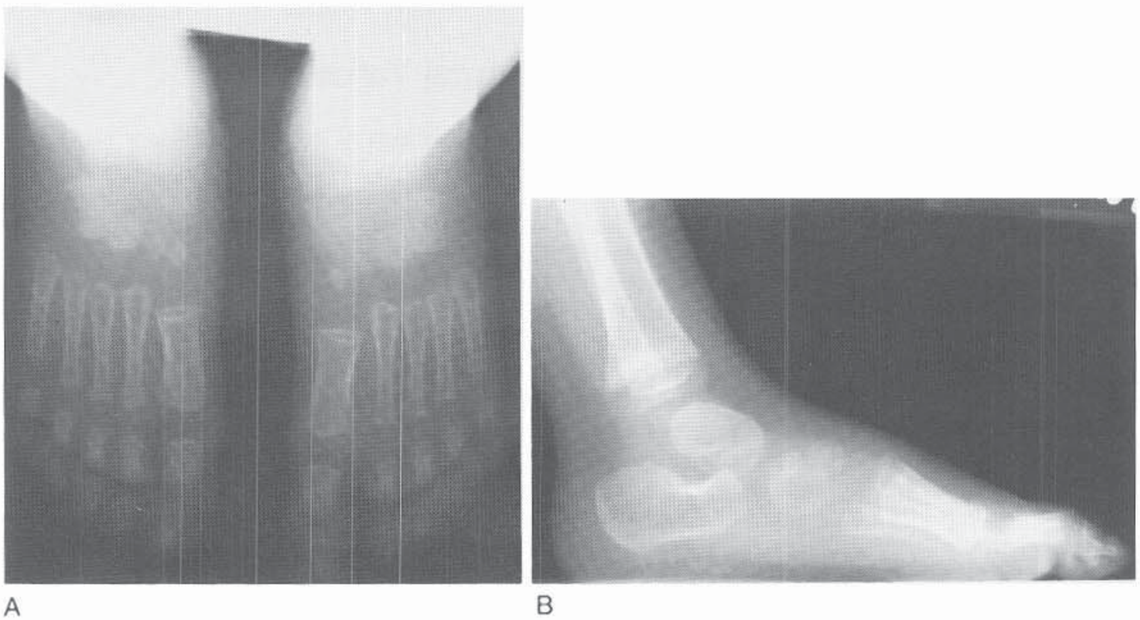
The os trigonum is formed from the lateral portion of the groove in the posterior talus, through which passes the flexor



A B

FIGURE 22–1 **A,** Lateral radiograph of a newborn foot. The cuboid is the only ossified midtarsal bone. Note the convergent talocalcaneal angle. **B,** AP radiograph of a newborn foot. The cuboid is ossified, but no ossification is present in the navicular or cuneiform.

hallucis longus (Fig. 22–8). Between 8 and 11 years of age, medial and lateral centers of ossification appear in the two portions of the walls of the groove for the flexor hallucis longus. These ossification centers normally fuse with the talus within a year. When the ankle is fully plantar flexed the tubercles contact the posterior edge of the distal tibia, and forceful plantar flexion may cause the lateral tubercle to break away from the talus. Radiographically the fractured os trigonum may be distinguished from one that has yet to fuse to the talus by the finding that the unfused os has a



A B

FIGURE 22–2 Ossification of the tarsal bones and metatarsals in a 3-year-old boy. **A,** AP radiograph of both feet. **B,** Lateral radiograph of the right foot. Note that the medial and intermediate cuneiform and navicular bones are ossified.

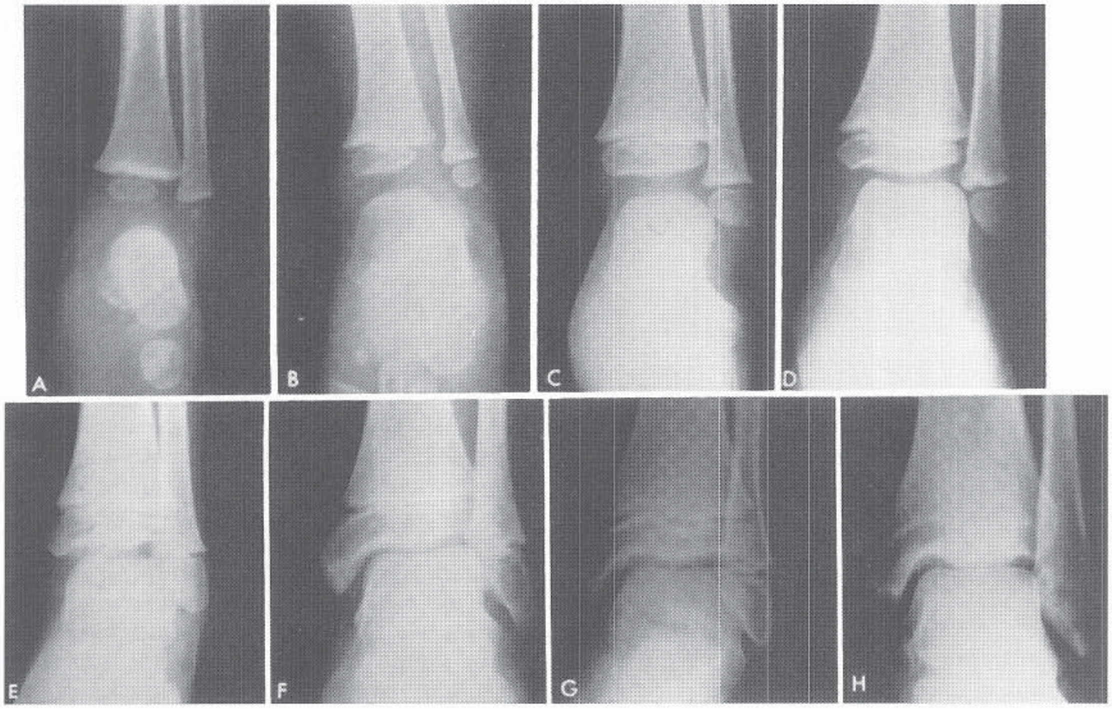


FIGURE 22–3 Ossification of the distal epiphyses of the tibia and fibula. **A,** One year of age. **B,** Two years. **C,** Four years. **D,** Six years. **E,** Seven years. **F,** Ten years. **G,** Twelve years. **H,** Adult.

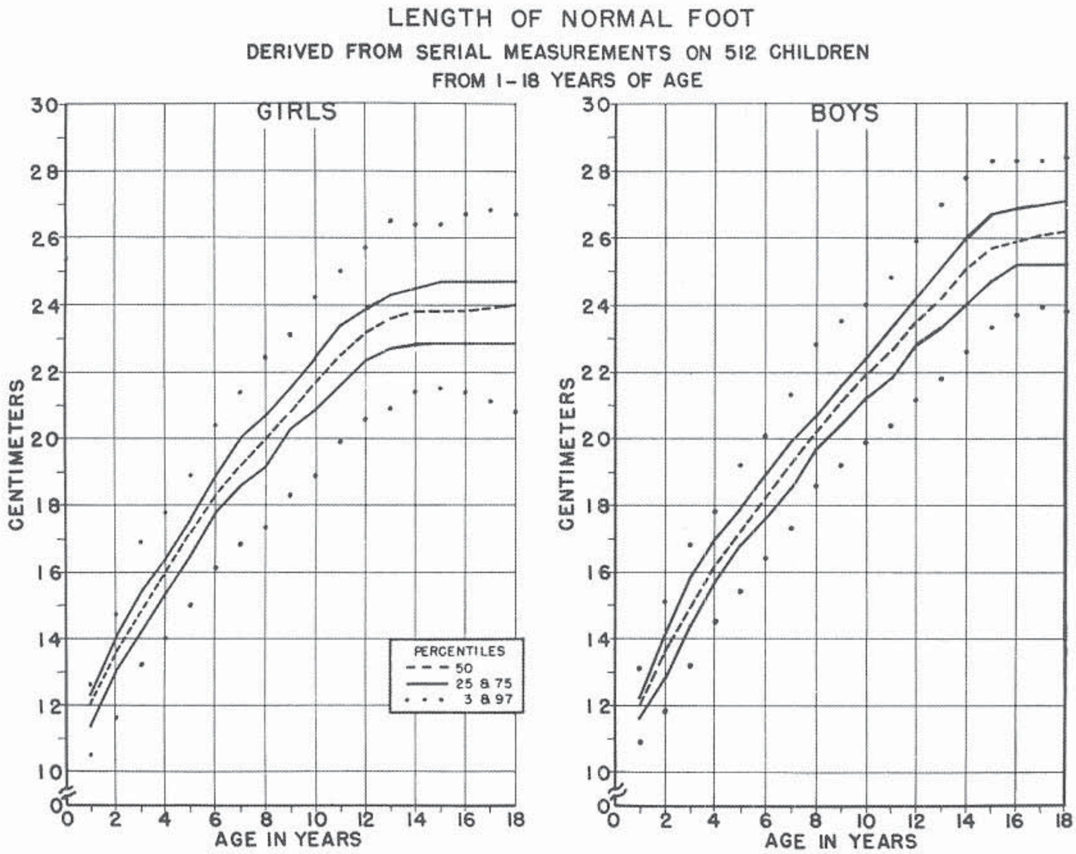


FIGURE 22–4 Length of the normal growing foot, derived from serial measurements made in 512 children from age 1 year to 18 years. (From Blais MM, Green WT, Anderson M: Length of the growing foot. J Bone Joint Surg 1956;38-A:998.)

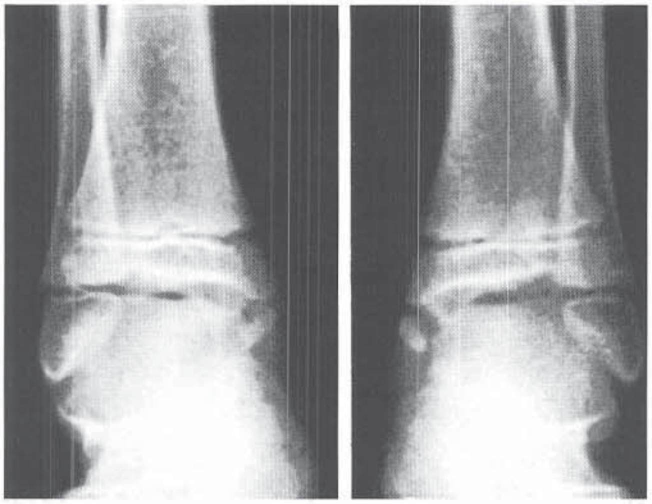


FIGURE 22–5 Accessory ossification center of the medial malleolus, a normal anatomic variation.

very smooth, regular, lucent area between it and the tibia. On the other hand, a fractured os trigonum has a rough border.

Injury to the os trigonum is seen in participants in activities that require extreme plantar flexion, especially ballet dancing (Fig. 22–9). Marotta and Micheli described a series of ballet dancers whose symptoms included pain localized to the posterior ankle, limitation of motion, weakness, swelling, and neurologic changes with dance activities. All were severely hampered in dance and had not benefited from nonsurgical therapy. All improved after excision of the ossicle, and although two-thirds still had occasional discomfort, all were able to return to dance. Time to full activity averaged 3 months.[12] Wakeley and colleagues found that magnetic resonance imaging (MRI) was best for demonstrating that the os trigonum was the site of symptoms when flexion and extension images were obtained to show the mobility of the fractured os.[25] Wredmark and colleagues described findings in 13 Swedish National Ballet dancers who underwent removal of the os trigonum. The main symptom was pain in the hindfoot while the dancer was actively plantar flexing the ankle. The flexor hallucis sheath was also incised if thickened.[27] Grogan and colleagues, in an anatomic study of 7-, 12-, and 14-year-old specimens, found the os trigonum to be part of the talus (i.e., in the cartilage anlage of the talus) as a secondary ossification center, similar to the posterior calcaneal apophysis. The chondro-osseous border of the synchondrosis was injured either as a chronic stress fracture or, less frequently, as an acute fracture, comparable to the injury patterns involving the accessory navicular.[6] Bone scan may show increased uptake in those symptomatic due to undisplaced fractures.[7] It should be remembered that when a patient presents with these symptoms, a trial of cast immobilization may relieve pain, and surgery should be reserved for those in whom conservative therapy fails.[13]

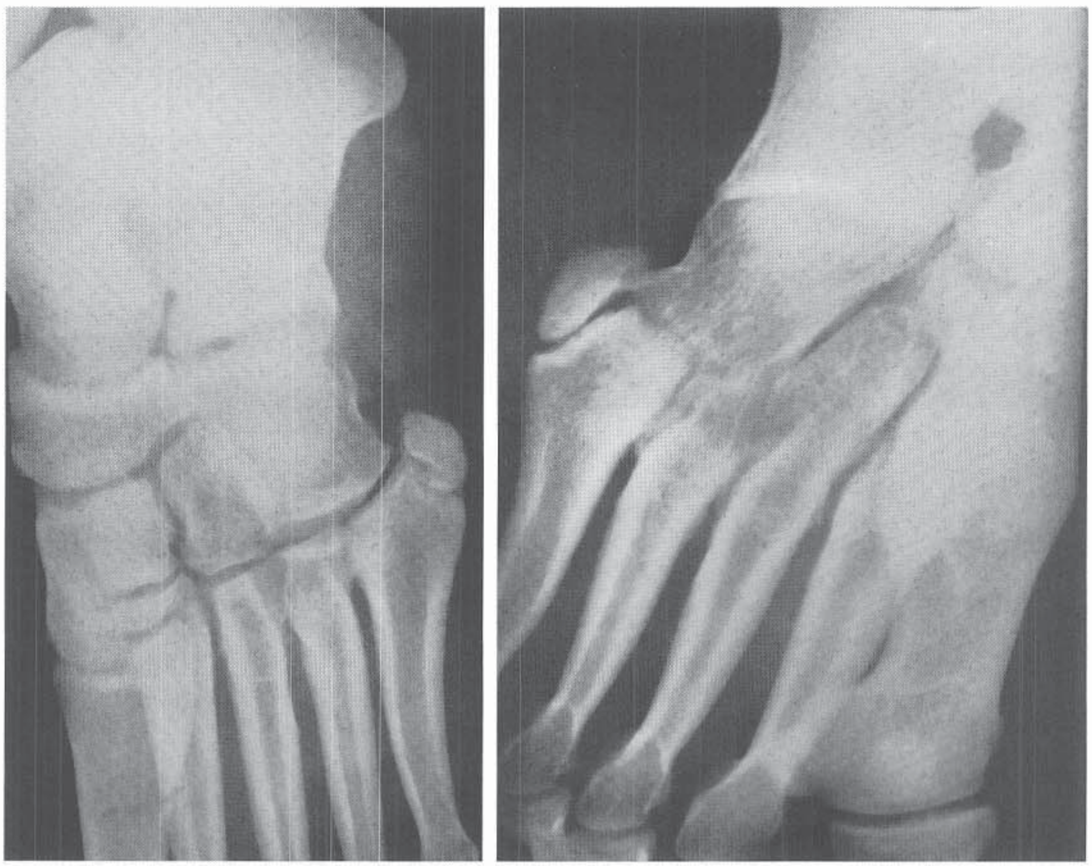


FIGURE 22–6 Accessory ossicle at the base of the fifth metatarsal (os vesalianum).

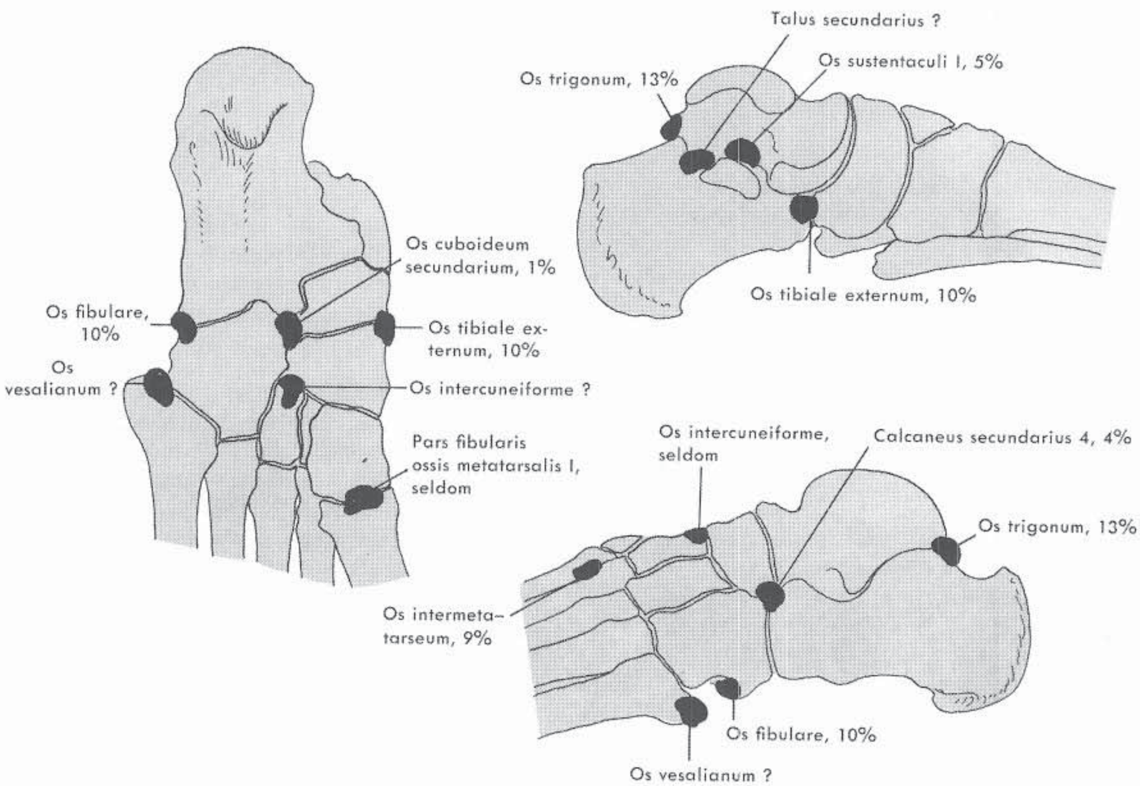


FIGURE 22–7 Accessory bones in the foot. (Adapted from von Lanz T, and Wachsmuth W: Praktische Anatomie, p 359. Berlin, Julius Springer, 1938. Copyright © 1938 by Julius Springer.)

ACCESSORY NAVICULAR

Accessory bones are common in the foot, but the accessory navicular is most often associated with symptoms. The condition was first described by Bauhin in 1605.[9,28] It has also been termed the prehallux, accessory scaphoid, os tibiale externum, os naviculare secundarium, and navicular secundum.[3–5,9] The estimated prevalence of accessory navicular bones in the general population ranges from 14 to 26 percent.[5,11,21]

Three types of accessory navicular bones have been described.[17,19] Type I is a small ossicle within the substance of the tibialis tendon. This type has also been called the os tibiale externum. Type II is an 8- to 12-mm ossicle, a medial

FIGURE 22–8 Os trigonum in a 12-year-old child. Note also the accessory navicular, visible in a lateral projection. The sclerosis of the apophysis of the os calcis is normal.

FIGURE 22–9 Fracture of a fused os trigonum.

and plantar extension of the navicular bone, that is connected to the navicular by a cartilaginous bridge. Type III is a cornuate navicular remaining after fusion of the accessory navicular with the primary navicular bone.

Pathology. In an examination of accessory naviculars removed from symptomatic patients, Grogan and associates[5] found areas of microfracture of the cartilaginous synchondrosis, hemorrhage, acute and chronic inflammation within and around the synchondrosis, and cellular proliferation in the tissues surrounding the fractures. All of the accessory naviculars exhibited chronic inflammation indicative of chronic injury with a prolonged inflammatory response. All of the fractures were partial separations, with no cases of complete separation of the accessory naviculars from the primary navicular. These changes were considered to be the result of chronic stress, as seen in overuse syndrome.

Bareither and associates studied 38 cadaveric feet that had a prominent navicular area.[1] Nineteen of the feet had an accessory navicular bone; the other 19 feet had a hypertrophic posterior tibial tendon. Some of the accessory navicular bones were true sesamoids, lying in the tendon before it split and separated from the navicular bone by a 3-mm distance. The majority of accessory naviculars were connected to the navicular by fibrous tissue and were within the main insertion of the tendon.

Kidner originally thought that the accessory navicular interfered with normal leverage of the tibialis posterior, resulting in a weak longitudinal arch and flatfeet.[9,10] Subsequent studies have shown no relationship between an accessory navicular and flatfoot.[22]

Imaging Studies. An accessory navicular is best visualized on an oblique radiographic, directed from medial to lateral (the external oblique view) (Figs. 22–10 and 22–11).[5] It may also be seen on a standard anteroposterior (AP) projection. The navicular is the last tarsal bone to ossify. Ossification occurs in girls between ages 1 and $3\frac{1}{2}$ years and in boys between ages 3 and $5\frac{1}{2}$ years.[8,26] The accessory navicular ossifies at an even later age. The radiographic diagnosis of the condition usually is made in later childhood or adolescence.

Computed tomography (CT) better delineates the extent of the true and accessory naviculars. Technetium bones scans may help identify symptomatic accessory naviculars (Fig. 22–12).[5,18] In a study of patients with focal pain over the navicular, MRI showed edema of the marrow.[14]

The bipartite navicular (which is a separate entity from the accessory navicular) (Fig. 22–13) appears on radiographs as a dorsally displaced, comma-shaped, separate segment of the navicular. Despite the dorsal displacement, the segment still articulates with the talus.[15]

Clinical Features. Controversy exists as to how often the entity is painful and how often its presence goes unnoticed. Many children have asymptomatic accessory navicular bones that may be noticed incidentally on clinical examination or on radiographs. In addition, the true navicular extends well medially and toward the plantar surface of the foot, and a prominence in this area may often be due to pressure over the normally large navicular.

The child with a symptomatic accessory navicular will present with pain over an enlarged area at the medial aspect of the navicular (see Fig. 22–10). The enlarged site is seldom larger than a centimeter in diameter, and usually is somewhat smaller. This area, which is just at the insertion of the tibialis posterior tendon, often is callused or red. The pain is aggravated by tight-fitting shoes, especially those worn for sports, and is alleviated by wearing less constraining footwear.

On examination, there will be some tenderness over the enlarged area. It may be possible to feel motion between the prominent accessory navicular and the primary navicular. Resisted inversion is sometimes painful, and there may be tenderness over the tibialis posterior tendon (see Fig. 22–10).

Treatment. Treatment varies from observation to excision. A child with an accessory navicular should initially be treated with soft pads between the foot and the sole of the shoes, and should avoid wearing tight, stiff shoes. Elevated arch pads are not beneficial for these patients because the pads may aggravate the pressure over the navicular. If these treatment measures fail and there is planovalgus deformity to the foot, a valgus-correcting shoe insert (such as the UCBL device) may be effective. The UCBL device relieves pressure over the navicular by inverting the patient's heel during gait rather than by pushing up on the arch of the foot.

In more recalcitrant cases, the surgeon may consider injecting the joint between the accessory navicular and the primary navicular with steroids and an analgesic agent. Immobilization in a short-leg cast has also been recommended.[5]

A number of surgical procedures have been used for this condition. We prefer simple excision of the navicular, shelling it out of the substance of the posterior tibial tendon. We have not found it necessary to detach the tendon from its broad insertions. The incision extends along the medial surface of the foot, directly over the accessory navicular from the mid-talus to the base of the first metatarsal. The subcutaneous tissue and the deep fascia are divided and the wound margins are retracted to expose the posterior tibial tendon and the medial portion of the navicular. The posterior tibial tendon inserts into the tuberosity of the navicular and into the plantar surfaces of the three cuneiform bones, as well as into the bases of the second through the fourth metatarsals and laterally into the cuboid. The accessory navicular is dissected free from the central portion of the posterior tibial tendon, leaving the remainder of the tendon insertions intact. The accessory navicular is excised and the medial and plantar portions of the navicular are resected until it is flush with or slightly depressed relative to the adjacent talus and cuneiform. If the primary navicular is still prominent after excision of the accessory navicular, the surgeon should consider completely removing the medial prominence of the true navicular. Bone wax may be pressed onto the exposed bony surface to help prevent regrowth of a medial prominence. The wound is then closed and soft dressings are applied. A short-leg walking cast may be used postoperatively for several weeks for ease of ambulation during healing.

Other authors prefer the Kidner procedure.[9,10,17,23] This operative approach entails rerouting the central slip of the tibialis posterior laterally onto the plantar surface of the navicular, where it is sutured under tension to the local ligaments. This is usually followed by a short-leg walking cast for 3 to 4 weeks postoperatively.

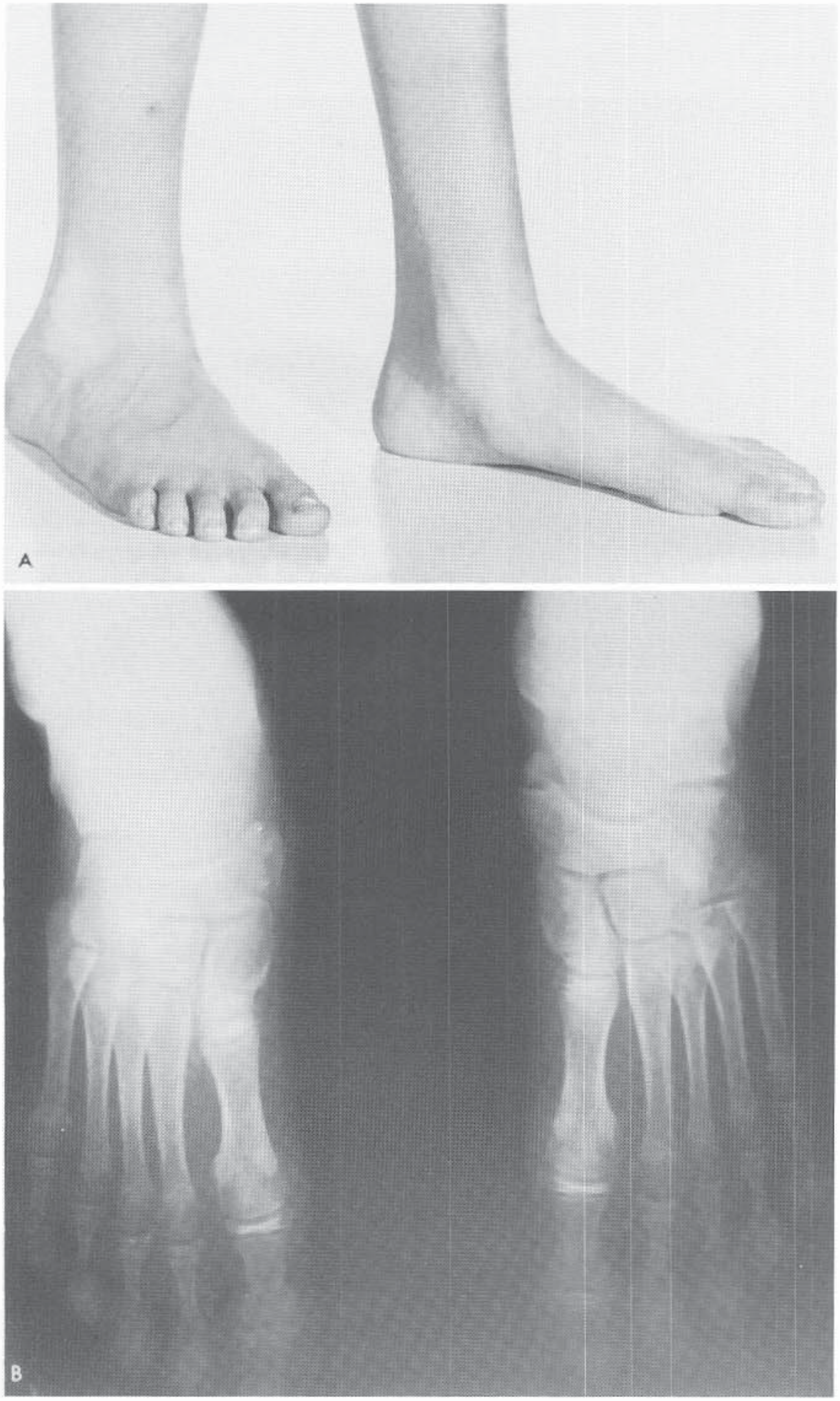


FIGURE 22–10 Accessory navicular of the left foot. **A,** Clinical appearance. Note the local swelling. **B,** Radiographic appearance. Note the smooth and rounded outline of the accessory ossicle.

Results and Complications. In many cases, patients obtain full relief of symptoms after excision of an accessory navicular.[19,22] Grogan and associates[5] reported complete relief in 16 of 17 feet treated by simple excision, and Bennett and associates[2] reported good results in 90 percent of 75 feet. Using the Kidner procedure, Prichasuk and Sinphurmsukskul obtained good results in 27 of 28 patients and noted no postoperative change in the arch.[16] In a comparison of the results of simple excision and the Kidner procedure, Tan and associates found no differences, and therefore recommended the simpler approach.[24]

In our experience, it is not unusual for symptoms to persist after surgical excision of the accessory navicular. If the primary navicular is prominent medially, pain and tenderness may continue over the area even though the accessory navicular has been removed. In other cases, the

FIGURE 22–11 Accessory navicular fused with the body of the tarsal navicular (cornuted navicular).

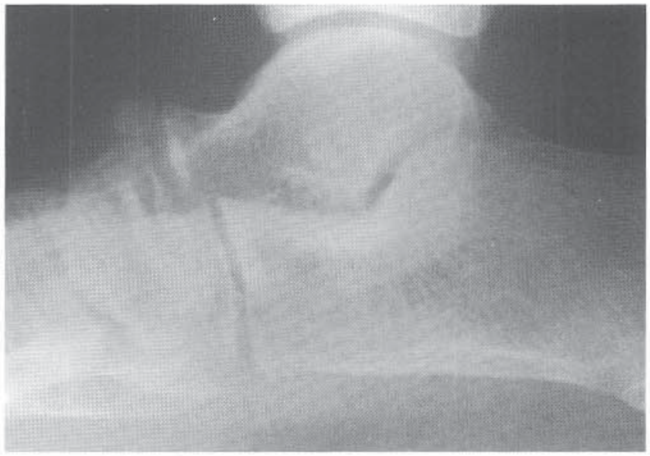
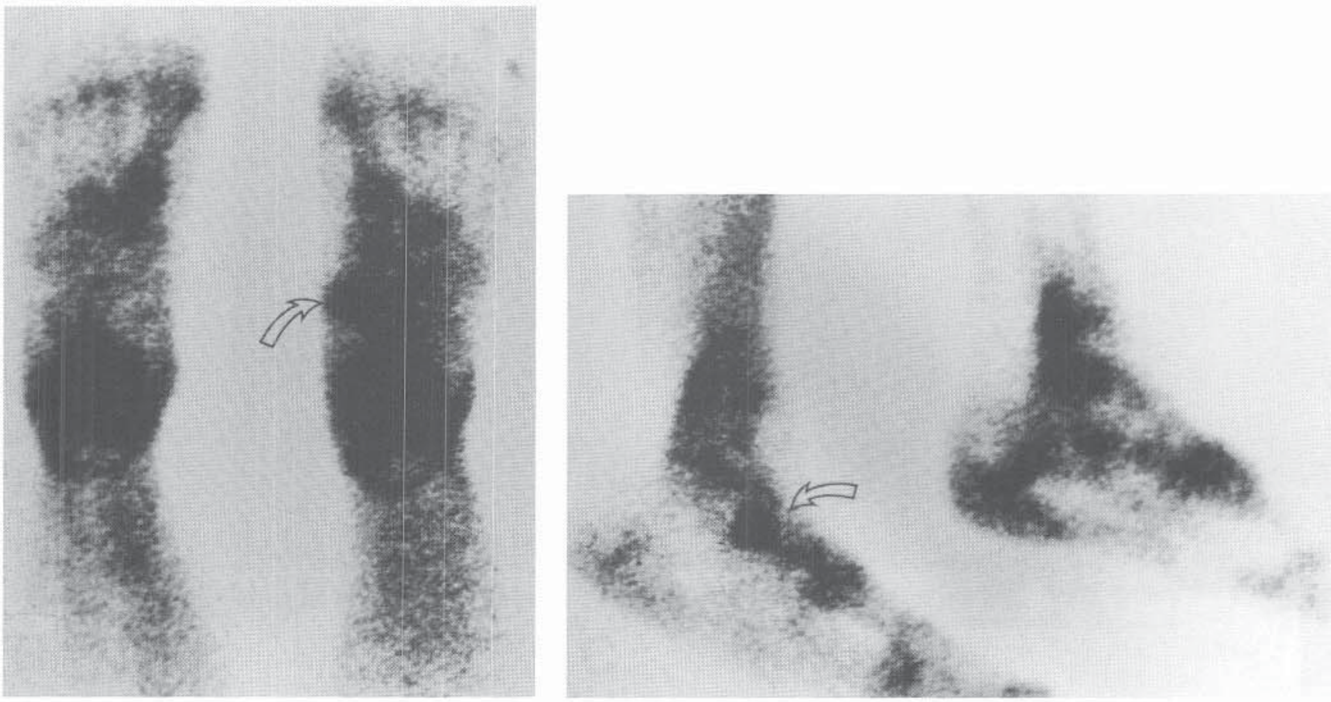


FIGURE 22–13 Lateral radiograph showing a bipartite navicular articulating with the talus.



A B

FIGURE 22–12 Accessory navicular. **A** and **B,** Technetium bone scans demonstrating increased tracer uptake in the symptomatic right foot (*arrow*) compared to the asymptomatic left foot. (From Grogan DP, Gasser SI, Ogden JA: The painful accessory navicular: a clinical and histopathological study. Foot Ankle 1989;10:164.)

scar itself and the area beneath the scar remain tender despite adequate removal of bone from the area. The cause of this is unclear. Over time these symptoms usually diminish, but they can be annoyingly persistent.

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Osteochondroses

KÖHLER'S DISEASE

Köhler's disease, described by Köhler in 1908,[4] is an osteochondrosis of the tarsal navicular. The disease is characterized by pain in the midfoot that is accompanied by radiographic changes of sclerosis, flattening, and irregular lucency of the tarsal navicular.

Etiology. The etiology of Köhler's disease is not known. It has been suggested that because of its late ossification relative to that of the other tarsals, the navicular is vulnerable to mechanical compression injury. In a study of normal navicular ossification, Karp found that ossification occurs much earlier in girls than in boys.[2] In more than half of the girls studied, the navicular had ossified by age 2 years. By age 3½ years, the navicular had ossified in all of the girls. In half of the boys, the navicular did not ossify until 3 years of age, and more than one-third were 3½ years old before the nucleus ossified. In those patients with slow ossification, the nucleus often appeared similar to Köhler's disease. Karp felt that the delay in ossification in boys predisposed them to the development of the disorder. Since ossification occurs later in boys than in girls, they would be more vulnerable to Köhler's disease.

Another possible cause of the disease process is avascular necrosis (AVN) due to periodic compression of the bone. The abundant blood supply of the navicular would allow for rapid, spontaneous healing, unlike AVN occurring in areas of marginal blood supply (e.g., the hip).[5] Blood is supplied to the navicular bone by a dense perichondrial network of vessels on the nonarticular surfaces. As a child matures, one or more penetrating arteries appear, and ossification begins around these vessels. When more than one vessel penetrates, there will be more than one ossification center. At maturity, the navicular is supplied by five or six arteries, which anastomose with the bone. In rare cases, a different pattern is found in which a single dorsal or plantar artery supplies most of the nutrition to the bone. Other tarsals seem to be similarly vascularized.[5] The vascular theories are supported by biopsy studies showing areas of necrosis, resorption of dead bone, and formation of new bone.[3]

Clinical Features. The disorder appears more often in boys than in girls, usually presenting between 2 and 7 years of age. The child will complain of pain in the midfoot and limping (Fig. 22–14). The duration of the symptoms ranges from a few days to more than a year. There is no apparent

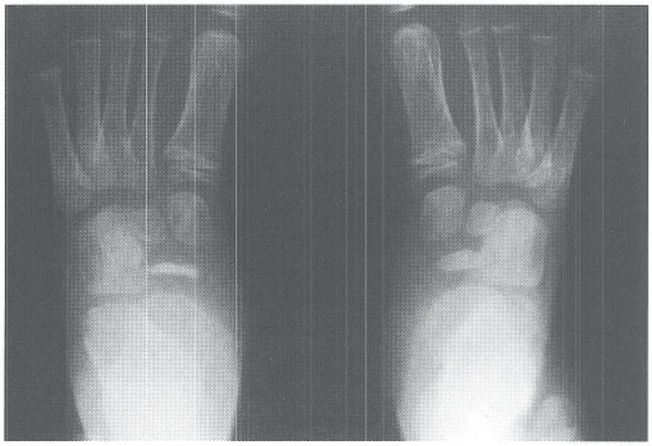

FIGURE 22–14 AP radiograph of both feet of a 4-year-old girl who complained of foot pain. The left navicular is sclerotic and wafer-thin, characteristic findings in Köhler's disease.

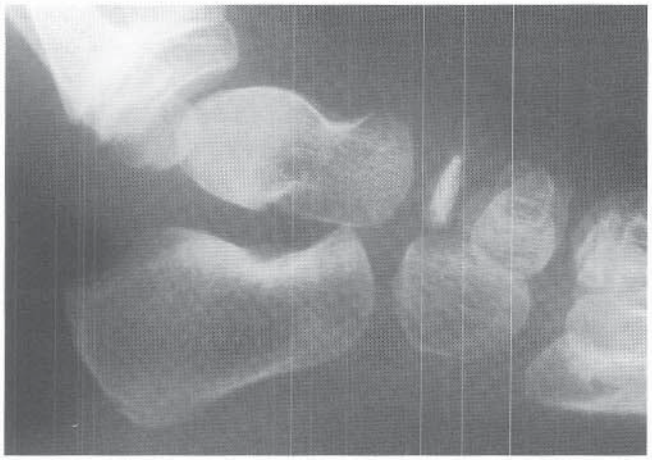

FIGURE 22–15 Köhler's disease. Lateral radiograph of the 4-year-old girl's left foot showing apparent compression of the navicular.

FIGURE 22–17 Lateral radiograph obtained 1 year later showing reconstitution of the navicular.

relationship between the duration of symptoms and radiographic changes. In one study, one-fifth of the patients had no symptoms, and the diagnosis of Köhler's disease was made incidentally from radiographs. A small number of patients will have a distinct history of trauma.

Physical signs include tenderness, swelling, and sometimes redness over the dorsum of the foot (occasionally this picture has been mistaken for an infection). The foot usually is held in pronation. Occasionally, however, it will be in supination as the child walks on the lateral side of the foot to relieve stress on the painful medial arch.

The natural history of the disorder is one of spontaneous resolution of clinical symptoms and radiographic abnormalities over a period ranging from 18 months to 3 years.[5]

Radiographic Findings. The radiographic findings in Köhler's disease are quite distinct (Fig. 22–15). Often there is dense sclerosis of the navicular, with narrowing and apparent flattening of the bone (especially on the lateral projection). On the AP view, both sclerosis and lucency of the navicular are seen. These changes gradually disappear over several years, with the radiographic appearance of the navicular returning to normal (Fig. 22–16).

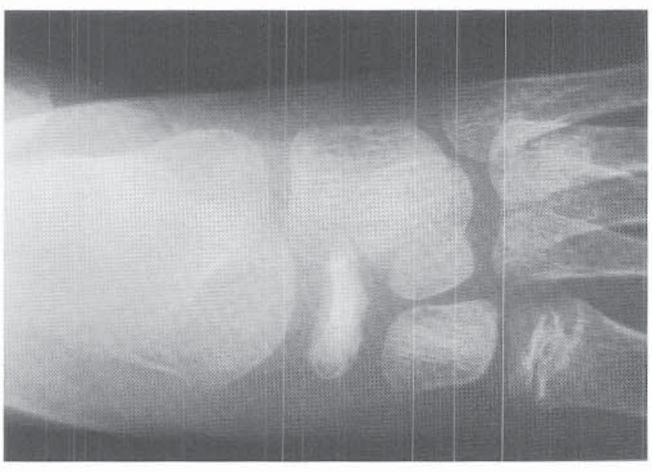

FIGURE 22–16 AP radiograph of the navicular obtained 1 year later showing partial reconstitution of the navicular with reduced sclerosis. This will go on to full healing.

Treatment. Karp in 1937 found that the mainstay of treatment was restriction of weightbearing.[2] Supportive measures, such as shoe inserts and casts, did not seem to affect the course of the disorder. Williams and Cowell[6] and, more recently, Borges and associates[1] have reported that symptoms resolved faster in patients treated in walking casts than in patients who were not casted. Cast treatment is currently recommended for patients with persistent symptoms that limit their activities.

Results. Köhler's disease is a self-limiting disorder that, in virtually all cases, resolves over time. Radiographic changes return to normal, and persistence into adulthood does not occur (Fig. 22–17). In a 31-year follow-up study, Borges and associates found that only two patients had persistent symptoms after being treated by cast immobilization.[1] One patient had a talocalcaneal coalition and the other patient had a large accessory navicular. The authors concluded that patients could expect to become normal with symptomatic treatment.

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FREIBERG'S INFARCTION

Freiberg's infarction is a disorder, usually seen in adolescence, that is characterized by pain over the plantar aspect of the second metatarsal head and associated destructive radiographic changes. Occasionally the disease involves other metatarsals. In the European literature, this disorder is known as Köhler's No. 2 disease, to distinguish it from

Köhler's disease of the navicular. Freiberg, however, was the first to describe the disorder,[3,4] and his name is more appropriately applied.

Etiology. The etiology of the disorder is not known. It is commonly thought to be due to AVN of the metatarsal head, and the histologic findings resemble those of AVN of other bones.[1] Smillie postulated that repetitive stress to the metatarsal head could be the etiology.[8] Binek and associates felt the disease was AVN caused by microfracture secondary to abnormal stress on the metatarsal head.[2] Katcherian proposed that the condition was related both to trauma and to abnormal circulation.[5] Stanley and associates, though, found no evidence to support trauma as the cause and, through pedobarographic studies, that pressure was not increased at the affected metatarsal head.[11] However, in their series of 33 feet, 85 percent of the affected metatarsals were the longest in the foot, and the authors believed this was an etiologic factor.

Clinical Features. The disorder appears most often in adolescents, usually after age 13 years. It occurs more often in girls than in boys. Pain under the second metatarsal head is the most common complaint, with resultant limping and a decrease in physical activities. An antalgic gait with poor push-off is usually present as well. Physical findings are normally limited to tenderness over the affected metatarsal head, with occasional swelling noted.

Radiographic Findings. Radiographs of the second metatarsal head show areas of lucency, and collapse with flattening and loss of the normal shape of the condyles (Fig. 22–18). This area shows increased uptake on technetium bone scans.

FIGURE 22–18 Freiberg's infarction. Radiograph of the metatarsals shows the typical flattening and sclerosis of the head of the second metatarsal.

Treatment. An initial trial of conservative treatment is strongly recommended. A period of time in a hard-soled shoe or a trial in a short-leg walking cast will often relieve symptoms. Subsequent use of a metatarsal pad in the shoe may reduce pressure on the metatarsal head.

Excision of the metatarsal head, which is recommended in very refractory cases, has been reported to relieve symptoms. Tachdjian advocated curettage of the metatarsal head, with a cancellous bone graft placed in the cavity in the head.[12] Several authors recommend a dorsiflexion osteotomy of the metatarsal head to relieve symptoms.[5–7] Spoul and associates described improvement in symptoms in 10 of 11 cases treated by metatarsophalangeal debridement.[10] Smith and associates reported that shortening of the second metatarsal provided excellent relief of pain in 16 feet, although most of the patients subsequently experienced persistent stiffness of the metatarsophalangeal joint.[9]

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Freiberg's Infarction

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Congenital Deformities

POSTURAL DEFORMITIES

Metatarsus Adductus. Occasionally, children are born with a foot deformity in which the forefoot is deviated inward relative to the hindfoot. The deformity may be very mild and resolve spontaneously, it may be slightly fixed and persist to walking age, or it may be rigid and associated with valgus of the hindfoot (Fig. 22–19). The milder deformities do not require treatment, and the moderate ones respond to manipulation and casting. Those rare cases in which there is severe, rigid deformation may require surgical correction.

The terminology of these conditions is confusing because different authors use various terms, with little agreement among authors. Tachdjian used the term *metatarsus adductus* for the milder cases and the term *metatarsus varus* for the more rigid deformities.[21] Some authors have called these

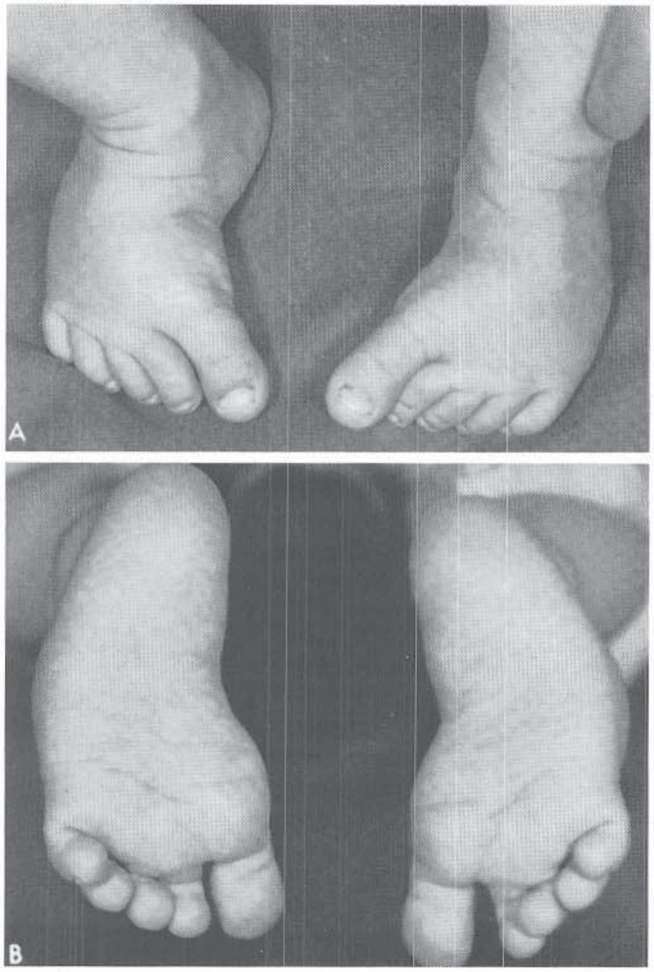

FIGURE 22–19 Bilateral mild metatarsus adductus. **A,** Dorsal view showing medial deviation of all the metatarsals. **B,** Plantar view showing the "bean-shaped" foot. This type of foot is easily corrected with serial casting.

conditions "a third of a clubfoot." This descriptive term is misleading, because the adductus of the forefoot is the only feature that resembles a clubfoot. The hindfoot is in valgus and easily dorsiflexes, which is the antithesis of a clubfoot. Other names that have been used include hook-foot, bean-shaped foot, serpentine foot, and congenital metatarsus adductus (Fig. 22–19). The term *skewfoot* has been used to describe feet with fixed hindfoot valgus and subluxation of the midfoot.

For simplicity, we will call most of these abnormalities metatarsus adductus, and qualify them as actively correctable, passively correctable, or rigid. In this text, the term skewfoot will be reserved for a foot with fixed adductus of the forefoot, increased valgus of the hindfoot, and lateral subluxation of the navicular on the talus.

ETIOLOGY. It has long been presumed, but never proved, that intrauterine compression produces the characteristic deformation of the foot. The condition is associated with torticollis and developmental dislocation of the hip to some degree, but it is not related to birth order.

INCIDENCE. Although flexible metatarsus adductus is a common neonatal problem, it is often overlooked. Thus, it is difficult to accurately determine the true incidence of the very mild forms of the abnormality. In a screening of 2,401 neonates, Widhe and associates noted foot abnormalities in 4 percent of the infants, with 1 percent having metatarsus adductus (by comparison, 0.7 percent had calcaneovalgus).[26] Hunziker and associates found that whereas metatarsus adductus was equally common in preterm and full-term infants, the condition was more likely to persist in the premature babies.[9] Wynne-Davies found an incidence of metatarsus varus of 1 in 1,000 births and reported that if one child was affected, the risk of deformity in a second child in the same family was 1 in 20.[27]

PATHOLOGY. Morcuende and Ponseti studied two fetuses (16 and 19 weeks of gestation) that had metatarsus adductus and found that the shape of the medial cuneiform was altered, with medial deviation of the articular surface.[15] There also was adduction of the metaphyses of the second through the fifth metatarsals. There were no joint subluxations or tendon abnormalities. Based on findings after dissection of stillborn babies' feet, Reimann and Werner concluded that the primary abnormality was medial subluxation of the tarsometatarsal joints in utero when the foot was in a dorsiflexed position.[18]

RADIOGRAPHIC FINDINGS. Radiographs show medial deviation of the metatarsals at the tarsometatarsal level, with some degree of valgus of the hindfoot. In older children with more severe forms of metatarsus adductus, there may be medial deformation of the metatarsal shafts.

CLINICAL FEATURES. The deformity usually is noted at birth, but it may present at any age. The clinical hallmark of the condition is medial deviation of the forefoot relative to the hindfoot. When the foot is viewed from the dorsal surface, the entire foot often appears to be turned inward. When the foot is viewed from the plantar surface, the sole of the foot has the shape of a bean. The base of the fifth metatarsal is prominent and the forefoot seems to be deviated away from it. The arch often appears higher than normal. In addition, the space between the first and second toe is wider than normal, and the great toe seems to be reaching medially. Kite listed six characteristic features of the disorder (Table 22–1).[12]

When the examiner grasps the heel and compares the alignment of the heel to the forefoot, the full extent of the deformity can be better appreciated. Although it is not seen on a casual examination, careful evaluation of the hindfoot reveals a slight valgus of the heel. There is always full range of ankle and subtalar motion.

It is important to establish the degree of flexibility of the deformity. In mild cases the foot will correct actively when

TABLE 22–1 **Characteristic Features of Metatarsus Adductus**

- Spontaneous active medial deviation of the foot by the child
- High arch
- Concave medial border of the foot
- Separation of the first and second toes
- Fixed adductus of the forefoot when the hindfoot is held in neutral
- Bean-shaped sole of the foot

These features were identified by Kite.[12]

the lateral border of the foot is stimulated. In less flexible cases the foot will not correct actively but can be easily corrected passively. The examiner should maintain the hindfoot in neutral during this maneuver, grasp the heel with one hand, and, with the web space of the hand placed against the head of the first metatarsal, push the foot laterally. A gentle push will align the metatarsals in most children. A rigid deformity has a medial soft tissue crease at the tarsometatarsal level and a medial soft tissue contracture that prevents passive correction of the foot. As the forefoot is abducted, the great toe will abduct and a tight abductor hallucis can be palpated medially (Fig. 22–20).

Many of these children will also have internal tibial torsion, which will contribute to an intoed appearance. This is the most common parental complaint. This component of the deformity should be noted separately.

TREATMENT. If the metatarsus adductus is flexible and spontaneously corrects as the foot is stimulated into active eversion, the condition does not need to be treated. These mild deformities will resolve gradually on their own. The parents should be reassured and shown how to gently stretch the foot and how to stimulate it to actively correct.

Metatarsus adductus of intermediate severity—that is, a deformity that does not actively correct but is easily corrected passively—can usually be treated, but there is considerable debate as to the need to treat. Many physicians believe that these deformities will self-correct over time without treatment. In a 7-year follow-up of 130 untreated feet, Rushforth found that 10 percent had moderate deformity but were asymptomatic, and that only 4 percent had residual deformity and stiffness.[19] Some physicians advocate passive

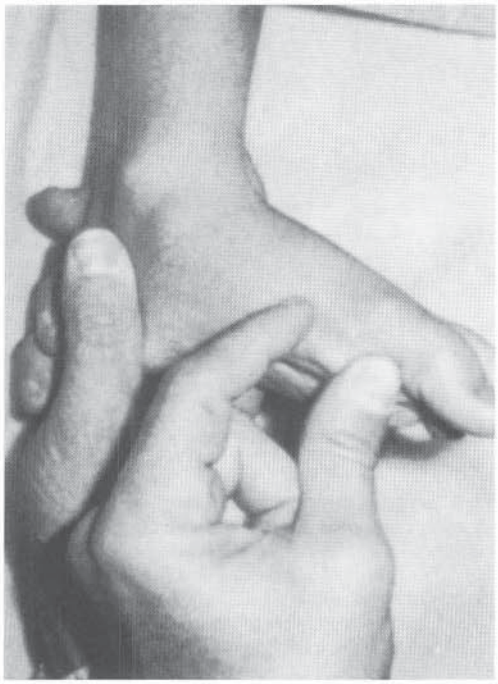

FIGURE 22–20 Lichtblau's test to demonstrate contracture of the abductor hallucis muscle often present in metatarsus adductus. (From Lichtblau S: Section of the abductor hallucis tendon for correction of metatarsus varus deformity. Clin Orthop 1975;110:228.)

stretching of the foot by the child's parent (Fig. 22–21). The parent is taught to hold the heel in a neutral position with one hand and abduct the forefoot with the web space of the other hand. Some physicians recommend that the child wear straight-last shoes as well. For a child less than 6 months old, a short series of short-leg plaster casts will easily correct the foot position, and this treatment approach may be warranted for patients with obvious deformity. Some authors prefer long-leg bent-knee casts, but find that only 10 percent of patients will need treatment.[17]

The technique of cast correction is similar to the stretching procedure taught to the parent. The child's heel is grasped and held in a neutral position while the forefoot is abducted. The thumb of the hand holding the heel should reach the cuboid so that the fulcrum for abducting the forefoot is at the level of the cuboid metatarsal joint. Eversion of the foot should be avoided. Nonrigid casts that are easily removed are preferable to rigid casting material. Kite recommended that three criteria be met before discontinuing cast treatment.[11–13] First, the convexity of the lateral border of the foot should be straightened or reversed. Second, the prominence of the base of the fifth metatarsal should no longer be noticeable. Third, active adduction should no longer occur when the child moves the foot.

Metatarsus adductus that cannot be passively corrected should be more vigorously treated.[2] These feet usually have significant rigidity and a medial crease with an overactive abductor hallucis. Most such feet can be corrected with serial stretching and cast application, which should be started as soon as possible.[13] If the foot is resistant to cast correction, surgical release of the abductor hallucis and capsulotomy of the first tarsometatarsal joint may be indicated, followed by further cast correction.[23] A recent report of 12 cases of metatarsus adductus treated by this procedure at a mean patient age of 3 years 6 months showed good results at an average follow-up of $3\frac{1}{2}$ years.[1]

The best way to manage older children who present with fixed adductus also is controversial. The Heyman-Herndon release of the tarsometatarsal joints has had mixed results: some authors have reported excellent outcomes and others have had numerous failures (Fig. 22–22).[8] Stark and associates reported good results in only half of their patients treated at a mean age of 5.4 years.[20] We occasionally recommend this procedure for treating severe adductus in children too young to undergo osteotomies. Children with significant deformity who are older than 3 years should undergo metatarsal osteotomies to realign the foot. Care must be taken, though, to avoid forcing the hindfoot into valgus and creating a skewfoot deformity.

Two other surgical procedures that have been recommended for treating metatarsus adductus are anteromedial release with capsulotomy of the medial metatarsocuneiform and naviculocuneiform joints,[6] and transfer of the posterior tibial tendon with capsulotomy of the naviculocuneiform joint.[3] We have no experience with either of these approaches.

Talipes Calcaneovalgus. This postural deformity of infancy presents as a sometimes dramatic hyperdorsiflexion of the foot that appears to be "plastered" up against the anterior surface of the tibia (Figs. 22–23 and Fig. 22–24). Plantar flexion of the foot is often limited as a result of

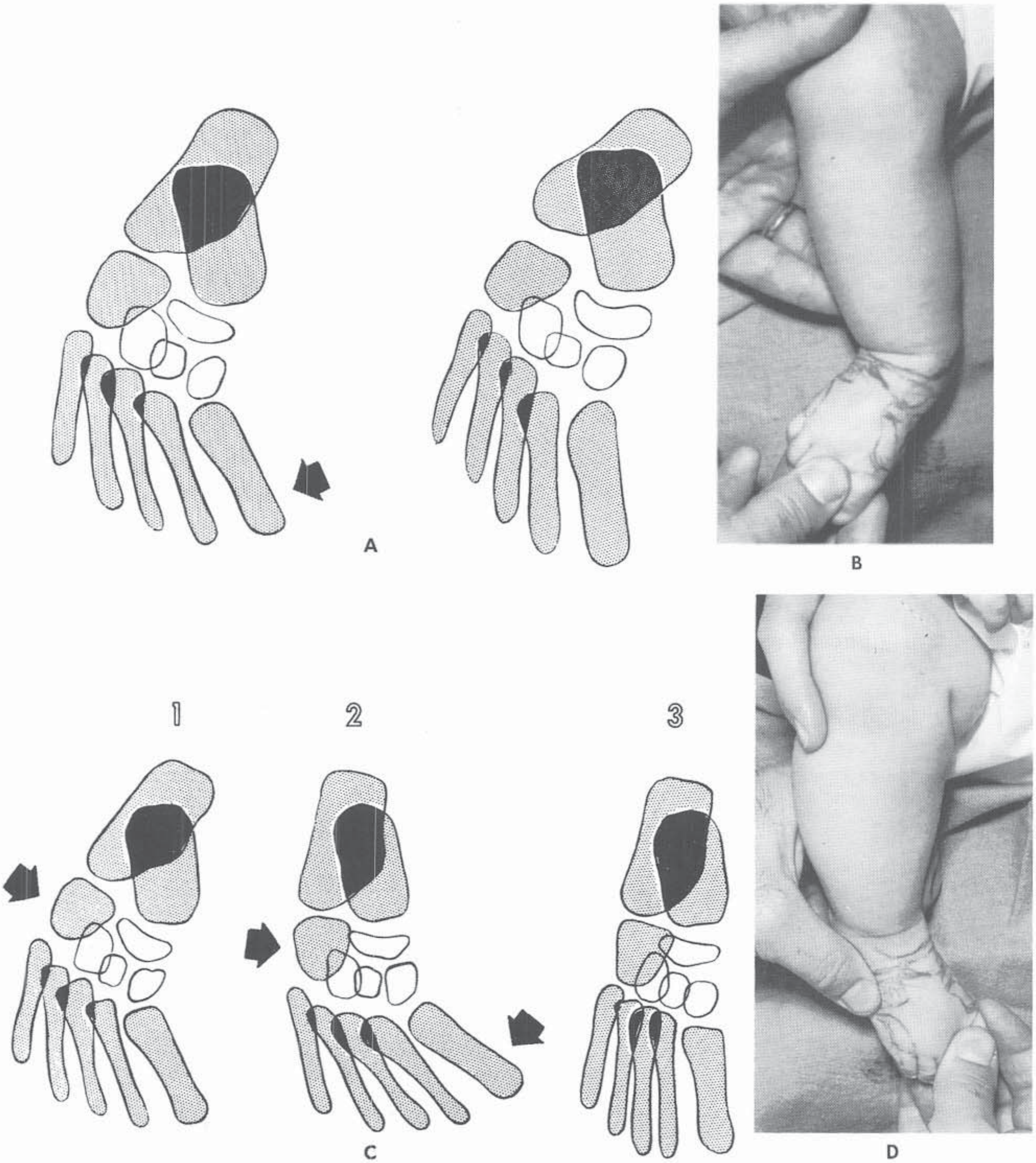

FIGURE 22–21 Correction of metatarsus adductus by passive stretching. **A** and **B**, The incorrect method of manipulation. The entire foot is abducted and everted by forcefully abducting and everting the forefoot without applying counterpressure to the hindfoot. The foot is simply being twisted at the ankle, with little corrective force being exerted at the metatarsotarsal joints. The diagram illustrates how the valgus deformity of the heel is increased and shows that the improved appearance of the varus deformity of the forepart of the foot is spurious and not real correction. **C** and **D**, Correct method of manipulation. The hindfoot is slightly plantar flexed and the anterior process of the talus is displaced medially underneath the head of the talus; the metatarsals are pushed into abduction while counterpressure is applied over the cuboid. The diagram illustrates the proper method.

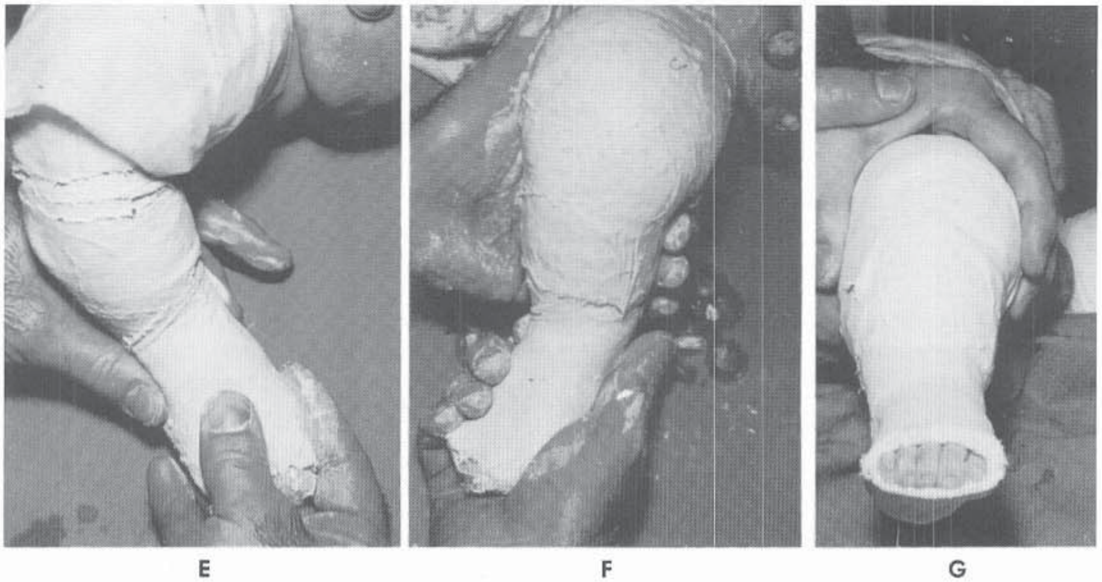

FIGURE 22–21 *Continued*. **E,** The foot points somewhat medialward while the first section of the plaster cast is applied. **F,** The foot and leg are in slight external rotation while the second section of the plaster cast is applied. **G,** Completed plaster cast. The heel and anterior part of the foot are immobilized in a position as near normal as possible. (From Ponseti IV, Becker JR: Congenital metatarsus adductus: the results of treatment. J Bone Joint Surg 1966;48-A:706.)

contracture of the anterior ankle and foot structures. This deformity results from abnormal in utero positioning of the foot, described as a "packaging" defect, in which all of the structures formed normally but then were deformed at the end of the pregnancy as the foot was abnormally positioned due to in utero crowding. Unlike a "manufacturing" or "parts" problem, such as congenital clubfoot, in which embryologic formation is defective, postural deformities such as calcaneovalgus resolve spontaneously in the vast majority of cases.

Not only is the foot hyperdorsiflexed, but the heel is often in marked valgus, with the forefoot appearing abducted. The calcaneus is palpable in the heel pad and is noted to be in the dorsiflexed ("calcaneus") position. This differentiates the calcaneovalgus foot from the more serious pathologic congenital vertical talus, where the heel is in equinus, giving the "rocker-bottom" appearance to the foot. The forefoot may be equally dorsiflexed, as in calcaneovalgus, but it is the heel equinus and rocker-bottom that distinctly differentiate this entity from the calcaneovalgus foot. It is important to look for associated problems that accompany the packaging problem of the foot—in particular, hip dysplasia. Although no initial instability may be appreciated during the newborn hip examination, the association of hip dysplasia with calcaneovalgus seems somewhat stronger when a contralateral metatarsus adductus is present, giving the feet a "windblown" appearance.[24] Although the association of hip dysplasia with metatarsus adductus is controversial, with some investigators reporting it as a close association[10,14,24] and others refuting such an association,[4] the simultaneous occurrence of calcaneovalgus of one foot and metatarsus adductus of the other seems to heighten the likelihood of at least silent dysplasia.[24]

Less subtle is the association of posteromedial bowing of the tibia with a calcaneovalgus foot. The deformation of the tibia is easily understood as a packaging defect associated with the hyperdorsiflexed foot (see Figs. 22–23 and 22–24). However, there is more to this "packaging" combination in that, as covered in Chapter 21, Disorders of the Leg, there is usually growth inhibition of the lower leg due to the posteromedial bow, and the growth inhibition usually mandates some form of limb equalization procedure later in childhood and adolescence. Thus, as opposed to a pure packaging defect, which should resolve without sequelae, calcaneovalgus with posteromedial bow of the tibia represents a more serious defect in morphogenesis.

Perhaps most commonly associated with calcaneovalgus is external rotation of the lower extremities. It becomes most obvious as the child begins to take weight on the lower extremities and the feet point outward, sometimes approaching 90 degrees externally. The source of the foot position may be persistent eversion and external rotation from the calcaneovalgus, or it may be an external rotation contracture of infancy at the hip.[16] The latter is destined to resolve spontaneously once the child begins walking, again with the caveat that silent hip dysplasia should be ruled out by some form of imaging study (radiography or ultrasound), especially if there is any suggestion of asymmetry in the hip examination and rotational profile of the infant.

The incidence of talipes calcaneovalgus has been reported to be as high as 30 to 50 percent of newborns.[25] This estimate is probably too high, as this same investigator found an increased incidence of flatfoot on long-term follow-up of patients who had calcaneovalgus in infancy. The study was skewed toward following patients with severe flatfoot. A more likely incidence is the 1 in 1,000 live births reported by Wynne-Davies.[27] The incidence, like that of congenital dislocation of the hip, is higher in first-born children and girls, again associating this foot deformity with hip dysplasia as alluded to above. Since calcaneovalgus foot position is either relatively common or extremely common, depending on which investigator is believed, the role of the orthopaedist

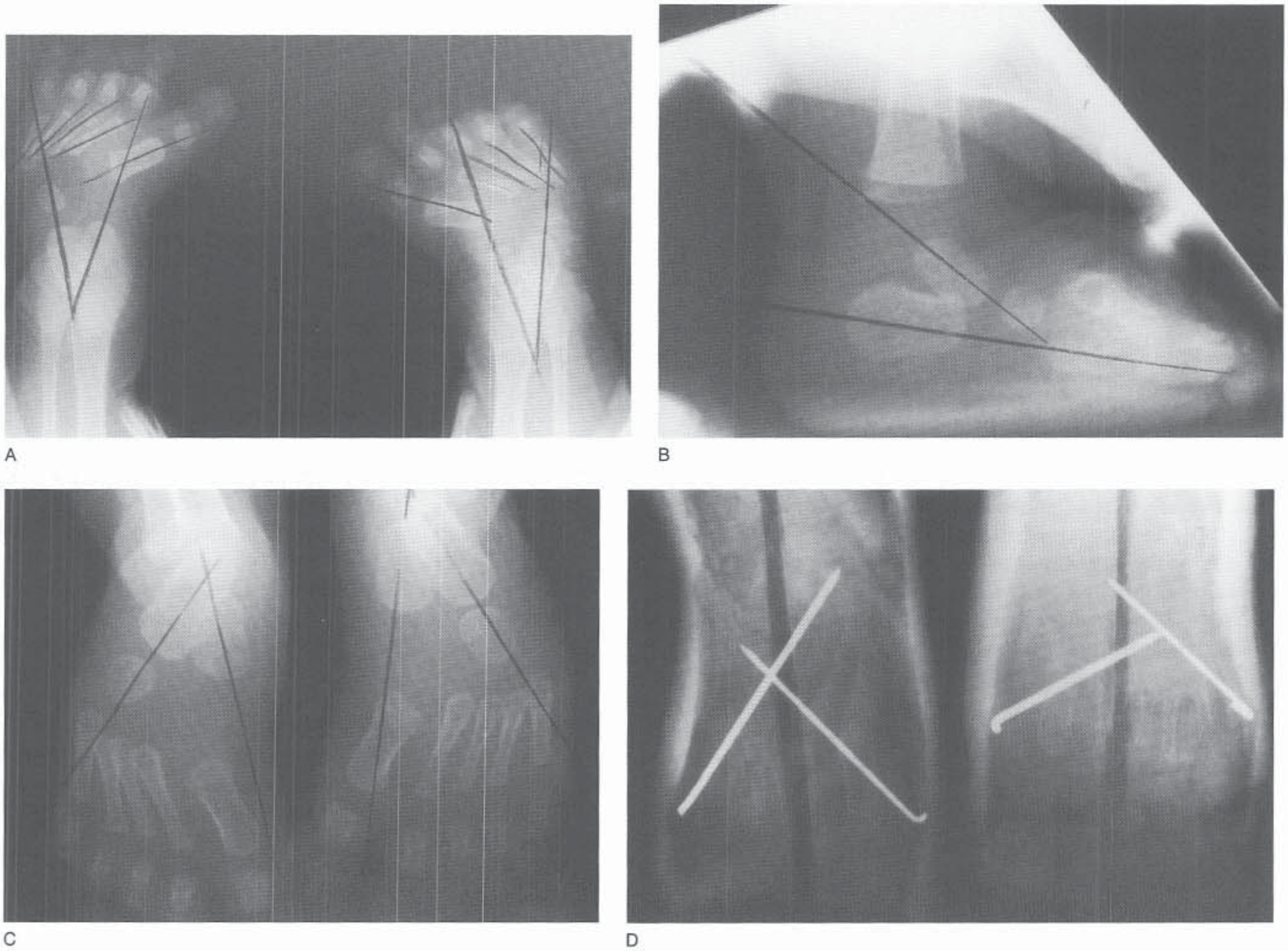

FIGURE 22–22 Very resistant metatarsus adductus in a young girl. **A,** Radiograph obtained at age 3 months showing marked medial deviation of the metatarsals at the tarsometatarsal joints. Note also the increased talocalcaneal angle, indicating valgus of the hindfoot. **B,** Standing lateral radiograph showing an increased talocalcaneal angle, indicating valgus of the hindfoot. **C,** AP radiograph obtained at age 15 months after a period of serial casting. There is still marked adduction of the forefoot, and the valgus of the hindfoot has increased. **D,** AP radiograph obtained after a tarsometatarsal soft tissue release and realignment stabilized with pins. This procedure is necessary only for the most severe cases of metatarsus adductus.

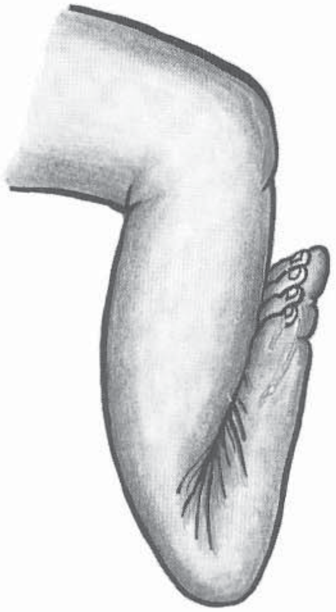

FIGURE 22–23 Severe talipes calcaneovalgus in a newborn. Note the foot "plastered" up against the anterior aspect of the tibia. The clinician should always examine the hips to rule out congenital dislocations.

in assessing what may be a normal variant of foot position is to eliminate true pathological foot conditions (congenital vertical talus), associated tibial anomalies (posteromedial bow of the tibia), and, most important, associated hip dysplasia.

TREATMENT. The prognosis for calcaneovalgus foot is excellent. Only in the most severe cases, with marked restriction of plantar flexion and supination/inversion, is anything more than gentle stretching exercises by the parents necessary. Generally the foot position normalizes within 3 to 6 months. In the more severe presentations, there may be a role for corrective casting and/or splinting in association with stretching exercises. The clinical experience of several authors has identified a correlation between talipes calcaneovalgus and a symptomatic form of hypermobile pes planovalgus in the older child.[5,7,22,25] There is thus little downside to two or three sets of corrective casts being applied in the newborn period when there is significant limitation of plantar flexion and inversion.[7] Casting can then be followed by stretching exercises and an ankle-foot orthosis (AFO) type of splint for a few additional months to ensure satisfactory foot position when the infant begins to pull to stand.

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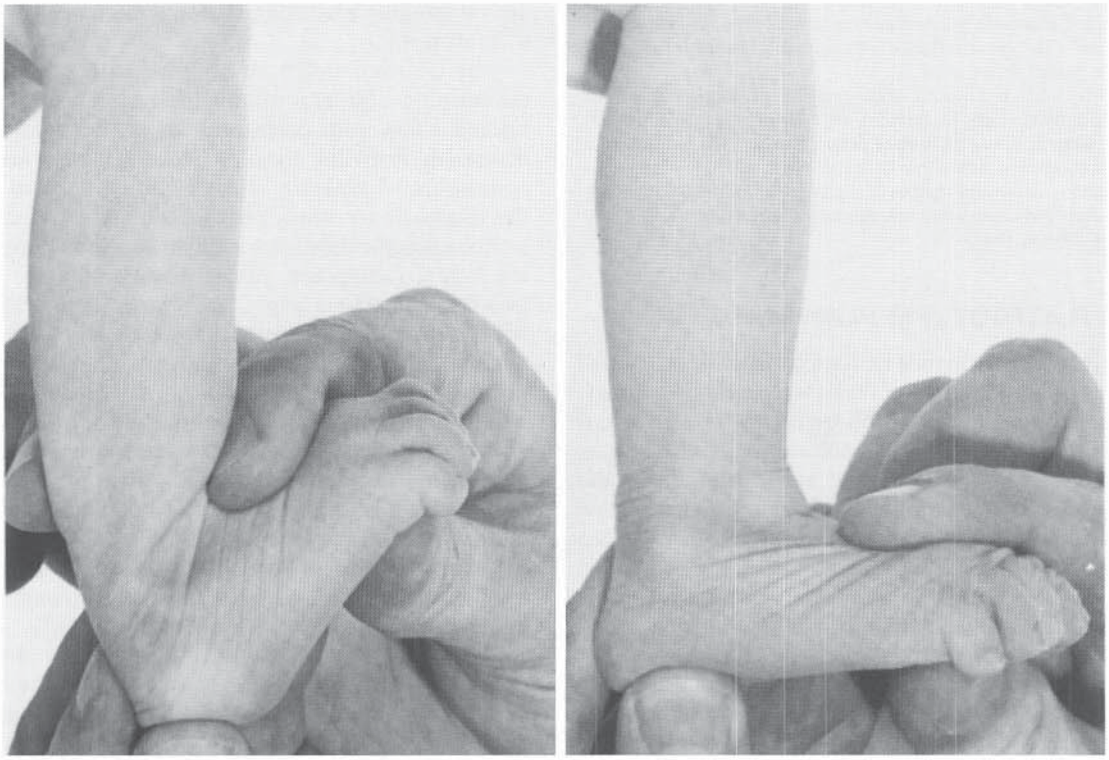

FIGURE 22–24 Talipes calcaneovalgus in an infant. The foot is dorsiflexed and everted. Plantar flexion is limited to neutral position.

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FLEXIBLE FLATFOOT (PES PLANOVALGUS)

Although the exact incidence of flatfoot in children is unknown, it is undoubtedly one of the most common "deformities" evaluated by pediatric orthopaedists. Whether or not flatfoot is an actual deformity is questionable. For example, Staheli and colleagues, in assessing and documenting the spontaneous development of the longitudinal arch, regarded flatfeet as "usual in infants, common in children, and within the normal range in adults."[40] Harris and Beath concluded from their study of Canadian army recruits that the flexible flatfoot "may be regarded as the normal contour of a strong and stable foot, rather than the result of weakness in foot structure or weakness of the muscles which motivate the foot."[17,18] Wenger has commented that "flexible flatfoot is the inevitable consequence of upright walking on normal foot bones which are connected by lax ligaments."[45] It may be surprising, therefore, that flatfoot is evaluated and treated, often prophylactically, with significant fervor by certain nonorthopaedic branches of medicine, where the controversy surrounding the "deformity" has not been resolved. On the other hand, because concern about abnormal-looking feet will never appreciably decrease among parents and pediatricians, especially in the more clinically severe-appearing feet (Fig. 22–25A), children with flatfoot will continue to be referred to the pediatric orthopaedist for the treatment of pain, perceived disability, and abnormal shoe wear (Figs. 22–25B and C).

There is no universally accepted clinical or radiographic definition of a flatfoot. The normal height of the longitudinal arch and the amount of excessive flattening that is "abnormal" are unknown.[29] Flatfoot as a reflection of generalized ligamentous laxity in the foot best describes the situation in which the foot has an abnormally low or absent arch. The heel shows excessive eversion during weightbearing, and the forefoot is usually abducted, producing a midfoot sag with lowering of the longitudinal arch (Fig. 22–25), so that the talar head and navicular tuberosity appear to be in contact with the floor and to participate excessively in weightbearing. The medial column of the foot appears longer than the lateral column. The entire foot is often described as pronated, although this description is misleading because the forefoot is actually supinated in relation to the hindfoot, a fact that is most appreciated when the hindfoot is corrected operatively or stabilized manually during a physical examination.[29,45] The relationship of forefoot to hindfoot may also be appreciated when one contemplates the cavovarus foot, the anatomic reverse of the flatfoot, in which the forefoot is pronated in relation to the hindfoot, producing the excessively *high* longitudinal arch.[45] Thus, when the medial border of the foot is abnormally in contact with the floor during weightbearing, this is traditionally described as a flatfoot, although there are no studies correlating radiographic anatomy with such a "footprint."

The clinician might be tempted to use radiography as the defining diagnostic examination, with flatfoot being considered a foot with measurements greater than 2 standard deviations from the mean.[29] Because flatfeet are relatively common and generally benign, radiographs to document the diagnosis are rarely obtained, thereby perpetrating the lack of a specific definition for flatfoot. Nevertheless, a standing lateral radiograph allows measurement of the lateral talo-first metatarsal angle, or Meary's angle (Fig. 22–26), which normally is a 0-degree (straight-line) angle but in the flexible flatfoot will have an apex-plantarward angle. The normal range of this angle also varies with age, with spontaneous improvement in the plantar sag seen between 0 and 8 years.[43] The location of the sag—talonavicular or naviculocuneiform—can be determined, which may suggest the cause (a tight heel cord producing a plantar-flexed talus and talonavicular sag, for example) (Fig. 22–27). The degree of plantar flexion of the talus—the angle formed by the longitudinal axis of the talus and the horizontal—can also be measured (normal, 26.5 ± 5.3 degrees[6]).

Perhaps the most compelling reason to obtain radiographs in cases of flatfoot is to rule out other causes of the "deformity" besides idiopathic. The differential diagnosis includes such bony abnormalities as tarsal coalition, congenital vertical talus (convex pes valgus), persistent talipes calcaneovalgus, an accessory navicular, and various arthritic and

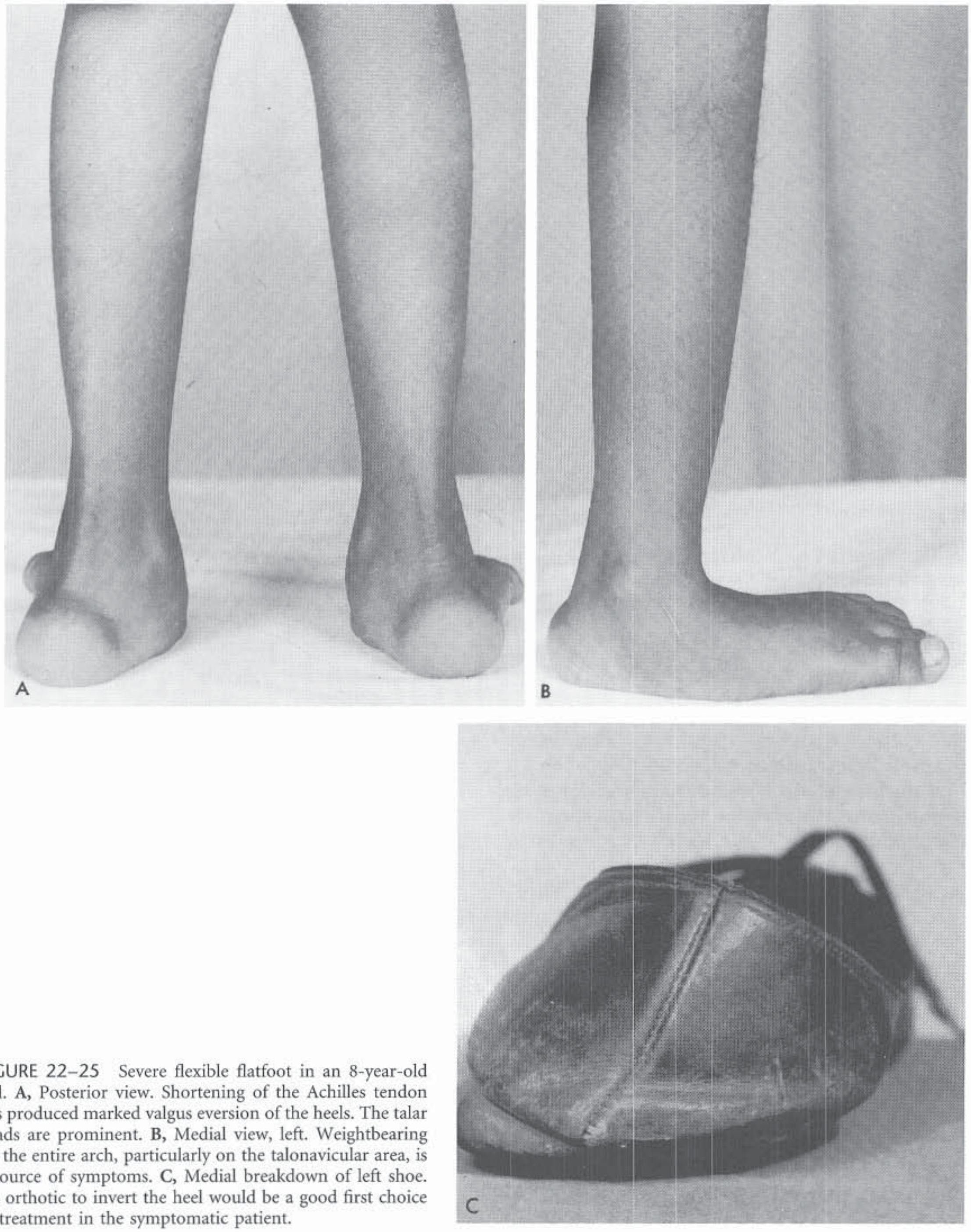


FIGURE 22–25 Severe flexible flatfoot in an 8-year-old girl. **A,** Posterior view. Shortening of the Achilles tendon has produced marked valgus eversion of the heels. The talar heads are prominent. **B,** Medial view, left. Weightbearing on the entire arch, particularly on the talonavicular area, is a source of symptoms. **C,** Medial breakdown of left shoe. An orthotic to invert the heel would be a good first choice of treatment in the symptomatic patient.

inflammatory conditions. Most of these conditions are diagnosed primarily from the history and physical examination findings, and radiographs are confirmatory. Not to be confused with diagnostic studies are radiographs obtained to appease parents—to "prove" that the flexible flatfoot is indeed just that. Such a radiograph may be important in aiding parental education, but in the otherwise typical case it is unnecessary for diagnosis.

Clinical Features. Regardless of the exact definition of flatfoot, it is estimated that a depressed longitudinal arch occurs in about 23 percent of the adult population.[17] Of this population, approximately two-thirds have the flexible, hypermobile flatfoot with normal or increased mobility of the subtalar complex and ankle joint. Approximately one-fourth of flatfeet exhibit a contracture of the triceps surae associated with an otherwise typical hypermobile flatfoot, and this form of flatfoot is a known cause of disability in army recruits.[17,18] The remainder of flatfeet are characterized by rigidity of the subtalar joint, typically seen with tarsal coalitions.

The height of the longitudinal arch is determined by bony structure and ligamentous laxity. Electromyographic (EMG) studies have documented that neither the intrinsic nor the extrinsic muscles of the foot have electrical activity during standing at rest.[5] Therefore, the static structure of the longitudinal arch is independent of any muscular activity.

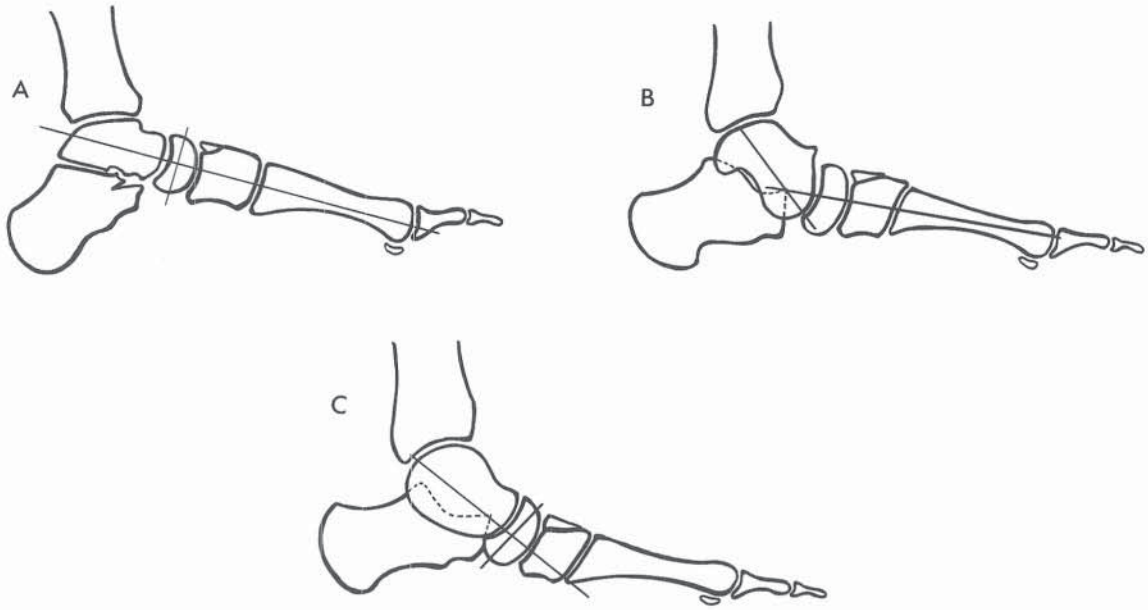


FIGURE 22–26 Measurement of Méary's angle on the lateral weightbearing radiograph. **A,** A line drawn through the longitudinal axis of the talus and first metatarsal is essentially straight on the normal foot. **B,** In talonavicular sag, the apex is plantarward, with the talar longitudinal axis intersecting only the most inferior tip of the navicular (due to its dorsal subluxation). **C,** In naviculocuneiform sag, the apex of the angle is still plantar, but the navicular remains located centrally on the head of the talus.

Obviously, with walking or more vigorous activity, both sets of muscles are active, suggesting dynamic stabilization of the arch. However, flatfoot is the well-known sequela of a lacerated or insufficient tibialis posterior tendon, suggesting that there must be some contribution to the static integrity of the arch from this muscle, since its absence results in the deformity.

The excessive laxity of the ligamentous structures is usually appreciated during the clinical examination. In the typical flexible flatfoot, the longitudinal arch is reconstituted when the foot is in a nonweightbearing position. The longitudinal arch is also reconstituted during active plantar flexion muscle activity, such as when the patient is asked to stand on tiptoes and the heels are viewed from behind (Fig. 22–28). The inversion of the heels and the arch reconstitution are among the findings required to make a diagnosis of flexible flatfoot. Because the arch reconstitutes during such active muscle function, it is tempting to prescribe mus-

A

B

FIGURE 22–27 Flatfoot. **A,** Standing lateral radiograph. Talonavicular sag and relative plantar flexion of the talus suggest an Achilles tendon contracture. **B,** Standing AP radiographs showing marked hindfoot and midfoot eversion and valgus as suggested by the divergent talocalcaneal axes and the lateral displacement of the navicular on the talar head.

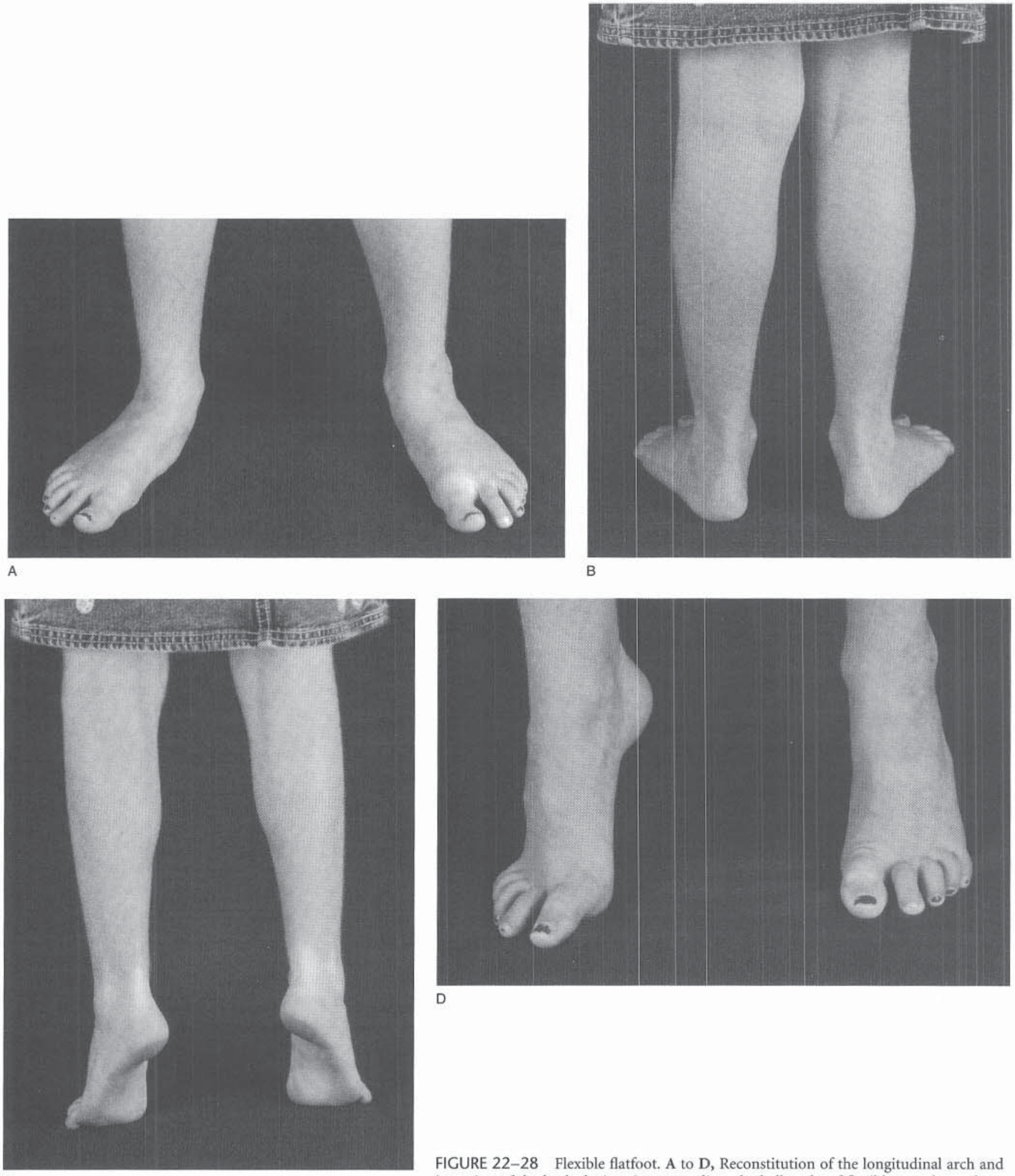


FIGURE 22–28 Flexible flatfoot. **A** to **D**, Reconstitution of the longitudinal arch and inversion of the heels during tiptoe standing, the hallmarks of flexible pes planovalgus.

cle strengthening exercises of the plantar flexors and invertors in a patient with a planovalgus deformity. Exercises such as grasping marbles or small balls with the toes of the foot, traditional therapy for the flatfooted child, is based on such a concept. Although Tachdjian in the second edition of this textbook denigrated such exercises,[41] there is no scientific study evaluating their effectiveness (or lack of it), and there may be some benefit for the child who is ligamentously lax and exhibits relative "hypotonia" in general somatotype.

Should flexibility of the hindfoot and arch reconstitution *not* be demonstrated on the tiptoe test, other conditions must be considered, especially if there is a pain complaint. The general neurologic assessment—observation of gait, coordination, and reflexes—will uncover neurologic or myopathic conditions associated with flatfeet in which the foot position may be due to weakness (poliomyelitis, peripheral neuropathy), weakness with Achilles tendon contracture (Duchenne's muscular dystrophy), or spasticity with equinus (cerebral palsy). The lack of hindfoot motion, especially if painful or rigidly resisted, suggests tarsal coalition or inflammatory arthritis, and a rigid rocker-bottom deformity with equinus suggests a congenital vertical talus. Specific areas of pain, such as the navicular, may point to an accessory navicular or osteochondritis. Radiographs or consultation with a neurologist or rheumatologist may be appropriate if an idiopathic, hypermobile flatfoot is ruled out by the history and physical examination.

Particular attention to the Achilles tendon is important, as a contracture tends to make hypermobile flatfeet symptomatic.[18] After tiptoeing to confirm subtalar flexibility, the child should be asked to walk on the heels. An Achilles tendon contracture will make this activity difficult. Passive dorsiflexion of the foot, with the heel locked in varus (inverted), will further demonstrate this contracture. Patients with such a contracture will divulge during the history taking that they have had progressive arch pain medially, sometimes with callus development and medial shoe breakdown (see Fig. 22–25). As with any lower extremity deformity, joint range of motion and torsional profile must be evaluated to assess more proximal causes that may have encouraged the development of the flatfoot.

Natural History. The arch is usually obscured in the infant's foot due to the subcutaneous fat, and spontaneous resolution of "fat foot" can be anticipated as the toddler and young child mature and such fat atrophies. Both footprint[27,40] and radiographic[43] studies of the child's foot demonstrate that the longitudinal arch develops during the first decade of life. As already discussed, measurement of the lateral talo-first metatarsal angle demonstrates a decrease in the amount of plantar sag of the midfoot from the ages of 0 to 8 years.[43] Such improvement in the sag of the medial ray of the foot would suggest that ligamentous laxity in the toddler spontaneously resolves, with the ligaments becoming more taut. This observation also leads to the overwhelming conclusion that prophylactic treatment for the typical flatfoot is unnecessary, with profound implications for the corrective shoe and insert-orthosis industry. The development of the arch is independent of the use of such external orthoses or the wearing of corrective shoes, and studies from countries where shoes are not worn at all tend to substantiate the opinion that children with flatfeet do not develop symptomatic deformities with aging.[31] The classic study of 3,600 army recruits by Harris and Beath documented that the presence or absence of the longitudinal arch did not correlate with disability and that a flatfoot was compatible with normal function unless an Achilles tendon contracture was present.[17,18] There has even been a suggestion that shoes may be detrimental to the development of the longitudinal arch.[33,37] Indeed, controlled prospective randomized studies on the effect of shoe modifications and inserts on the development of the arch have failed to demonstrate any effect.[16,46] The use of such devices for correction of such a "deformity" must be considered ineffective and probably unnecessary. It is therefore the orthopaedist's main function to educate parents seeking treatment of their child's flatfeet that the condition is both benign and unaffected by prophylactic treatment with such devices.

The development of bunions has also been related to the presence of a flatfoot. However, in Kilmartin and Wallace's longitudinal study of children with flatfeet, bunion development was independent of the longitudinal arch.[19] More significant in the discussion of shoe wear and foot deformities is the tendency for children wearing closed-toe or "fashionable" shoes to actually have arch development inhibited while placing a valgus stress at the first metatarsophalangeal (MTP) joint.

Treatment. In the typical case of hypermobile (postural) flatfoot, no treatment is indicated in the asymptomatic pediatric patient. Education and reassurance are the mainstays of "treatment."

Orthopaedic shoes, including various heel modifications, molded heel cups and other orthoses, and medial arch supports, have traditionally been prescribed, though there is no scientific evidence that such modifications are efficacious. Although some studies claim that such methods restore a normal longitudinal arch and reduce pathologic pressure on the weightbearing area of the longitudinal arch in a flexible flatfoot by introducing a medial heel wedge,[3,6,7] it is worth repeating that the only controlled studies of the effect of corrective shoes or foot orthotics failed to demonstrate any influence on the development or restoration of the longitudinal arch.[16,46] With the evidence that flatfooted army recruits and children who do not wear shoes have essentially normal function as adults, and with the lack of objective studies demonstrating a lasting change in the radiographic anatomy of the foot with the use of corrective devices, there is no medical indication for the treatment of asymptomatic flatfeet. In light of the not insubstantial cost of some of the custom-molded inserts and orthoses, there is little justification for prescribing such devices, and the tradition of prescribing special shoes or inserts for the orthopaedic management of the child's foot should be abandoned.

If Achilles tendon contracture is present, then stretching exercises—both manually by the parents and actively by the child, if old enough to cooperate—are an appropriate form of management. These children may have symptomatic callosities under the head of the plantar-flexed talus associated with the Achilles tendon contracture. Emphasis should be placed on stretching the heel cord with the heel inverted and the knee straight, and in the case of the older child, exercises using an elastic theraband and dorsiflexion stretching with the heel maintained on the ground (with the patient leaning forward with the hands supported on the wall) are recommended (Fig. 22–29). If the heel valgus is excessive and the patient has ligamentous laxity as determined by examination of other joints,[47] strengthening of the tibialis posterior by such methods as toe-walking and performing multiple repetitions of toe-ups can be recommended. Such exercises may help minimize medial plantar "strain" associated with the pressure on the talar head.

In symptomatic patients, arch supports and orthoses may be of benefit. Typical symptoms include medial arch pain and fatigue, and cramping at night. In addition to stretching and muscle-strengthening exercises, we have found that the footwear sold in sporting goods stores, especially that designed for running, is often more readily accepted for social reasons by children and adolescents than the more traditional devices placed inside shoes. Running shoes designed for the "hyperpronated" foot have significant heel and arch support built into the shoe itself, making the prescription of additional orthotics superfluous. Since running shoes usually support the relaxed portion of the arch or hindfoot, the suggestion to use such footwear may be all that is necessary to resolve the problem.

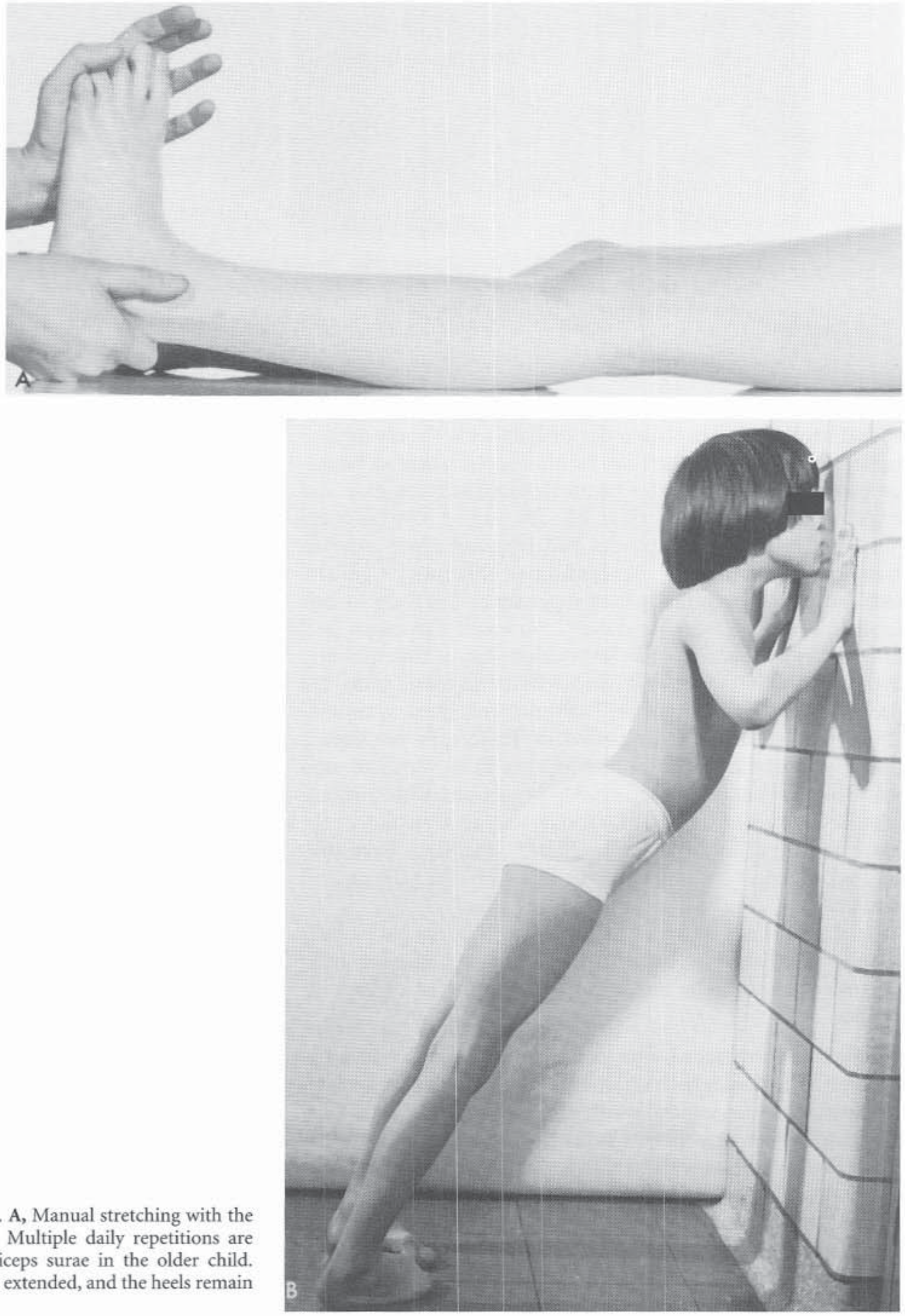


FIGURE 22–29 Exercises to treat flatfoot. **A,** Manual stretching with the knee extended and the hindfoot inverted. Multiple daily repetitions are prescribed. **B,** Passive stretching of the triceps surae in the older child. Note that the feet are inverted, the knees are extended, and the heels remain on the floor.

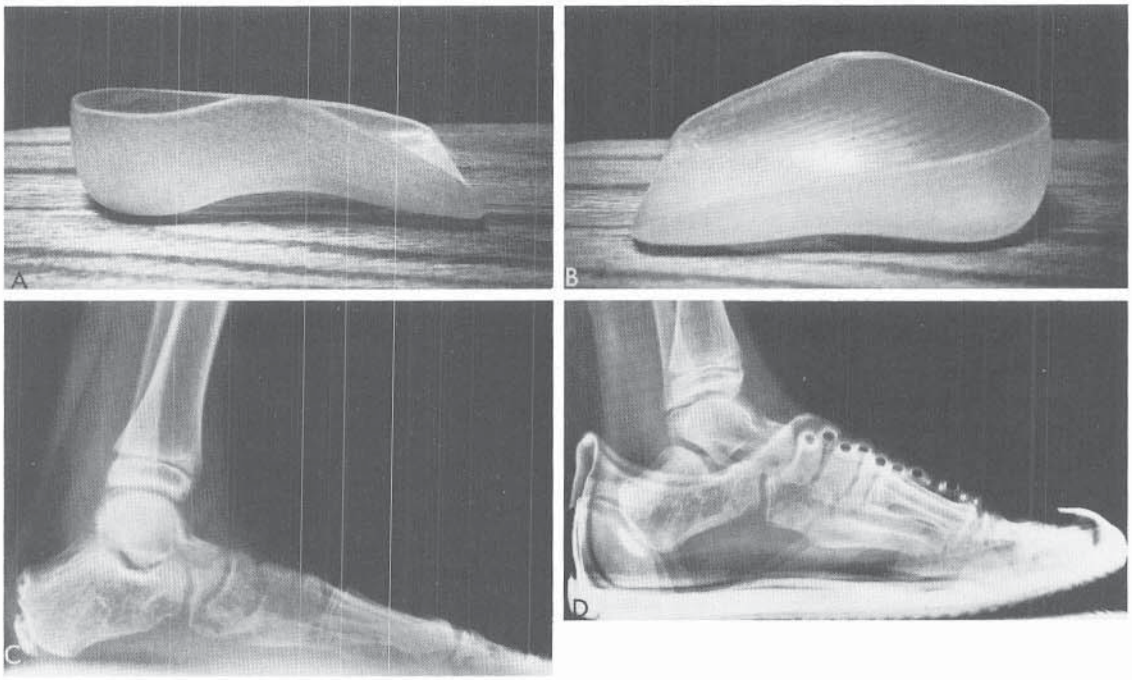


FIGURE 22–30 **A** and **B,** The UCBL orthosis, used in the treatment of flatfoot. **C,** Standing lateral radiograph showing naviculocuneiform sag. **D,** Standing lateral radiograph with UCBL orthosis. Naviculocuneiform sag and calcaneal dorsiflexion ("pitch") are improved.

In more recalcitrant cases, formal orthotic management with devices such as a University of California Biomechanics Laboratory (UCBL) insert can be attempted. Such an orthosis can acutely change the talonaviculocuneiform axis and improve calcaneal pitch (Fig. 22–30), and it has been reported to alleviate symptoms and improve shoe wear in symptomatic patients.[6,25] Acceptance of this more rigid device—the orthosis is made from a plaster cast of the patient's foot, and molded from rigid plastic to invert the valgus heel and support the arch—may be problematic, in that the rigid orthotic can be somewhat uncomfortable, similar to the proverbial "rock in the shoe," and because there is no evidence that it has any lasting effect on flatfeet, its use should not be pursued if an initial prescription fails. We have used soft inserts (Plastizote; Fig. 22–31) in symptomatic patients who have rejected the UCBL-type device, with better acceptance and probably the same efficacy, and thus have prescribed the rigid formal orthosis less over the past several years.

It should once again be emphasized that no permanent change in either foot anatomy or arch structure has been documented with the use of any orthosis or shoe modification.

Surgical Management. Surgical management of a true hypermobile flatfoot is reserved for the patient who has intractable symptoms unresponsive to shoe or orthotic modifications and who is unable to modify the activities that produce pain. Thus, patients with talonavicular callosities and medial arch "strain" whose daily activities are limited by foot pain are the only true candidates for surgical management. Although surgery can alter the shape of the arch by reconstructing it with either soft tissue imbrication or bony fusion procedures, with generally good short-term results, long-term evidence of continued foot health after such procedures is generally lacking.

Indeed, *unsatisfactory* results from surgery have been reported in 49 percent to 77 percent of longer-term stud-

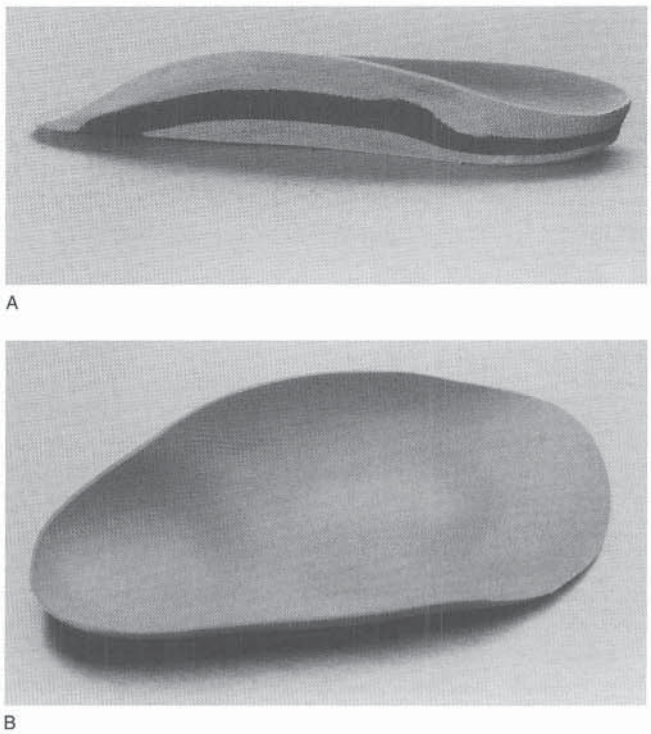


FIGURE 22–31 **A** and **B,** Soft molded insert with arch support and medial heel "wedge" to invert the hindfoot.

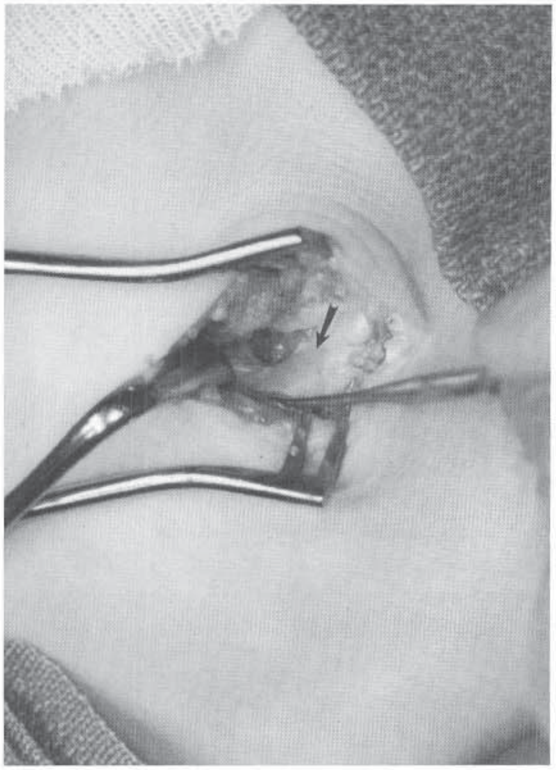


FIGURE 22–32 Intraoperative view of a STA-peg implant (*arrow*), which was removed because of unrelenting subtalar pain. Note the synovitis associated with the device.

ies,[8,11,36] and all longer-term follow-up studies of limited tarsal fusion procedures have described osteoarthritic changes at adjacent unfused joints.* Because such changes are known to occur de novo in untreated tarsal coalitions, it should come as no surprise that the creation of an iatrogenic coalition in the reconstruction of the longitudinal arch by tarsal bone fusion, however limited, produces the same degenerative changes at the adjacent joints. Thus, surgical correction of flatfoot must emphasize joint-*sparing* procedures, usually combining extra-articular osteotomy with soft tissue imbrication.

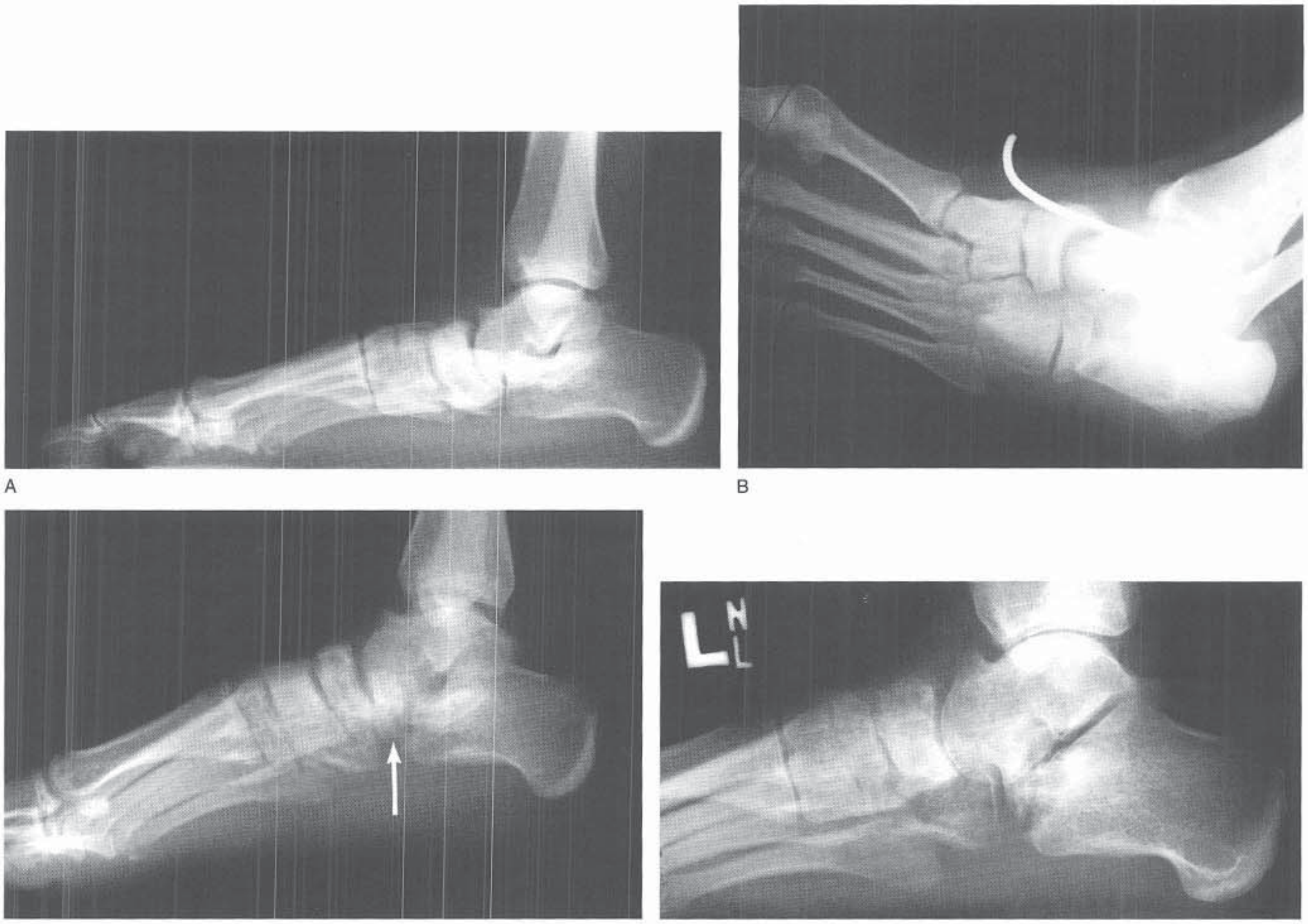
Arthroereisis of the subtalar joint, using a silicone or Silastic implant, has been reported as an alternative to more complex joint reconstruction. The rationale of the procedure is to limit the amount of valgus motion in the subtalar joint using an interposition peg. Long-term results of this procedure are lacking, and because of potential complications of intra-articular placement of Silastic material, especially in the normal cartilaginous surfaces of a child's hindfoot, this procedure is not warranted, given that the natural history of a flexible flatfoot is generally benign.[1,22,42,44] Nevertheless, the use of silicone or Silastic material interposed in the subtalar joint is common in the podiatric literature.[1,23,38,42] The potential for synovitis, necessitating implant removal, is real (Fig. 22–32).

An Achilles tendon contracture should always be considered and addressed in any surgical treatment of a flatfoot. If the patient has severe enough symptoms to warrant surgery, then heel cord lengthening should be part of a comprehensive procedure to reconstruct the longitudinal arch. The traditional surgical procedures for flatfeet include "limited" arthrodesis of midfoot joints, the subtalar joint alone, triple arthrodesis, or the more physiologic osteotomies to realign the foot and not sacrifice joint motion, such as lateral column lengthening alone or in combination with medial soft tissue imbrication.

Subtalar fusion as a primary procedure for hypermobile flatfoot should probably be condemned. Although there is no question that the excessive heel valgus can be corrected, which in turn will restore the longitudinal arch in the typical relaxed flatfoot, the sacrifice of subtalar motion for this purpose is not indicated. The mechanics of the hindfoot are completely altered by subtalar fusion, and the mobility of the remaining midfoot joints—talonavicular, calcaneocuboid, indeed of the entire midfoot complex—is irretrievably altered by subtalar fusion. The more extensive triple arthrodesis eliminates all hindfoot mobility, and thus, while deformity is effectively corrected by such a procedure, it is again only indicated as a salvage procedure in a foot in which other surgical procedures have failed and after selective joint injection under local anesthesia has demonstrated that it is the mobility of the hindfoot-midfoot complex that is producing the pain and disability (Fig. 22–33).

Lateral column lengthening by insertion of a bone graft into an osteotomy of the calcaneal neck is currently the most attractive procedure to correct the deformity and not sacrifice joint motion.[14,28,34] The lateral column is lengthened by inserting a tricortical iliac crest graft between the anterior and middle facets of the calcaneus (Fig. 22–34). A transverse osteotomy of the neck of the calcaneus, approximately 1 to $1\frac{1}{2}$ cm proximal to the calcaneocuboid joint, is gently spread apart to receive a graft of the same length. The technique of spreading the osteotomy is crucial to the success of the procedure, as forceful spreading with a lamina spreader, for example, can crush the cortical edges of the two sides of the osteotomy, making it difficult to prop the osteotomy open. One technique is to use threaded Steinmann pins in each osteotomy segment placed transversely so that the osteotomy can be opened using the pins as a handle. This has the advantage of not obstructing the osteotomy site during the insertion of the graft. Once the graft has been properly impacted into place, additional internal fixation using screws, pins, or a staple can be selected if the osteotomy is not stable or if there is dorsal displacement of the distal calcaneal fragment producing calcaneocuboid subluxation (Fig. 22–33).[28] Usually soft tissue tensioning will hold the graft in place, but if calcaneocuboid subluxation occurs, longitudinal pinning prior to graft insertion will aid in avoiding this complication, although no disability has been recognized in patients who healed with this malunion.[28] The foot is usually maintained in plaster for 8 to 12 weeks to ensure healing of the opening-wedge osteotomy. Results of calcaneal lengthening have been considered satisfactory when there is relief of medial arch pain and callosities, correction of heel valgus and limited dorsiflexion, improvement in the appearance of the arch, radiographic restoration of Meary's angle and the lateral talocalcaneal angle, and maintenance of subtalar motion. This outcome is achieved in more than 90 percent of patients in short-term reviews,[20,28] and in 75 percent the satisfactory outcome is maintained

* See references 2, 4, 8, 11, 12, 32, 35, 36, 39.



A B C D

FIGURE 22–33 Painful flatfoot in a 17-year-old girl. **A,** Preoperative lateral radiograph showing naviculocuneiform sag primarily. An osteophyte on the talar neck suggests early arthritic changes. **B,** Intraoperative radiograph obtained during calcaneal lengthening and medial imbrication procedure. No true lateral radiograph was obtained. **C,** Dorsal displacement of the distal calcaneal fragment with subluxation of the calcaneocuboid joint was diagnosed at 8 weeks postoperatively. Note correction of the talonaviculo-cuneiform axis. **D,** At 6 months postoperatively, calcaneal nonunion was evident. A triple arthrodesis was eventually performed for pain.

without evidence of degenerative changes in longer-term follow-up.[30]

Imbrication of the talonaviculocuneiform complex medially can be performed in combination with calcaneal lengthening. It is not recommended as a single procedure alone, owing to progressive stretching of the medial repair with weightbearing, especially when the lateral column has not been addressed. The technique has evolved from limited fusions of the talonavicular and naviculocuneiform joints with tendon imbrication[26] to "tightening" of the naviculocuneiform joint[15] with plantar imbrication, to opening-wedge osteotomy of the cuneiform to recreate the arch.[24] Our technique involves an initial detachment and later imbrication of the tibialis posterior tendon, and the raising of an osteoperiosteal flap of the cuneiform-navicular capsules by sharply dissecting a tongue of the medial capsules from proximal talonavicular to distal, leaving the flap attached at the cuneiform (Fig. 22–35).[9,10] The talonaviculocuneiform alignment is corrected (usually after the lateral column lengthening), and the osteoperiosteal flap is advanced proximally and plantarward and reattached to the talar neck with heavy suture. We usually protect the medial reconstruction with a smooth K-wire that is removed at the time of cast removal (see Fig. 22–33B). As mentioned, the tibialis posterior should be shortened and advanced to restore appropriate tension following the medial column "shortening" by soft tissue imbrication. Postoperative care for the soft tissue usually requires an additional 4 to 6 weeks of casting to allow complete healing of the repair. Although excellent results have been reported with this medial reconstruction alone,[9,10] we continue to use it only in conjunction with a calcaneal osteotomy.

Along these same lines, medial imbrication can also be combined with sliding calcaneal osteotomy.[13,21,45] Although one may argue that displacement of the posterior half of the calcaneus medially, to reestablish the weightbearing line in the center of the ankle-subtalar coronal plane, merely creates a compensatory varusization for talocalcaneal valgus,[29] the effect of such a shift seems to be to help support the plantar-flexed talus and decrease overall eversion and midfoot abduction (Fig. 22–36). Thus, when combined with the medial column shortening-imbrication, good results are achieved (Fig. 22–37). The need for Achilles tendon lengthening, peroneal lengthening, and corrective metatarsal or

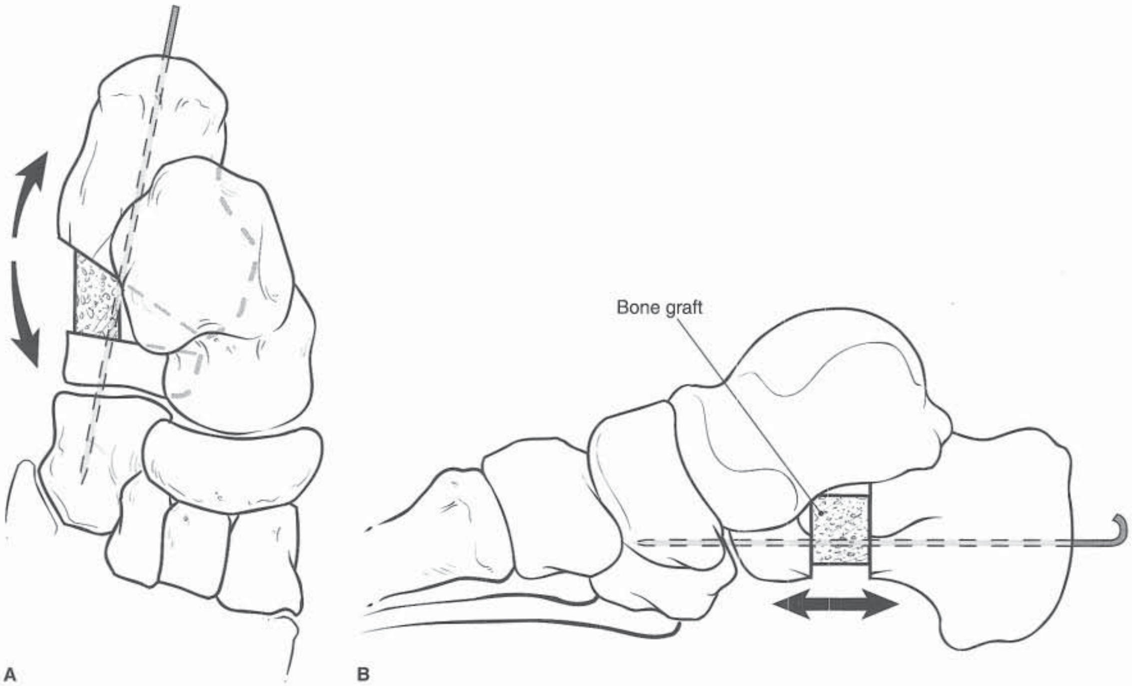


FIGURE 22–34 **A** and **B,** Lateral column lengthening to treat flatfoot. K-wire fixation can be useful to prevent displacement of both the graft and the distal osteotomy fragment.

medial cuneiform osteotomy if forefoot supination is excessive following calcaneal osteotomy-medial imbrication must be assessed during the combined procedures.[45]

Complications of the combined procedure include nonunion of the calcaneal graft, displacement of the graft, requiring revision, displacement of the calcaneocuboid joint (see Fig. 22–33), and recurrence of the deformity or pain with time and continued weightbearing. Nonunion may be particularly difficult to address because it is usually accompanied by calcaneocuboid joint degeneration, producing pain requiring a triple arthrodesis. It goes without saying that such a salvage procedure is an extremely untoward outcome for a "joint-sparing" procedure, and thus attention to the sizing and stability of the graft, to avoid nonunion, cannot be overemphasized.

In summary, hypermobile flatfoot is a normal variant of foot structure and does not require prophylactic treatment. Should an Achilles tendon contracture be present, it should be stretched vigorously, because of the possibility that symptoms might arise later. Nonoperative management of painful flatfeet in adolescents is usually successful and entails shoe modifications (running shoes suffice for this purpose), orthotics, and stretching and strengthening exercises. Surgical correction, truly a last resort for this normal variant, should emphasize joint-sparing procedures, including lateral column lengthening or a calcaneal medial-slide osteotomy, often combined with medial soft tissue imbrication, to provide symptomatic relief by realigning the subluxated talonaviculocuneiform complex.

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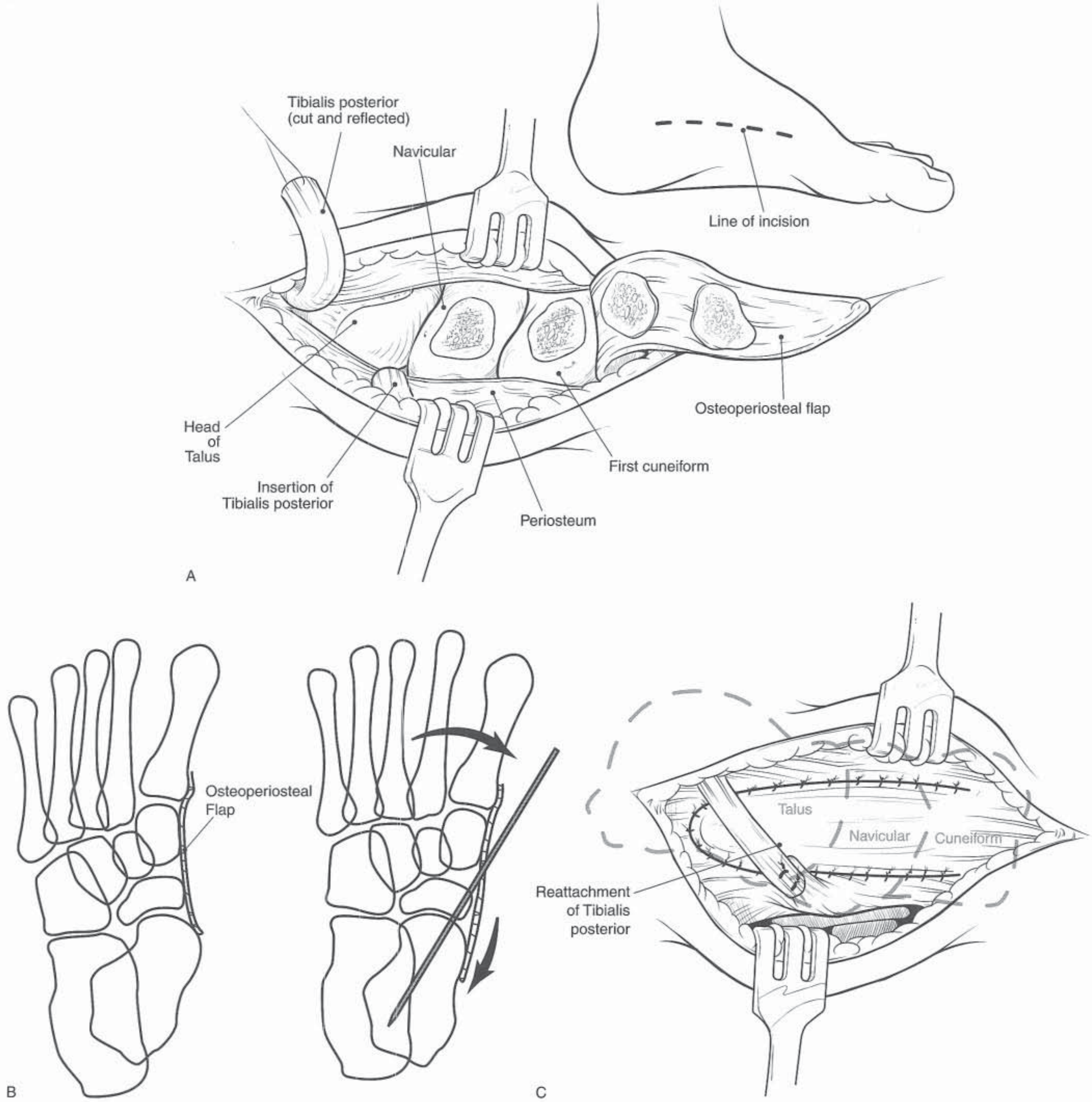

FIGURE 22–35 Medial imbrication of the talonavicular-cuneiform joints in the surgical treatment of flatfoot. **A,** The tibialis posterior is divided (for later imbrication). The osteoperiosteal flap is raised from proximal to distal. **B,** After reduction of the talonavicular displacement by translating the navicular medially and plantarward, the osteoperiosteal flap is advanced proximally. Internal fixation is recommended. **C,** The tibialis posterior is shortened/imbricated after proximal reattachment of the osteoperiosteal flap.

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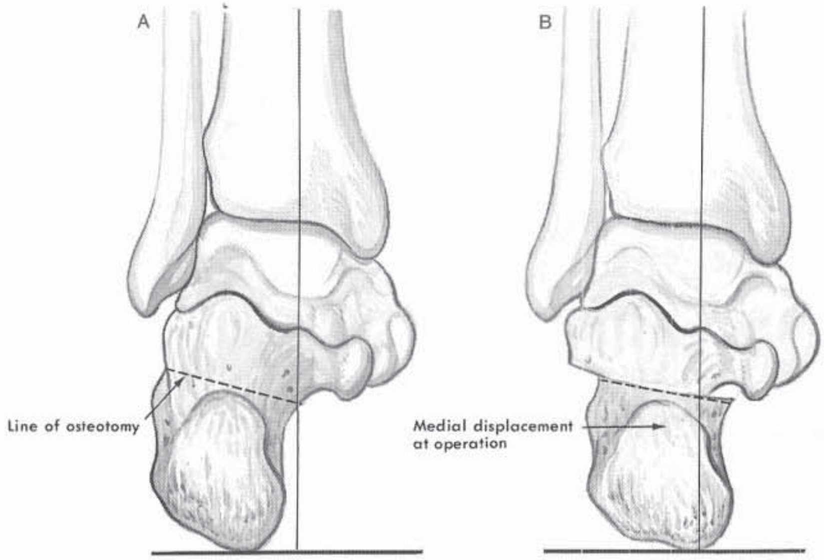

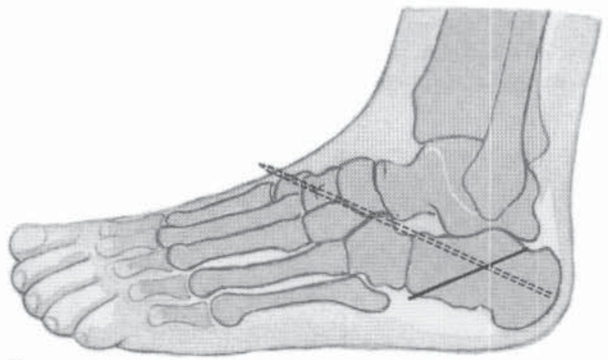

FIGURE 22–36 Medial displacement osteotomy of the calcaneus in severe pes planovalgus. The weightbearing line and relation of the talus to the calcaneus are seen from the posterosuperior aspect. **A,** The axis of weight transmission passes medial to the calcaneus. The line of the osteotomy parallels the subtalar joint. **B,** Appearance after medial displacement osteotomy of the calcaneus. The medial margin of the distal calcaneal fragment is placed in line with the sustentaculum tali, and a normal weightbearing axis is restored. **C,** Threaded Steinmann pin transfixing the osteotomized calcaneus.

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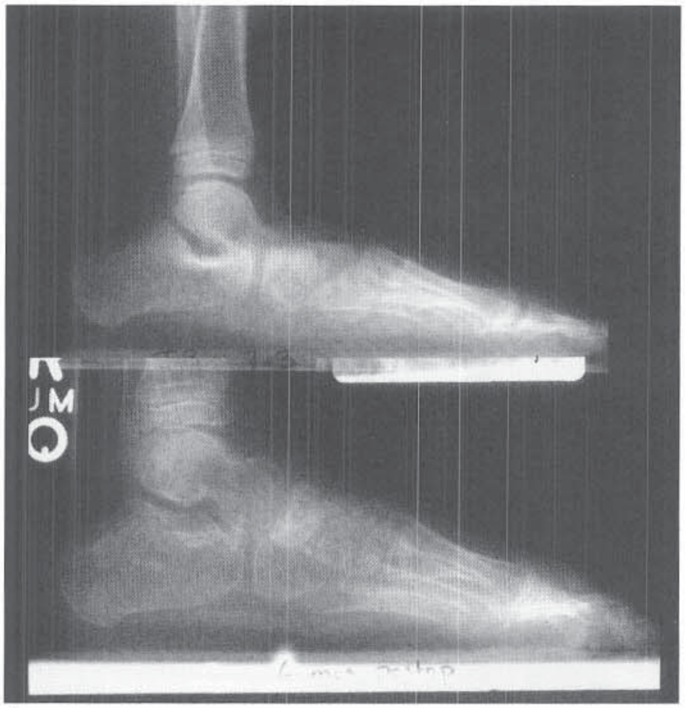
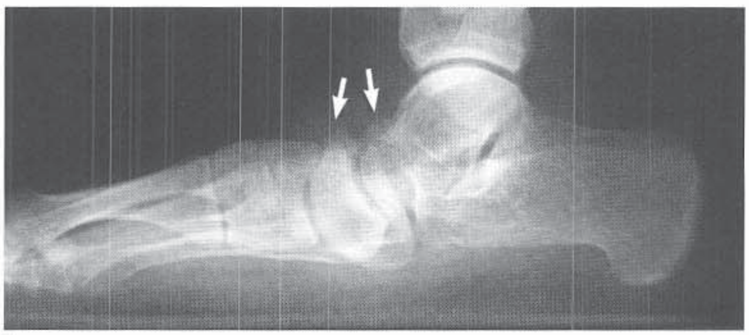


A

B



C

D

FIGURE 22–37 Result of medial imbrication/calcaneal sliding osteotomy. **A,** Lateral radiographs of both feet of the patient shown in Figure 22–27, 6 months after correction of the right foot (*bottom*). The talonaviculocuneiform axis is normally aligned. **B,** Lateral radiograph of the right foot obtained at 10-year follow-up. The correction is maintained and the foot is asymptomatic. **C** and **D,** The left foot of the same patient, which was not operated on. Note the arthritic changes in the talonavicular joint. This foot was only mildly symptomatic and was never treated surgically, although the degenerative changes provoke some concern.

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SKEWFOOT

Skewfoot is a condition that resembles metatarsus adductus but in which the elements of adductus of the forefoot and valgus of the hindfoot are more severe and rigid. In addition, there is lateral subluxation of the navicular on the talus. Skewfoot is also called the S-shaped foot, the serpentine foot, or the Z-foot (Fig. 22–38). A similar deformity is sometimes seen in a clubfoot that has had inadequate mid-foot release and excessive hindfoot release. Unfortunately, there are no criteria that establish how much metatarsus adductus is necessary to reclassify flatfoot as skewfoot, or how much hindfoot valgus is required to reclassify metatarsus adductus as skewfoot.[14] This lack of defining criteria often makes the diagnosis subjective.

By all reports, skewfoot is a rare deformity.[3,8,9,16] The first accurate description in the American literature was published in 1933.[15] Its rarity is underscored by Peterson, who was able to find only 50 cases in the literature,[16] and Kite,

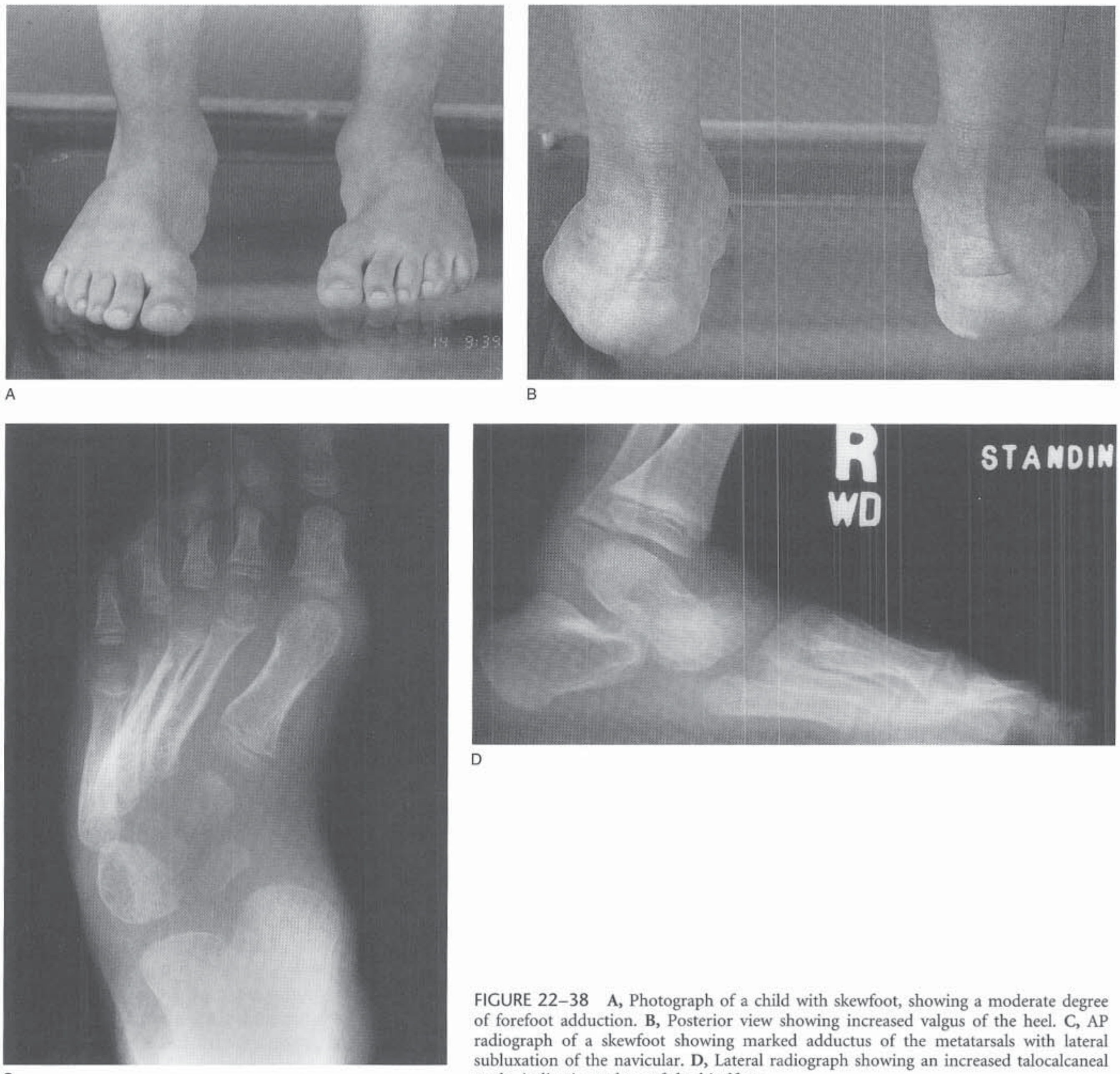

FIGURE 22–38 **A,** Photograph of a child with skewfoot, showing a moderate degree of forefoot adduction. **B,** Posterior view showing increased valgus of the heel. **C,** AP radiograph of a skewfoot showing marked adductus of the metatarsals with lateral subluxation of the navicular. **D,** Lateral radiograph showing an increased talocalcaneal angle, indicating valgus of the hindfoot.

who found only 12 skewfeet out of 2,818 cases of forefoot adduction.[8]

The natural history of skewfoot is unknown. In some patients the deformity undoubtedly resolves, just as metatarsus adductus and flexible pes planovalgus resolve. On the other hand, with more rigid feet, callosities on either side of the foot (talar head, cuboid-fifth metatarsal) may become symptomatic at around age 10[7,9,11,14,16] or after.

Radiographic Findings. Radiographically, there is an increase in the talocalcaneal angles in both the AP and lateral projections,[6] along with a lateral and dorsal subluxation of the navicular on the talus. On the AP radiograph, a line drawn along the first metatarsal through the navicular, then to the head of the talus and through the body of the talus, makes a Z shape. Similarly, on the lateral radiograph, there is dorsiflexion of the talonavicular joint (probably secondary to increased talar plantar flexion) and plantar flexion of the tarsometatarsal joints, another Z deformity.

Treatment. Because of the lack of criteria for definition of the deformity and its relative severity, early treatment of skewfoot often is based on a perception of need to treat the metatarsus adductus. Thus, a more severe adductus that is not passively correctable and had rigidity can be treated by stretching and serial casting. The cast technique must carefully mold the hindfoot into varus, to avoid exacerbating the existing valgus while correcting the forefoot.[1,8] Since the hindfoot valgus cannot be corrected by casting, this treatment basically converts skewfoot into flatfoot, a not unreasonable outcome, since flatfeet are generally less symptomatic and responsive to nonoperative treatment, if necessary, in the older child. Reverse-last shoes and the Denis Browne bar are probably contraindicated in true skewfoot, as they can exacerbate hindfoot valgus.

In older children (second decade), symptomatic skewfeet usually have an Achilles contracture, not unlike the symptomatic flatfoot. Stretching and orthoses can be tried, but because of the more inflexible deformities (compared to pes planovalgus), these approaches are usually unsuccessful. Thus, operative treatment, ideally the last resort, is often resorted to for lack of a useful alternative.

Earlier reports of extensive soft tissue releases (tarsometatarsal capsulotomies) combined with subtalar fusion[16] or calcaneal osteotomy[2] included only small numbers of patients, and because of recurrence and degenerative arthritis arising from the joint-damaging procedures,[17,18] they have fallen into disfavor. Mosca has proposed Evans's calcaneal lengthening combined with a Fowler-type opening-wedge osteotomy of the medial cuneiform and Achilles tendon lengthening, to avoid completely any joint incursion and correct all the different deformities.[4,5,10,12–14] With this combination, nine of ten feet were well corrected, with joint motion preserved. This approach would appear to be procedure of choice in the skewfoot requiring surgical correction.

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Skewfoot

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CONGENITAL TALIPES EQUINOVARUS (CLUBFOOT)

The deformity known as clubfoot is probably the most common (1 to 2 in 1,000 live births) congenital orthopaedic condition requiring intensive treatment. It most likely represents a congenital dysplasia of all musculoskeletal tissues (musculotendinous, ligamentous, osteoarticular, and neurovascular structures) distal to the knee. This conclusion is based on multiple investigators' observations of a myriad of different abnormal anatomic findings and on the functional outcome of patients believed to have had optimal nonoperative or operative treatment and who nevertheless always had subsequently some degree of impairment. Evans has pointed out that as E. H. Bradford so prophetically noted in 1889,[26] the treatment of clubfoot is often described in "glowing terms," with very satisfactory results in the short term, but in practice recurrent or persistent deformity is common, having defied correction and ultimately producing a less than good result. The so-called satisfactory or "good" result, when studied *objectively*,[61] presents a more contradictory picture, as the affected foot invariably has restricted motion, particularly in the ankle; diminished muscle strength and power generation of the triceps surae, possibly secondary to the ankle dysfunction or to the aforementioned primary dysplasia affecting all tissues of the lower leg; and kinetic and kinematic abnormalities (recurvatum, valgus, and quadriceps and hamstring weakness) of the ipsilateral knee and thigh, which probably predispose to degenerative arthritis. Whereas neonatal developmental dysplasia of the hip, when recognized and treated appropriately and early, usually resolves completely, resulting in a normal hip, such is not the case for the neonatal clubfoot treated appropriately and early, for a completely normal extremity and foot are essentially precluded by the underlying congenital dysplasia.

Etiology. Clubfoot has long been associated with neuromuscular diseases and syndromes, and therefore an underlying neuromuscular or syndromic/dysmorphic etiology for all "idiopathic" clubfeet has always been suspected. In the second edition of this book, Tachdjian listed arthrogryposis, diastrophic dysplasia, Streeter's dysplasia (constriction band syndrome), Freeman-Sheldon syndrome, Möbius' syndrome, and other conditions resulting from chromosomal deletions as just a few of the more recognizable systemic conditions with associated clubfeet.[99] On the other hand, idiopathic clubfoot commonly presents as a single musculoskeletal deformity in an otherwise normal infant. Since the final outcome in this latter situation often is diminished function,[5,61] the conclusion that idiopathic clubfoot represents a primary but *local* dysplasia of all tissues of the affected extremity from the knee down is supported by the historical inability of treatment to completely reverse this congenital dysplasia and produce a normal foot and extremity. That is not to say that current treatment modalities, including surgery, cannot produce a functional foot and extremity that serve the patient well. It simply recognizes that treatment of true idiopathic clubfoot can never produce a *normal* extremity.

Numerous theories on the etiology of congenital clubfoot have been proposed, including an arrest in embryonic development.[9] In normal fetal development of the lower limb, the foot in the 6- to 8-week-old fetus has many characteristics of a congenital clubfoot, including equinus, supination, and forefoot adduction and medial deviation of the talar neck.[62] Bohm proposed that an arrest of fetal development at this stage was responsible for the clinical deformities seen at birth.[9] Normally the supinated, adducted, and equinus position seen in the 8-week embryo gradually corrects with continued development, and the fetal foot becomes normal at 12 to 14 weeks.[62] If this theory is accepted, then the true congenital clubfoot has already existed for approximately 7 months in utero by the time the full-term infant is actually born. However, the characteristic dysmorphic talar head and the medial dislocation of the navicular have never been observed at any stage of normal fetal development.[13,60,71,103] Thus an arrest of normal fetal development fails to account for this primary dysplasia.

The innate stiffness of clubfeet was clarified by Zimny and colleagues, who identified myofibroblastic *retractile* tissue in the medial ligaments.[111] This finding confirmed earlier studies by Ippolito and Ponseti, who identified an increase in collagen fibers and fibroblastic cells in the ligaments and tendons of the clubfoot.[52] Thus a second hypothesis about the etiology of clubfoot proposes a retractive fibrotic response, not unlike Dupuytren's contracture, as a primary factor. This hypothesis is supported by studies demonstrating abnormal ligamentous and fascial restraints in "soft" tissues that inherently resist deformity correction.[28,88] These histopathologic findings help explain the maintenance of a clubfoot deformity and resistance to correction, if not the actual cause. However, the association of clubfeet with syndromes of inherent ligamentous laxity (Down, Larsen's) confounds the hypothesis that fibrotic retractile tissue is a primary etiology.

Studies of stillborn and fetal clubfeet have led some authors to propose a primary germ plasm defect in the cartilaginous talar anlage, producing the dysmorphic neck and navicular subluxation.[10,53,92,96] Such a proposal is consistent with the observation that the dysmorphic talar head and navicular position are *not* seen in normal embryonic development and so must be present from initial limb bud differentiation in an affected extremity. The association of clubfoot with various neurologic entities is well known, with some of the most severe clubfeet being associated with paralytic disorders such as arthrogryposis and spina bifida. Not surprisingly, a theory postulating localized neuromyogenic imbalance, especially involving the peroneals, has been proposed.[39,55] Congenital fiber-type disproportion, with an imbalance between type I and II muscle fibers and atrophy of type I fibers, has been found in both peroneal and triceps surae histopathologic specimens.[40,67,79] A peroneal muscle deficit would certainly allow any medial contracture, fibrosis, or cartilaginous dysplasia to worsen and would also explain a high incidence of recurrent deformity if the imbalance cannot be corrected.[67]

It is safe to conclude that the etiology of idiopathic clubfoot is multifactorial and is modulated significantly by developmental aberrations *early* in limb bud development. Investigation of the genetic sequences of early embryonic limb development will eventually yield a more unified etiologic picture as well as possible new therapeutic avenues in interrupting or correcting these aberrations.

Pathoanatomy. Descriptions of the pathologic anatomy in clubfoot can be found in some of the earliest orthopaedic writings and continue to be essentially correct today, even as we have more sophisticated methods of imaging to quantitate that deformity. Scarpa in 1803 reported the medial and plantar displacement of the navicular, cuboid, and calcaneus around the talus. Displacement of the navicular and calcaneus produces an inverted or varus hindfoot, and the entire complex rests in equinus. Contracture of the soft tissue (ligaments, joint capsules, and tendons) maintains this pathologic malalignment of joints, described as equinovarus.[82] Multiple subsequent authors have added to the body of knowledge, describing deformities that can be separated into *intra*osseous deformities, or deformities within the bone itself, and *inter*osseous deformities, or deformities resulting from the relationship of one bone to another. Scarpa, Adams, in 1866, and Elmslie, in 1920, did not implicate the talus as the main pathologic structure but emphasized the midtarsal subluxation—the navicular and cuboid displaced medially, with plantar and medial rotation of the calcaneus.[24,82] Others have emphasized the talonavicular subluxation[26] and the dislocation of the head of the talus out of its "socket" ("acetabulum pedis").[10,25] Actual deformity of the talar body and neck has been described in more recent literature, based on intraoperative observations and imaging studies.[12,44,48,100] Finally, Ponseti, in defining clubfoot, has emphasized the cavus component, especially how it relates to nonoperative correction.[82]

The deformity in the talus itself includes medial and plantar deviation of the anterior end, with a short talar neck projecting medially from a dysmorphic, small body that is malplaced within the ankle joint. The talar neck–body declination angle is invariably decreased, with the neck axis approximating 90 degrees to the axis of the body in some specimens, compared to the normal 150 to 160 degrees (Fig. 22–39).[93,99] The articular surface of the talar head is found so close to the body that a true neck is not present (Fig. 22–40). On the inferior aspect of the talus, the anterior and medial facets of the subtalar joint are either absent, fused, or significantly misshapen, so that the overall impression of talar development is consistent with the proposed primary cartilaginous anlage defect.[10,54,92,96] Delayed radiographic appearance of the ossification center of the talus further supports the hypothesis of a primary germ plasm defect. Intraosseous deformity in the calcaneus, navicular, and cuboid, while similar to the dysplasia of the talus, is generally much less severe. The contour of the calcaneus, for example, is generally normal, although the calcaneus is often small in size. The sustentaculum tali is usually underdeveloped, consistent with the dysplasia of the talar facets above,[53] and the anterior articular surface of the calcaneus is medially deviated and deformed, owing to the interosseous deformity of the calcaneocuboid joint (Fig. 22–40).[24,26] Both the navicular and the cuboid tend to have more normal shapes and are misshapen only by their interosseous relationships with the talus and calcaneus. The medial tuberosity of the navicular may be hypertrophied as a result of the excessively thick ligamentous structure tethering the navicular to the medial malleolus and calcaneus.[48]

Controversy exists concerning the presence or absence of excessive medial or internal tibial torsion. Evidence for[50,64,67] and against[18,43,98] this deformity has been reported, and it is the author's experience that true medial tibial torsion can exist in the presence of clubfoot but is generally unusual. More important is the intra-articular (*inter*osseous) deformity known as medial, or internal, spin. This latter deformity, which involves both the talus and the calcaneus within the mortise, is also a source of controversy. In the transverse plane, the talus has been described as medially rotated and supinated within the mortise, laterally rotated in its body, and neutrally rotated.[13,35,36,74,98] The controversy arises from the difficulty of observing the interosseous deformity at surgery. Exposure of the tibiotalar joint and the other structures of the clubfoot will necessarily eliminate some of the interosseous deformity because the joint capsules and ligaments are cut to expose these deformities. It is therefore not surprising that surgical observations by different investigators have been somewhat contradictory, which underscores the need for preoperative imaging to evaluate the *inter*osseous deformities noninvasively, and thus not disturb the deformity while in the process of observing it.

The study of Herzenberg and colleagues was a landmark in this regard.[44] By digitizing multiple microtome slices of normal and clubfoot fetal specimens, the investigators were able to create three-dimensional reconstructions of the deformities by computer, similar to what would ultimately become three-dimensional CT reconstruction technology. The significantly dysmorphic talus (Fig. 22–40) was found to have a neck-body axis of 60 degrees in their specimen. More important, the talar neck was found to be internally rotated 45 degrees relative to the tibia-fibula axis (ankle mortise), while the calcaneus was internally rotated 22 degrees. Both of these rotations were about 20 degrees more than normal. Herzenberg and colleagues further commented that the body of the talus appeared *externally* rotated within the mortise, but noted that the overall axis gave the impression of internal rotation because of the marked intrinsic deformity of the talar neck and medial displacement of the articular surface.

In the coronal plane of the ankle, deformity of the talus around its longitudinal axis has been found to actually be a pronation or "intorsion" deformity,[51,57] reminiscent of the deformity seen in embryonic specimens.[62] The calcaneus, often described as inverted or supinated in surgical observations, has also been found to be intorted or pronated, especially its posterior segment,[25] although not nearly to the same degree as the talus. Although it may be difficult to visualize a pronation deformity being responsible for what is anatomically an inversion or varus of the heel, the presence of this deformity, perhaps exacerbated by the equinus positioning of both hindfoot bones, cannot be denied, as it has been observed both arthrographically and visually at surgery (Fig. 22–41). As will be described later, ligamentous release of both tibiotalar and the subtalar joints to correct this intorsion is just as important in the surgical correction of the clubfoot as the ligamentous release necessary to correct the equinus and the lateral rotation of the talar body.[13]

The navicular is consistently displaced medially and plantarward on the talar head and has a false articular relationship to the medial malleolus (see Figs. 22–39 and 22–40).

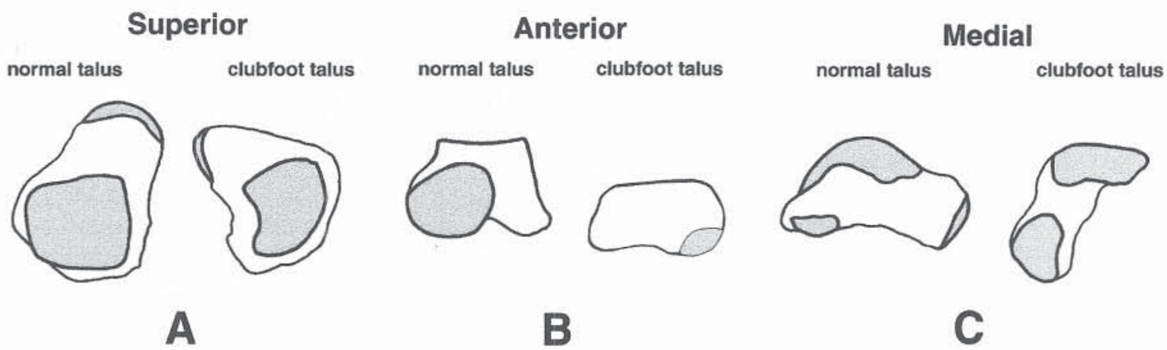


FIGURE 22–39 Schematic illustration of the clubfoot talus. (Adapted from Shapiro F, Glimcher MJ: Gross and histological abnormalities of the talus in congenital club foot. J Bone Joint Surg 1979;61-A:522.) **A,** Top view. The neck is shortened and deviated medially, so that true distinction from the body of the talus is questionable. The articulation with navicular is on the medial side of the misshapen talar head. **B,** End-on view. The medial and plantar deviation of the navicular articulation is apparent. **C,** Equinus of the neck in relation to the tibiotalar articular surface is significant.[4]

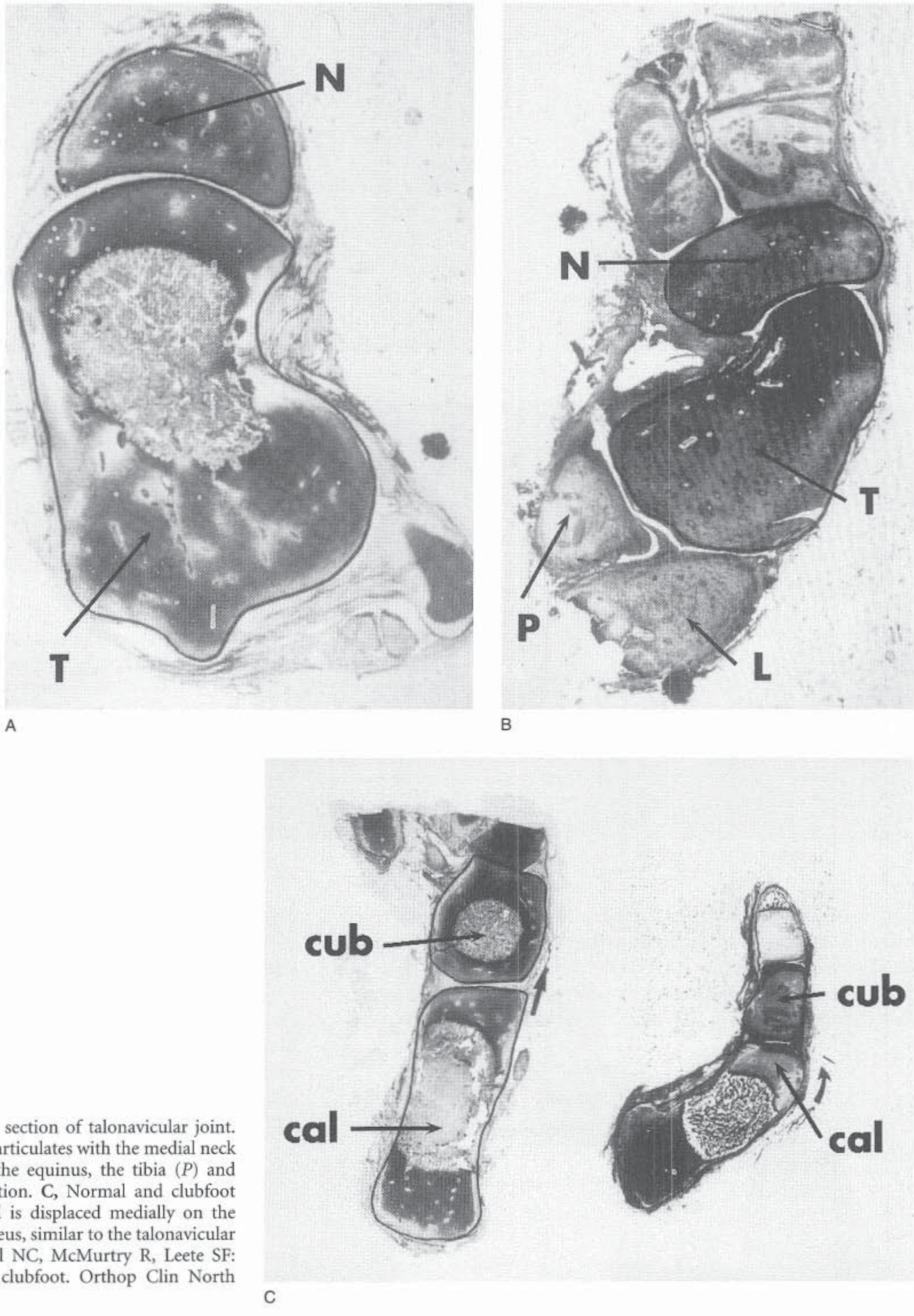


FIGURE 22–40 **A,** Normal foot, section of talonavicular joint. **B,** Clubfoot section. The navicular articulates with the medial neck of the dysmorphic talus. Due to the equinus, the tibia (*P*) and fibula (*L*) are included in the section. **C,** Normal and clubfoot calcaneocuboid joints. The cuboid is displaced medially on the dysmorphic distal end of the calcaneus, similar to the talonavicular alignment. (Modified from Carroll NC, McMurtry R, Leete SF: The pathoanatomy of congenital clubfoot. Orthop Clin North Am 1978;9:225.)

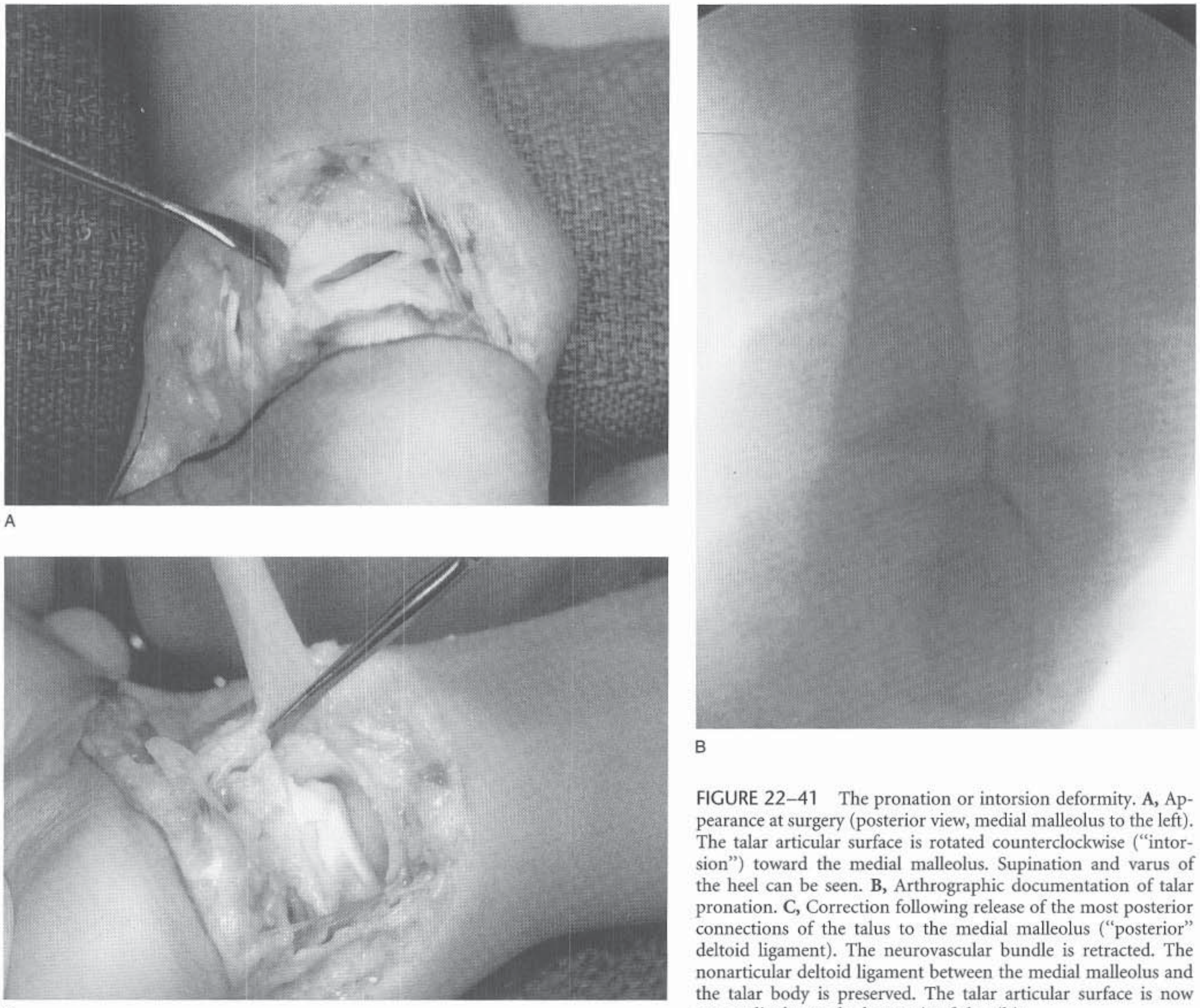


FIGURE 22–41 The pronation or intorsion deformity. **A,** Appearance at surgery (posterior view, medial malleolus to the left). The talar articular surface is rotated counterclockwise ("intorsion") toward the medial malleolus. Supination and varus of the heel can be seen. **B,** Arthrographic documentation of talar pronation. **C,** Correction following release of the most posterior connections of the talus to the medial malleolus ("posterior" deltoid ligament). The neurovascular bundle is retracted. The nonarticular deltoid ligament between the medial malleolus and the talar body is preserved. The talar articular surface is now perpendicular to the long axis of the tibia.

The articular cartilage of the talar head may be uncovered laterally as a result of the medial displacement of the navicular.[59] Release of the so-called tibionavicular (anterior deltoid) ligament is always necessary to correct this major interosseous deformation (Fig. 22–42).[24,101] Usually the tibialis posterior tendon must also be lengthened to allow reduction of the navicular to a normal position on the head of the talus.

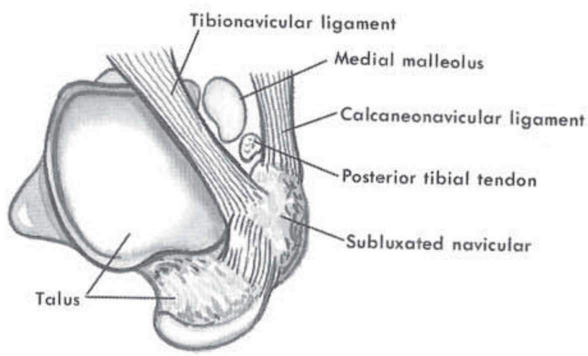


FIGURE 22–42 The tibionavicular ligament must be released along with the calcaneonavicular ligament to allow the subluxed navicular to be reduced on the head of the talus.

As mentioned, the cuboid is similarly displaced medially on the anterior end of the calcaneus (see Fig. 22–40C).[24,26] Because the calcaneus is also medially rotated in the ankle mortise in the transverse plane, this contributes to a significant midfoot "varus" or adductus. This midfoot joint must usually be released, similar to release of the tibiotalar-navicular complex, in order to restore the midfoot joints to anatomic alignment.[13,94] As will be described later, release of the calcaneocuboid and calcaneonavicular ligaments, associated with release of ligaments between the neck of the talus and the anterior calcaneus, is required to fully restore both talocalcaneal and midfoot alignment.

Finally, contractures of the periarticular soft tissues must also be eliminated to successfully restore clubfoot anatomy to normal. Thickening and contracture of tendon sheaths and ligaments (as well as of inelastic muscle tissue) have been reported by multiple investigators and are an expected surgical finding. From studies of muscle, evidence of neurogenic disease has been described,[55,67] as well as a fibrotic (collagen-producing) protein synthesis.[41,82] Although it is difficult to separate out the effects of presurgical treatment (i.e., casting) and the underlying histologic abnormalities found at biopsy, denervation and neuromyogenic changes in the tibialis posterior, peroneals, triceps surae, and long toe flexors appear to be a result of the condition itself. Shortened musculotendinous units are a consistent finding at any stage of clubfoot treatment and are well-known obstacles to the correction of the bony deformity described earlier. In addition, fibrosis of tissues such as the plantar fascia, the calcaneonavicular ("spring") ligament, the tibionavicular ligament, and the so-called master knot of Henry (which engages the flexor hallucis longus and flexor digitorum longus at their decussation) is suspected in the pathogenesis,[51,88] and all must be addressed, either by manipulative stretching or by surgical release. Mobility of the navicular depends on achieving lengthening of the tibialis posterior and the master knot; mobilizing the talus and calcaneus out of equinus often requires lengthening the Achilles tendon; and ability to internally rotate the body of the talus and to externally rotate the calcaneus to restore the normal talocalcaneal divergence requires peripheral subtalar capsular stretching or release circumferentially, including anteriorly (by severing the connections between the talar neck and the anterior end of the calcaneus). The increased fibrosis and contractile myofibroblasts in these "soft" tissues, the *inter*osseous restraints maintaining deformity, must be successfully lengthened or released if there is to be any remodeling following anatomic correction of the bony dysmorphic structures.

Diagnostic Features/Differential Diagnosis. It is rarely difficult to identify a true clubfoot in a newborn (Fig. 22–43). The classic appearance of the heel in marked equinus with the foot and inverted on the end of the tibia, with the foot approaching an upside-down appearance in the more severe cases, is difficult to mistake for anything else. Lack of correctability separates the true clubfoot from the milder *postural* clubfoot. The milder presentations represent an in utero postural deformity, identified by the fact that it is fully (or nearly fully) correctable passively and by the conspicuous absence of the significant contractures and deep skin creases of a true clubfoot. The postural clubfoot exhibits none of the atrophy and rigidity of true talipes equinovarus. Postural deformity can frequently be eliminated at the first presentation by several minutes of gentle stretching.

In addition to distinguishing the severity, it is also essential to search for associated anomalies and neuromuscular conditions that define a nonidiopathic deformity. The prognosis for a nonidiopathic, syndromic clubfoot is generally worse than the prognosis for idiopathic clubfoot, although there are certain exceptions, such as Down syndrome or Larsen's syndrome. In these syndromes, because of the significant ligamentous laxity underlying the syndrome itself, release of the clubfoot must be done judiciously rather than aggressively or completely, as the foot will have a propensity to be overcorrected, and overcorrection will result in an equally severe and perhaps unreconstructable calcaneovalgus deformity if the laxity of the underlying syndrome is not taken into account. On the other hand, patients with arthrogryposis, diastrophic dysplasia, Möbius' or Freeman-Sheldon syndrome, spina bifida and spinal dysraphism, and fetal alcohol syndrome have clubfeet that are notorious for defying correction and subject to severe recurrence. Such techniques as primary bone resection (e.g., lateral column shortening, talectomy) and complete division of tendons rather than lengthening are often employed in the management of these syndromic type of clubfeet. The importance of the presurgical diagnosis of these conditions cannot be overemphasized, as it will affect eventually the technique of management.

Some evaluation of the severity of the deformity is recommended, both for prognostic value and to follow the progress of treatment. Methods of treatment cannot be compared for efficacy if the initial severity of the deformity is not known or described. Determining the initial severity index is an important assessment of each component of talipes equinocavovarus, alerting the surgeon and family to the need for an earlier surgical release, for example, because nonoperative management is not likely to succeed. Although Goldner and Fitch,[35] Carroll and colleagues,[13] and others[31,42,68] have proposed evaluation schemes, we favor the method of Dimeglio[21] because of its more objective and reproducible method of scoring (Fig. 22–44).

Nonoperative Treatment. Almost all orthopaedists agree that the initial treatment for idiopathic clubfoot should be nonoperative. Most also agree that the earlier the treatment is begun, the more likely that it will be successful, due to the relatively viscoelastic character of the newborn foot and, to some degree, its resistance to atrophy or stiffness from immobilization (a frequent form of nonoperative treatment). The underlying philosophy of the advocates of nonoperative treatment is that this should be a definitive method, eliminating or significantly reducing the incidence and amount of surgery that might eventually be required. Much of this philosophy is predicated on observations in earlier surgical experience that neonatal clubfoot surgery almost always produced a more scarred and stiff foot.[22,83] With this in mind, nonoperative treatment proposes to gradually correct the deformity of clubfoot without producing the scar tissue that inevitably diminishes the result. The detrimental role of retracting fibrosis and the observations of myofibroblasts in retractile tissue[28,82,88,111] certainly add histologic confirmation to the clinical observations concerning the results of neonatal surgery.

Perhaps the most determined protagonist of nonoperative treatment was J. H. Kite, who in the period of 1924 to 1960 treated more than 800 patients at the Atlanta Scottish Rite Hospital nonoperatively. The average treatment time was around 6 months, but the patient population included some children as old as 8 years at the time of their cast correction and who were casted longer. When patients began treatment before age 1, their cast treatment ranged from 26 to 49 weeks, and in Kite's private patients, manipulated and casted by him alone, the deformity was usually corrected in 36 weeks. In his 1964 monograph *The Clubfoot,* Kite described in great detail the method of manipulation and cast correction that had served him well over many years.[63] Kite corrected each component of the clubfoot deformity separately and in order, beginning with the forefoot adduction and proceeding to correction of the heel varus (inversion) and finally to correction of the equinus. He was adamant that one could not proceed to correct the next deformity until the previous one had been corrected. For example, the forefoot had to be abducted until the cuboid was in front of the calcaneus and the navicular in front of the head of

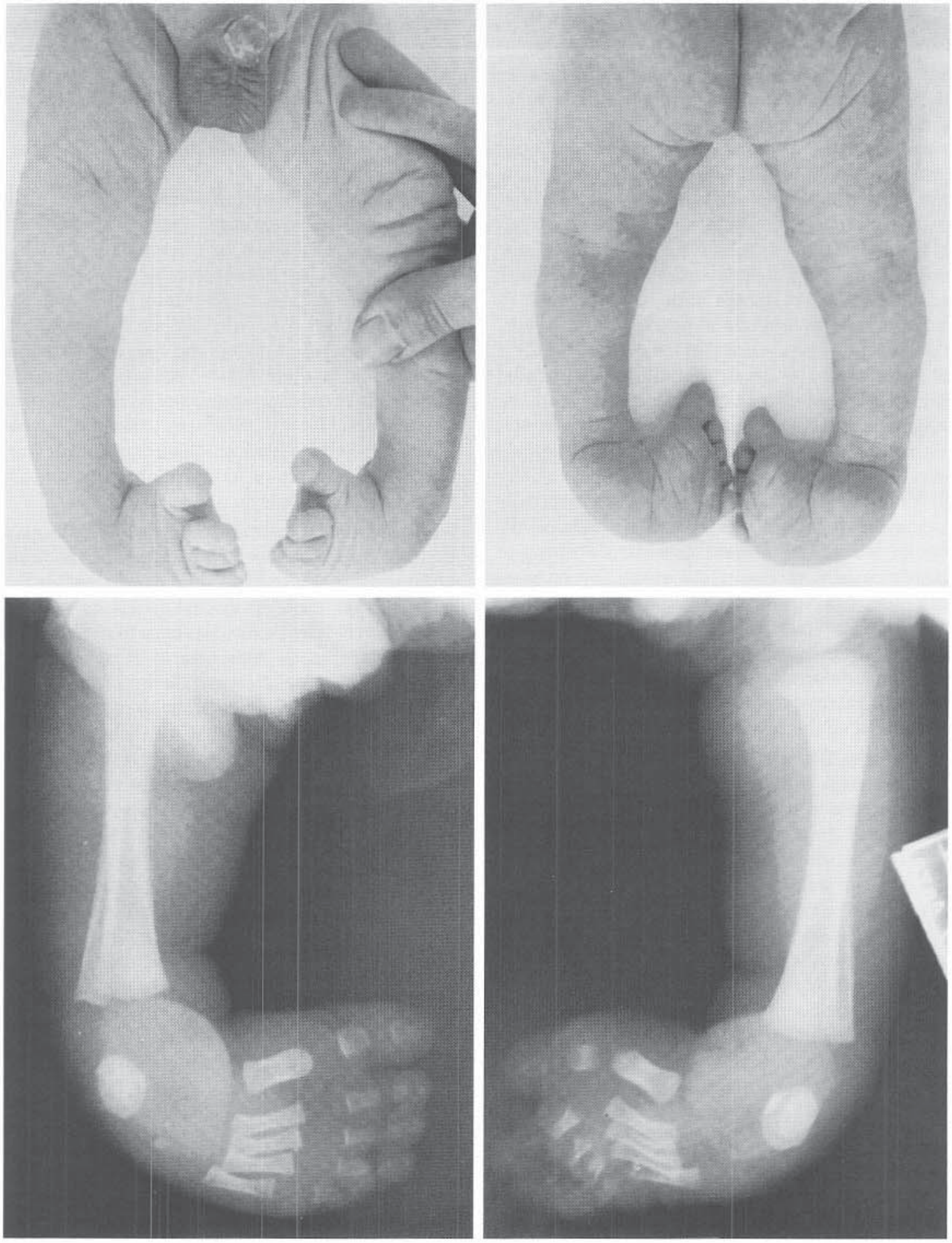


FIGURE 22–43 Bilateral talipes equinovarus in a newborn. Note the deep creases above the heel and medially in the midfoot.

the talus. If the foot was dorsiflexed before this occurred, the navicular would remain on the medial side of the talar neck and would then never achieve normal anatomic position. Ideally, Kite believed, the forefoot should be slightly overcorrected into a mild flatfoot position before the foot was brought up out of equinus. It was also necessary to roll the calcaneus out of inversion prior to attempting to correct equinus. Kite achieved this by placing the plantar surface of the "slipper" cast on a glass plate to flatten the sole, which then allowed the foot to be everted as a unit. Once forefoot and calcaneal abduction/eversion were achieved, the foot could be dorsiflexed, often with wedging casts, to achieve the plantigrade position.

Ponseti was equally as adamant in obtaining correction nonoperatively, and by correcting all components of the clubfoot simultaneously, he was more efficient in obtaining correction, which was usually accomplished in ten casts or less. Ponseti's significant departure from Kite's technique involved the realization that the calcaneus could in fact be corrected out of varus/inversion by abducting the forefoot.[82] Ponseti felt that Kite's technique of correcting the forefoot adduction first and individually interfered with the correction of the heel varus because the necessary abduction and lateral rotation of the calcaneus were intentionally blocked. Ponseti felt that Kite achieved calcaneal correction only with great patience and a long treatment time by not allowing the heel to correct simultaneously with the forefoot. In addition, Ponseti emphasized the correction of forefoot cavus (Fig. 22–45), cautioning against the natural tendency to want to correct the "varus," supinated foot by pronating the entire foot, which would only exacerbate the forefoot cavus. The forefoot should be corrected by supinating it (dorsiflexion of the first metatarsal) and then shifting the navicular, cuboid, and calcaneus in relation to the talus by abducting the foot in the supinated position. The heel was not constrained (as it was with Kite's technique), so the calcaneus could evert during these maneuvers (Fig. 22–45).

Ponseti's correction of equinus frequently included a percutaneous Achilles tendon lengthening done at the time of the final casting. The goal was to achieve 15 degrees of dorsiflexion by this maneuver. In 85 percent of patients a percutaneous tenotomy was performed. After the final casting, patients were placed in a Denis Browne bar, which was worn full-time and then part-time for up to age 6 years.

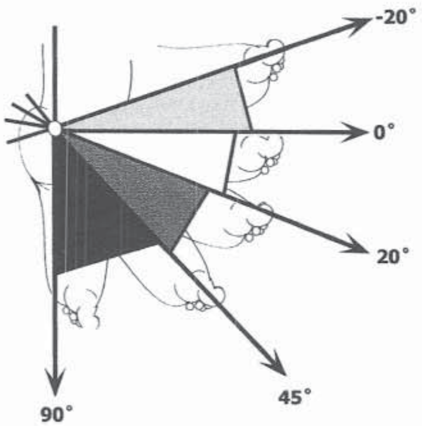
An interesting theoretical concept elaborated by Ponseti was the proposition that the contracted soft tissues and capsules of the congenital clubfoot not only could be stretched by manipulation, but would then yield further to allow more correction by means of immobilization in plaster. Following the identification of retracting fibrosis and increased collagen synthesis histologically, Ponseti claimed that the soft tissues of the clubfoot exhibited a kind of inflammatory response, especially in the newborn infant, that was only exacerbated by daily manipulations and vigorous stretching. It was therefore necessary to put this inflammatory response, similar to Dupuytren's contracture, to rest following stretching by immobilization in plaster. Repetitive vigorous stretching would only produce a more proliferative cellular response, producing retracting fibrosis. This theory, of course, is counter to the usual concept of motion being beneficial to the healthy stretching of soft tissues and the health of cartilaginous surfaces.[11,86,107,108,109] Nevertheless, the 30-year follow-up of 71 of Ponseti's patients[16] revealed excellent or good results in 78 percent of feet, with at least a portion of the evaluation including functional motion (dorsiflexion, plantar flexion, inversion, eversion) and muscle strength, particular of the triceps surae. The overall results, challenged by some as being difficult to believe,[34] are noteworthy for the apparent lack of stiffness and the excellent function of the triceps surae, results that are not reproduced on objective analysis of surgically corrected feet.[5,61,68]

Several centers in France have developed a program for clubfoot treatment consisting of manipulative therapy using a minimum of immobilization. We have used this technique, and our studies over a follow-up averaging 2 years have been most encouraging. We have seen a dramatic decrease in the need for surgical correction, and those requiring surgery have been corrected with simpler releases. Masse is credited with originating this functional or manipulative correction method, which emphasizes daily corrective manipulation by trained physiotherapists during the first 8 weeks of life.[70,91] Following manipulation, which generally includes maneuvers to disengage the navicular from the medial malleolus, derotation of the calcaneus and foot as a unit (the calcaneopedal bloc) (Figs. 22–45C and 22–46), downward traction to lengthen the heel cord and distract the hindfoot joints, and forefoot correction into abduction combined with eversion of the heel, the foot is immobilized with elastic bandages, similar to the original strapping technique described by Robert Jones,[59,99] sometimes supplemented by the use of a rigid footplate and, as the child gets older, a thermoplastic ankle-foot orthosis (AFO) or other orthosis to maintain position (Fig. 22–46). Bensahel and colleagues in 1990 reported good results in nearly 50 percent of patients, a figure that eventually improved to 86 percent with subsequent complementary surgery, in a review of 338 cases treated functionally.[6] The expertise and gentleness of manipulation were emphasized, including the importance of maintaining the foot position during nonmanipulative periods by bandaging or bracing, with the latter being less attractive because of the possibility of forcing the foot into an extreme position. Seringe and Atia at St. Vincent de Paul Hospital, in Paris, added active solicitation of peroneal muscle function to the program of Masse and Bensahel, as well as use of the footplate with an adhesive bandage and later a custom brace. A 1990 report on 157 feet in 105 children with a minimum 4-year follow-up indicated that good results were achieved in 87 percent by using functional therapy and surgery as necessary, with nearly half obtaining this result without any surgery.[91] The functional method alone was particularly successful in clubfeet that were initially mild (two-thirds obtaining a successful result without surgery) and in feet treated by trained, specialized physiotherapists, who achieved 100 percent success in mildly affected and 44 percent success in severely affected feet *without* surgery. Seringe and Atia pointed out that the success of the conservative treatment depended on the initial severity of the foot, the skill and motivation of the physiotherapist, and social factors affecting compliance with treatment and regularity of treatment by the family.[91]

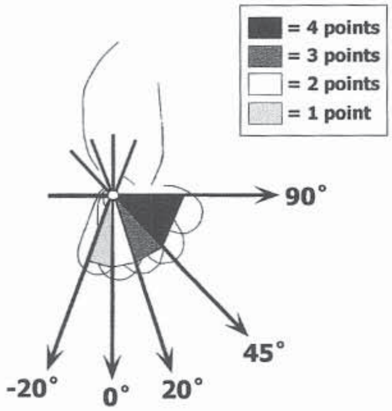
Further studies recently reported from Montpellier by Dimeglio and colleagues show even better results with the addition of a continuous passive motion (CPM) machine during the earliest portion of the treatment (Figs. 22–46K and L).[22,23] When CPM during the first month of life was added to the treatment of 241 clubfeet, only 28 percent of feet eventually required operation. More impressive, only 54 percent of feet considered to be the most severely affected required surgery. The most common persistent deformity, requiring "limited posterior release," was residual hindfoot equinus (Fig. 22–47), which was also the most common residual deformity following cast treatment. In 60 feet treated by home manipulation but without CPM, 78 percent of all feet and 100 percent of the 21 severely affected feet required surgery. Although these results are preliminary and lack the long-term follow-up reported by Cooper and Dietz,[16] it seems that the functional treatment as practiced in France, utilizing frequent (daily) manipulations and CPM when possible, is producing results, especially in the more severely affected feet, comparable to or better than the results achieved with any form of casting, even though the technique of treatment—active stretching and daily manipulation—would be expected (according to Ponseti) to incite

Classification of Clubfoot

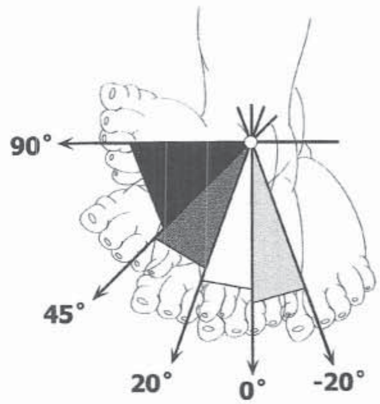
Classification Grade	Type	Frequency (%)	Score
I	benign	20	(<5)
II	moderate	33	(=5<10)
III	severe	35	(=10<15)
IV	very severe	12	(=15<20)



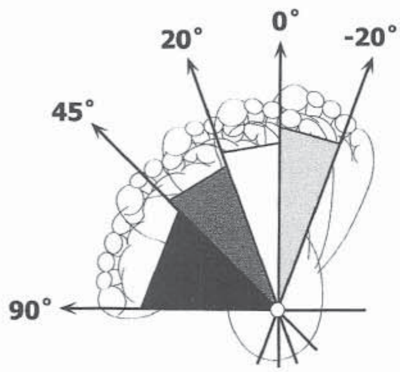
Sagittal plane evaluation of equinus.



Frontal plane evaluation of varus.



Horizontal plane evaluation of derotation of the calcaneopedal block.



Horizontal plane evaluation of forefoot relative to hindfoot.

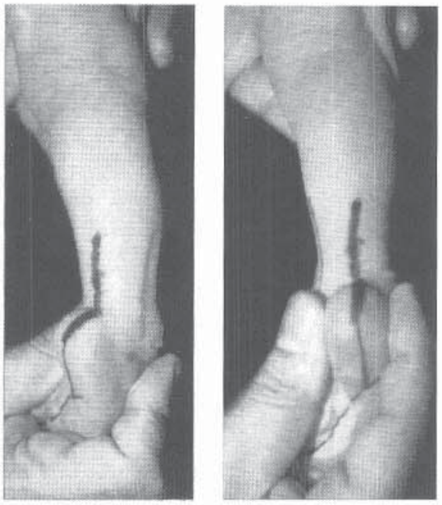
Assessment of Clubfoot by Severity Scale

Characteristics	Points	Characteristics	Points
Reducibility		Other parameters	
90°-45°	4	Posterior crease	1
45°-20°	3	Medial crease	1
20°-0°	2	Cavus	1
<0° to -20°	1	Poor muscle condition	1

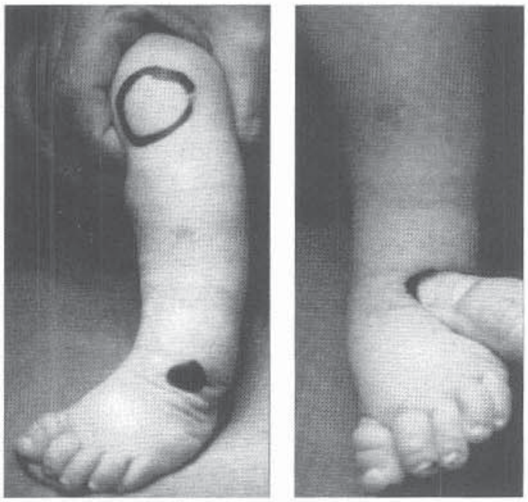
FIGURE 22–44 Classification of clubfoot according to Dimeglio. Each major component of clubfoot (equinus, heel varus, medial rotation of the calcaneo-pedal "block," and forefoot adductus) are graded clinically from 4 to 1 (most severe to most mild). Additional points are added for deep posterior and medial creases, cavus, and poor muscle function. The total score is stratified into four groups of severity (benign to very severe, grades I to IV). (From Dimeglio A, Bensahel H, Souchet P, et al: Classification of clubfoot. J Pediatr Orthop B 1995;4:129; Dimeglio A, Bonnet F: Reeducation du piet bot varus equin. Encyclopédie Médico Chirurgicale. Paris, Elsevier, 1997.)

Equinus: 3 points

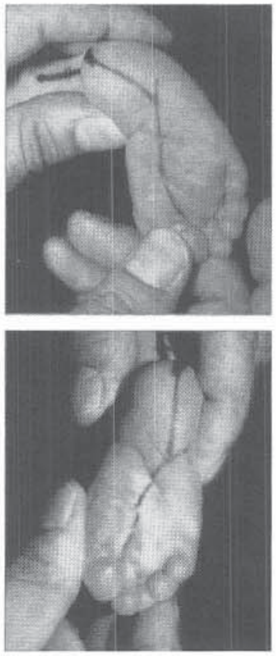
Equinus: 2 points



Varus deviations, reducibility to 0°: 2 points

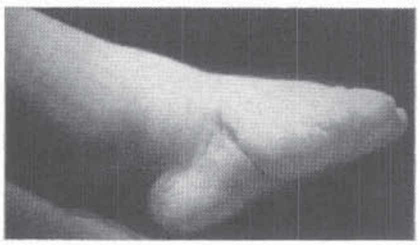


Derotation of calcaneopedal block: reducibility to -10°: 1 point

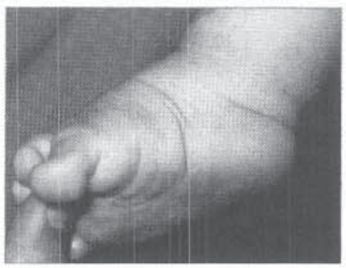


Adduction of forefoot, reducibility to 0°: 2 points

Posterior crease: 1 point



Medial crease: 1 point



Cavus foot: 1 point (for this foot, 1 more point for the medial crease and another point for the posterior crease)

FIGURE 22–44 *Continued.* *See legend on opposite page*

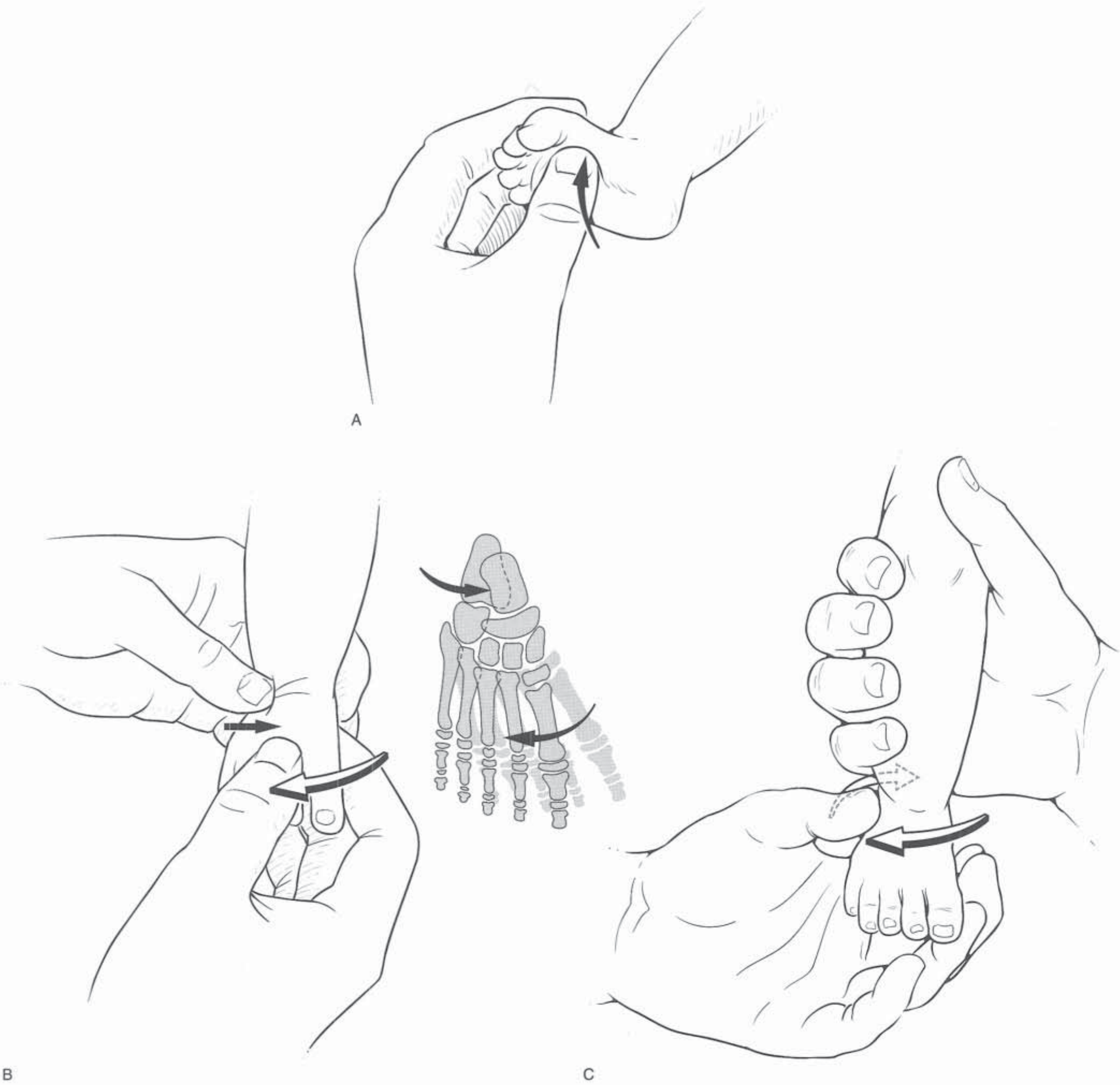
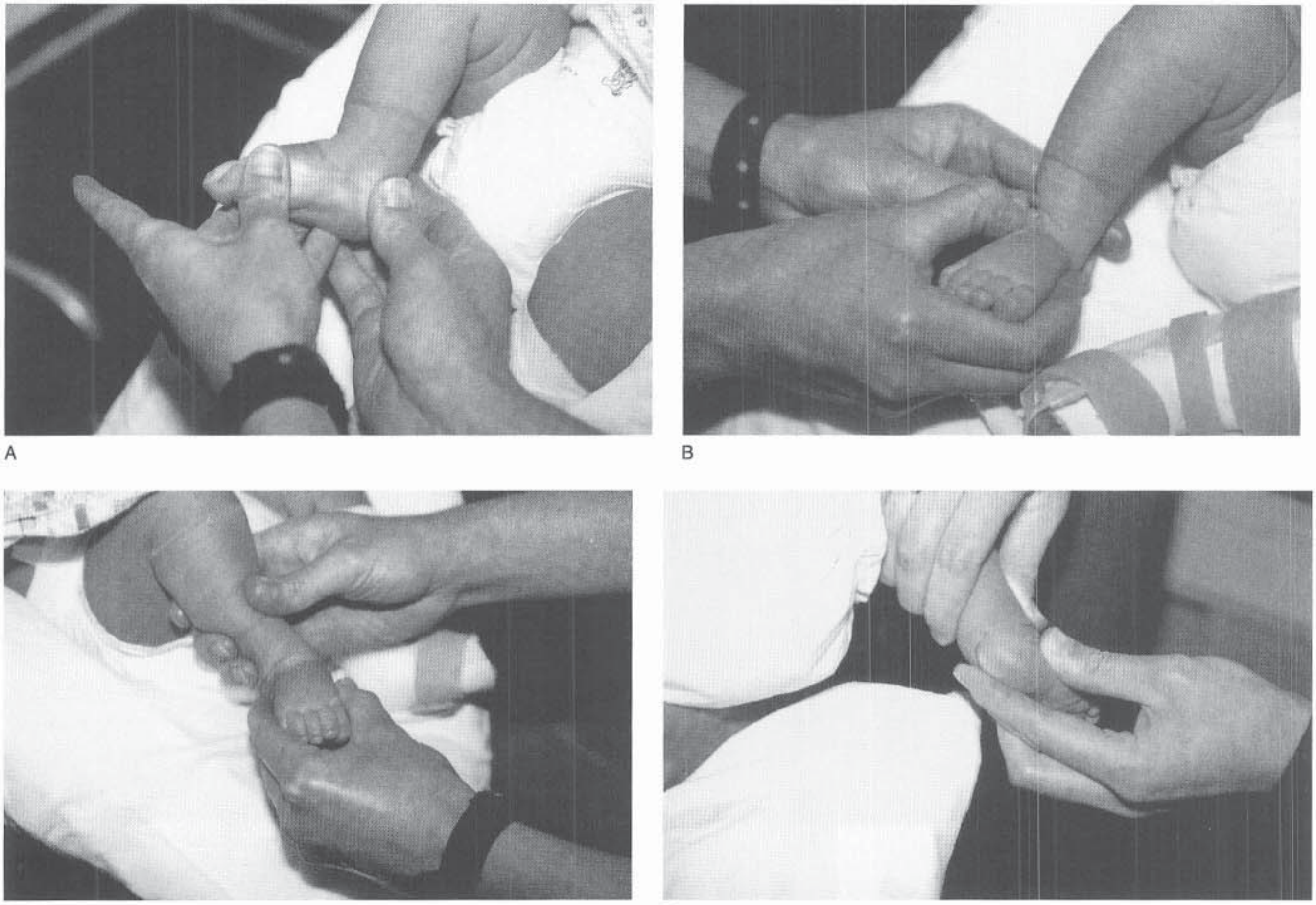


FIGURE 22–45 Technique of nonoperative correction of clubfoot. **A** and **B,** Dorsiflexion of the first ray while maintaining thumb pressure on the lateral aspect of the talar neck. The forefoot is abducted while dorsiflexion of the first ray is maintained. The heel is *not* constrained (Modified from Ponseti IV: Congenital Clubfoot: Fundamentals of Treatment. Oxford, Oxford University Press, 1996.) **C,** Derotation of calcaneo-pedal "block." The heel is pushed away from the fibula posteriorly while the tibia/fibula are immobilized.

retractive fibrosis and jeopardize the result. Longer-term functional results, especially emphasizing motion and muscle strength, will be eagerly awaited in the French patients avoiding surgery or having minimal procedures.

The success of any nonoperative treatment, whether weekly stretching and casting or daily physiotherapy and CPM, depends heavily on the factors identified by Seringe and Atia.[91] Additionally, it is obviously not possible to treat all patients with the intensive Montpellier program, simply because not all families have the logistical or economic resources to travel daily to the physical therapist or submit to inpatient treatment for the first month of the baby's life. Such logistical and economic factors, coupled with the lack of efficient correction with weekly visits, have produced the general feeling, echoed by North American surgeons, that nonoperative treatment is rarely successful—perhaps in only 10 to 20 percent of feet—and that feet in which the deformity does correct with nonoperative treatment, primarily casting, are the mildest of clubfeet or are even postural clubfeet and thus not truly a congenital deformity of significance. Because of the number of clubfeet in which nonoperative treatment fails, for whatever reason, surgical treatment remains the



A B C D

E

FIGURE 22–46 French technique of manipulation and taping. **A,** Manipulation to correct forefoot adductus and heel varus. **B** and **C,** Derotation of calcaneo-pedal block and reduction of talonavicular displacement. **D,** Manipulation of heel varus. The calcaneus is then rotated medially away from the fibula while the forefoot is externally rotated (as in B and C). **E,** Manipulation of equinus. The taping technique maintains correction of the forefoot.

Illustration continued on following page

mainstay of clubfoot management in the majority of orthopaedic centers. For those centers with the patience, physiotherapy resources, and patients able to comply with daily treatment, nonoperative treatment should not only be considered a viable option, it should be given every opportunity to succeed prior to acquiescing in a surgical solution.

Surgical Treatment. Surgical management of resistant or persistent clubfoot deformity requires more judgment and skill from the surgeon than any other orthopaedic condition amenable to surgical correction. The decisions that have to be made pre- and intraoperatively—the timing of surgery, its extent, and, regretfully, how to plan a salvage procedure—compose a challenging treatment algorithm for which there is no "standard" method or reference.

Surgical correction is the last resort for a deformity that is resistant to nonoperative treatment. Although for many orthopaedists surgical treatment is in fact the method of

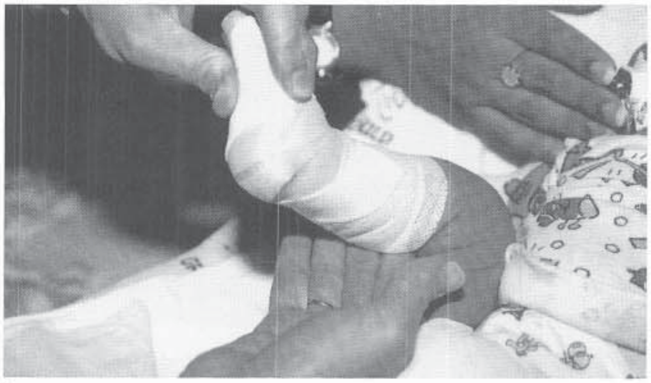

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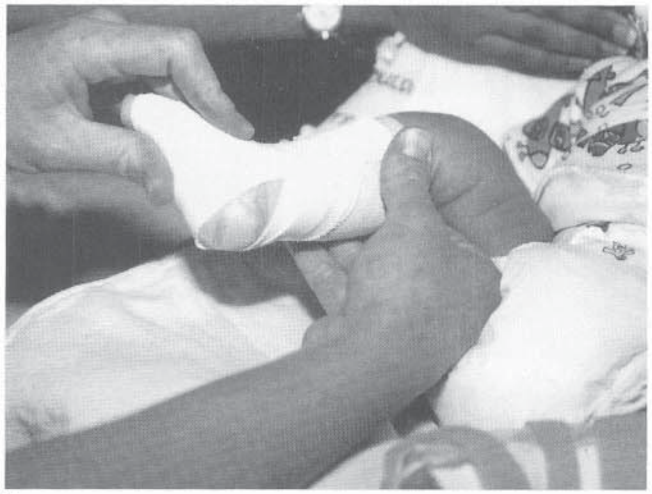
FIGURE 22–46 *Continued.* **F** to **H,** Taping to maintain forefoot eversion and midfoot dorsiflexion. **I** and **J,** Additional taping maintains external rotation and dorsiflexion. **K** and **L,** Footplate and CPM machine.


G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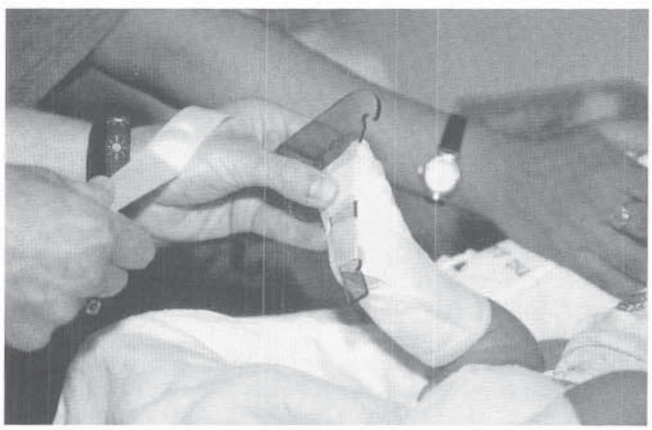
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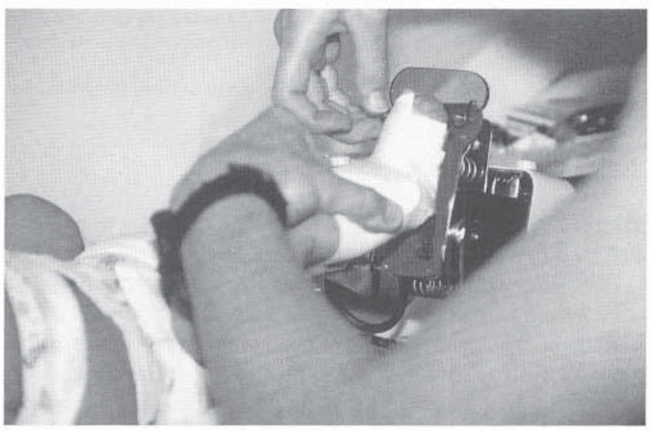
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FIGURE 22–47 Persistent equinus of the hindfoot following manipulative therapy and splinting. This patient had severe (grade III) clubfeet bilaterally. All components of the deformity were clinically corrected, but as shown here on stress dorsiflexion, the correction of equinus is incomplete, with "rockering" through the midfoot. A limited posterior release is indicated.

choice, and the method with which they feel most likely to be able to correct a clubfoot, one cannot discuss surgery as the most effective method of treatment without again recalling the warning of E. H. Bradford that "the literature on the treatment of clubfeet is, as a general rule, that of unvarying success . . . yet in practice there is no lack of half-cured or relapsed cases, sufficient evidence that methods of cure are not universally understood."[10] Certain principles of surgery are self-evident, yet bear repeating because of Bradford's warning. Multiple operations are to be avoided: the increasing stiffness, the deepening of scars and hardening of tissue from repeated surgery, and the atrophy introduced by immobilization are relative contraindications to repeat surgery. The surgeon who performs the first operation has the best chance of achieving permanent correction, and thus this surgeon's responsibility is increased. The most frequent cause of repeat surgery is incomplete or inadequate correction, revealing that the so-called "limited" release is often a euphemism for an incomplete or inadequate operation. The ability of the French school of treatment to achieve improved results with limited releases following the physiotherapy/nonoperative treatment discussed in the previous section is a measure of the practitioners' skill and experience in knowing how much surgery, short of a comprehensive release, is necessary to produce appropriate correction that is additive to the correction achieved by the nonoperative manipulation.

In the infant previously untreated or in whom nonoperative treatment fails, the age at which to perform surgery varies from about 3 months at the earliest to as late as 12 months or later (once the child has achieved walking status).[31,68,82,91,101] At one time, Pous and Dimeglio were performing surgical releases between 1 and 6 weeks of age, reasoning that the earlier the fibrous medial and posterior contractures were released, the better.[83] They subsequently abandoned such a program because of excessive scarring and recurrent fibrosis, which was attributed to the hypermetabolic reaction of the connective tissue in such a young infant.[83] Turco recommended surgery at the age of 1 year or older, citing primarily the advantages that the structures were larger, the anatomy more easily evaluated and corrected, and the repairs of tendon lengthenings more secure.[101] Some surgeons will treat severe clubfeet with percutaneous Achilles tendon lengthening or other limited releases and continue the nonoperative treatment for several more months. Consensus indicates that while surgery can be done as early as 5 months by an experienced surgeon, there is little advantage to doing it at this time, because weightbearing and achieving the standing position will be delayed by the postoperative immobilization. The lack of weightbearing in the 6-month-old infant requires that the plantigrade position be maintained in retention braces or some other external device to prevent recurrence until such time as the child achieves the standing position. There is therefore little advantage to performing the surgery prior to 9 to 10 months, an age which assures that the child will be weightbearing when the postoperative cast immobilization is completed.

The surgical release must address *all* of the pathoanatomic structures, including a complex release of the hindfoot and midfoot, possibly one of the more complicated procedures performed in all of orthopaedics. A variety of surgical procedures and techniques have been described to achieve the goal of complete anatomic restoration. Turco is credited with describing the first complete one-stage posteromedial release.[101] He emphasized correcting the deformity of the calcaneus beneath the talus, which required complete subtalar release (lateral, posterior, and medial), as well as release of the calcaneofibular ligaments. The surgery is performed with patient supine, through a curved posteromedial incision beginning alongside the Achilles tendon above the ankle joint. All medial neurovascular structures and tendons are identified, with the posterior tibialis tendon being lengthened or released, the talonavicular joint opened dorsally, medially, and inferiorly, and the calcaneonavicular spring ligament released. The Achilles tendon and long toe flexors are lengthened and repaired. The talonavicular joint is reduced and pinned (Fig. 22–48).

Release of the interosseous talocalcaneal ligament so that the calcaneus can be everted and rotated, moving the anterior end laterally and the posterior tuberosity downward, was part of Turco's original description, although it is generally avoided in other techniques. Turco immobilized his patients for a total of 4 months, removing the K-wires at 6 weeks. Night splints were used for an additional year following the end of cast immobilization.

Other surgeons modified Turco's basic procedure to address different pathoanatomic aspects that were also considered important. Carroll emphasized the plantar fascial release and capsulotomy of the calcaneocuboid joint,[12,13] since forefoot adduction and supination (actual cavus) were not addressed by Turco's procedure. Thus, through a medial inci-

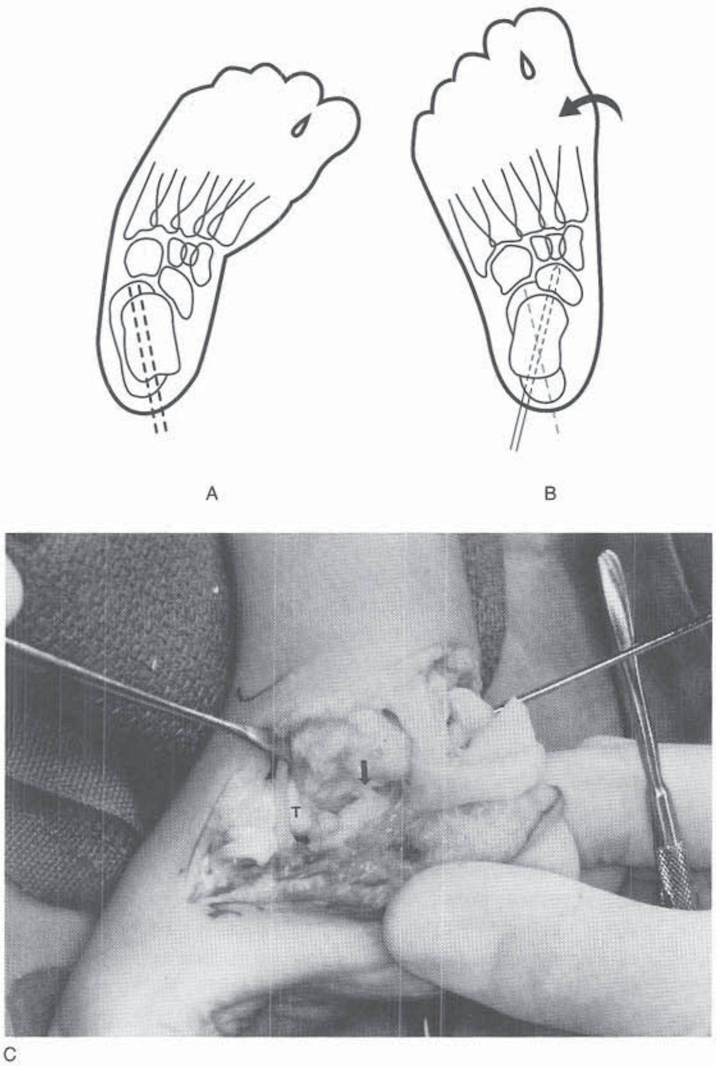


FIGURE 22–48 **A** and **B,** Pinning of the talonavicular joint. The pin is drilled from the posterolateral corner of the talus and, once firmly in the body of the talus, can be used as a joystick to rotate the talus medially while the forefoot is *ab*ducted to effect reduction. Parallelism of the talus and calcaneus, indicative of hindfoot deformity, is corrected by the rotation with the axis at the interosseous ligament. (After Carroll NC, McMurtry R, et al: The pathoanatomy of congenital clubfoot. Orthop Clin North Am 1978;9:225.) **C,** The pin is drilled across the talonavicular joint under direct vision. The interosseous ligament (*arrow*), which is usually released as part of several published techniques (Turco, McKay, Simons), is preserved. The anterior talocalcaneal ligaments connecting the talar neck and anterior end of the calcaneus are released to allow complete talocalcaneal divergence. (Note space between the interosseous ligament and the talar head (*T*).)

sion with the patient prone, the abductor hallucis is identified and released, and deep to it, the plantar fascia is divided: after sufficient dissection of the inferior talonavicular and anterior talocalcaneal area, the peroneus longus tendon is protected and the calcaneocuboid joint is opened from the medial side and fully released (Fig. 22–49). This follows posterolateral release through a posterior longitudinal incision paralleling the lateral edge of the heel cord, through which the Achilles tendon is Z-lengthened and a posterior capsulotomy of the ankle joint, including the medial and lateral ligaments, is performed to mobilize the talus and to reduce the talonavicular joint, which is done by internally rotating the talus using a longitudinal K-wire as the "handle" to perform this derotation (Fig. 22–48).[13] This maneuver is consistent with the three-dimensional analysis showing that the body of the talus is externally rotated in the ankle mortise and must be internally rotated to produce correction.

Goldner, also emphasizing correction of the talar rotation as the primary deformity, performs complete release of the tibiotalar joint, including deep medial deltoid ligaments, and subsequently repairs these ligaments if necessary, in order to correct the talar rotation through the ankle joint.[33] Subtalar capsulotomy is minimized to protect against valgus overcorrection. The medial and plantar structures (all tendons) are lengthened, as is the Achilles tendon, through a medial incision, and through a separate lateral incision in the sinus tarsi region, calcaneocuboid capsulotomy is performed and calcaneonavicular impingement preventing midfoot abduction ("the rug in front of the door") is assessed. A lateral talonavicular capsulorrhaphy to obviate internal fixation is then performed. Deemphasizing the subtalar circumferential release and replacing it with the more complete tibiotalar and midfoot release makes Goldner's approach unique among the modern clubfoot procedures. The results of clubfoot release without formal subtalar release indicate more *under*correction (residual internal foot progression angle), but rarely valgus overcorrection, a more difficult deformity to reconstruct.[110]

More extensive procedures are employed by McKay[74] and Simons,[94] using the Cincinnati incision[16] with the patient supine. The majority of peritalar structures, including all hindfoot and midfoot joints, are released. A medial and lateral circumferential talocalcaneal release is performed, with the latter being released from the attachment of the calcaneocuboid joint laterally to the sheath of the flexor hallucis longus posteromedially. Complete release of the talonavicular and

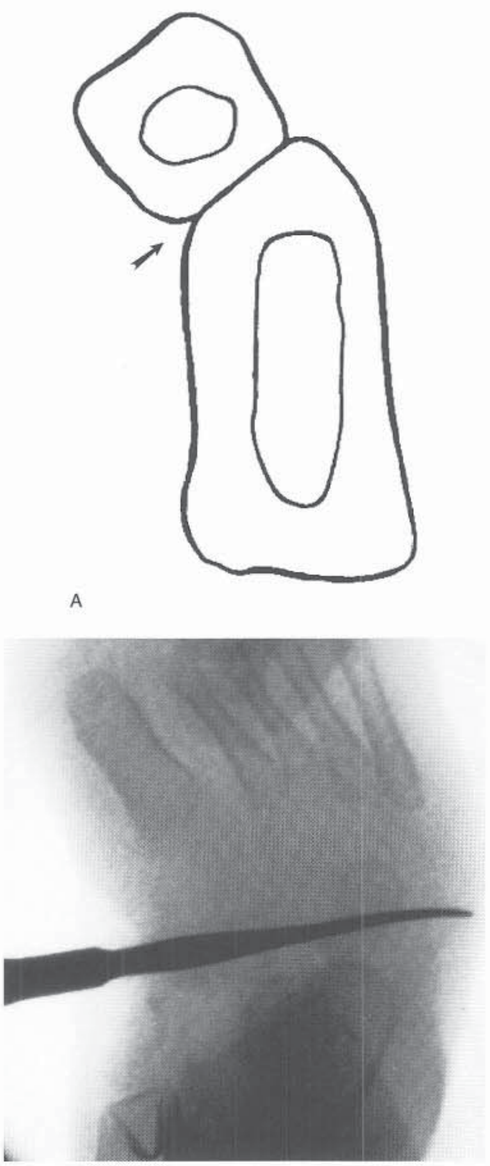


FIGURE 22–49 **A,** The calcaneocuboid deformity. Release from the medial approach (*arrow*). **B,** Fluoroscopic confirmation of calcaneocuboid release from the medial side.

calcaneocuboid is included, and both of these are pinned. The subtalar release includes the interosseous ligament. Once the calcaneus has been adequately derotated by pushing the anterior end laterally and the posterior tuberosity medially and downward, the interosseous ligament is internally fixed. McKay also introduced the concept of an articulated "cable cast," in which the hinge is centered at the ankle joint for immediate postoperative movement, with the connection between the foot and the leg portions of the cast being large-gauge telephone wire. This was intended to increase hindfoot (ankle) motion, with 30 to 60 degrees of total motion being reported,[74,94] although dorsiflexion is usually limited to 10 to 15 degrees. Wound complications from the early motion of the cable cast have decreased the acceptance of this method of postoperative management. In spite of the use of multiple K-wires to internally fix the three joints, late motion does not appear to diminish if early motion is commenced, a theme more emphatically repeated as long-term outcome studies are completed.

Current Operative Technique. The comprehensive procedure favored at our institution includes portions of all of the above procedures. Prone positioning allows better assessment of hindfoot release, while a supine position probably improves medial and plantar exposure. We utilize the Cincinnati incision and occasionally add a longitudinal arm paralleling the Achilles tendon, placed perpendicular to the transverse portion of the Cincinnati incision over the calcaneal tuberosity, if additional proximal exposure for Achilles tendon lengthening is needed.

As in any procedure involving multiple anatomic steps, exposure is key to a successful comprehensive release, and there is no better place to begin emphasis of exposure than with the posterolateral corner of the ankle, where, after the sural nerve has been identified and protected, the peroneal sheath is opened to allow full anterior retraction of the two tendons (Fig. 22–50). This permits a precise and *complete* release of calcaneofibular and lateral subtalar ligaments anteriorly to the sinus tarsi area[74] under direct vision, avoiding blind peroneal tendon injury. Longitudinal exposure of the Achilles tendon permits a long Z-lengthening in the coronal plane (Fig. 22–50C), so that two wider strips (anterior and posterior) are created for later *competent* repair under tension. Because of concern over late triceps surae insufficiency, Achilles tendon lengthening can be deferred until late in the procedure, when it can be definitively determined that Achilles tendon lengthening is required to achieve neutral dorsiflexion. Extensile longitudinal exposure of the Achilles tendon allows it to be mobilized and retracted *without* lengthening, but we do not hesitate to lengthen it to aid exposure if it is undeniably contracted. The flexor hallucis longus sheath medially is opened so that the tendon can be retracted. Posterior and medial release of the subtalar and tibiotalar joints can be performed at this stage (Fig. 22–50D). The neurovascular bundle is mobilized and protected with a Penrose drain, and the posterior tibialis and flexor digitorum longus sheaths also incised for retraction and/or lengthening. In mobilizing the bundle, it is elevated off the medial subtalar area completely, and release of the flexor hallucis longus sheath all the way to the decussation with flexor digitorum longus distally provides easy visual access to release the medial subtalar capsule *peripherally* to the sustentaculum without cutting the interosseous ligament (Figs. 22–48C and 22–50E).

Moving medially, the key to wide exposure is to dissect the plane dorsal to the abductor hallucis, and effectively release it from its origins on the flexor tendon sheaths. The plantar exposure depends on this and on incision/release of all tibialis posterior insertions in the plantar aspect of the foot, leaving only the insertion on the navicular. The tibialis posterior is usually Z-lengthened at this stage, for exposure as well as to allow navicular reduction later. The two long flexors and the neurovascular bundle can then be retracted as a group in the Penrose drain and the most anterior part of the medial subtalar joint incised, leading dorsally to talonavicular capsule and laterally under the neck of talus for release of the anterior talocalcaneal attachments (Fig. 22–48C). The flexor tendon sheaths, now empty, at the level of the medial malleolus should be incised transversely to

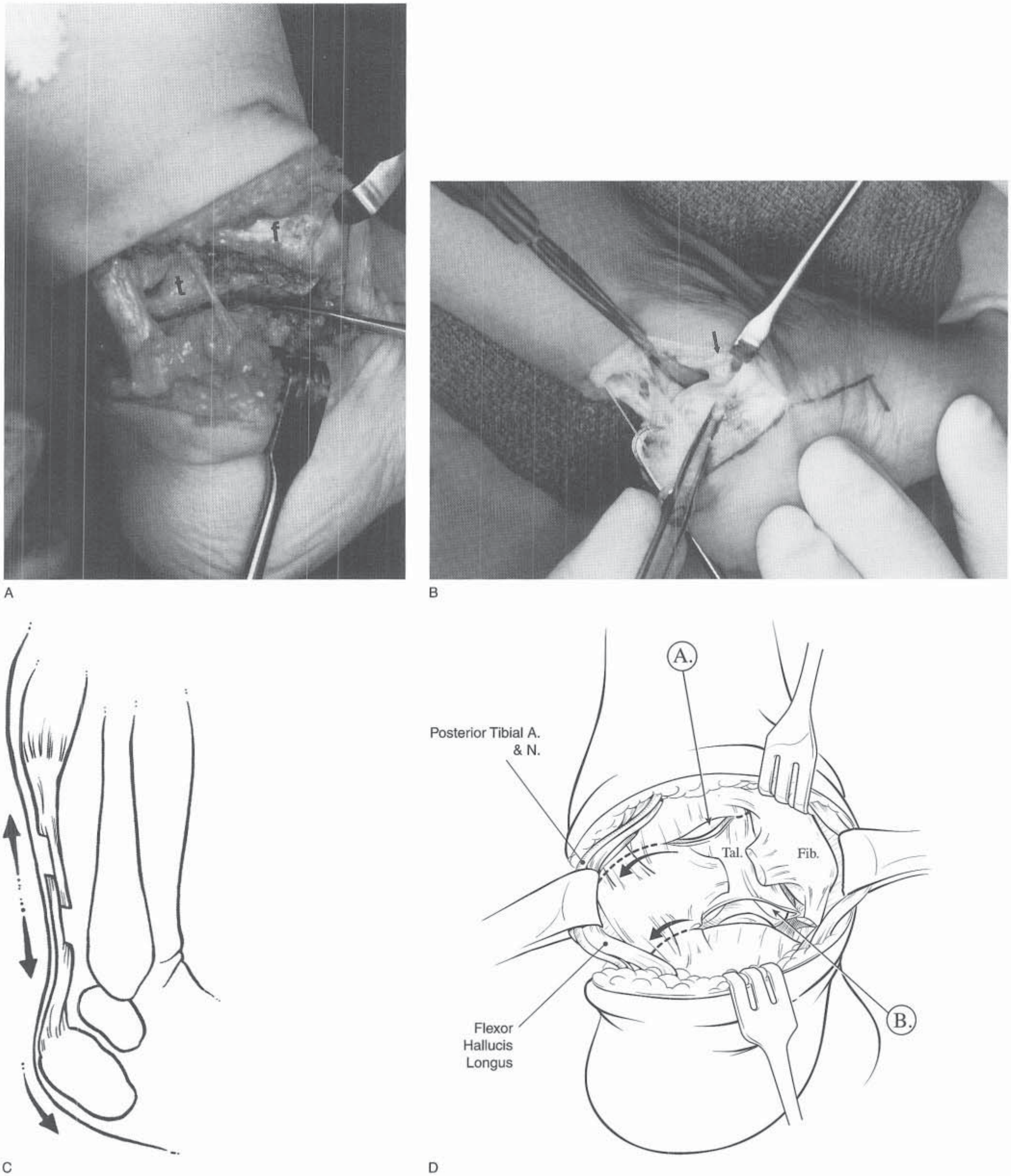


FIGURE 22–50 **A,** Posterolateral corner. The peroneal tendons are retracted to allow complete calcaneofibular and lateral subtalar release (elevator in the subtalar joint, t = talus, f = fibula). The Achilles tendon and sural nerve are preserved. **B,** Deep calcaneofibular (*arrow*) release. Improved exposure by peroneal retraction makes complete posterolateral release feasible under direct vision. The Achilles tendon has already been lengthened in this example. **C,** Z-lengthening of the Achilles tendon in the *coronal* plane. **D,** Retraction of the flexor hallucis longus tendon allows exposure for posteromedial release of the tibiotalar (*A*) and subtalar (*B*) joints.

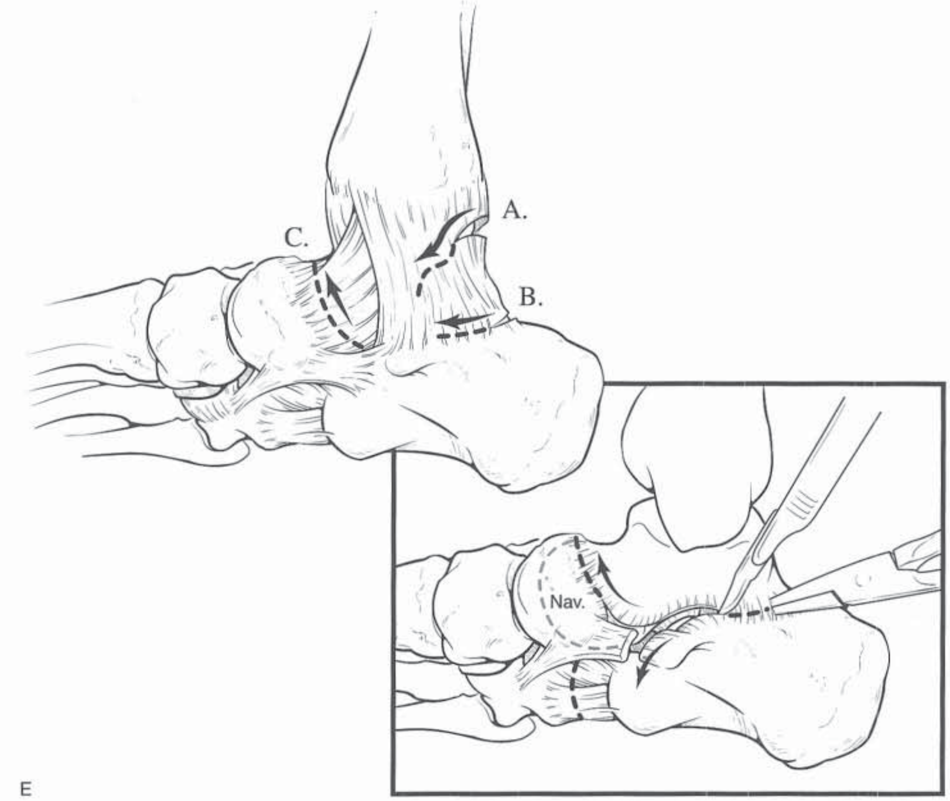


FIGURE 22–50 *Continued.* **E,** Release of the posteromedial ankle (*A*) and medial subtalar (*B*) joints (neurovascular bundle and tendons removed). The stump of the lengthened tibialis posterior remains attached to the navicular, which acts as a guide to continue subtalar release distally into the talonavicular joint (*C*).

the posterior edge of the tibialis posterior, to eliminate their functioning as a supinating contracture.

Talonavicular release must be done carefully, owing to the medial displacement of the navicular. The dorsal structures should be elevated close to bone off the talar neck and navicular, with complete release of the tibionavicular (superficial deltoid) ligament (Fig. 22–50E). The tibialis *anterior* tendon should be identified and followed partially to its cuneiform insertion, to aid in directing dissection to the talonavicular joint and avoid misrecognition of the naviculocuneiform joint. With the distal stump of the Z-lengthened tibialis posterior used as a guide, talonavicular capsulotomy medially, dorsally, and plantarward is performed. With a curved elevator, the *lateral* talonavicular capsule is stripped from the talus and the navicular is mobilized so that it can be accurately reduced on the talar head. The elevator is then passed under the neck of the talus to strip/incise anterior calcaneal connections with the talus (Fig. 22–51). This will ensure full rotatory mobility of the talocalcaneal joint, with the only remaining structure between the two bones being the interosseous ligament, the center of rotation. Failure to release the talar neck from the anterior end of the calcaneus means that the rotatory divergence necessary to correct hindfoot inversion and medial rotation is impaired, as the calcaneus is still "locked" to the talus anteriorly (Fig. 22–51). Division of the calcaneonavicular (spring) ligament completes the medial release. With the navicular retracted distally using a two-prong rake, blunt dissection distal to the end of the calcaneus will lead to the calcaneocuboid joint, which is incised and mobilized with a Freer elevator, stripping and opening the capsule to allow cuboid reduction laterally (Fig. 22–49).

The plantar fascia should be transversely divided by returning to the plane superficial to the plantar edge of abductor hallucis. The muscle belly is bluntly dissected and retracted dorsally to locate the fascial edge. An elevator is passed deep and superficial to the fascia to clear all soft tissue before the fascia is cut with scissors. If the first ray remains tethered, resisting dorsiflexion and abduction, the tendon of the abductor hallucis can be released distally.

The talonavicular joint is now reduced and pinned using Carroll's technique of passing a K-wire from the posterior lateral corner of the talus (in the posterior part of the incision) longitudinally toward the talar head (Fig. 22–48). The pin is used as a joystick to rotate the talar body internally while the navicular is pushed into abduction and onto the true talar head. The reduced joint is pinned, and the pin is pushed out onto the dorsal aspect of the foot (for later removal). The forefoot is now reduced to the talus. The surgeon must ensure that the reduction is anatomic and that no rotation of the navicular has occurred due to pivoting on lateral soft tissue or calcaneal obstruction. Otherwise a dorsal subluxation of the navicular will ensue, leading to recurrent cavovarus.[65]

Normally, accurate talonavicular reduction produces calcaneocuboid reduction, which is recognized from the flattened or overcorrectable lateral column and border of the foot. If the lateral border is not straight but remains with

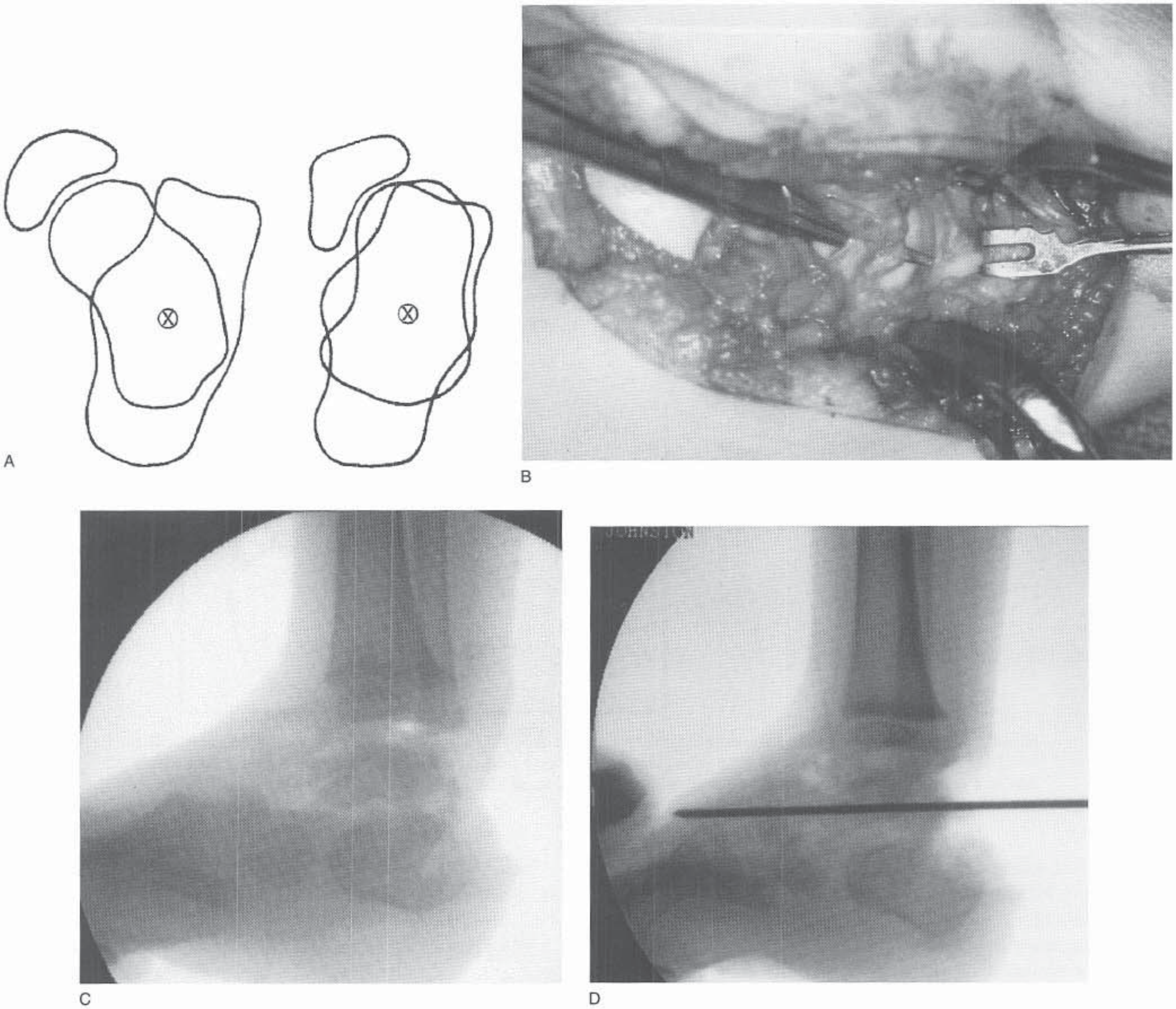


FIGURE 22–51 **A,** Uncorrected deformity, right. In order to gain rotatory divergence of the talus and calcaneus, all peripheral connections between the two bones, except the interosseous ligament (⊗), must be released, including the anterior talocalcaneal ligaments. **B,** Release of anterior talocalcaneal structures dissected by the hemostat (heel is to left, toes to right). The talonavicular joint is distracted by the two-prong rake. The talar head is visible. **C,** Intraoperative radiograph obtained after posterior and talonavicular release but before complete medial and anterior talocalcaneal release. As evidenced by the shortened-appearing calcaneus and posterior displacement of the fibula, the talus and calcaneus are still medially rotated together, with inadequate rotatory divergence. The fibula appears more posterior because the hindfoot remains medially rotated, and the foot and ankle are positioned for a true lateral view of the foot. **D,** Radiograph obtained after medial and anterior talocalcaneal release and reduction of the talonavicular joint. The calcaneal projection now shows normal length and pitch, indicating a true lateral rather than medially rotated position. This is confirmed by the normal lateral view of the fibula, which is no longer posteriorly displaced in external rotation. The talocalcaneal relationship has now diverged appropriately.

forefoot-medial curve, a calcaneocuboid capsulotomy from the lateral side and a Lichtblau[66] or other resection to shorten the lateral column must be considered (Fig. 22–52). Similarly if dynamic forefoot cavovarus is present, long flexor tendon lengthenings should be performed. Once these tendons are lengthened (typically 1 to 1.5 cm), the forefoot can be passively corrected. The lengthened and repaired tendons are replaced in their original sheath grooves (the flexor hallucis longus posterior to the bundle, the flexor digitorum longus anterior) and the sheaths are partially repaired with 5-0 suture to act as a checkrein and keep the tendons in place (Fig. 22–53).

The tibialis posterior is repaired with tension and replaced in its sheath. Repair of a checkrein here is helpful to avoid adhesions. Finally, the Achilles tendon, if lengthened, is repaired with the foot in slight ("gravity") equinus, to encourage maximal triceps function. Several interrupted sutures between the overlapped surfaces of the tendon are tied to prevent stretching or rupture of this most important of tendons.

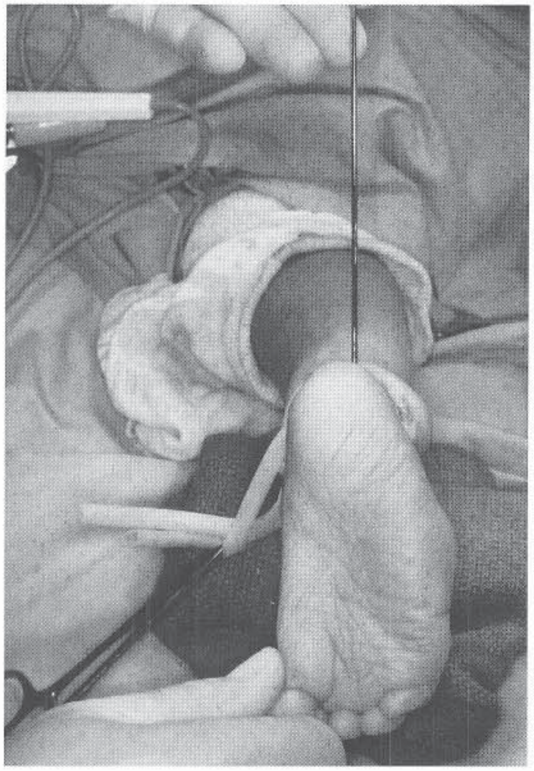


FIGURE 22–52 Incomplete correction of the midfoot in spite of calcaneocuboid capsulotomy. The lateral column remains too long. A lateral column shortening is indicated.

Skin closure is done with minimal tension, allowing the wound to gap open, up to 10 mm, if necessary, to avoid necrosis of skin edges. If the original deformity was severe, the foot must be left with residual equinus in the immediate postoperative cast or splint, with the cast changed in 10 to 14 days (usually under anesthesia) to correct residual equinus once the wound has healed partially.

Immobilization of the freshly operate clubfoot is as important as the operative technique itself, as a foot poorly positioned in a cast or splint will heal in that position. Although it may be necessary to leave some residual equinus because of skin tightness posteriorly, the other components of the deformity must be correctly positioned to avoid early recurrence. Thus the foot should be externally rotated by simultaneously abducting the forefoot and pushing the posterior calcaneus away from the fibula. The surgeon holding the correction must immobilize the tibia-fibula unit in *internal* rotation by grasping just distal to the knee and externally rotating the foot against the tibia-fibula position while the plaster hardens. As the cast or splint hardens, additional molding against the first metatarsal medially helps correct forefoot adduction, and additional molding against the cuboid plantar surface everts the midfoot. At the 2-week cast change, the foot is dorsiflexed to neutral by pushing the first metatarsal upward (to correct cavus) while maintaining the externally rotated position of the entire foot.

Because of concern about immobilization stiffness and atrophy, casting is continued only until soft tissues have healed, generally 4 to 6 weeks. Pins are removed at 3 to 4 weeks, with ambulation encouraged immediately in a short-leg walking cast.

Postoperative Complications

LOSS OF CORRECTION. Loss of correction with recurrence of deformity is a recognized scenario in clubfoot surgery. Wound or cast complications may necessitate a change in the postoperative plan of treatment. Most commonly the loss of correction involves inadequate postoperative position due to a cast becoming too loose once postoperative swelling has resolved. Failure to recognize and change a loose or inadequate cast—usually with equinus positioning—will invite recurrence. The surgeon must be prepared during the first 3 to 4 postoperative weeks to change the cast, under anesthesia if necessary, to maintain the corrected position. After the first 4 weeks, cast changes will likely be ineffective in regaining dorsiflexion or external rotation that has been lost, and so if a cast change is done in an attempt to regain position successfully, it must be done early.

Maintaining position in spite of wound complications follows the principle of obtaining maximum correction by the first cast change. Although wound dehiscence and infection

FIGURE 22–53 Replacement of the lengthened flexor hallucis longus tendon in its original sheath location at the posterior margin of the talus. A check rein repair (*arrow*) maintains the biomechanical function of the lengthened tendon. (The neurovascular bundle is under the retractor to the right.)

(including infection of the pin tracks) are uncommon in a first-time operation, if there is occasion to need wound access for dehiscence with secondary infection, the foot position must be maintained while wound healing by secondary intention proceeds. If a cast window threatens the integrity of the foot positioning, the cast should be replaced, with a new window then created. If the foot position is maintained during resolution of wound problems, recurrent deformity, and an even more difficult revision operation, may be avoided.

Pin tract infection can be a real dilemma, because premature pin removal obviously can lead to loss of correction. Dorsal subluxation of the navicular can be traced to premature talonavicular pin removal, especially if lateral tethers to the navicular have not been adequately released.[65] The ensuing shortening of the medial column, forefoot equinus, and supination usually produce a cavovarus deformity that will need revision. Thus, if a pin track becomes infected within the first 4 weeks of postoperative management and retention of the pin is felt to be critical to maintain correction, vigorous pin care and antibiotics should be administered to try and maintain the fixation until the normal time for removal.

DORSAL SUBLUXATION OF THE NAVICULAR. This condition, producing a shortened cavovarus foot, has been reported frequently following Turco procedures.[75,89] Simons[95] and Tachdjian[99] blamed failure to release the tibionavicular ligament and dorsolateral talonavicular capsule for the complication. Kuo and Jansen, however, found this complication just as frequently following a Carroll-type subtalar and calcaneocuboid release as following Turco procedures.[65] It should be apparent that failure to release the important tethering structures of the navicular, failure to accurately reduce the navicular head of the talus, or loss of talonavicular reduction as a result of premature pin removal are technical errors that have nothing to do directly with the type of procedure performed. Anatomic analysis of the deformity has shown it to be a rotatory subluxation, with the medial edge of the navicular rotated superiorly while the lateral edge is tethered (by talonavicular, naviculocuneiform, and cubonavicular ligaments).

Should revision surgery be indicated for the resulting cavovarus foot, an attempt at reduction of the navicular is appropriate under the age of 6. A midfoot release, beginning on the lateral side (calcaneocuboid, cubonavicular, and lateral, dorsal, and medial talonavicular), combined with repeat plantar release and possibly tibial anterior lengthening, will be necessary to mobilize the navicular for reduction. Derotation following dorsolateral release with inferior pressure may allow reduction, and the talonaviculocuneiform alignment should be maintained by internal fixation. In the older child, reduction and concomitant medial column lengthening may not be possible without more extensive surgery, including bone resection laterally for shortening.

VALGUS OVERCORRECTION. A so-called overcorrected foot, with excessive hindfoot valgus and usually forefoot abduction and pronation, is a significant complication of surgical release that unfortunately leads to further surgery due to pain (Fig. 22–54). The typical background for this complication involves two scenarios, both of which illustrate what *not* to do in clubfoot treatment. First, the surgeon is unable to obtain or is dissatisfied with the intraoperative correction, and cuts the interosseous ligaments in the presence of a severe internal talar rotational persistence; second, the postoperative position is deemed unsatisfactory due to forefoot adduction or heel inversion, and aggressive casting to evert the hindfoot and abduct the forefoot is carried out to redress the unsatisfactory position. Overcorrection has also been seen in feet treated by external fixation, in which powerful deforming force could be applied. In our opinion, the overcorrected foot results from a horizontal breach in the foot, primarily through the subtalar joint, where the talus is still relatively tethered in internal rotation due to incomplete posterolateral and posteromedial talofibular and deltoid release, and the subtalar joint is completely released and unstable. The calcaneus is then excessively translated laterally during improper cast maneuvers postoperatively, where the heel is everted only from medial molding, rather than derotated by pushing the tuberosity away from the fibula from a lateral position (Fig. 22–54D). Additional deformity is produced by vigorous abduction molding, which subluxes the midfoot laterally, especially when the talonavicular joint has either not been internally fixed or when one final aggressive cast is applied following pin removal. Finally, since equinus may also appear uncorrected in this postoperative period in which the surgeon is desperately trying to obtain more correction, the forefoot may be dorsiflexed selectively, adding dorsal subluxation to the horizontal breach, and possibly rupturing the heel cord as well. The result is the valgus, hyperabducted foot, with weak plantar flexion due to heel cord rupture or weakening due to loss of height of the heel in a laterally subluxed, breached subtalar joint.

Correction of the overcorrected foot is usually required because of pain from excessive medial arch weightbearing, lateral ankle impingement, or weakened triceps. Restoring heel height and triceps function can be attempted by an opening wedge lateral subtalar fusion. Forefoot realignment requires medial column shortening and lateral column lengthening through the subluxed joints, and thus the entire procedure may best be accomplished via a triple arthrodesis. Through a traditional lateral approach, a bone graft is required to prop open the subtalar joint and restore heel height and alignment. The navicular is usually in a fixed position lateral to the talar head, which may still be internally rotated as a result of talar undercorrection. Thus a second medial incision over the prominent talar head is recommended so that adequate exposure can be obtained to *shorten* the medial column by resecting part of the talar head and reducing the navicular by medial displacement. Since the lateral column may also need lengthening to correct midfoot abduction, a bone graft in the calcaneocuboid joint may also be required. Internal fixation of all three joints is recommended, especially if significant opening wedge grafts are being used (Fig. 22–55).

Triple arthrodesis for valgus deformity is required when the midfoot is stiff and simple hindfoot correction or lateral column elongation will not correct the excessive forefoot abduction and pronation. Symptomatic weightbearing on the medial arch should be unresponsive to nonoperative methods before the surgeon resorts to triple arthrodesis. Although pseudarthrosis is less common for valgus triples than for varus, there is a definite tendency to undercorrect a valgus triple,[15,90,106] probably due to a conscious avoidance of overcorrection into varus and the "settling" of opening wedge grafts as they incorporate. Despite a tendency to undercorrect, however, most patients are improved with

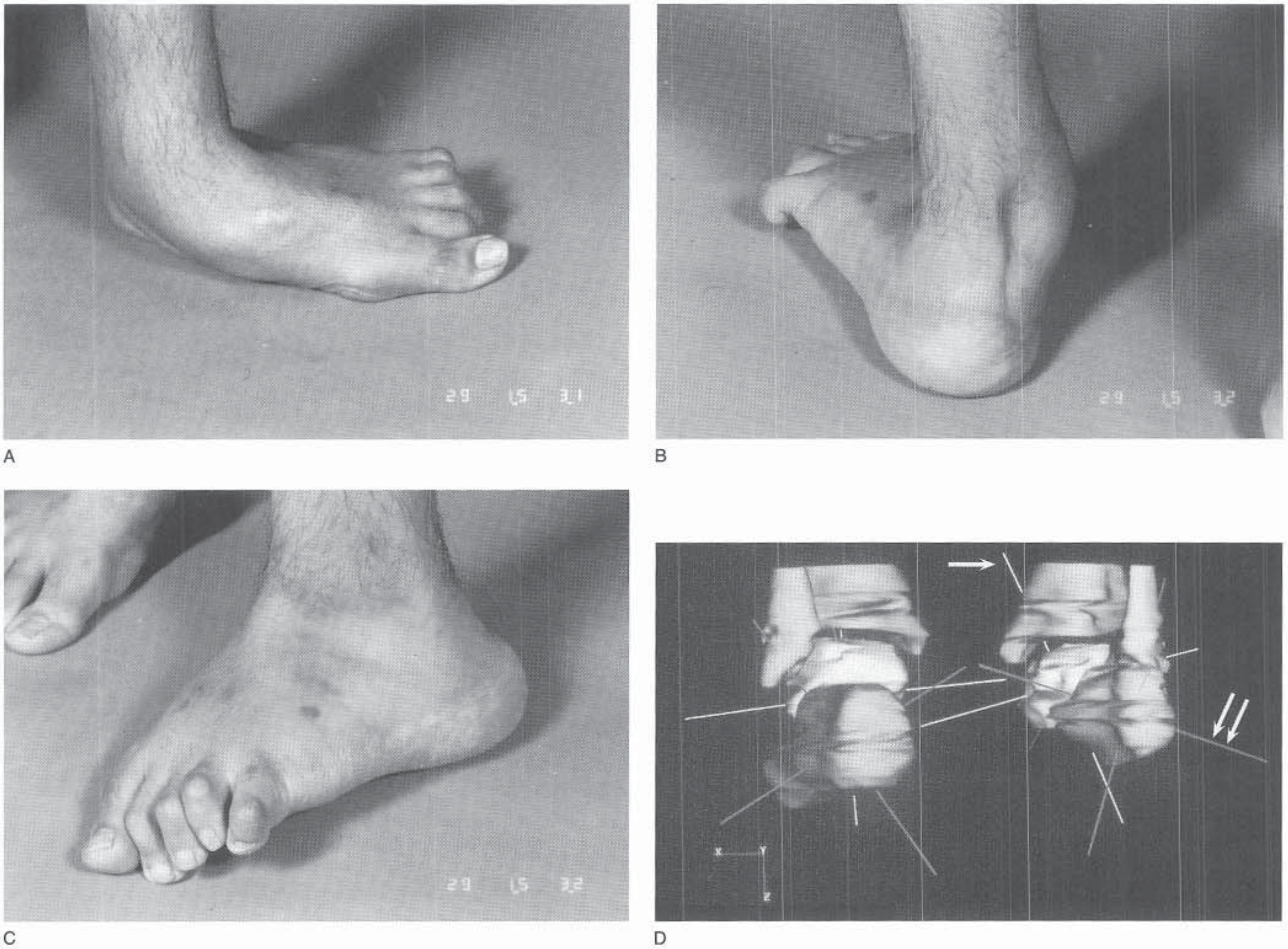


FIGURE 22–54 **A** to **C**, Valgus overcorrection. Due to hyperpronation and abduction of the midfoot and forefoot, weightbearing is excessive on the medial border of the foot. Lateral ankle impingement at the fibula is caused by horizontal translation of the calcaneus under the talus. The resulting loss of normal heel height produces additional triceps surae weakness over that caused by TAL. **D**, Three-dimensional reconstruction (from CT images) of the right foot valgus overcorrection (posterior view). The loss of heel height and the horizontal translation of the calcaneus laterally are obvious. Note the talus vertical moment of inertia (*arrow* points to axis) indicates persistent pronation-intorsion—that is, the tibiotalar clubfoot deformity is uncorrected. Similarly, the longitudinal axis of the calcaneus (*double arrows*) is internally rotated, an uncorrected clubfoot position exacerbated by eversion/valgus corrective force applied during casting, pushing the calcaneus toward the fibula rather than rotating the posterior tuberosity away from it.

relief of weightbearing on the medial arch and decompression of the lateral impingement.

Ultimately, any foot subjected to a triple arthrodesis will exhibit radiographic degenerative changes in adjacent joints, particularly the ankle,[3,4] after 10 years of follow-up or more. For this reason, extra-articular correction of valgus deformity has been recommended by Rathjen and Mubarak, combining medial column shortening, lateral column lengthening, and medial translation of the calcaneus, in an effort to avoid fusion of any joints.[84] Although radiographic correction was noted, functional follow-up was insufficient to determine whether joint sparing surgery was beneficial as hypothesized. Interestingly, varus foot correction was not as successful the same three sites of osteotomy were used in reverse (medial column lengthening, lateral column shortening, and lateral translation of the calcaneus; Fig. 22–56), because of the relative stiffness of varus feet compared to the relative flexibility in valgus deformities. The valgus deformities addressed successfully by extra-articular osteotomies were less rigid than the stiff, symptomatic, overcorrected clubfeet discussed here, which may still be best managed by triple arthrodesis.

DORSAL BUNION. This deformity can be considered a complication of clubfoot surgery because the underlying muscle imbalance required to produce the deformity is caused by some of the surgical maneuvers for clubfoot correction gone awry. Traditionally, dorsal bunion (hallux flexus) is thought to occur when the depressing strength of the peroneus longus on the first metatarsal is lost, either through disease (e.g., poliomyelitis) or iatrogenetically (scarring or division), in the face of an unopposed first metatarsal elevation by the tibialis anterior. In clubfoot, dorsal bunion probably occurs due to overpull of the long and, especially, short great toe flexors in the foot with weak plantar flexion (calcaneus gait). McKay has emphasized the flexor hallucis brevis and

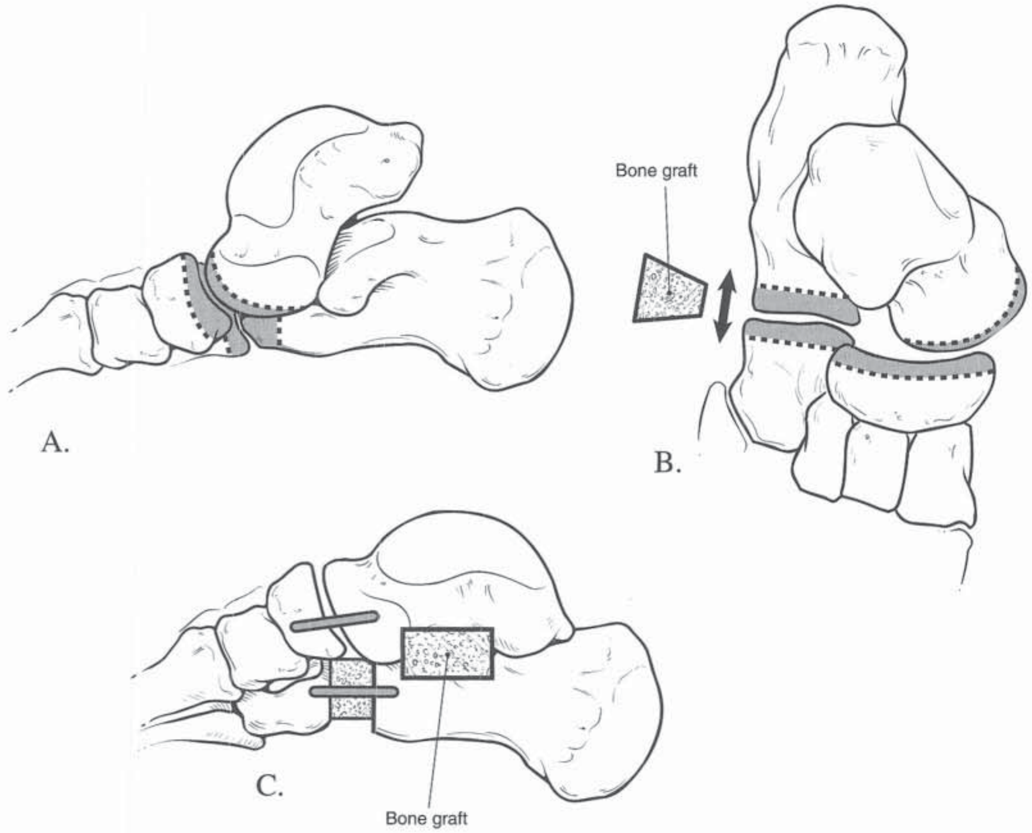


FIGURE 22–55 **A** to **C**, Triple arthrodesis for overcorrected valgus deformity. For an overcorrected clubfoot, opening wedge bone grafts in the lateral column and subtalar joints may be required to restore heel height and lateral column length.

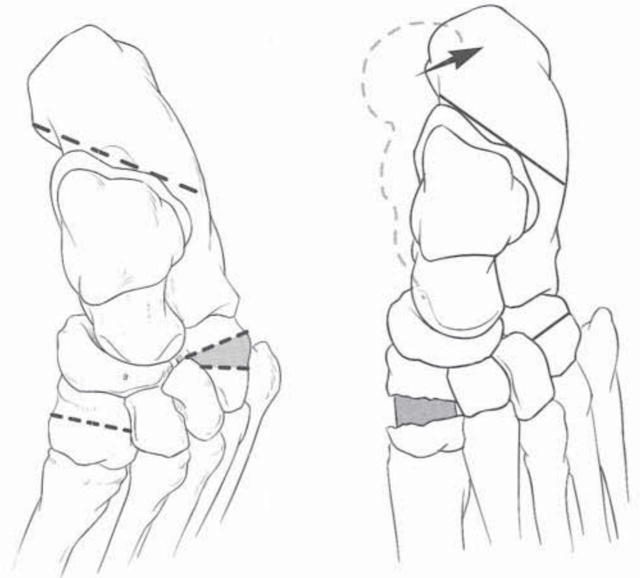


FIGURE 22–56 Cuboid wedge removal/reversal to first cuneiform can be combined with calcaneal osteotomy for correction of varus deformities after age 6 years. (After Rathjen KE, Mubarak SJ: Calcaneal-cuboid-cuneiform osteotomy for the correction of valgus foot deformities in children. J Pediatr Orthop 1998;18:775.)

abductor hallucis as responsible for the hallux flexus.[73] Thus, in the postoperative clubfoot with weak triceps, the flexors halluces (longus and brevis) overact as part of an effort to compensate for weak plantar flexion, and with a sectioned or scarred peroneus longus, the flexed great toe MTP joint worsens if the tibialis anterior is functioning unopposed by adequate plantar flexors and first metatarsal depressor.[58]

In the clinical scenario of calcaneus gait, a dorsal bunion often presents in the overcorrected valgus foot with poor triceps and a horizontal breach deformity. The peroneus longus may be functionally inadequate in the rigid abducted foot because of lack of excursion. Alternatively, it may have been inadvertently divided during an earlier procedure when the latter is performed in a supine patient and the posterolateral release is performed through a posteromedial incision. In a fixed hallux flexus, abnormal pressure on the tip of the flexed great toe or a painful callus on the dorsum of the first metatarsal head (the "bunion") is likely to produce symptoms before such problems as excessive medial arch weightbearing, lateral impingement, or calf weakness become symptomatic.

Treatment for a dorsal bunion involves realignment of the first ray, both proximally and distally. The flexed MTP joint is released by volar, medial, and lateral capsulotomy, lengthening of the flexor hallucis longus tendon, and release or dorsal transfer of the flexor hallucis brevis (to become an MTP *extensor*). The elevated metatarsal shaft is depressed by a proximal plantar closing wedge osteotomy (Fig. 22–57).

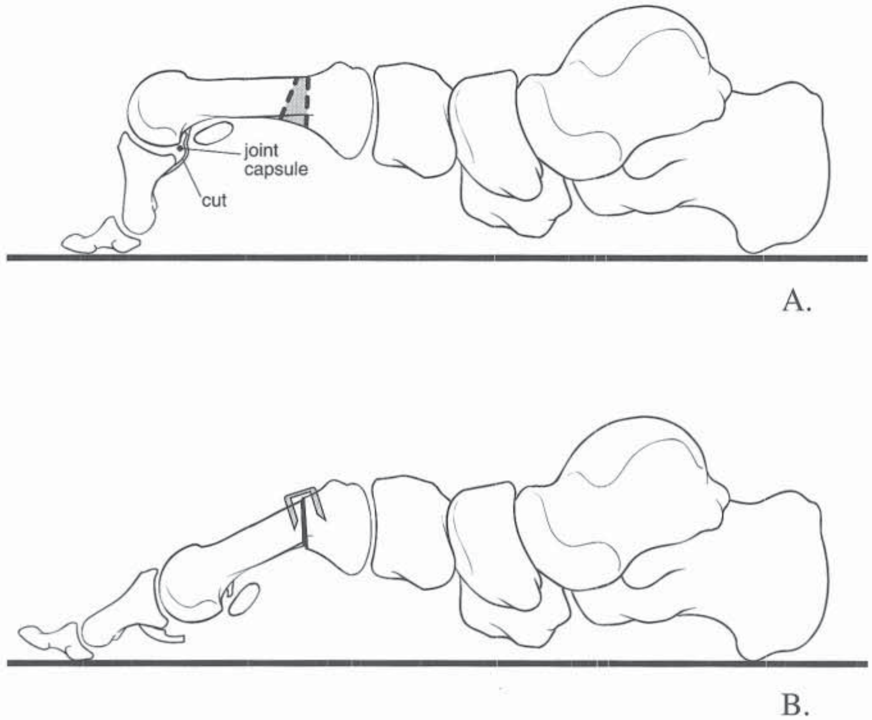


FIGURE 22–57 **A** and **B**, Dorsal bunion correction. The MTP joint is released to reduce on the first metatarsal head. The first ray is corrected by a plantar closing wedge osteotomy.

The tibialis anterior, if obviously contracted or overpulling, is either lengthened or transferred laterally to the second metatarsal. This first ray realignment must be done in conjunction with whatever reconstruction may be necessary for the rest of the foot, since it is likely that there will be other postoperative residua (calcaneus, hyperpronation, horizontal breach with valgus) that will need to be addressed.

Revision/Secondary Procedures. Recurrence of clubfoot deformity following what appeared to be initially a satisfactory outcome is a discouraging event for all concerned. Faced with what seems to be true recurrence, the surgeon must candidly self-assess the original procedure, because of the generally accepted belief that the majority of "recurrences" are merely a *persistence* of deformity that was never completely corrected in the first place. If this self-assessment concludes that there was a complete correction followed by true recurrence, then nonidiopathic causes, such as a neurologic disorder (e.g., occult spinal dysraphism), must be considered and diagnostic workup undertaken in an effort to prognosticate and even correct the neurologic imbalance responsible. Electrophysiologic testing or MRI of the spinal cord are two such tests to be considered if true recurrence is present.

The prevalence of repeat surgery following the initial soft tissue release in infancy is estimated to be in the range of 10 percent. Obviously, not all feet with residual deformity or muscle imbalance undergo additional surgery, as the surgeon's and parents' perception and acceptance of the postoperative result can differ. In selecting revision procedures, the surgeon must strongly consider the inevitable additional stiffness and muscle weakness that result from repeat surgery and immobilization, and thus the primary goal of additional procedures must be to achieve the eventual *realistic* foot position with the least possible number of procedures. It is therefore justified to accept, for example, recurrent deformity in a 3-year-old for several years, and if possible delay for one definitive final surgery at 10 years of age, when bony correction can be accomplished without significant further soft tissue dissection and immobilization.

In general, revision surgery should address a specific problem or deformity that has become unacceptably symptomatic, producing functional problems and pain. Functional problems include poor foot position (such as supination/inversion) or an excessive internal foot progression angle (producing painful lateral ray weightbearing), and muscle imbalance/weakness, such as triceps surae incompetence (producing calcaneus gait and calf pain). Revision surgery has a greater likelihood of success if such a single problem can be identified and addressed, rather than simply taking the patient to the operating room for the nebulous "repeat clubfoot release," a euphemism for a second less than complete correction.

ANTERIOR TIBIAL TENDON TRANSFER. Transfer of the tibialis anterior insertion—either the entire tendon or as a split transfer—is indicated when there is dynamic inversion/supination of the midfoot, especially in swing phase, that produces weightbearing on the lateral aspect of the foot at initial stance. The goal is to eliminate the supinated position for the initiation of stance. Ponseti and Goldner have advocated this be routinely performed as part of the index operative procedure, as well as using it as a secondary procedure.[82] The dynamic deformity should be demonstrated statically by observing midfoot supination or excessive first ray elevation when the patient attempts to dorsiflex the foot voluntarily, and can be further identified by formal gait analysis. If the foot is otherwise mobile and can be placed plantigrade for stance, this may be the only procedure necessary. Fixed deformity of the foot must be corrected prior to consideration of anterior tibial tendon transfer.[29,30]

If the split transfer is used, the lateral arm of the transfer should be rerouted subcutaneously from the ankle retinaculum at the distal tibia and reinserted in the cuboid or lateral cuneiform area through a drill hole and anchored over a button on the plantar surface.[45] Tension in the lateral arm should be sufficient to produce slight eversion/pronation statically. If the entire tendon is transposed, the insertion should be moved to the midline or just slightly lateral to midline, to give dorsiflexion without excessive abduction. Transfer of the entire tendon will produce a loss of dorsiflexion strength of one grade, which may become functionally important if there is residual equinus, and thus assessment of posterior ankle contracture and the need for its release must be made, or else a more significant drop foot may result.

Anterior tibialis transfer with *lengthening* may be indicated as part of revision for a postoperative dorsal bunion, where the first ray is excessively dorsiflexed. In this situation, the overpull of the tibialis anterior must be balanced with the plantar flexing strength of the peroneus longus on the first ray, which will most likely be weak or absent. See the previous discussion of dorsal bunion.

TRANSFER FOR INSUFFICIENT TRICEPS SURAE (CALCANEUS GAIT). Overlengthening of the Achilles tendon, or triceps insufficiency secondary to inadequate excursion from scarring, is notoriously difficult to reconstruct and is best *prevented* rather than reconstructed. Careful attention to the technique of heel cord lengthening and tensioning for repair at the index procedure and avoidance of heel cord rupture during postoperative manipulation and casting are essential to avoid this complication. However, as long-term functional evaluations[5,61,68] have shown, plantar flexion weakness is universal following even the most meticulous technique in an otherwise "good" result. The plantar flexion insufficiency may not be apparent for years, until the child has grown sufficiently in body size so that the weakness is clinically exposed, at which time the strength of the muscles available for tendon transfer may be inadequate to replace the missing triceps. The surgeon must diagnose plantar flexion weakness as early as possible if muscle transfer is to have any chance of being effective.

Muscles available for transfer to reconstruct the triceps-Achilles complex should be phasic with the latter, and thus include the peroneals, tibialis posterior, and long toe flexors. Laterally, the peroneus brevis can be divided distal to the fibula and the proximal end rerouted to the calcaneus tuberosity (Figs. 22–58A to C). Tendon-to-bone transfer is preferred, anchoring the tendon through a drill hole in the calcaneal tuberosity to a button on the plantar surface of the heel. The distal stump of the brevis should be tenodesed side-to-side to the longus to maintain eversion power. Medially, the tibialis posterior or flexor hallucis longus should be rerouted in a similar fashion and interwoven with the residual Achilles tendon, if present, or anchored to bone (Figs. 22–58D to F). The tibialis posterior is often scarred and nonfunctional as a result of the index procedure, and a discrete tendon may not be discernible. However, the author has found that by dissecting proximally above the medial malleolus, the original tibialis posterior tendon can in fact be located, mobilized, and transferred distally toward the calcaneal insertion of the original Achilles. If the tibialis posterior is unavailable, the flexor hallucis longus is rerouted from above the medial malleolus to the Achilles insertion. Due to the intertendinous connections between flexor hallucis and flexor digitorum longus distally at their decussation, flexion power of the great toe does not appear to suffer from this transfer.

Traditionally the tibialis *anterior* has been transferred to the heel (through the interosseous membrane) in cases of paralytic/neuromuscular calcaneus.[8,81,102] In clubfeet, this procedure has been reported sparingly but with some success.[105] The author has found, however, that although the calcaneus gait is improved by such a transfer in clubfeet, the resulting foot drop from the lack of active dorsiflexion becomes an unacceptable secondary problem, often making the patient brace dependent to avoid tripping and awkward due to the steppage gait (increased hip and knee flexion in swing phase). Consequently, transfer of the tibialis anterior in nonparalytic conditions such as clubfoot is *not* recommended.

If no other procedures (for other residual deformity) are to be performed, transfer of the peroneal and tibialis posterior or flexor hallucis longus tendons to the heel is best performed with the patient prone through one longitudinal incision centered over the Achilles, or alternatively through separate posterolateral and posteromedial incisions spaced appropriately. Anterior ankle capsulotomy and lengthening of a contracted tibialis anterior may be necessary if passive plantar flexion to 20 degrees is not present. The transfers should be tensioned so that the foot is passively held in equinus, and immobilized for 6 to 8 weeks of nonweightbearing. Thereafter a solid AFO with dorsiflexion stopped at neutral should be continued for an additional 4 months, to attempt to prevent stretching out of the transfers, with active plantar flexion exercises performed nonweightbearing with the brace off. Although *normal* plantar flexion strength can never be realized by tendon transfer, several patients have developed toe-up ability, especially when triceps reconstruction has been performed prior to age 6. Usually a transfer performed after this age must also be accompanied by bony procedures, such as calcaneal osteotomy or subtalar fusion for hindfoot valgus, which frequently accompanies calcaneus deformity. Combining some posterior displacement of the calcaneal tuberosity,[76,87] where the transferred tendons will be anchored, with the medial displacement to address heel valgus is an additional feature that theoretically will increase the plantar flexion moment arm of the transferred muscles (see the discussion under Calcaneal Osteotomy, below).

Bony Procedures

LATERAL COLUMN SHORTENING. A "recurrence" of clubfoot deformity following earlier surgical release requires an analysis

FIGURE 22–58 Muscle transfers for calcaneus gait (triceps insufficiency). **A** to **C**, The peroneus brevis is exposed distal to the fibula, released from its insertion, mobilized proximally and rerouted through a drill hole in the calcaneal tuberosity, and sutured under tension to a button on the heel. **D** to **F**, The tibialis posterior (or flexor hallucis longus) is identified above the medial malleolus in relatively unscarred tissue. It is dissected distally as far as possible, cut, and transferred to the Achilles tendon, where it is sutured under maximum tension.

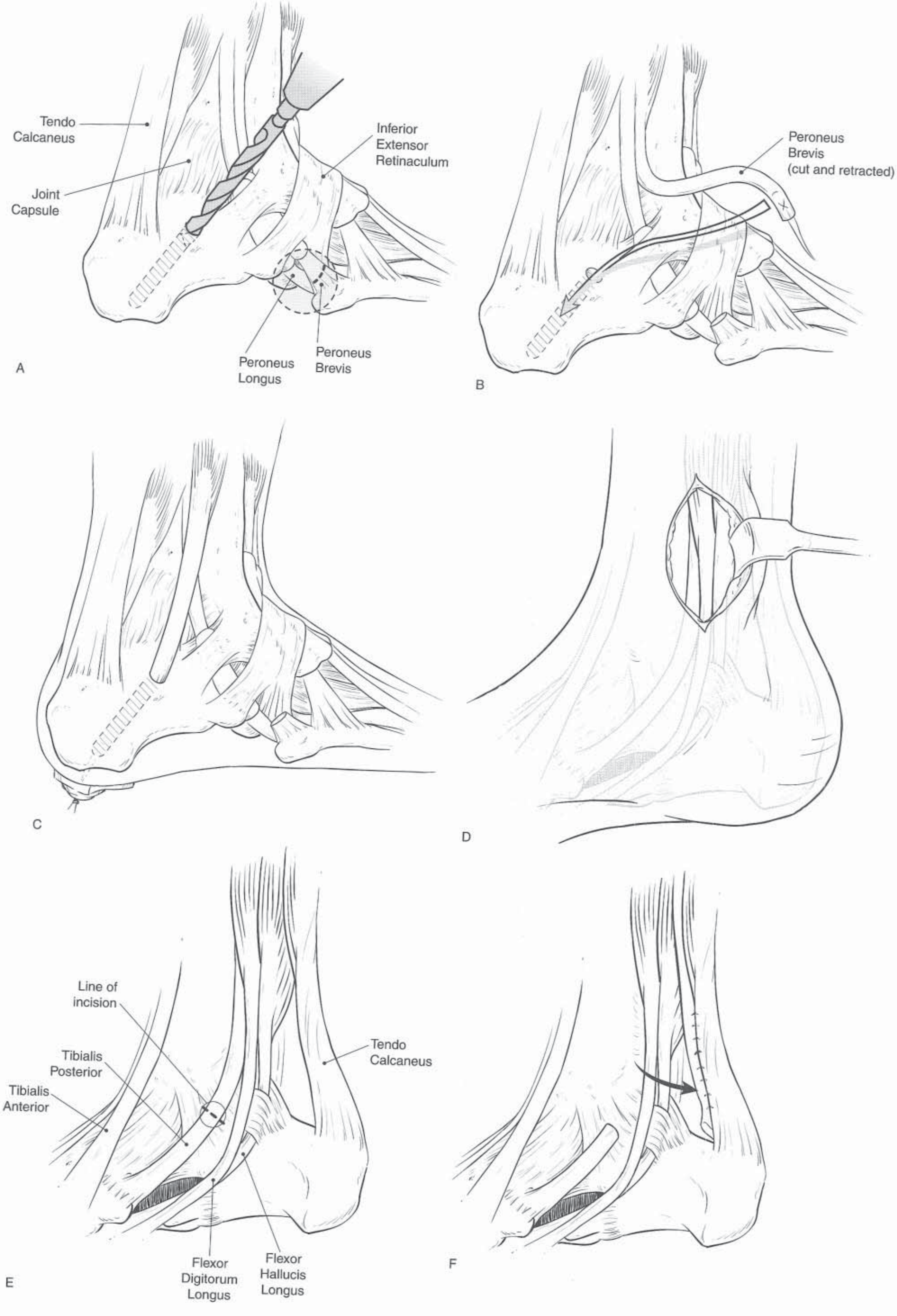


FIGURE 22–58 *See legend on opposite page*

of this secondary deformity. Frequently all components of the equinovarus deformity seem to have recurred, but the "essential" deformity consists of a length disparity between the medial and lateral borders of the foot. Any attempt to abduct and externally rotate the forefoot in relation to the hindfoot, as well as any attempt to correct forefoot supination, is resisted by medial contracture and excessive length of the lateral ray, where the cuboid is displaced medially on the anterior end of the calcaneus. Evans suggested that this obstruction to forefoot positioning by the length and adaptive obliquity of the calcaneocuboid joint was the essential lesion of clubfoot, and described a wedge resection of the calcaneocuboid joint to shorten the lateral column as part of the treatment of the relapsed deformity.[26] This approach, which combines a posteromedial release and lateral column shortening in one stage, is probably the most common procedure for recurrent clubfoot. Evans's procedure relies on concepts of midfoot (talonavicular and calcaneocuboid) dislocation[24,56] and by allowing reduction of the navicular on the talar head by the lateral column shortening, further relapse/recurrence was avoided. Heel varus was also corrected adequately in Evans's original series, although most investigators find that heel varus must often be formally corrected by either repeat subtalar release or calcaneal osteotomy.

The Evans procedure has become a standard technique for the recurrent clubfoot deformity in which the midfoot is clearly in varus due to talonavicular and calcaneocuboid medial displacement. It is also an ideal index procedure for the late presenting clubfoot. Other reviews[1,2] have confirmed this to be the procedure of choice between 4 and 8 years. Prior to age 4, calcaneocuboid fusion is more difficult to achieve, because of the large amount of cartilage in the two bones. In this instance, simple resection of the anterior end of the calcaneus as described by Lichtblau[66] or a shortening of the calcaneal neck proximal to the calcaneocuboid joint will achieve the desired shortening. Lichtblau's operation, essentially a calcaneocuboid arthroplasty, can be used whenever the lateral column is too long. Alternatively, cuboid decancellation[56] will preserve the joint, but it limits the amount of lateral column shortening if the cuboid is small. The actual amount of lateral shortening is determined intraoperatively and should be sufficient to allow talonavicular reduction, after adequate medial and/or posterior release, with little effort. The lateral edge of the foot should become a straight border rather than the rounded, "kidney bean" shape of the varus forefoot (see Fig. 22–52) after a lateral column shortening. The lateral wedge can be removed through a separate longitudinal incision over the calcaneocuboid joint (Fig. 22–59) or by simple extension of the lateral extent of the Cincinnati incision. The closure of the lateral wedge should be maintained by internal fixation, either with longitudinal pins that are removed, or with staples or lag screws for the formal calcaneocuboid fusion. Although formal fusion is generally believed to result in better long-term maintenance of correction,[37] long-term results of Lichtblau's procedure have also been gratifying, with the additional benefit of maintaining some calcaneocuboid motion in the majority of patients.[32]

Some patients with recurrent deformity have sufficient scarring that medial soft tissue release and subtalar release will be ineffective. In the patient more than 6 years old with varus forefoot position, lateral column shortening has been combined with medial column lengthening by removing a wedge of bone from the cuboid and transferring it to an opening wedge osteotomy in the first cuneiform (Fowler procedure) (see Fig. 22–56).[46,72] This approach has the advantage of avoiding formal fusion of joints and maintaining the relative length of the overall foot by dividing the correction between the two columns, rather than taking all of the correction from a lateral wedge resection and possibly shortening the foot excessively. Midfoot supination can be improved by utilizing a quadrilateral graft to obtain an opening wedge plantarward as well as medially. Because of the cartilaginous nature of the midfoot bones, the opening wedge medially can be technically difficult prior to age 6 to 8 years.[46,84] However, if technically feasible, this approach does not require soft tissue release to achieve correction of the bean-shaped foot.[72]

CALCANEAL OSTEOTOMY. In the foot with fixed heel varus with or without other significant residual deformity, an opening or closing wedge osteotomy or a lateral displacement osteotomy can be used for this specific indication. The essential prerequisite for an opening wedge osteotomy is that there be sufficient ossification of the calcaneus to stabilize a bone graft. The advantage of the calcaneal osteotomy as proposed by Dwyer is that subtalar motion is preserved, although in a recurrent deformity following soft tissue release, it is debatable whether significant subtalar motion will ever exist. Additionally, calcaneal osteotomy can be combined with other procedures, and it does not hinder the performance of a future triple arthrodesis, for example.

The opening wedge technique theoretically increases the height of the heel and may therefore require Achilles tendon or other posterior release to avoid producing equinus (Figs. 22–60A and B). Practically speaking, wound closure on the medial ankle can be compromised, especially in a recurrent situation where the osteotomy is being performed through a previous incision.[99] For this reason, a lateral closing wedge technique with some lateral displacement (Fig. 22–60C) is associated with less wound healing morbidity, but because it decreases the height of the heel to some degree, it also risks lateral impingement problems between the calcaneus and fibula. For effective lateral displacement—or a posterior displacement,[76,87] if calcaneovarus or valgus is being addressed (see discussion of triceps insufficiency, above)—the plantar fascia and muscle origins must be stripped or divided to fully mobilize the distal osteotomy fragment.

The osteotomy should be made roughly parallel to the subtalar joint and internally fixed, usually with threaded pins that are later removed. If an opening wedge technique is used, weightbearing must be delayed or risk collapsing the graft and losing correction. Tibial bone can be used, but tricortical iliac crest graft, due to its cancellous nature, is preferred because of faster incorporation.

Debate rages about the efficacy of calcaneal osteotomy, both in terms of effective heel position correction and in terms of the effect on the midfoot. Dwyer claimed that correction of heel varus alone could produce gradual correction of forefoot equinus and midfoot varus. Other investigators[20,27,99] have not confirmed this, and calcaneal osteotomy as an isolated procedure for recurrent or residual varus is rarely performed at our institution, as the dynamic gradual

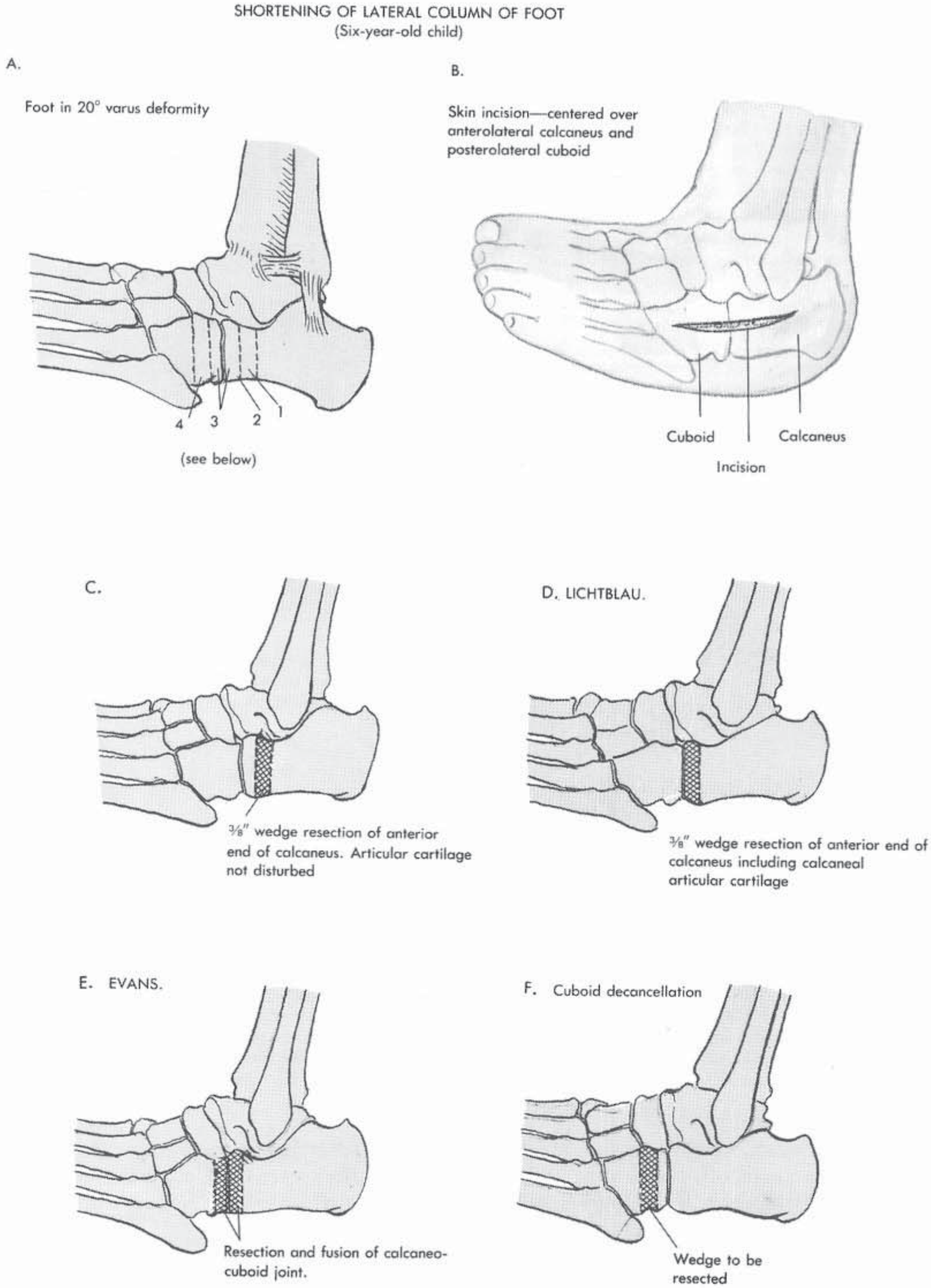


FIGURE 22–59 Shortening of the lateral column of the foot. **A,** Lateral view of the foot showing different levels of osteotomy: (*1*) vertical osteotomy of the anterior part of the calcaneus; (*2*) excision of the anterior end of the calcaneus (Lichtblau procedure); (*3*) excision of the calcaneocuboid joint and fusion (Evans operation); (*4*) wedge resection and enucleation of cuboid bone. **B,** A 4-cm incision is made that is centered over the dorsolateral aspect of the calcaneocuboid joint. The peroneus brevis tendon is identified and retracted plantarward. The extensor digitorum brevis muscle is elevated off the cuboid bone and retracted dorsally and medially. **C,** Vertical osteotomy with a wedge resection of bone based laterally for shortening the calcaneal neck. The calcaneocuboid joint is preserved. **D,** Calcaneocuboid arthroplasty (Lichtblau procedure). This is indicated in children less than 6 years old who need lateral column shortening. **E,** Wedge resection and fusion of the calcaneocuboid joint (Evans procedure). **F,** Cuboid decancellation and wedge resection in older children preserves the articular surfaces. Lateral column shortening procedures should be internally fixed with smooth pins or staples to maintain alignment until healing.

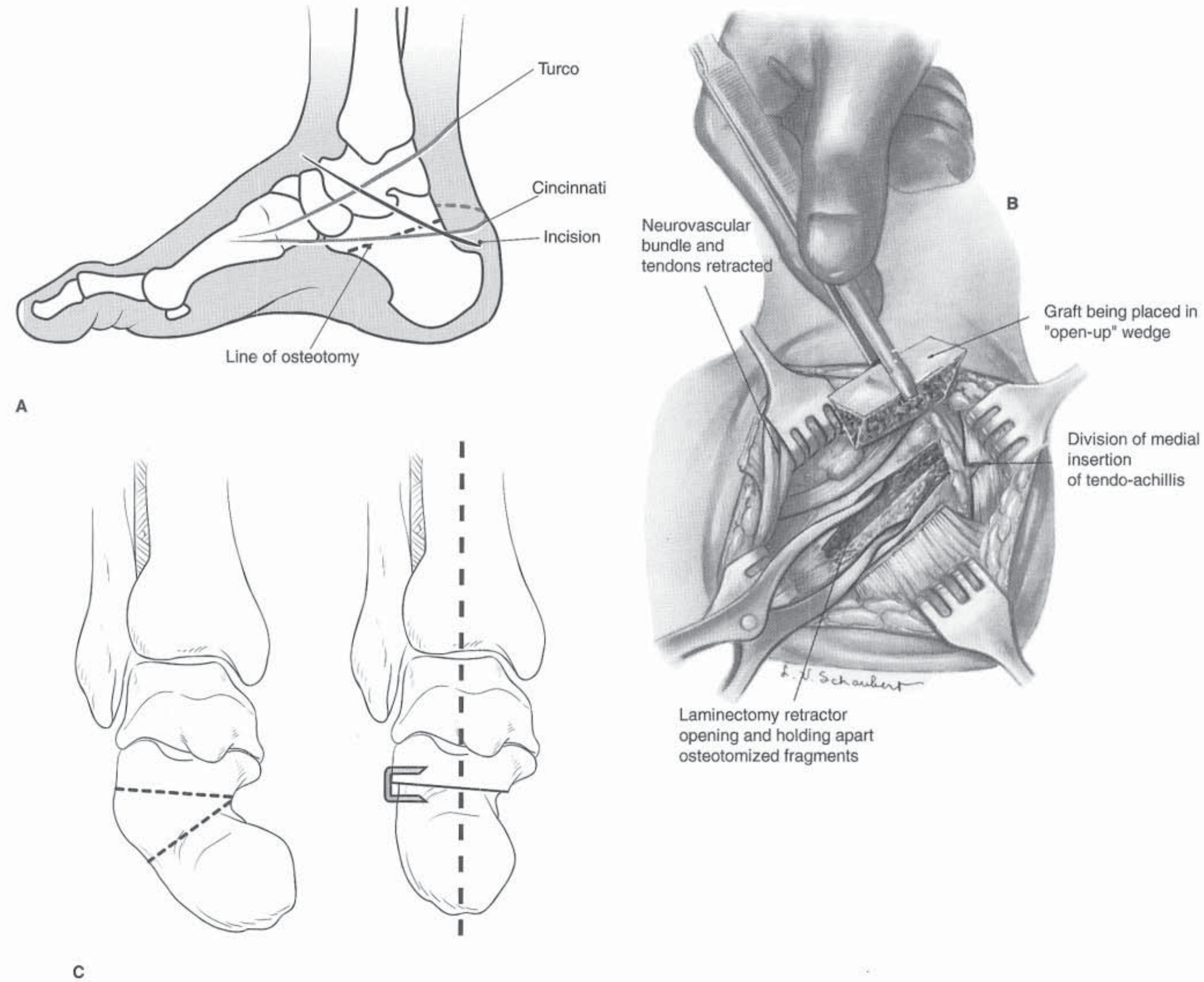


FIGURE 22–60 Medial osteotomy of calcaneus with bone graft wedge for corection of varus hindfoot—Dwyer technique. **A,** The skin incision is usually made perpendicular to previous incisions due to closure problems if made parallel (Cincinnati, Turco). The posterior extent should be to the superior edge of the calcaneal tuberosity (to allow TAL if necessary). The calcaneus is osteotomized roughly parallel to the subtalar joint. **B,** The osteotomy is opened with the aid of a lamina spreader. Achilles tendon lengthening and stripping of the plantar fascia and muscle origins from the distal fragment may be necessary to allow adequate correction by opening wedge osteotomy. Tricortical iliac crest graft is recommended. **C,** Calcaneal closing wedge osteotomy for varus of the heel.

correction is minimal at best. Correction of heel position in the older child is more permanently obtained by subtalar fusion (as part of a triple arthrodesis, for example), and so every effort should be made to delay interim procedures, such as calcaneal osteotomy, until an age at which definitive correction can be achieved. Although the author uses calcaneal osteotomy in conjunction with midfoot and forefoot procedures (such as first metatarsal or cuneiform osteotomy to correct supination/cavus and lateral column shortening to correct midfoot varus; see Fig. 22–56) in the child deemed too young for triple arthrodesis,[72] such a combination is rarely performed because of the "interim" nature of the procedure. It is better to eliminate the interim procedure and perform definitive correction at age 10 if possible. Thus, calcaneal osteotomy as an isolated procedure has limited utility, and calcaneal osteotomy in combination with other procedures for interim correction of recurrent hindfoot and midfoot deformity is unattractive, though sometime necessary, for the reasons just discussed.

SUPRAMALLEOLAR OSTEOTOMY. Persistence of a toe-in gait in an otherwise plantigrade foot is common, regardless of the surgical technique employed. Postoperative intoeing more than 2 standard deviations from normal has been documented by Yngve and colleagues in 48 percent of patients[110] and has been the indication for further surgery in 8 to 25 percent of postoperative patients in other series.[49,67] In patients with abnormal peroneal muscle histopathology, Loren and colleagues reported an increased incidence of internal torsional deformity, presumably due to the muscle imbalance.[67] An internal foot progression angle can result from several sources: true internal tibial torsion; medial spin of the hindfoot in the ankle mortise; and medial deviation of the forefoot, with or without true metatarsus adductus, where the medially deviated talar neck directs the forefoot into an internal foot progression angle.[7] In the younger child, residual internal foot progression angle should be observed for spontaneous correction, but if the toe-in gait persists for 2 years following clubfoot surgery, there is justification for correction to avoid the long-term secondary deformity at the knee—primarily valgus—seen in association with excessive internal foot progression angle.[5,61,68]

Should intoeing gait be severe and not resolve with 2 years of observation, correction by supramalleolar external rotation osteotomy can be effective,[49] and because it avoids further foot dissection, it does not contribute to further stiffness. The deformity should be secondary to persistent internal tibial torsion or hindfoot medial spin in the patient with previous subtalar release but with persistent medial rotation of the talus and calcaneus. In such a patient another subtalar release would likely be ineffective due to fixed bony position. Should the internal foot progression angle be due to medial forefoot deviation with talar neck deformity, or midfoot medial deviation, correction by foot procedures, such as lateral column shortening[26,66] and/or plantar fascia release,[13,100] directly addresses the pathoanatomy. Supramalleolar osteotomy generally is *not* effective for multiple plane corrections of residual clubfoot due to remodeling of the distal tibia by continued physical growth causing the deformity to recur. It should be reserved only for rotational correction.[77]

Goldner recommends 35 degrees of external rotation correction at a level just proximal to the distal tibial physis (Fig. 22–61). The fibula usually does not have to be osteotomized unless greater correction is needed. The tibial osteotomy is fixed with an oblique pin. Prior to wound closure, the vascular status of the foot *must* be ascertained by deflating the tourniquet, with adjustment of the amount of rotation should there be any sign of ischemia. In Goldner's series, two of 66 patients required a decrease in the amount of correction due to sluggish circulation, with no sequelae following refixation with less correction. All patients had successful union and maintained their rotational correction at follow-up.

Supramalleolar osteotomy to correct intoeing gait in an otherwise plantigrade foot has been a useful procedure at our institution. Because of the late knee valgus that is seen on follow-up of club feet, this procedure should probably be considered more frequently. It is unknown, however, whether earlier correction of an internal FPA foot progression angle will change the degree of knee valgus documented in follow-up studies.[5,61,68]

TRIPLE ARTHRODESIS. After the age of 10, management of residual deformity requires bony stabilization, not only to correct the remaining deformity, which will be resistant to soft tissue procedures, but also to *maintain* the correction. Triple arthrodesis has been standard orthopaedic procedure for producing and maintaining correction since it was first described in the 1920s.[47,85] A triple arthrodesis may be regarded as the ultimate salvage procedure, in that the surgeon is capitulating in fusion of movable joints, but older children with symptomatic recurrent deformity generally will not have foot joints that are functionally acceptable. Hence their sacrifice tends to be a moot point, especially if correction and a plantigrade, if stiff, foot is finally achieved.

Triple arthrodesis can be used for either varus or overcorrected valgus feet. The varus foot, with heel varus and midfoot supination, is inadequate for a weightbearing platform,[69] producing pain due to excessive lateral ray pressure, and often instability with lateral ankle pain or giving way due to rotational stress on ligaments.[97] For a valgus foot, the deformity per se may be fairly well tolerated because of a broad plantar weightbearing surface. Eventually pain over the plantar medial surface—the talar head—and lateral impingement symptoms due to the loss of the heel height and valgus translation of the calcaneus become the indications for surgical correction of the valgus foot.

Removal of appropriate wedges, based laterally, corrects the varus foot (Fig. 22–62). A classic lateral incision over the sinus tarsi and curved dorsomedially toward the talar head provides adequate exposure to align the foot with the ankle mortise. Care must be taken not to align the foot with, for example, the knee, because this will place the foot in a relatively supinated position if there is external tibial deformity and knee valgus.[80] Internal fixation is recommended, especially of the talonavicular joint, which is the most frequent joint to go on to pseudarthrosis.[15,80,106] In addition, internal fixation can maintain the corrected position in the face of postoperative cast loosening/splitting, required by the not infrequent swelling and sluggish circulation following triple arthrodesis in the multiply operated foot.

The most crucial aspect of correction of varus is to avoid *under*correction and leave the foot fixed in residual varus/supination. Thus, slight correction to valgus and pronation is favored over an undercorrected position.[80,97] Although radiographic pseudarthrosis has been reported in as many as 23 percent of cases,[3] a single pseudarthrosis may remain asymptomatic if the *other* joints are successfully fused. Meticulous attention to bony contact between joint surfaces and careful preparation and cartilage removal are the best insurance against pseudarthrosis. Precision in the performance of triple arthrodesis will be rewarded with a stable, corrected foot and a functional patient.

See the previous discussion under the section Valgus Overcorrection concerning valgus deformity after clubfoot surgery.

CORRECTION USING THE ILIZAROV TECHNIQUE. Since stretching and elongation of contracted tissue is fundamental to the management of clubfoot, it is not surprising that the methods of Ilizarov have been applied to the neglected or recurrent deformity, especially in the face of severe scarring. Several investigators in addition to Ilizarov himself[14,19,38,78,104] have reported the technique applied to clubfoot, combining multiple plane corrections by use of hinged distraction between a tibial frame and a foot frame, usually treating hindfoot and forefoot separately. Skeletal fixation is achieved via proximal and distal tibial rings; hindfoot fixation is achieved via crossed or nonparallel transverse wires in the calcaneus and/or talus, supplemented as necessary by an axial calcaneal half-pin fixed to a semicircular ring controlling the heel; and forefoot fixation is achieved transversely with wires through the metatarsals or cuneiforms via a semicircular ring over the dorsum (Fig. 22–63). Angular correction with lengthening can then be achieved between the forefoot and hindfoot, between hindfoot and tibia, or the entire foot can

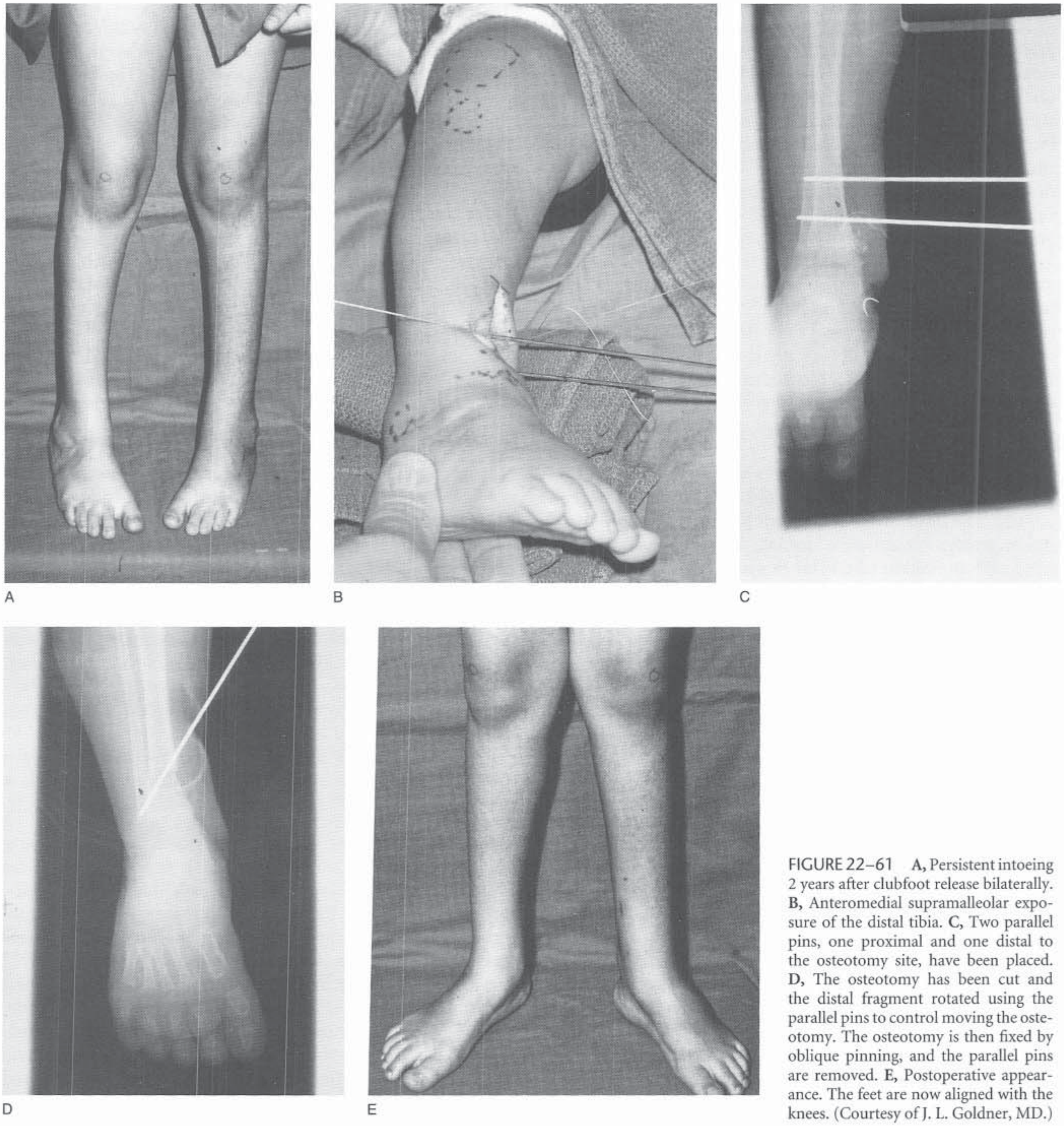


FIGURE 22–61 **A,** Persistent intoeing 2 years after clubfoot release bilaterally. **B,** Anteromedial supramalleolar exposure of the distal tibia. **C,** Two parallel pins, one proximal and one distal to the osteotomy site, have been placed. **D,** The osteotomy has been cut and the distal fragment rotated using the parallel pins to control moving the osteotomy. The osteotomy is then fixed by oblique pinning, and the parallel pins are removed. **E,** Postoperative appearance. The feet are now aligned with the knees. (Courtesy of J. L. Goldner, MD.)

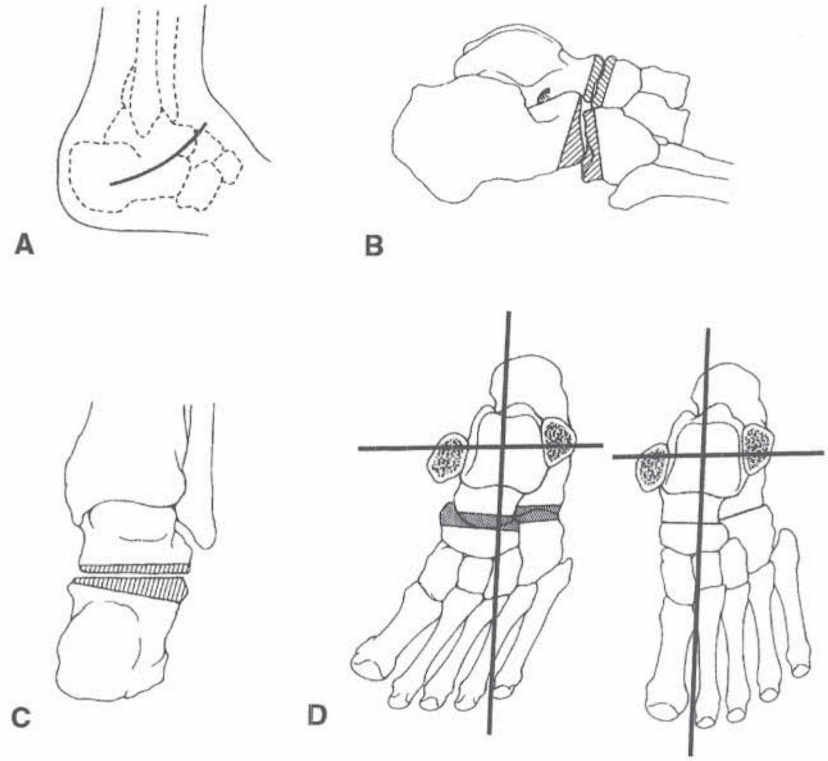


FIGURE 22–62 **A** to **D,** Triple arthrodesis for clubfoot. The wedges, based laterally, can be safely removed from the standard sinus tarsi incision. The foot must be aligned with the ankle mortise and not with the knee axis. (From Coleman SS: Complex Foot Deformities in Children. Philadelphia, Lea & Febiger, 1983.)

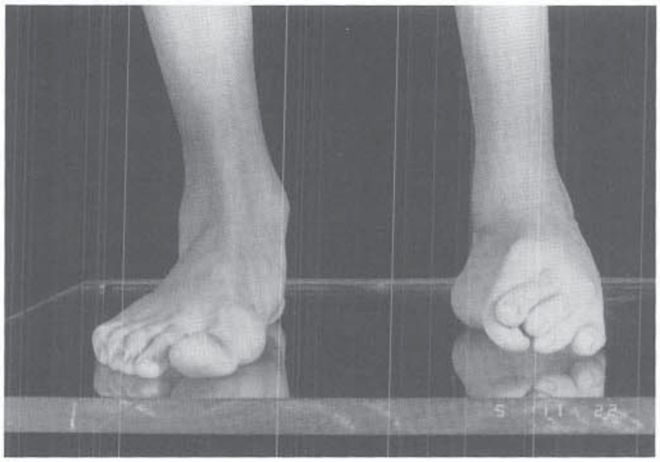
be moved as a unit. The ability to achieve lengthening and correction in a foot destined to be shorter due to a deformity is another attraction of the method.

Major issues surrounding foot correction include the role of soft tissue "release" before or after deformity correction, and the need for osteotomy to allow bony correction. Because of the lengthy treatment time often required by severe or neglected deformities, it is attractive to attempt partial acute correction by soft tissue release, with the intention of decreasing the total time necessary in external fixation. Unfortunately, preliminary dissection can produce ischemic skin complications when poorly vascularized or scarred areas are impaled by transfixation wires, leading to necrosis (Fig. 22–64). Thus, in previously unoperated, neglected clubfeet, and in severely scarred or multiply operate tissue, correction by tissue distraction *only,* with no preliminary surgery, has been recommended.[19,78,104] It is usually necessary to hold the foot in the corrected or overcorrected position for 2 to 4 months, either in a frame or a cast, once the plantigrade position is reached, to avoid relapse. In feet with previous surgery with significant ankylosis, correction through osteotomies (crescentic or through multiple joints) with no acute correction is preferred (Fig. 22–63).

Experience has shown that correction of deformity by soft tissue lengthening may be transient, and the surgeon must be prepared to perform soft tissue release, tenotomies, and/or bony stabilization *following* frame correction. Since surgery in the foot made stiff and atrophic by frame immobilization may be complicated by infection and poor wound healing, a waiting period is recommended between frame removal and further surgery, opting for functional bracing while tissue health and muscle and joint function recover to some degree.

Rehabilitation is a major problem following frame removal, with all reports universally documenting ankle and subtalar stiffness (less than 20 degrees of motion with no dorsiflexion), even when only tissue distraction is utilized. These results[19,38,104] are undoubtedly due to the long (5 to 9 months) treatment period, the underlying ankylosis of the deformity itself, and the stiffness induced by cartilage pressure as, for example, recurrent equinus is corrected by posterior distraction. Controversy exists concerning use of ankle hinges to control the talus, which may sublux anteriorly if a nonconstrained correction of equinus is attempted. Some authors report correction without constrained ankle correction,[38,78,104] while others recommend hinges,[14] and also an *anterior* motor rod to aid in dorsiflexion between the forefoot and distal tibial ring (Figs. 22–63J and K). Distraction of the ankle joint during equinus correction has been alluded to, without evidence that the range of motion at follow-up is improved.

The disadvantages of external fixation/distraction for recurrent clubfoot also involve psychological adaptation to the lengthy and admittedly uncomfortable process. In our experience, patients are uniformly unable to bear weight on a foot frame, and osteopenia, soft tissue edema, and trophic changes are universal while the foot is being corrected. Minimizing overall time-in-frame, with early removal and cast application once deformity correction is completed, is the best compromise for this dystrophic morbidity. Athough patients may not fully bear weight for months following treatment, rehabilitation must be vigorous to resolve the treatment-induced morbidity. Because there are few other options for the late-recurring or neglected deformity with poor skin coverage, the Ilizarov technique remains extremely useful to correct such deformities without further shortening



A

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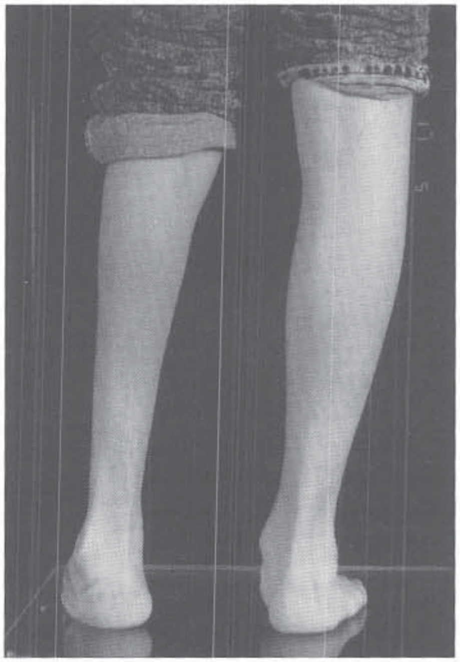

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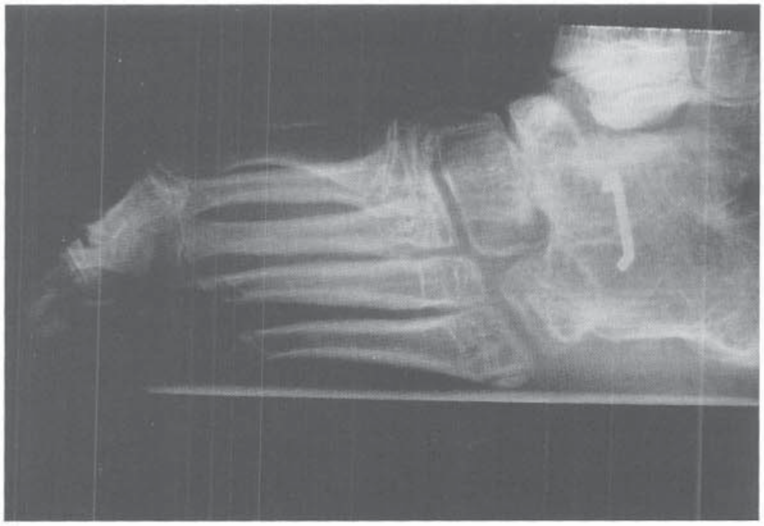

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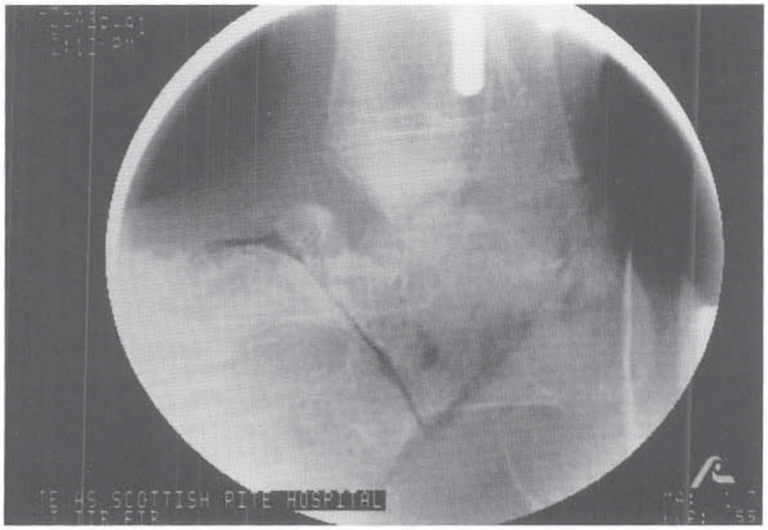

E

FIGURE 22–63 Ilizarov correction of rigid varus foot deformity. **A** to **C**, Clinical appearance of a 12-year-old boy with a multiply operate left clubfoot. Following triple arthrodesis complicated by vascular insufficiency and infection, he is left with a rigid, painful supination deformity of both hindfoot and forefoot. **D**, Radiograph showing triple arthrodesis in supination. **E**, Separate correction of hindfoot and forefoot position was done through two osteotomies, one through the talonavicular and calcaneocuboid fusions and one through the subtalar fusion to allow calcaneal correction.

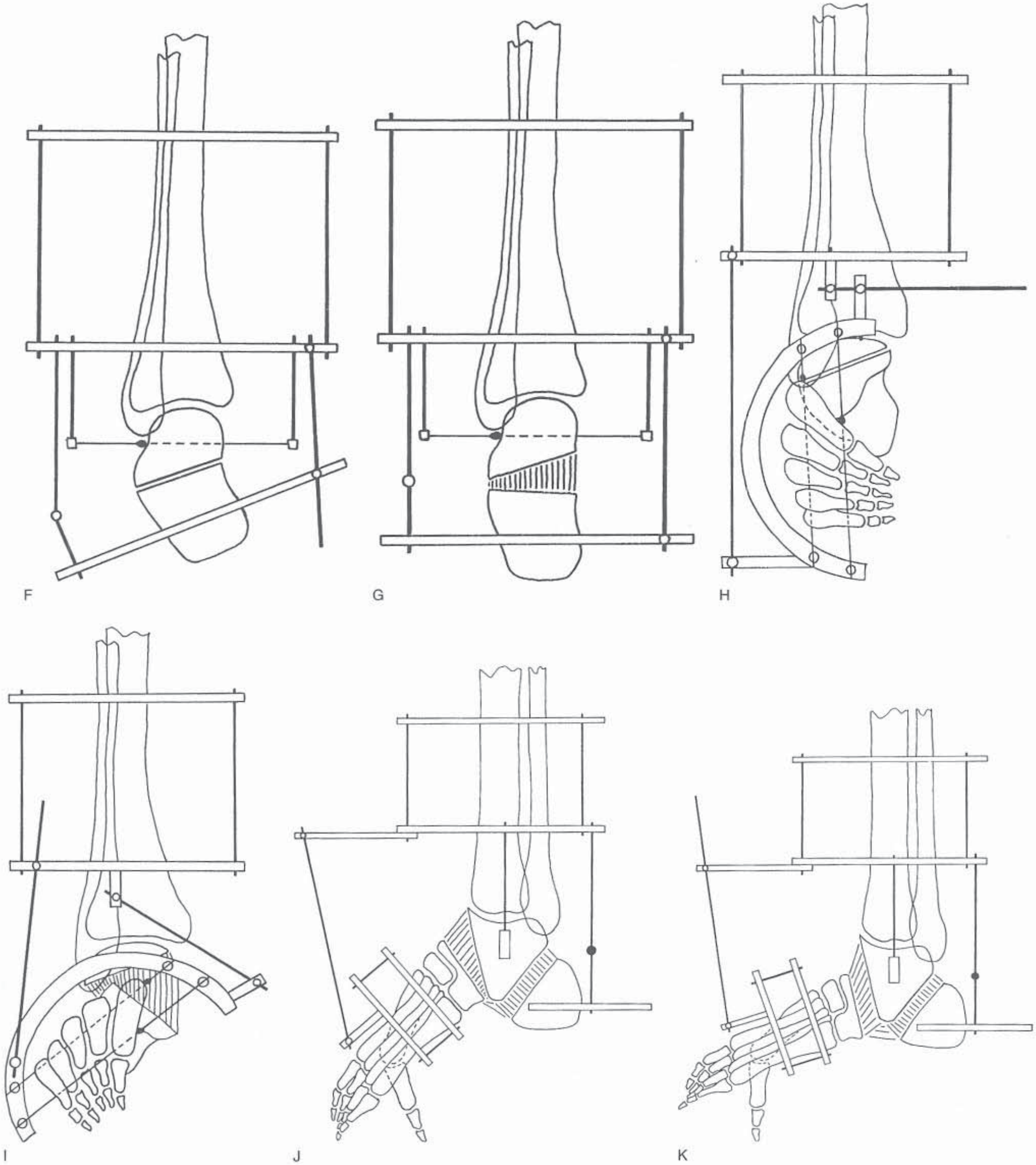

FIGURE 22–63 *Continued.* **F** and **G,** Correction of hindfoot varus by angular correction, distracting medially between the distal tibial ring and the distal calcaneal ring. Note olive wire fixation of the main talar fragment to prevent ankle subluxation. **H** and **I,** Correction of forefoot supination. The metatarsals are fixed to a forefoot ring by two olive wires. The forefoot is rotated in relation to the distal tibial ring, which incorporates the ankle via the fixation of the main talar fragment (see F). Correction occurs via the midfoot osteotomy. **J** and **K,** Correction of the forefoot equinus following correction of supination. The forefoot ring is dorsiflexed in relation to the main talar fragment, which is fixed to the distal tibial ring.

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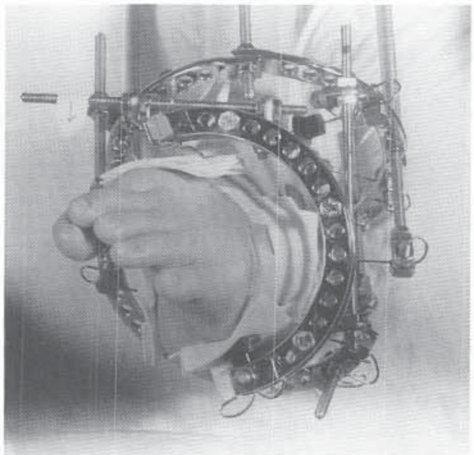
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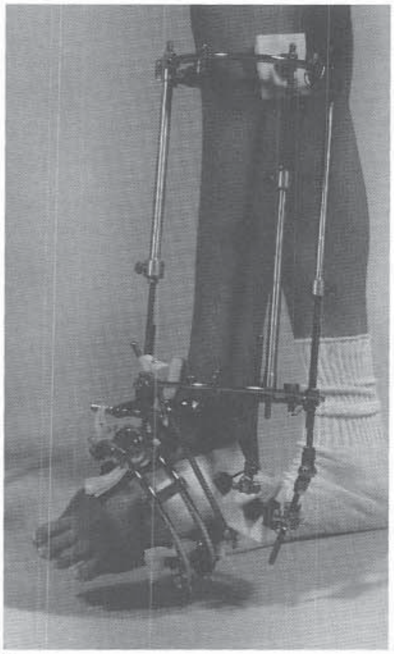
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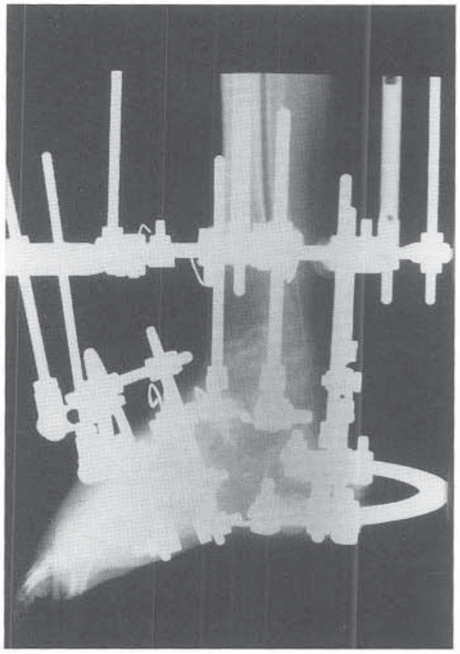
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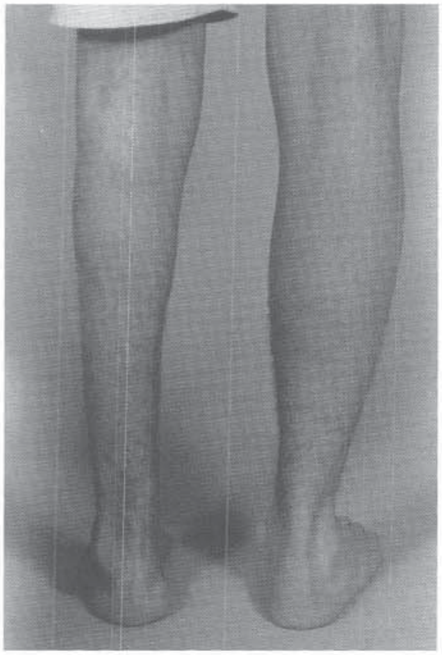
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FIGURE 22–63 *Continued.* **L** and **M,** Clinical appearance of forefoot ring prior to rotational correction. **N,** Radiograph obtained at completion of correction of supination. The frame is modified to begin forefoot dorsiflexion. **O** to **Q,** Final result 2 years later. The foot is now nearly plantigrade, with all toes in contact with the floor. (The great toe underwent flexion contracture release following frame correction.) Compare the weightbearing surfaces of the involved foot with those of the normal foot. Total time in frame was 8 months.

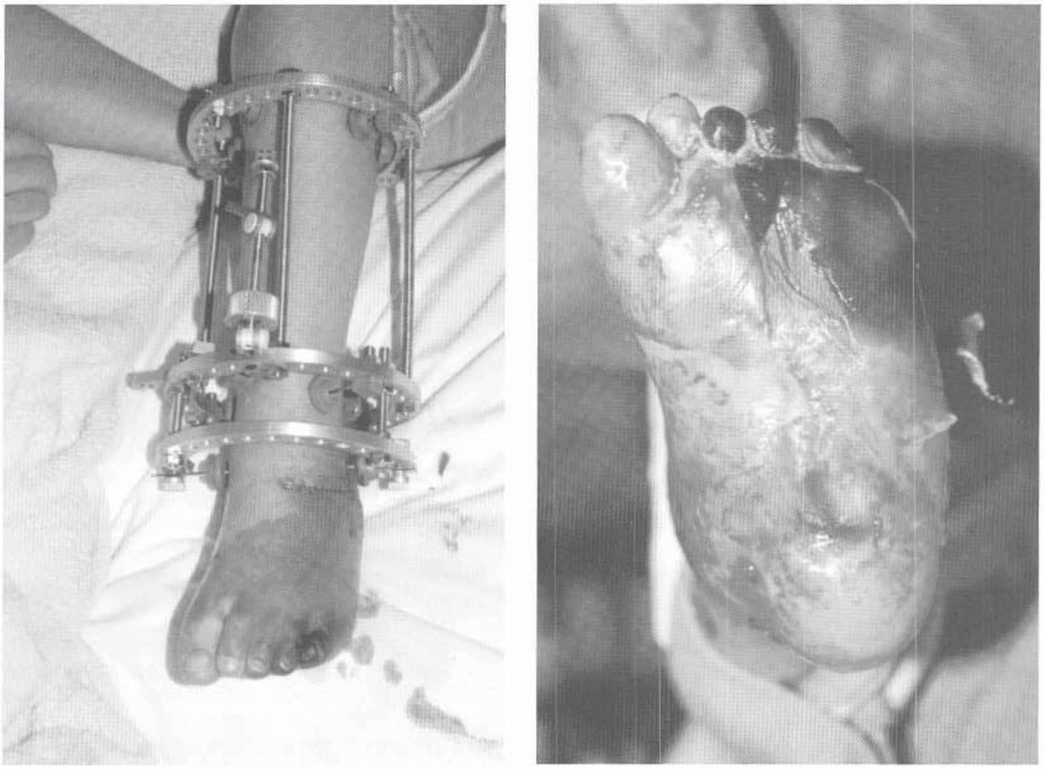

FIGURE 22–64 Ischemic necrosis of the lateral toes and midfoot in a 6-year-old with arthrogryposis. He underwent complete posteromedial and lateral soft tissue release *prior* to foot frame application. Acute correction was attempted but relaxed within hours due to ischemia, which did not recover on removal of the foot portion of the frame.

the foot by more bone resection. However, the physical and psychological disturbances and the lengthy treatment time must not be underestimated.

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VERTICAL TALUS

Definition. Vertical talus is a condition that produces a rocker-bottom deformity of the foot. The term *vertical talus* should be reserved for feet with fixed equinus of the calcaneus and dorsal dislocation of the navicular on the talus.[3,12,13,15,20] It has also been called congenital convex pes valgus.[16,11] The most appropriate name for the condition is teratologic dorsolateral dislocation of the talocalcaneonavicular joint.[23] The first description was by Henken in 1914,[9] and the characteristic features were well defined by Lamy and Weissman.[11]

Clinical Features and Associated Conditions. Vertical talus may be present alone or more commonly in association with other neuromuscular conditions. It is most often present in association with myelomeningocele and arthrogryposis, but it has been found in prune belly syndrome, spinal muscular atrophy, neurofibromatosis, congenital dislocation of the hip, and with trisomy 13–15 and 18.[7,24,25] In one large series, 10 percent of children with myelomeningocele who had foot deformities had vertical talus.[19] The neuraxis should be studied in cases without other apparent pathology to rule out occult neurologic dysfunction.[4]

The classic appearance is of a rocker-bottom foot, which is a foot with a convex plantar surface with the apex of the convexity at the talar head (Fig. 22–65). The calcaneus is fixed in equinus and the Achilles tendon is contracted. The peroneal and anterior tibialis tendons are taught, and the foot is everted into a valgus, externally rotated position. The navicular is palpable as it lies on the talar neck where it abuts the anterior tibial surface at the front of the ankle joint. There may be some flexibility of the foot, but passive correction of the deformity is not possible.

Coleman and colleagues distinguished two types of vertical talus, the first with talonavicular dislocation and the second with concomitant dislocation of the cuboid on the calcaneus.[3] Lichtblau defined three groups.[12] In his group I, which he termed teratogenic, there was a positive family history, the condition was often bilateral, and the condition was associated with developmental dysplasia of the hip and mental retardation. A rigid deformity was present at birth, with very tight extensors and heel cords. In group II, termed the neurogenic type, the deformity was associated with muscle imbalance. These cases were associated with myelomeningocele or neurofibromatosis, and there was a variable degree of deformity and rigidity. He noted that type II vertical talus may be more correctable than is first suspected. His group III, termed the acquired type, was attributed to intrauterine malposition. There were no other defects, the condition was unilateral, and it was not too severe clinically and was partially correctable. The calcaneus was not necessarily fixed in equinus in group III.[12]

A less severe manifestation of the deformity has been called the oblique talus.[10] In this variant there is a rocker-bottom deformity of the foot and an equinus contracture of the hindfoot, but the navicular can be reduced when the forefoot is plantar flexed.

Severe flatfoot or valgus deformities are sometimes confused with vertical talus because the talus is vertical on a lateral radiograph. The clinical and radiographic difference between the conditions is that in a severe flatfoot, the calcaneus can be easily dorsiflexed, and there is no fixed dislocation of the navicular.

Etiology. The exact etiology of vertical talus is unknown. Possible causes include muscle imbalance, especially overpull of the anterior tibial tendon in paralytic disorders, and intrauterine compression, especially when coupled with arthrogryposis. Autosomal dominant transmission through three generations of a Honduran family has been reported,[21] as well as transmission from parent to child.[11,17] It has been suggested that the deformity represents an arrest of fetal development of the foot occurring between the 7th and 12th weeks of gestation.[1,27]

Pathologic Anatomy. Several investigators have carefully described the pathoanatomy of vertical talus.[1,4,8] The skeletal anatomy is most characteristic (Fig. 22–66). The navicular articulates with the dorsal aspect of the neck of the talus and is locked there. The proximal articular surface of the navicular is tilted plantarward. The head of the talus is flattened superiorly and is ovoid in its length. The calcaneus is displaced posterolaterally in relationship to the talus, is in contact with the distal fibula, and is tilted into equinus. The angle between the axes of the talus and calcaneus is markedly increased. The subtalar joint is abnormal, with

FIGURE 22–65 Congenital vertical talus. **A,** Pronation of the forefoot. **B,** Valgus of the heel. **C,** Absence of an arch, the "rocker-bottom" deformity. **D,** Elevation of the lateral toes and tight peroneal tendons.

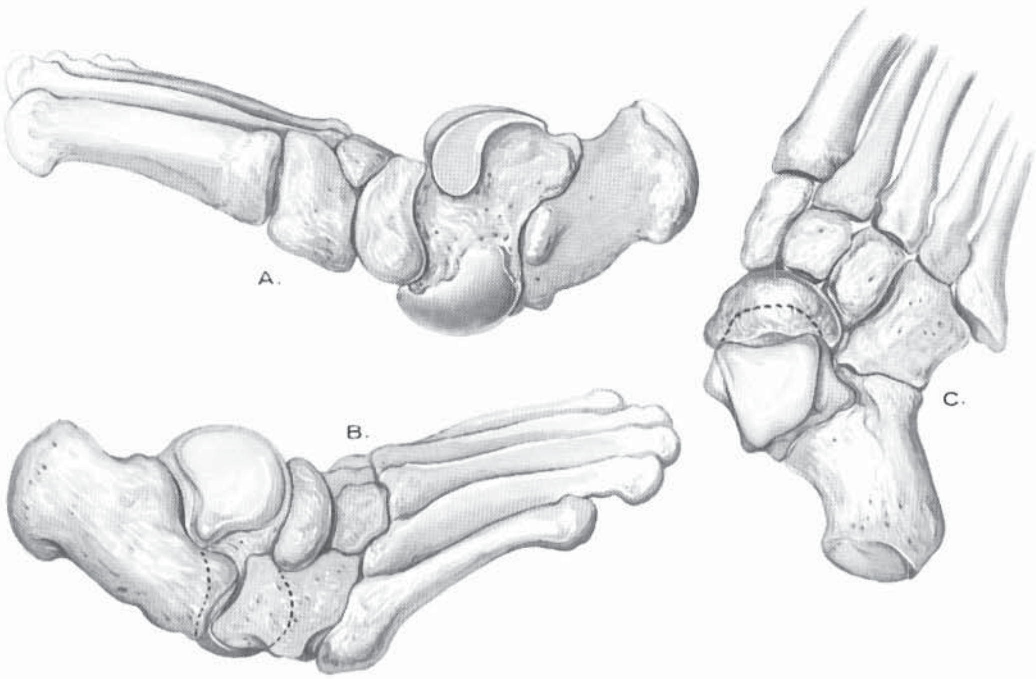

FIGURE 22–66 Bone and joint changes (pathoanatomy) in congenital vertical talus. **A,** Medial aspect of the right foot showing dorsiflexion of the forefoot at the midtarsal joint; vertical talus, producing a rocker-bottom convexity; subluxation of the navicular on the neck of the talus, locking the talus vertically; and calcaneus in 20 to 25 degrees equinus. **B,** Lateral aspect of the right foot. *Dotted lines* indicate the displaced head of the talus. **C,** Dorsal aspect showing abducted forefoot beginning at midtarsus. *Dotted lines* indicate head of talus subluxed below the navicular bone (From Tachdjian MO: Congenital convex pes valgus. Orthop Clin North Am 1972;3:133.)

the anterior facet usually absent and the middle facet hypoplastic. The articular facet of the calcaneus for the cuboid is inclined dorsally and laterally, and there is a variable degree of subluxation of the calcaneocuboid joint. These abnormalities result in elongation of the medial column and shortening of the lateral column of the foot.

Ligamentous abnormalities mirror the bony deformities (Figs. 22–67 to 22–69). The spring ligament is attenuated while the tibionavicular portion of the superficial deltoid is markedly contracted, holding the navicular in a dislocated position. Other contracted ligaments include the bifurcated ligament between the calcaneus and the navicular and cuboid, the calcaneofibular ligament, and the interosseous talocalcaneal ligaments. There are corresponding contractures of the tibialis anterior, long toe extensors, peroneus brevis, and triceps surae (Figs. 22–70 and 22–71). The posterior tibial and peroneal tendons may be displaced anteriorly so that they act as dorsiflexors rather than plantar flexors.

Radiography. There is a characteristic appearance of the foot with a vertical talus.[15,26] On the lateral projection the talus appears in a near vertical position, almost parallel to the tibia (Fig. 22–72). The calcaneus is also in an equinus posture, and the talocalcaneal angle is increased. In the young child the navicular is not ossified, but its position is inferred from the direction of the metatarsals and the position of the ossified cuneiform(s). The metatarsals and the cuneiform align with the neck of the talus, well dorsal to the head of the talus. The navicular is always dislocated dorsally on the talus and, when ossified, is seen to abut the anterior tibia. A line through the axis of the talus on a lateral radiograph passes posterior to the cuboid rather than through it, as in a normal foot (Figs. 22–73 to 22–75). A standing radiograph shows the same deformities. On a forced dorsiflexion radiograph the talus and calcaneus remain plantar flexed. In forced plantar flexion the navicular fails to reduce on the talus, either as viewed directly or as inferred from the position of the metatarsals and cuneiforms.[6] Should all elements exist (calcaneus in equinus, navicular dislocated in neutral position) except that the navicular is reducible on a radiograph obtained with the feet in plantar flexion, the condition is termed an oblique talus.[15]

Differential Diagnosis. In early infancy, a calcaneovalgus deformity can be mistaken for a vertical talus. In that condition the forefoot is dorsiflexed against the tibia, but there is dorsiflexion of the hindfoot (as opposed to equinus) and the plantar aspect of the foot is flat rather than convex. The deformity is not as rigid as a vertical talus.

The term oblique talus is given to an intermediate deformity, neither a true vertical talus nor yet a flexible flatfoot. The diagnostic finding is subluxation of the navicular on the talus in a standing position, with reduction of the navicular in maximum plantar flexion.[10]

At walking age the flatfoot with a heel cord contracture may also be mistaken for a vertical talus. The difference is that in the flatfoot, the navicular, which may be dorsally displaced (but not dislocated) in the standing position, is easily reduced on the talar head with plantar flexion. In fact, the navicular is never truly dislocated in a flexible flatfoot. The valgus deformity in flatfoot is also flexible and will reduce on inversion of the foot, which restores the arch contour. There is no such flexibility or reducibility in a vertical talus deformity.

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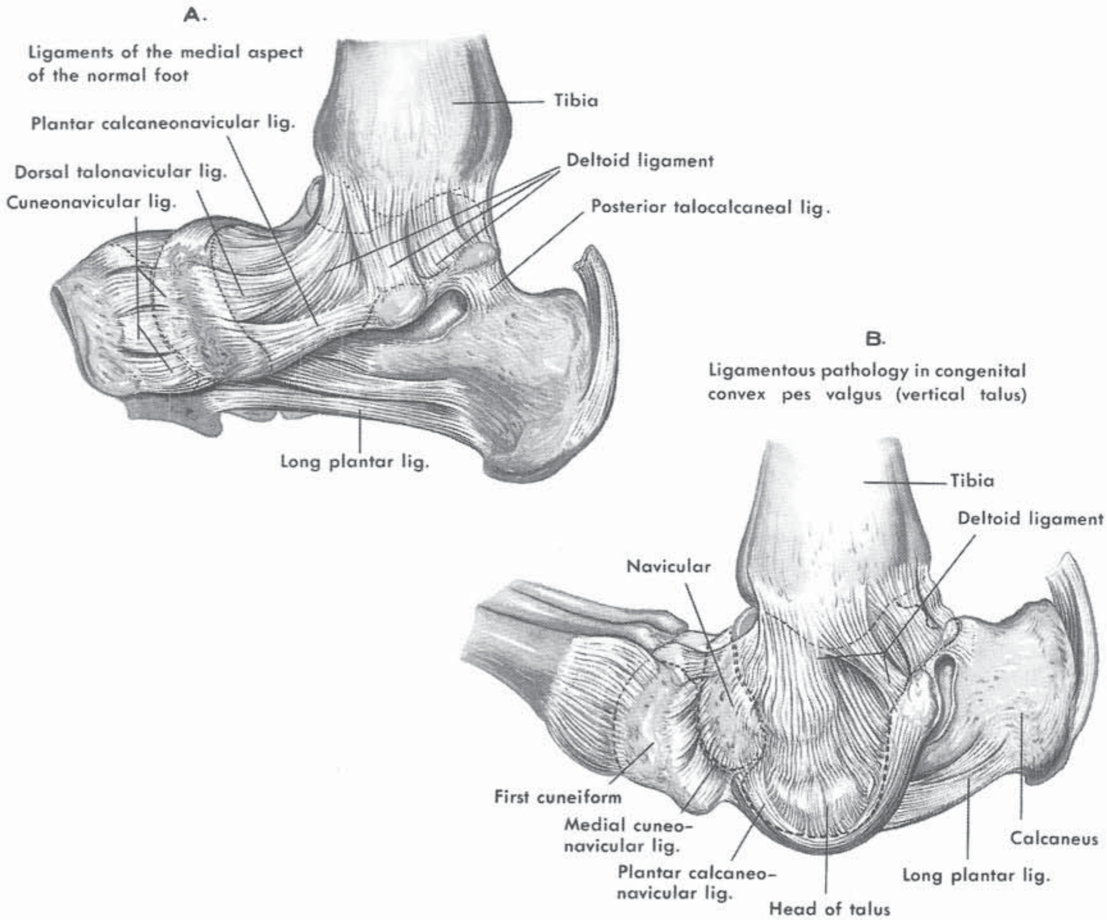

FIGURE 22–67 Ligamentous pathologic changes in congenital vertical talus—medial view. **A,** Normal foot. **B,** Malformed foot with congenital vertical talus.

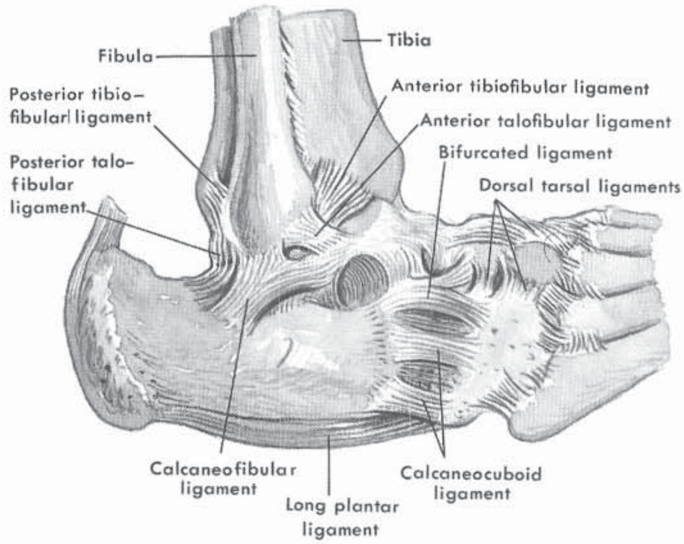

FIGURE 22–68 Ligamentous pathologic changes in congenital vertical talus—lateral view.

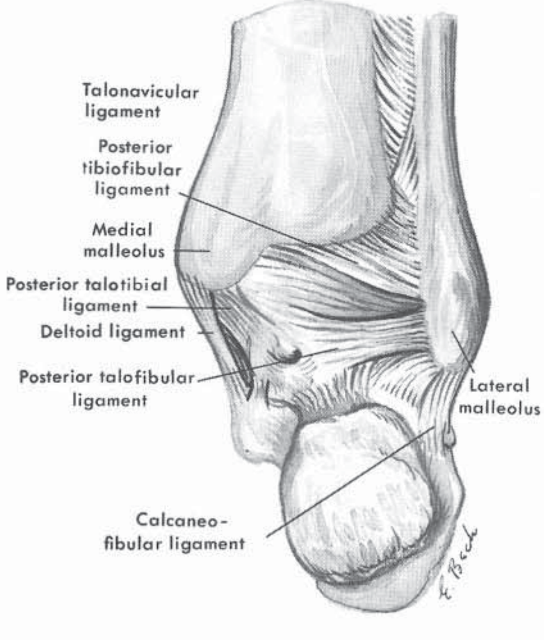

FIGURE 22–69 Ligamentous pathologic changes in congenital vertical talus—posterior view.

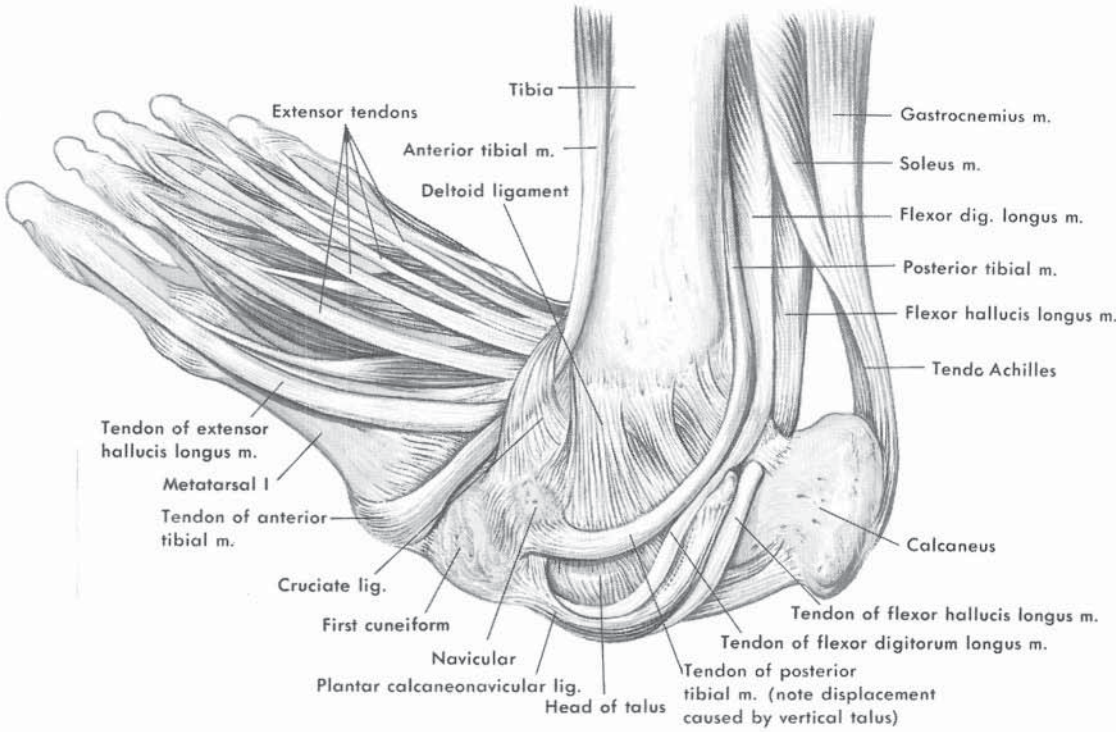

FIGURE 22–70 Abnormalities of muscles and tendons in congenital vertical talus.

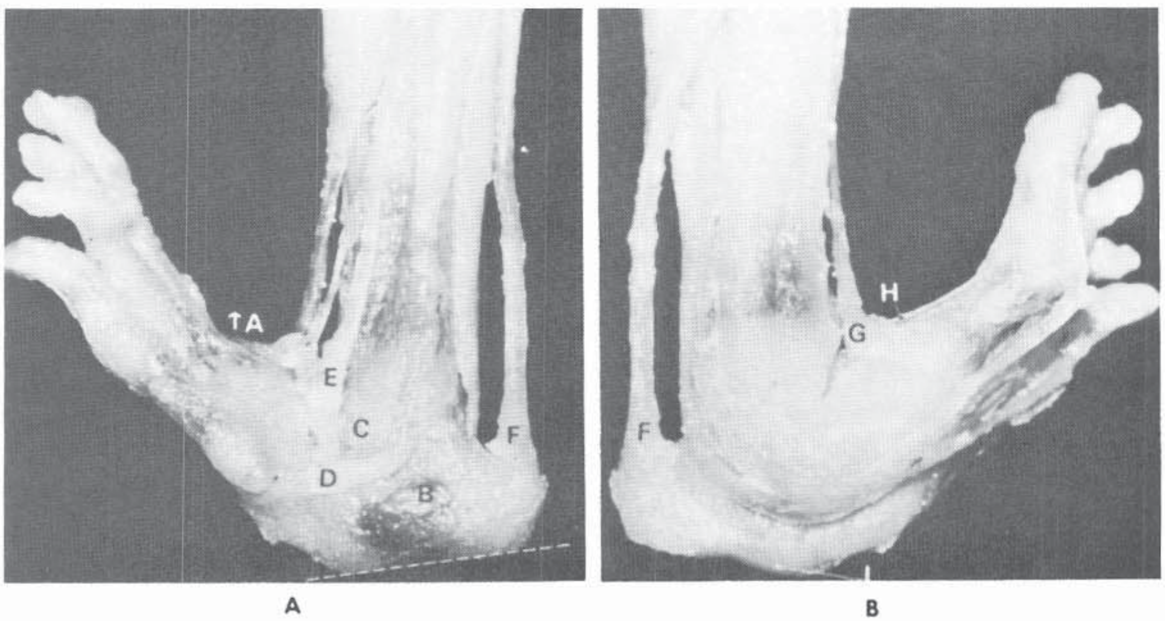

FIGURE 22–71 Pathologic soft tissue changes in congenital vertical talus. Anatomic findings in an infant who died 8 hours after birth. **A,** Lateral view. Note the rocker-bottom foot with dorsiflexion of the forefoot (*A*) and equinus deformity of the heel. The apex angulation of the lateral column is at the calcaneonavicular joint. The calcaneus (*B*) is displaced laterally under the talus, lying in close proximity to the distal end of the fibula (*C*). The triceps surae (*F*) is contracted, holding the calcaneus in plantar flexion. The peroneus longus (*D*) and peroneus tertius (*E*) are shortened. **B,** Medial view. The anterior tibial (*G*) and extensor hallucis longus (*H*) muscles are shortened. (The extensor digitorum longus is also contracted, but it does not show in this photograph.) The triceps surae muscle (*F*) is shortened. These musculotendinous contractures are secondary obstacles to anatomic alignment of the talocalcaneonavicular joint. (From Campos da Paz A Jr, De Souza V, deSouza DC: Congenital convex pes valgus. Orthop Clin North Am 1978;9:210.)

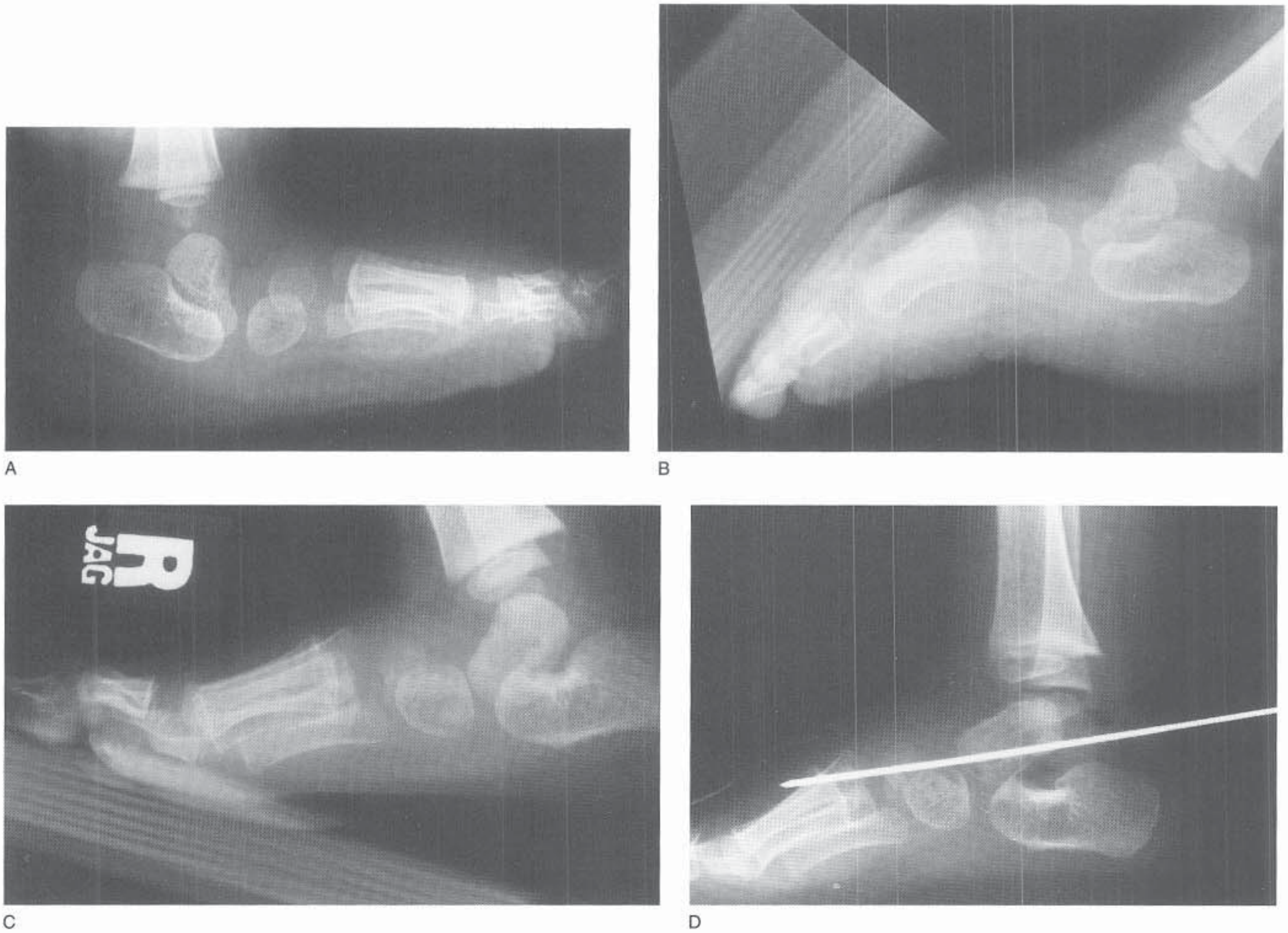

FIGURE 22–72 A child with congenital vertical talus. **A,** Lateral radiograph showing the increased talocalcaneal angle, equinus of the calcaneus, and dorsal dislocation of the navicular. The bone ossified over the talus is the medial cuneiform, which indicates the position of the navicular, which is not ossified yet in this child. **B,** Plantar flexion lateral radiograph. The navicular is still dorsally displaced over the talar neck (again indicated by the location of the cuneiform). **C,** Dorsiflexion lateral radiograph. The hindfoot remains in neutral position and lacks true dorsiflexion. **D,** Lateral radiograph after open reduction of the dislocated navicular and soft tissue release. The pin secures the navicular in a reduced position opposite the talus.

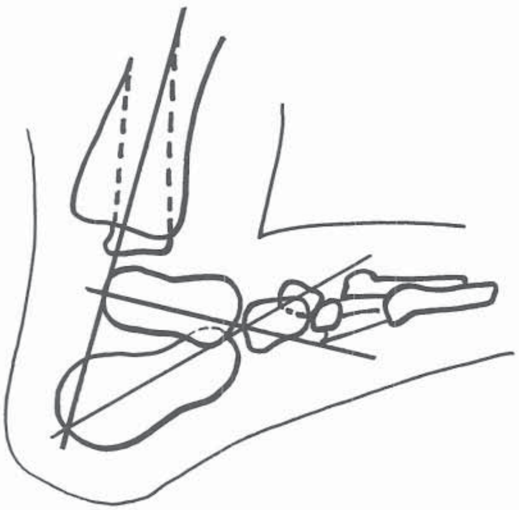

FIGURE 22–73 Line drawing showing alignment of normal structures on the lateral radiograph. The long axis of the talus cuts the lower half of the cuboid, whereas the long axis of the calcaneus passes through the upper half of the cuboid.

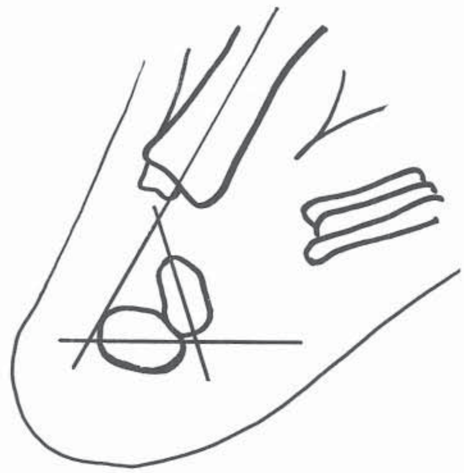

FIGURE 22–75 Relationship of structures as seen on a lateral radiograph of a foot with congenital vertical talus. The long axis of the talus passes very close to the anterior end of the calcaneus.

Treatment. Serial cast treatment has been recommended and tried by many but is largely ineffective, due to the rigidity of the deformity. Manipulation and casting may be effective in milder cases, most of which are not true vertical talus deformities. In these cases the forefoot is first stretched into plantar flexion and inversion by applying distal traction to the metatarsals. An upward push on the calcaneus and a downward pull on the heel may stretch the equinus deformity. The foot is casted in the most corrected position. If the navicular reduces, Tachdjian has performed blind pinning of the talonavicular joint to maintain reduction while gradually correcting the equinus contracture.[23]

Surgical correction is the mainstay of treatment. The difficulty of surgical correction depends on the severity of the deformity, the associated diagnoses, and the age of the patient. Milder cases require less extensive releases, while rigid, arthrogrypotic deformities require circumferential releases and will probably never achieve normal mobility.

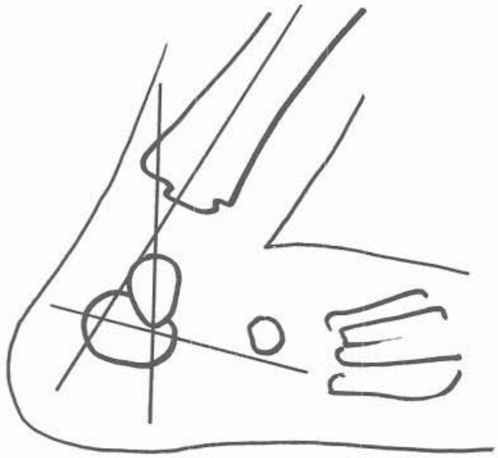

FIGURE 22–74 Relationship of structures as seen on a lateral radiograph of a foot with congenital vertical talus. The long axis of the talus passes below and behind the cuboid bone and cuts through the anterior part of the calcaneus, and the long axis of the calcaneus passes plantar to the cuboid.

Single-stage releases, two-stage releases, soft tissue release with navicular excision, and Grice-Green subtalar fusion after release have all been reported to be effective.[3,5,6,11,15,18,19,27] We prefer a single-stage release performed at about age 1 year, as described in Plate 22–1. A modified Cincinnati incision is used, with extension across the dorsum of the foot as necessary to lengthen the toe extensors and peroneals. There are four components to the release. First is the reduction of the navicular on the talus by release of the anterior tibialis tendon and the tibionavicular and talonavicular ligaments and capsule. The reduction is stabilized by a pin placed across the talonavicular joint and by reconstruction of the spring ligament. The second portion is a lengthening of the toe extensors and peroneals to allow reduction of the forefoot. The cuboid is reduced on the calcaneus with release of the bifurcate ligament and the calcaneocuboid joint capsule. The third stage is release of the equinus contracture, lengthening the Achilles tendon and the ankle and subtalar joint capsules. The fourth component is transfer of the anterior tibialis tendon to the talus to dynamically stabilize the correction. In older children with resistant deformities, excision of the navicular may be necessary to achieve the first step of correction.[17]

It must be noted that this is a very extensive exposure and release. In milder deformities some components may not be required. For example, if the extensor tendons and peroneals are only mildly contracted, they should not be lengthened unless their length does not allow the foot to rest in neutral at the end of the procedure. When the entire release is necessary, it is imperative that the surgeon preserve as many superficial veins as possible so that venous drainage of the forefoot is not compromised. Sometimes percutaneous tenotomy of the extensors can be done with a less extensive skin incision.

A postoperative splint or a bivalved cast is used to maintain correction, and the foot should be elevated. Any cast or dressing that constricts the foot must be released, as swelling is often a problem after the surgery. A cast change under anesthesia may be required at a week or two to convert to a solid cast. The cast should be maintained for 6 to 12 weeks, depending on the severity of the deformity.

Children between 3 and 4 years of age may be best managed with a concomitant Grice extra-articular arthrodesis at the time of the release.[3] Older children may also require excision of the navicular to shorten the medial column and allow reduction of the forefoot.

Results and Complications. Stricker and Rosen reported on 20 feet treated with a one-stage release and reduction, with good results in 17 and fair results in 3. They suggested that the surgery be done before age 27 months. At follow-up all feet had some residual midfoot sag and forefoot abduction, and some had decreased motion.[22] Wirth and colleagues also reported results of single-stage correction procedures done between 3 and 6 months of age. Of 13 feet, results were good or excellent in 10, two had recurrent deformity, and one was overcorrected. They found that correction at an early age allowed the surgeon to avoid tendon lengthenings and gave good functional results.[27] Marciniak reported good clinical results in 5 feet treated by Grice fusions and lateral column lengthening.[14] Clark and colleagues reported success with navicular excision in 15 feet.[2]

Napiontek reported results in a large series of 32 feet, of which 8 had been treated by Grice procedures primarily and 8 had been treated by Grice procedures as part of a reoperation. Results were excellent in 5 feet, good in 12, fair in 9, and poor in 6. Those with severe deformity and less extensive surgery had worse results. Some of the Grice procedures resulted in overcorrection. The commonest reason for surgical failure was inadequate reduction of the navicular.[15]

Correction of a neglected deformity in an older child is difficult. In severe deformities full correction is not possible, and later reconstruction with a triple arthrodesis is appropriate for the symptomatic foot. The deformity is not easily corrected with the triple arthrodesis, and a supplemental bone graft is necessary to correct the valgus deformity.

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TARSAL COALITION

A tarsal coalition is an abnormal connection between two or more bones of the foot that may produce pain and limitation of motion of the foot. The most common coalitions are talocalcaneal and talonavicular, but many different combinations of fusions between tarsal bones have been reported. The condition has also been called *peroneal spastic flatfoot.* The reported incidence of tarsal coalitions has varied from 0.03 percent to 1.0 percent (Table 22–2).[15,33,39,45]

Historical Aspects. Buffon, in 1769, was the first to recognize a tarsal coalition.[4] Cruveilhier in 1829 reported the first calcaneonavicular coalition,[7] and Zuckerkandl in 1877 described the first talocalcaneal coalition.[51] Sir Robert Jones provided the first clinical description of peroneal spastic flatfoot, in 1897,[18] but it was not until the 1920s that Slomann[40] and then Badgley[1] showed that rigid pes planovalgus and peroneal spasm are caused by calcaneonavicular coalitions. In 1948, Harris and Beath reported the correlation between medial talocalcaneal bridge and peroneal spastic flatfoot.[16]

Etiology. The etiology of tarsal coalition is unknown. The most likely cause is failure of segmentation of fetal tarsal bones, as connections between tarsal bones have been observed in fetal specimens (Fig. 22–76).[14] In a study comprising 142 feet from 7-week to 20-week fetuses, 16 feet had talocalcaneal bridges.[19] These bridges were not found in older fetuses, and some may resolve spontaneously. Although a coalition may be present at birth, symptoms appear later as the child matures (Fig. 22–77). The cause of pain from a coalition is also conjectural. The coalition may produce abnormal motion between the complex of bones of the hindfoot and midfoot that becomes painful and induces

Text continued on page 974

Open Reduction of Dorsolateral Dislocation of Talocalcaneonavicular Joint (Congenital Vertical Talus)

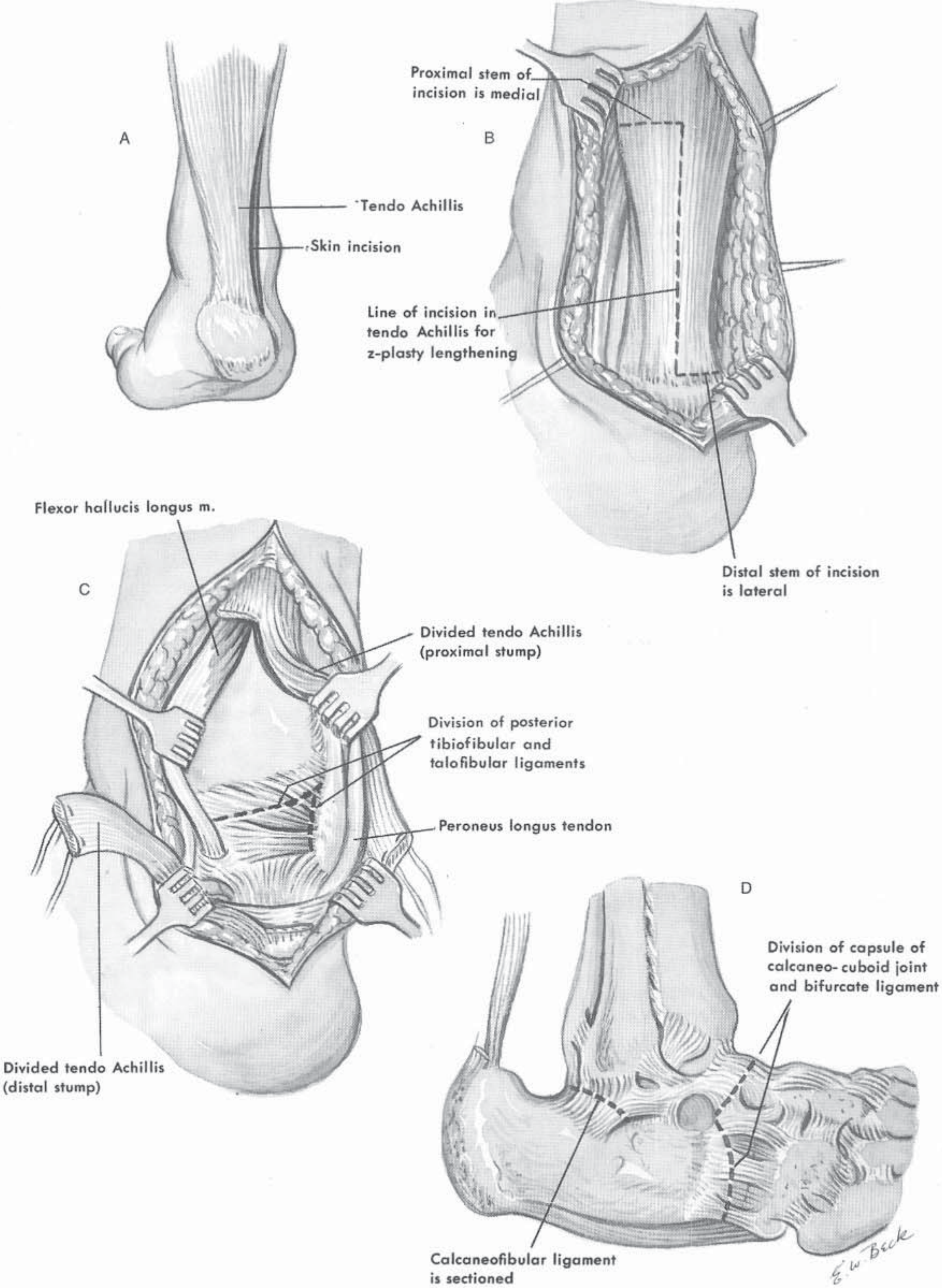
OPERATIVE TECHNIQUE

A, A longitudinal incision is made lateral to the tendo calcaneus, beginning at the heel and extending proximally for a distance of 7 to 10 cm. The subcutaneous tissue and tendon sheath are divided in line with the skin incision, and the wound flaps are retracted, exposing the Achilles tendon.

B, Z-plasty lengthening is performed in the AP plane. With a knife, the Achilles tendon is divided longitudinally into lateral and medial halves for a distance of 5 to 7 cm. The distal end of the lateral half is detached from the calcaneus to prevent recurrence of valgus deformity of the heel; the medial half is divided proximally. When the equinus deformity is not marked, sliding lengthening of the heel cord is performed.

C and **D,** A posterior capsulotomy of the ankle and subtalar joint is performed if necessary. The calcaneofibular ligament is sectioned. The thickened capsule of the calcaneocuboid joint and the bifurcated ligament are divided through a separate lateral incision. The Cincinnati transverse incision is an alternative surgical approach; it is preferred by this author.

PLATE 22–1. Open Reduction of Dorsolateral Dislocation of Talocalcaneonavicular Joint (Congenital Vertical Talus)



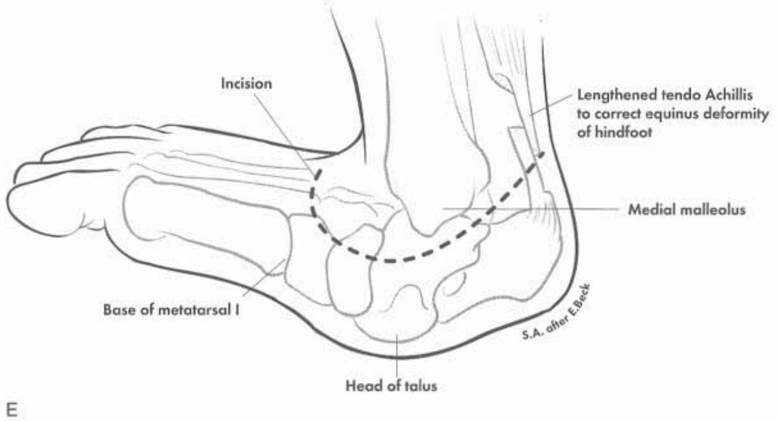
Open Reduction of Dorsolateral Dislocation of Talocalcaneonavicular Joint (Congenital Vertical Talus) *Continued*

E, The incision is a modified Cincinnati incision that passes beneath the medial malleolus just past the Achilles tendon posteriorly and proceeds dorsally over the navicular just past the extensor tendons.

F and **G,** The posterior tibial tendon is identified, dissected, and divided at its insertion to the tuberosity of the navicular. The end of the tendon is marked with 0 Mersilene suture for later reattachment. The articular surface of the head of the talus points steeply downward and medially to the sole of the foot and is covered by the capsule and ligament. The navicular will be found against the dorsal aspect of the neck of the talus, locking it in vertical position. The pathologic anatomy of the ligaments and capsule is noted and the incisions are planned so that a secure capsuloplasty can be performed and the talus maintained in its normal anatomic position. Circulation to the talus is another important consideration; it should be disturbed as little as possible by exercising great care and gentleness during dissection. Avascular necrosis of the talus is always a potential serious complication of open reduction. The plantar calcaneonavicular ligament is identified and divided distally from its attachment to the sustentaculum tali, and a 00 Mersilene suture is inserted in its end for later reattachment. The talonavicular articulation is exposed by a T-incision. The transverse limb of the T is made distally over the tibionavicular ligament (the anterior portion of the deltoid ligament) and over the dorsal and medial portions of the talonavicular ligament. A cuff of capsule is kept attached to the navicular for plication on completion of surgery. The longitudinal limb of the incision is made over the head and neck of the talus inferiorly.

The articular surface of the head of the talus is identified, and a large threaded Kirschner wire is inserted in its center. With a skid and the leverage of the Kirschner wire, the head and neck of the talus are lifted dorsally and the forefoot is manipulated into plantar flexion and inversion, bringing the articular surfaces of the navicular and head of the talus into normal anatomic position.

PLATE 22–1. Open Reduction of Dorsolateral Dislocation of Talocalcaneonavicular Joint (Congenital Vertical Talus)



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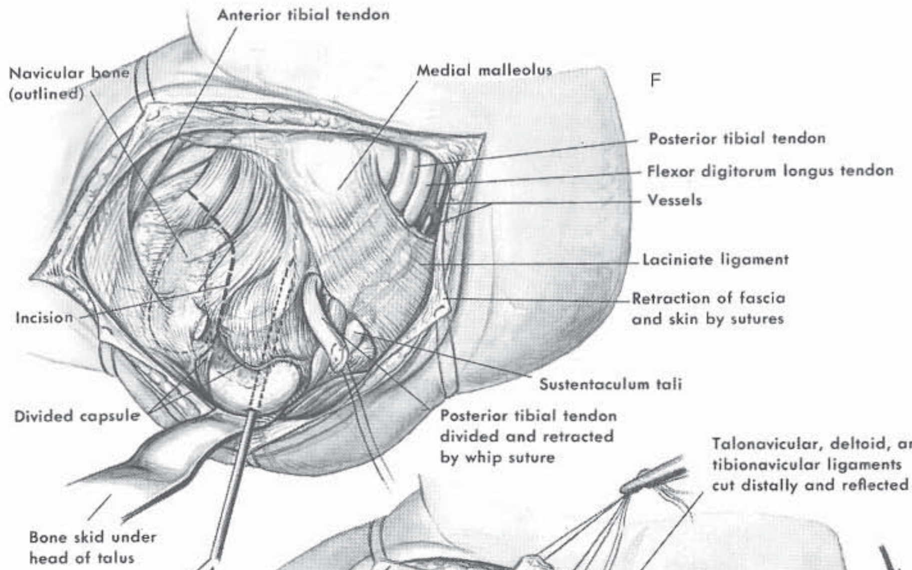

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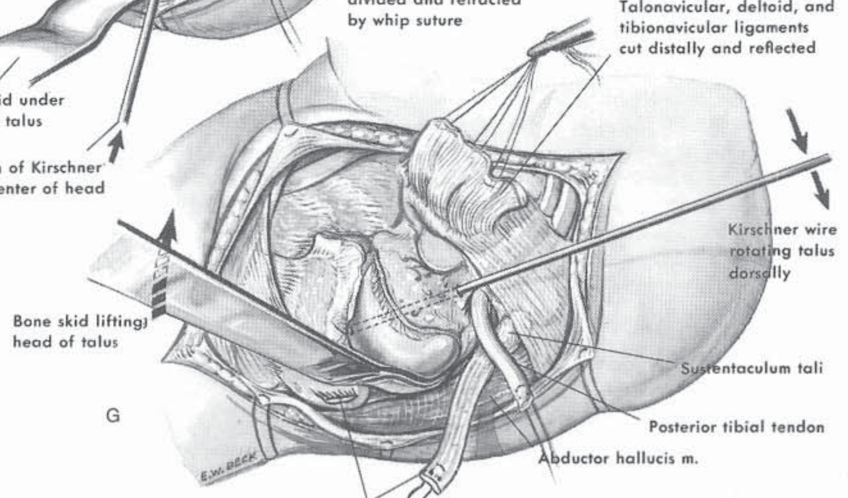


G

Open Reduction of Dorsolateral Dislocation of Talocalcaneonavicular Joint (Congenital Vertical Talus) *Continued*

H, The Kirschner wire is drilled retrograde into the navicular, cuneiform, and first metatarsal bones, maintaining the reduction. Radiographs of the foot are obtained at this time to verify the reduction.

In severe cases the calcaneocuboid and talocalcaneal interosseous ligaments may prevent reduction of the laterally subluxated Chopart's and subtalar joints. They are divided when necessary to allow reduction. In addition, the extensor hallucis, extensor digitorum longus, and occasionally the peroneals may be contracted. They should be Z-lengthened to allow reduction of the foot into plantar flexion. These releases require extension of the incision over the dorsum of the foot.

I and **J,** A careful capsuloplasty is very important for maintaining the reduction and the normal anatomic relationship of the talus and navicular. The redundant inferior part of the capsule should be tightened by plication and overlapping of its free edges. First, the plantar-proximal segment of the T of the capsule is pulled dorsally and distally and sutured to the dorsal corner of the inner surface of the distal capsule. Next, the dorsoproximal segment of the T is brought plantarward and distally over the plantar-proximal segment of the capsule and sutured to the plantar corner on the inner surface of the distal capsule. Then interrupted sutures are used to tighten the capsule on its plantar and medial aspects by bringing the distal segment over the proximal segments.

The plantar calcaneonavicular ligament is sutured under tension to the base of the first metatarsal. To tighten the posterior tibial tendon under the head of the talus, it is advanced distally and sutured to the inferior surface of the first cuneiform.

The anterior tibial may be transferred to provide additional dynamic force for maintaining the navicular in correct relationship to the talus. The tendon is detached from its insertion to the medial cuneiform and first metatarsal bone, and dissected free proximally and medially for a distance of 5 cm. Then it is redirected to pass along the medial aspect of the neck of the talus and beneath the head of the talus, where it is fixed to the inferior aspects of the talus and navicular with 00 Mersilene sutures. Normally the lower end of the anterior tibial tendon may be split near its insertion. Often the author leaves intact the attachment to the first metatarsal, dividing only the insertion to the medial cuneiform. The tendon is split (if not normally bifurcated), and the portion to the medial cuneiform bone is transferred to the head of the talus and the navicular. Sometimes, following adequate capsuloplasty, the reduction of the talonavicular joint is so stable that anterior tibial transfer is not necessary to restore support to the head of the talus.

K, The wounds are then closed in routine fashion. The Kirschner wire across the talonavicular joint is cut subcutaneously. To maintain the normal anatomic relationship of the os calcis to the talus, a Kirschner wire is inserted transversely in the os calcis and incorporated into the cast. An alternative method is to pass the wire from the sole of the foot upward through the calcaneus into the talus. The author prefers the former, as it controls the heel in the cast and prevents recurrence of both equinus deformity and eversion of the hindfoot. An above-knee cast is applied with the knee in 45 degrees of flexion, the ankle in 10 to 15 degrees of dorsiflexion, the heel in 10 degrees of inversion, and the forefoot in plantar flexion and inversion. The longitudinal arch and the heel in the cast are well molded.

POSTOPERATIVE CARE

The Kirschner wires are removed after 6 weeks. The foot is placed in a walking cast for another 4 to 6 weeks to maintain correction. Further splinting is necessary only in children with neurologic abnormalities or those with arthrogryposis.

PLATE 22–1. Open Reduction of Dorsolateral Dislocation of Talocalcaneonavicular Joint (Congenital Vertical Talus)

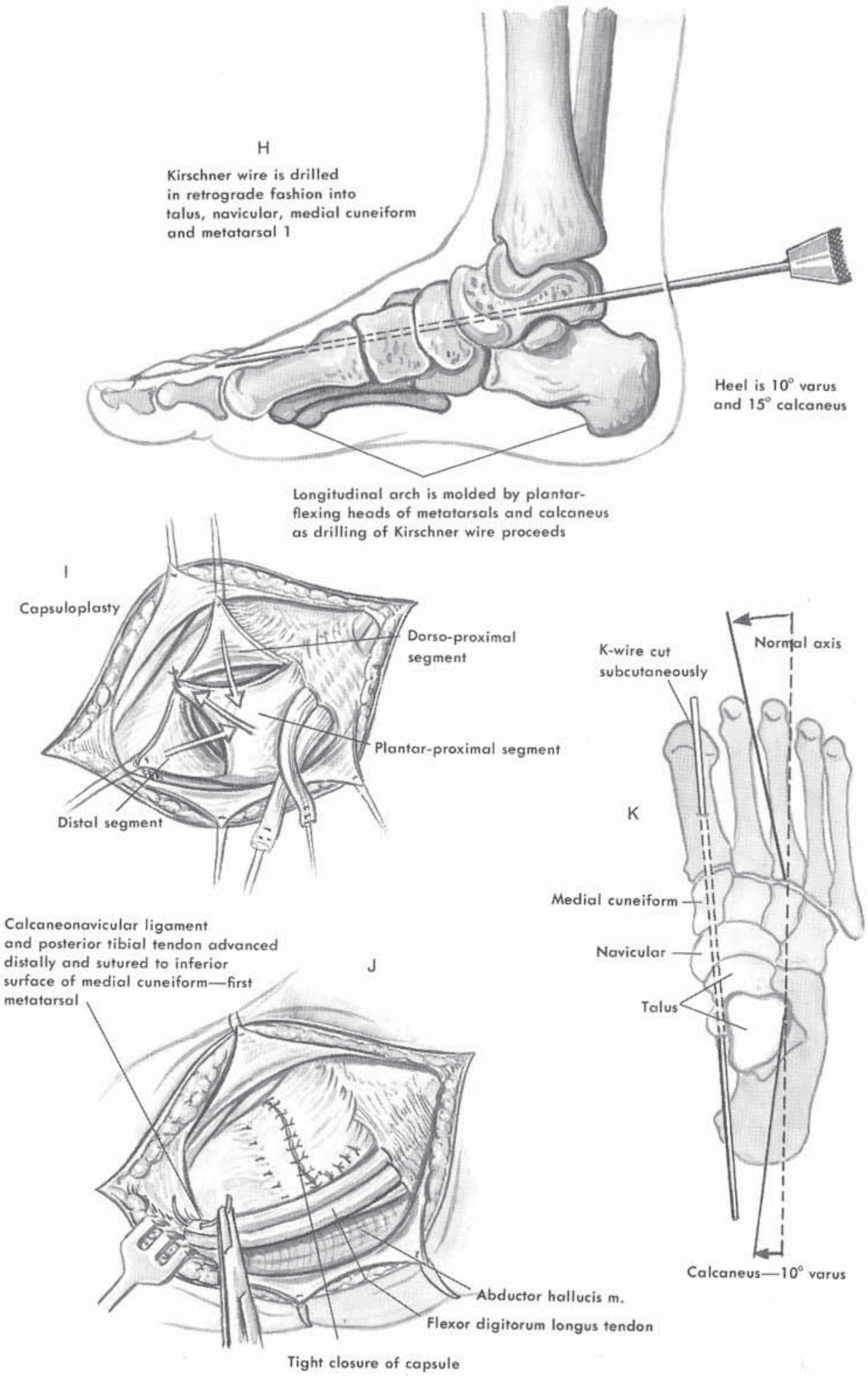


TABLE 22–2 **Reported Incidence of Tarsal Coalition**

Study	Material	Incidence (%)
Pfitzner[33]	Autopsy	0.38 (2 of 524)
Harris & Beath[15]	Army recruits	0.03 (1 of 3,619)*
Vaughan & Segal[45]	Army personnel	1.0 (21 of 2,000)
Shands & Wentz[39]	Children's clinic	0.9 (11 of 1,232)

*Of 3,619, 72 (2%) had peroneal spastic flatfoot.

peroneal muscle spasm. Partial and cartilaginous coalitions may be painful because of stresses across the incomplete fusion. A case has been reported of pain after fracture of a solid calcaneonavicular bar in a previously asymptomatic 17-year-old adolescent.[36]

There are a number of reported cases of familial occurrence of coalitions. Boyd described a family with bilateral talonavicular bars in three generations,[3] and there are several other reports of familial incidence of this coalition.[2,37] Wray and Herndon described calcaneonavicular coalition in three generations of a family, and proposed that this phenomenon represented autosomal dominant inheritance with reduced penetrance.[50] Harris found calcaneonavicular bars in identical twins and also in a father and son.[13] Several pairs of monozygotic twins with identical coalitions have been reported.[10,35] Leonard conducted an extensive family survey of 31 patients with known coalitions and found that 39 percent of first-degree relatives had some type of coalition.[23] This finding was considered to be evidence of almost complete penetrance of autosomal dominant inheritance of the disorder.

Clinical Features. Tarsal coalitions usually become symptomatic in adolescence, with patients typically presenting between ages 12 and 16 years. Some coalitions have been documented in children as young as age 6 years,[46] but these are rare cases. Pain is usually the presenting complaint (Fig. 22–78). The pain often is over the tarsal sinus, but it may also be localized beneath the medial malleolus, along the arch of the foot, or occasionally on the dorsum of the foot. The pain is exacerbated by vigorous sports activities, particularly running on uneven surfaces. Patients may also notice external rotation of the foot and stiffness of the hindfoot. Progressive flatfoot also is an occasional complaint.

Many patients have asymptomatic coalitions in the contralateral foot when a coalition is identified (50 to 60 percent of coalitions are bilateral). The other foot may remain asymptomatic, or it may become painful and stiff later on. This lack of correlation between symptoms and radiographic findings has led to considerable speculation as to the pain-producing mechanism when a coalition is present. Some investigators suspect that a partial coalition produces symptoms as a result of stress on the fibrous coalition or, possibly, from a fracture of a coalition.

The predominant physical finding is decreased range of motion of the subtalar joint in a patient with a flatfoot. Passive inversion and eversion of the calcaneus is limited or absent. Gentle efforts to move the subtalar joint may result in some motion, but the motion is limited by a grab or spasm of the peroneals when the patient feels pain with the movement. When the patient walks, there may be fixed external rotation of the foot with a larger than normal foot progression angle. When the patient does a toe rise, there is no inversion of the heel (normally, the calcaneus inverts as a person rises on the toes). This is best viewed from the back. A lack of tibial external rotation during a toe rise has also been noted in patients with tarsal coalitions (Table 22–3).[20] A useful test is to have the patient attempt to walk on the lateral border of the foot, an impossible task if there is limited inversion of the foot.

A tarsal coalition is easily distinguished from a flexible flatfoot simply by the range of motion of the hindfoot. By definition, the flexible flatfoot has a full or excessive range of inversion and eversion, although there may be an occasional patient with a tight heel cord.

A difficult clinical problem is the child or adolescent who presents with classic symptoms and findings of a tarsal coalition, but no coalition is identified on radiographs. In many of these cases, surgical exploration of the feet has yielded variable findings. Some feet have fibrous coalitions, while others have unexplained inflammatory changes in the subtalar joint. The clinician should remember that the subtalar joint often is involved in children with pauciarticular juvenile arthritis, and the joint may be the presenting site of the disease. Thus, other joints should be carefully examined to rule out systemic arthritis.

FIGURE 22–76 Complete medial talocalcaneal bridge in the foot of a 72.3-mm fetus (coronal section). (Courtesy of Barbara Anne Harris Monie. From Harris RI: Retrospect-peroneal spastic flat foot. J Bone Joint Surg 1965;47-A:1658.)

A

B

FIGURE 22–77 Radiographic findings in a child with mild complaints of foot pain. **A,** AP radiograph showing a medial bony connection between the talus and navicular. **B,** Oblique radiograph showing the talonavicular coalition. Surgical treatment was not required.

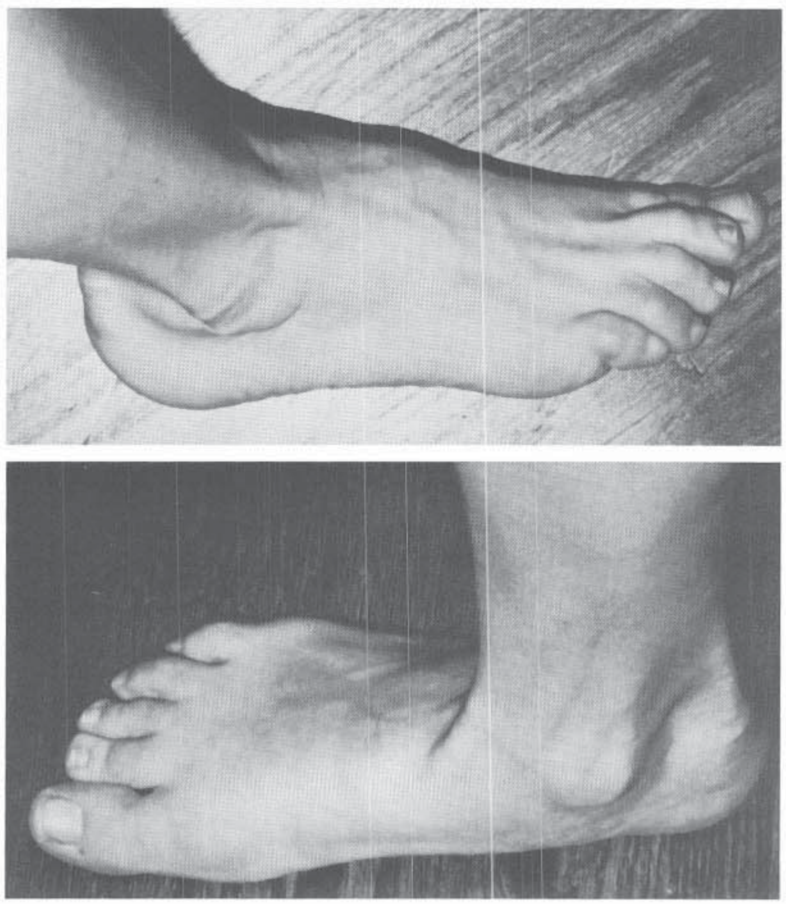


FIGURE 22–78 Clinical findings in peroneal "spastic" flatfoot. Note the severe pes planovalgus with abduction of the forefoot. The peroneal tendons are taut, and there is marked restriction of subtalar motion.

TABLE 22–3 **Clinical Findings Associated with Peroneal Spastic Flatfoot**

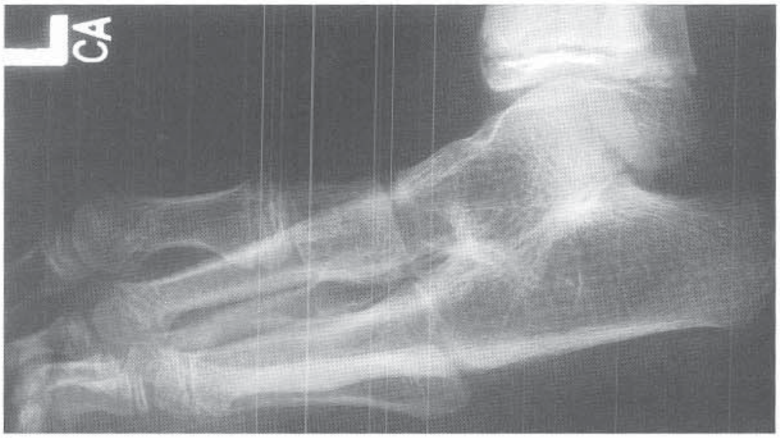
- Restricted subtalar motion
- Hindfoot valgus deformity
- Abduction of the forefoot
- Tightness of the peroneal tendons

Occasionally other foot deformities, such as cavovarus deformity[42] and talipes equinovarus,[41] are noted in patients with coalitions. Patients with fibular hemimelia usually have asymptomatic tarsal coalitions. Coalitions that are associated with a limb deficiency are present at birth and are compensated for by the development of a "ball-and-socket" ankle, which allows inversion and eversion to take place at the tibiotalar joint. These are almost always asymptomatic and do not require treatment.[22,29] The Nievergelt-Pearlman syndrome is a rare, heritable condition in which there are massive tarsal and carpal coalitions (Fig. 22–79).[31] Massive synostosis of the tarsal bones may also be seen in patients with Apert's syndrome (Fig. 22–80).

Imaging Studies. The presence of tarsal coalitions may be suspected from plain radiographs of the foot, but a definitive diagnosis is usually made with CT.

The subtalar joint is a complex consisting of three parts: the anterior and middle facets, in the anterior compartment, and the posterior facet, in the posterior compartment. The two compartments are separated by the interosseous talocalcaneal ligament. All three parts of the subtalar joint as well as the calcaneonavicular area should be studied to rule out the presence of a coalition.

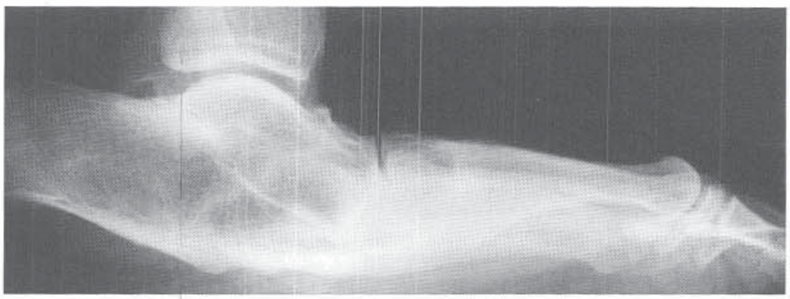
Plain radiographs should include AP, lateral, oblique, and Harris views (Figs. 22–81 and 22–82). The standing oblique view (Fig. 22–83), obtained at a 45-degree angle from lateral to medial, is the best view for identifying a calcaneonavicular coalition (Fig. 22–81). This condition ap-



A

B

C



D

FIGURE 22–79 A 10-year-old girl with Nievergelt-Pearlman syndrome. **A,** Lateral radiograph of left foot showing massive coalition of the talus to the calcaneus. Note also the talonavicular and calcaneocuboid-fourth metatarsal coalition. **B,** Lateral radiograph of the right foot showing coalitions between the talus-calcaneus-cuboid and metatarsals. **C,** Radiograph of the hand showing fusion of the distal carpal row. There is also fusion of the first MCP joints of all four fingers. **D,** Lateral radiograph of the foot of the mother, who was also treated for the Nievergelt-Pearlman syndrome. Both mother and daughter underwent corrective osteotomies through the hindfoot bony mass to improve the weightbearing surface of the foot.

FIGURE 22–80 Massive tarsal coalition in Apert's syndrome. **A** and **B,** Radiographs of feet. **C** to **E,** Characteristic clinical appearance of the feet.

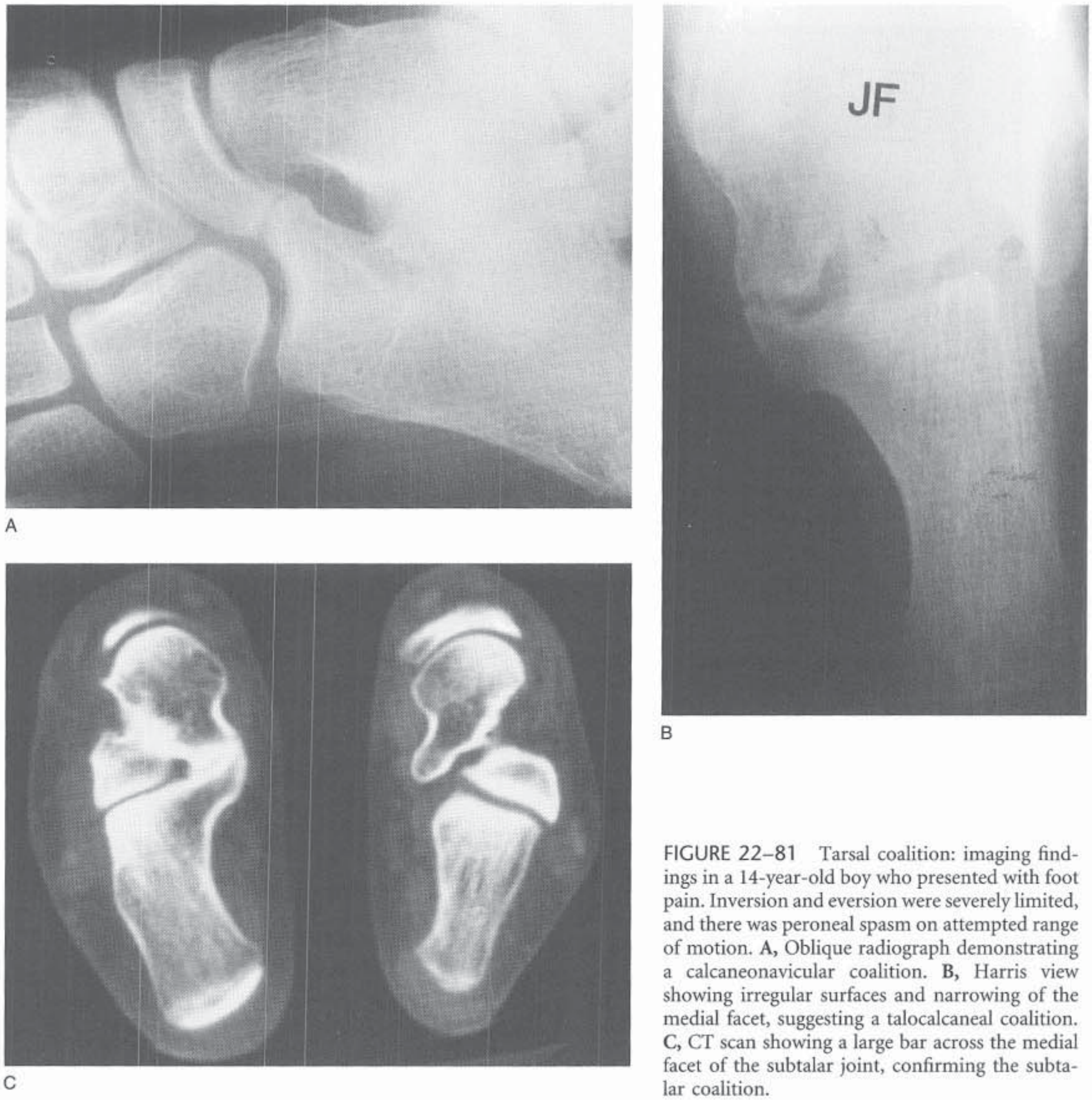


FIGURE 22–81 Tarsal coalition: imaging findings in a 14-year-old boy who presented with foot pain. Inversion and eversion were severely limited, and there was peroneal spasm on attempted range of motion. **A,** Oblique radiograph demonstrating a calcaneonavicular coalition. **B,** Harris view showing irregular surfaces and narrowing of the medial facet, suggesting a talocalcaneal coalition. **C,** CT scan showing a large bar across the medial facet of the subtalar joint, confirming the subtalar coalition.

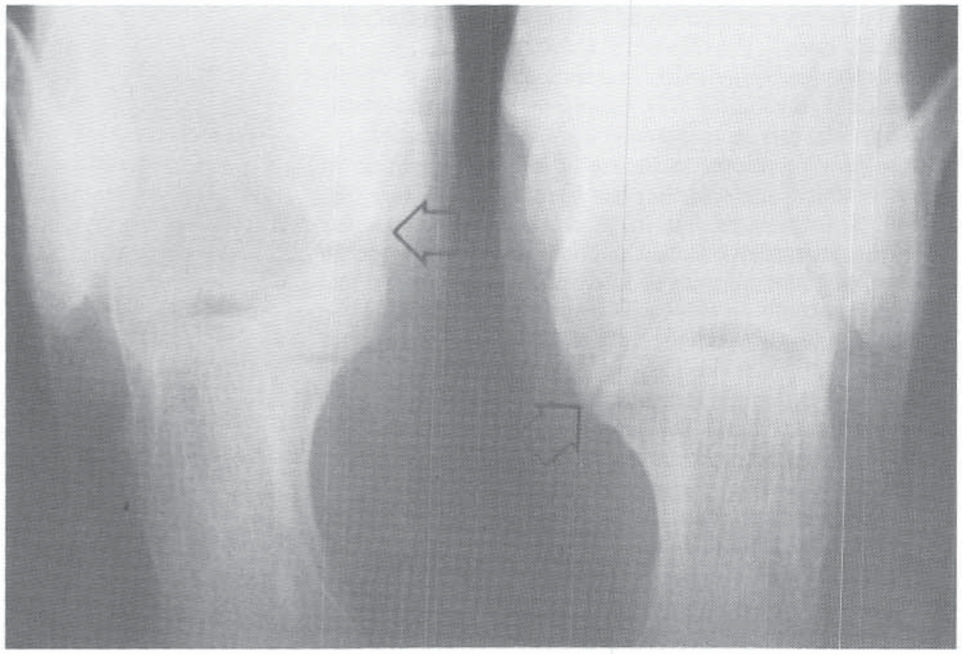


FIGURE 22–82 Tarsal coalition: penetrated axial view of both feet. *Left:* The middle facet joint is normal; note that the radiolucent articular cartilage space lies horizontally. *Right:* fibrocartilaginous coalition of the middle facet joint. Its radiolucent line is tilted medially and downward, with irregular margins that lack cortication.

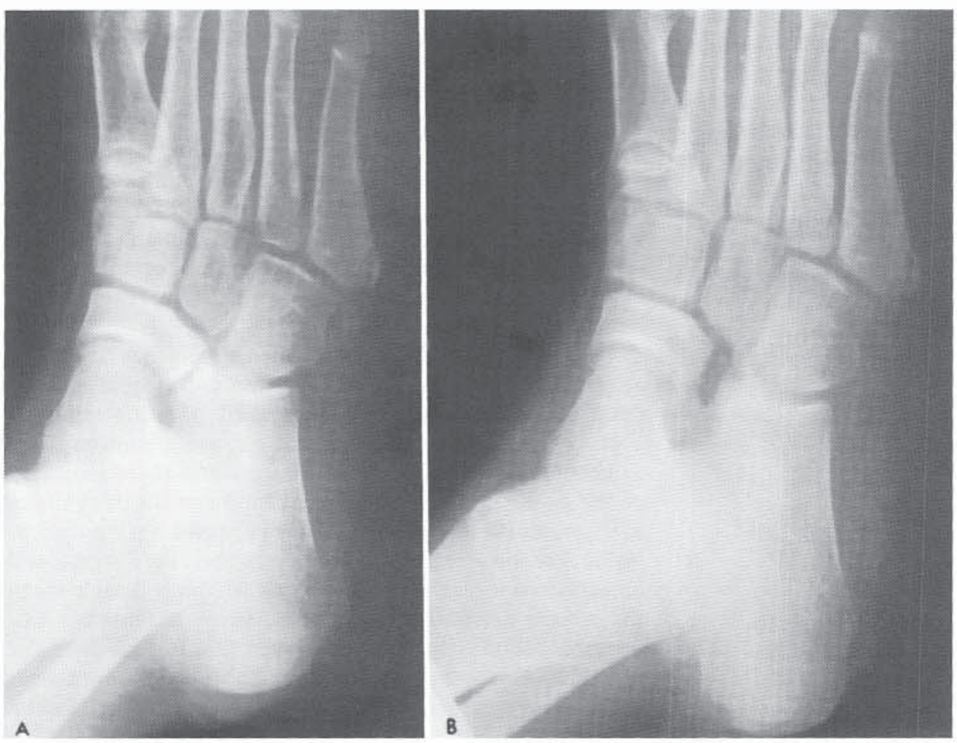


FIGURE 22–83 Calcaneonavicular coalition. **A,** Oblique view of the foot showing a cartilaginous bar. Note the flattened ends of the two bones on either side of the cartilaginous bridge. **B,** Postoperative radiograph obtained after the bar was excised. Following surgery, peroneal spasm disappeared and full range of motion of the subtalar joint was achieved.

pears either as a solid bony connection between the anterior projection of the calcaneus and the navicular or as an irregular lucent line at the junction of the projection of the calcaneus and the navicular. The diagnosis can usually be corroborated with a lateral radiograph, which will show an elongated anterior projection of the calcaneus, the so-called "anteater's nose" (Fig. 22–84).[27] Other coalitions, such as talonavicular, cubonavicular,[49] naviculocuneiform,[12,26,38] calcaneocuboid (see Fig. 22–77), cuboidmetatarsal, and multiple coalitions, can be recognized on plain radiographs.

There is a significant incidence of a second coalition in a foot in which one coalition has been identified (see Fig. 22–81). Thus, any patient in whom a calcaneonavicular coalition is identified on plain radiography should undergo CT of the subtalar complex to rule out any additional coalitions. In any coalition there may be an anterior beak on the talus, which is believed to be a traction spur caused by abnormal motion at the ankle due to the lack of subtalar motion (Fig. 22–85).[48] It has also been proposed that the spur or beak is caused by impingement of the navicular against the head of the talus, which in turn is due to abnormal motion caused by the coalition.[30]

A talocalcaneal coalition may appear on the Harris view as a bony bridge across the medial subtalar joint. The Harris view is a posterior oblique projection in which the beam is directed through the posterior facet of the talocalcaneal joint.[6,16] Narrowing of the joint and obliquity of the joint surface of the medial facet of the subtalar joint may also indicate the presence of a coalition. CT of the hindfoot is the best study for assessing a bony talocalcaneal coalition. A narrowed medial facet joint suggests a fibrous coalition. CT also allows the extent of the coalition to be determined.[17,34,44,46,47] If the coalition involves more than 50 percent of the posterior facet, the prognosis for regaining motion after resection may be poor.[48]

MRI is even more accurate than CT in demonstrating fibrous tarsal coalitions, but MRI is not necessary to diagnose most coalitions.[47] A technetium bone scan has also been shown to help in the diagnosis of a fibrous coalition when the CT appearance is equivocal.[9,24] Increased uptake in the area of the subtalar joint indicates that the coalition is responsible for the patient's symptoms.

When a bony coalition is present at birth, such as in some cases of fibular hemimelia, the ankle will adapt to allow inversion and eversion motions to occur at the ankle joint. The talus will become dome-shaped and the distal tibia will be spherically shaped, like an acetabulum. This is termed the "ball-and-socket" ankle.[22]

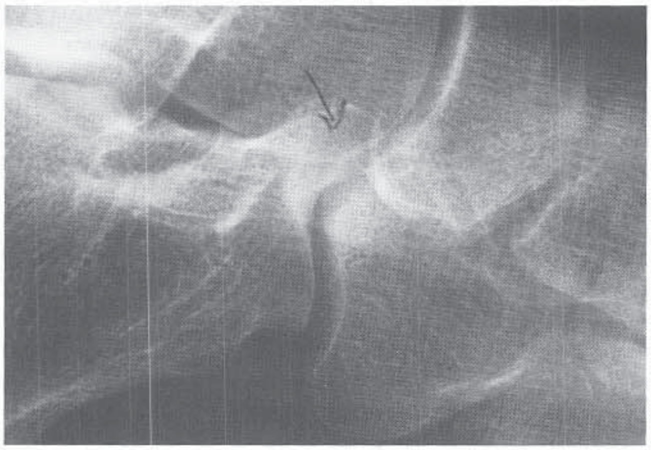


FIGURE 22–84 "Anteater's nose," associated with a calcaneonavicular bar a prominent dorsal process of the calcaneus.

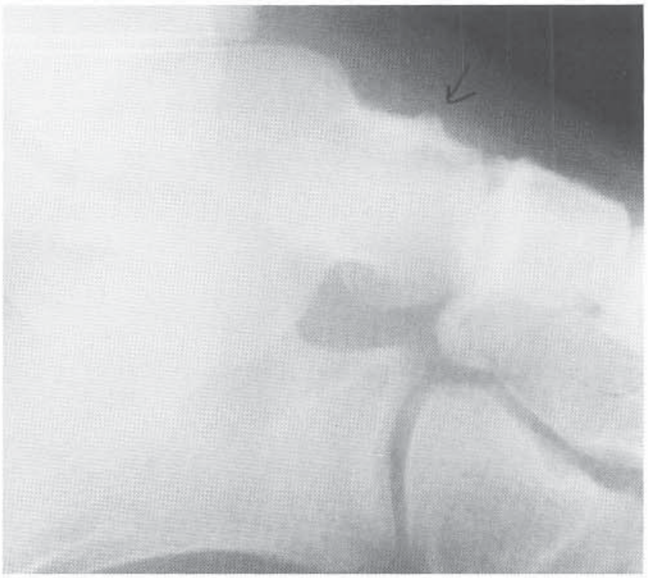


FIGURE 22–85 Talar beaking, a prominence on the dorsum of the talus, is often associated with tarsal coalitions. It is probably a traction spur from abnormal tarsal mobility.

Treatment. All authors agree that an initial trial of conservative treatment is necessary and that it may be successful in relieving the pain associated with a tarsal coalition. The most common nonoperative approach is the use of a firm orthosis, flattened on the bottom to reduce inversion and eversion stresses on the foot. To achieve this reduction in motion, we prefer to use the UCBL (University of California Biomechanical Laboratory) orthosis. The addition of high-top shoes may also be helpful. For those children who do not experience relief of symptoms from an orthosis, a 4- to 6-week period of immobilization in a short-leg walking cast may significantly alleviate symptoms, and the relief may be long-lasting.

Patients who continue to have symptoms that limit their activities despite conservative therapy are appropriate candidates for surgical excision of the coalition. A contraindication to surgery is the presence of massive coalitions, such as those that involve the medial facet and more than half of the posterior facet. Beaking of the talus is not evidence of degenerative disease of the ankle joint and is not a contraindication to surgical resection.[48] At times, the surgeon will be surprised to find that a rigid foot becomes flexible when the patient is anesthetized. These patients often have a fibrous coalition with peroneal spasm limiting motion. The presence of foot motion under anesthesia, however, does not necessarily obviate the need for resection of the coalition.

CALCANEONAVICULAR COALITION. A calcaneonavicular coalition is excised through a dorsolateral tarsal sinus incision.[11] Care must be taken to excise the entire coalition. The most plantar extent of the bar is the most difficult to remove. After the coalition is resected, the origin of the extensor digitorum brevis is pulled into the space between the calcaneus and navicular. The muscle is secured there with a suture passed through the plantar surface of the foot and tied over a button (see Plate 22–1). We prefer to immobilize the foot in a short-leg walking cast for 3 or 4 weeks, followed by range-of-motion exercises designed to regain subtalar motion.

MEDIAL TALOCALCANEAL COALITION. Resection of a talocalcaneal coalition is more complex than the excision of a calcaneonavicular bar. The coalition is approached medially through a short, horizontal incision extending from the anterior margin of the Achilles tendon to the talonavicular joint just below the sustentaculum tali (Fig. 22–86). The incision represents the medial half of a Cincinnati incision. The flexor digitorum and the flexor hallucis tendons are identified and released from their sheaths. The coalition usually lies deep to the flexor hallucis. Because it is difficult to distinguish the bar by direct dissection, it is helpful to identify the subtalar joint posteriorly and anteriorly so that the bar lies between two recognizable joint surfaces.

A dissection is made between the Achilles tendon and the neurovascular bundle to identify the posterior facet of the subtalar joint. The exposure of the posterior subtalar joint is similar to the approach for a clubfoot correction. The joint is followed medially until the coalition is encountered. Anteriorly, the talonavicular is identified. The anterior subtalar facet is located just behind the talonavicular joint. Just posterior to the anterior subtalar facet, the surgeon will encounter the anterior margin of the coalition. The coalition is identified and is then resected until the nonfused subtalar joint surfaces are seen. High-speed burs, rongeurs, and curet are used to excise the bar. Bone wax is interposed on the exposed bone surfaces to prevent refusion. An autogenous free fat graft may be used as an interposition.[8,28] A split portion of the flexor hallucis tendon has also been used as an interposition graft between the margins of the coalition.

Once the coalition has been removed, the subtalar joint is taken through a range of motion to assess the completeness of the resection. If motion is still limited, the surgeon should further expose the subtalar joint complex and inspect it to be certain that the entire coalition has been removed.

Fusion of the hindfoot may be necessary to alleviate symptoms in patients with extensive coalitions and those in whom excision of a coalition does not provide relief. In these situations, we prefer to perform a Grice-type extra-articular fusion. Peterson has reported using a dowel technique, which may be used to fuse the talocalcaneal, talonavicular, or calcaneocuboid joint or all three joints.[32] Others recommend a triple arthrodesis in cases where excision of the coalition does not provide relief of symptoms.[6]

The unusual coalitions of the calcaneocuboid, cubonavicular, naviculocuneiform, and other joints are usually treated symptomatically. If further treatment is necessary, arthrodesis of the subtalar joint may be indicated, but there are few reports of treatment of these entities.[12,26,38,49]

Results and Complications. Many authors have reported almost uniformly successful results after resecting calcaneonavicular and talocalcaneal coalitions. Swiontkowski and

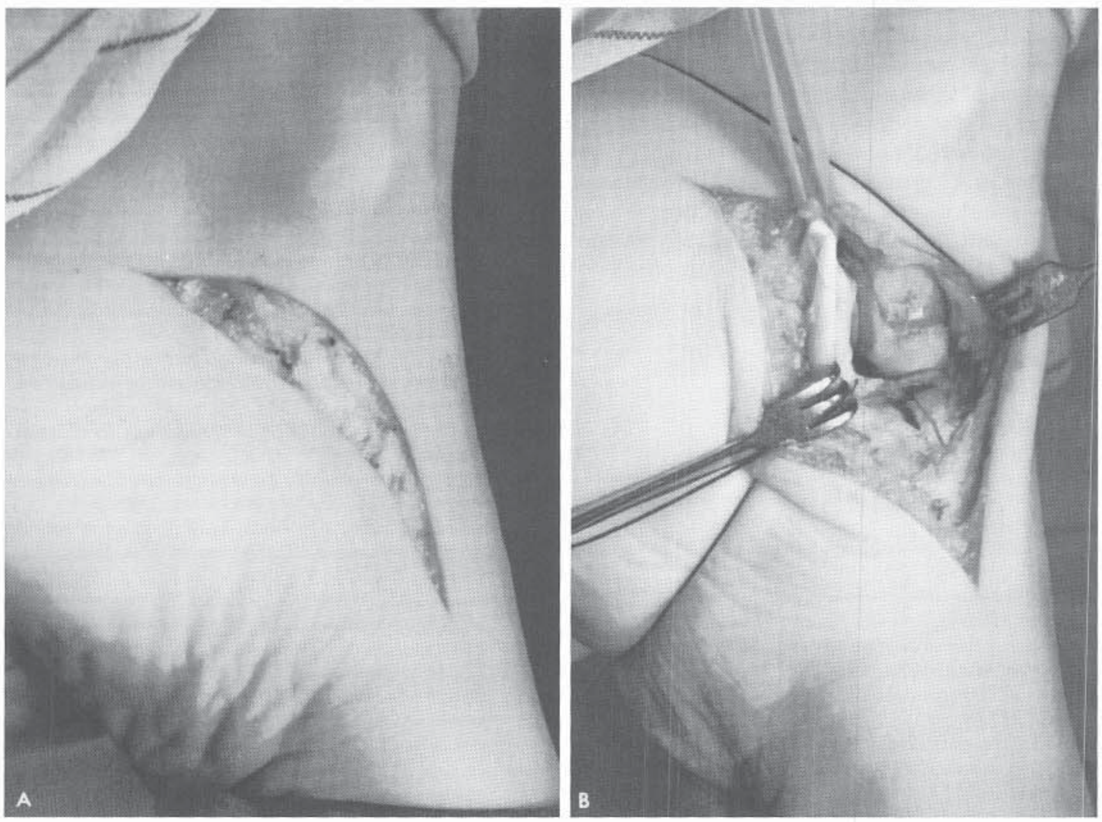


FIGURE 22–86 Medial approach to the subtalar joint for resection of a talocalcaneal coalition. **A,** The skin incision is begun at the base of the first cuneiform bone and ends 2 cm inferior and posterior to the tip of the medial malleolus (the bony prominence in the photograph). **B,** The posterior tibial tendon is elevated and retracted inferiorly and posteriorly, exposing the subtalar joint.

associates had only two failures in 57 operations for coalition, which included both resections and fusions.[43] Olney and Asher reported one fair and one poor result following resection of 10 coalitions of the talocalcaneal joint.[28] In both unsatisfactory results, reexploration revealed a lack of complete resection during the primary procedure. McCormack and colleagues followed Olney and Asher's patients for a minimum of 10 years after their surgeries and found that eight of nine patients continued to do well.[25] Gonzalez and Kumar reported only five poor results after resection of calcaneonavicular coalitions in 75 feet.[11] Kumar and associates had only one poor result following middle facet resections of the talocalcaneal joint.[21] The authors identified three types of coalition: type I was an osseous bridging, type II a cartilaginous coalition, and type III a fibrous coalition. The type of coalition, however, did not influence the outcome.

Other authors have reported residual pain and stiffness after resection, and have performed arthrodeses for these patients. Wilde and associates found that if the coalition involved 50 percent or less of the area of the posterior facet of the calcaneus, the outcome following resection was good.[48] When fusion of the posterior facet was greater than 50 percent, the postoperative results were not good. These feet had heel valgus greater than 16 degrees, and most had narrowing of the posterior talocalcaneal joint and impingement of the lateral process of the talus on the calcaneus. Comfort and Johnson reported a 77 percent success rate resecting talocalcaneal coalitions when the coalition involved one-third or less of the total subtalar joint surface.[5] Peterson described the use of a dowel fusion for those coalitions that were unresectable or had failed resection.[32]

In our experience, large coalitions and, at times, average-size coalitions in large patients may remain symptomatic after resection. Most of these cases have subsequently been treated with either a subtalar fusion or a triple arthrodesis. We have also encountered a small number of patients with the clinical features of a peroneal spastic flatfoot in whom no coalition was found during surgical exploration. These patients have persistent symptoms postoperatively, and to date we have not identified the cause of their problem.

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CLEFT FOOT

Cleft foot is a congenital anomaly characterized by a deficit in the central rays of the foot. The first report of the disorder was from South Africa in 1770.[5] The unfortunate term "lobster claw" deformity was first used by Cruveilhier in 1829.[4]

Etiology. The disorder is very rare, estimated to occur in one in every 90,000 births. Inheritance is autosomal dominant with variable penetrance.[1,8,10,12] Cleft foot is probably caused by a functional defect in the apical ectodermal ridge, which induces limb bud formation in the embryo.[1,8] Further work has suggested that the locus of the defect may be at chromosomal region 7q21.2-q21.3.[3]

Clinical Features. The majority of cases are bilateral, but occasionally a single cleft foot will occur.[6,7] The hands are often cleft as well, and the cleft foot may also be associated with triphalangeal thumb.[9]

The degree of defect varies considerably among patients, but the defect itself is usually conical in shape with the base being distal. In more severe cases, as progressively more rays are deleted, the deletions move from the tibial side toward the fibular side, with the fifth ray the last to be affected. The first metatarsal may be of normal size, or it may be broad and connected toward the center of the defect.

The different degrees of deformity have been nicely classified by Blauth and Borisch.[2] Their classification system is based on the study of the radiographic morphology of 45 cleft feet from their own patient population and 128 cleft feet from the literature. Types I and II are feet with minor deficiencies, both with five metatarsals. In type I the metatarsals are all normal, whereas in type II they are partially hypoplastic. As the degree of deformity increases, the number of metatarsals decreases: there are four metatarsals in type III, three in type IV, two in type V, and one in type VI. The authors also reported polydactylous and monodactylous cleft feet with distal tibiofibular diastasis.

Treatment. Treatment of the disorder is somewhat controversial. Some authors propose that the feet be left untreated if they are plantigrade and capable of shoe wear, whereas others advocate closure of the defect before walking age. We have found that most widely split feet function well without surgical closure and the patients usually are able to wear shoes that are smaller than usual but are not modified. Occasionally, custom-molded orthotics are necessary to improve shoe wear or to relieve excessive pressure over prominent areas on the sole of the foot. We recommend surgical closure if shoe wear cannot reasonably be expected because of the degree of deformity present.

Based on a series of 42 operations in 15 feet, Wood and associates[14] have recommended a closure technique (Fig. 22–87), with the procedure possible only if at least two metatarsals are present. First, rectangular flaps are raised until the entire skin of the cleft is open. These flaps are then approximated and metatarsal osteotomies are performed, if

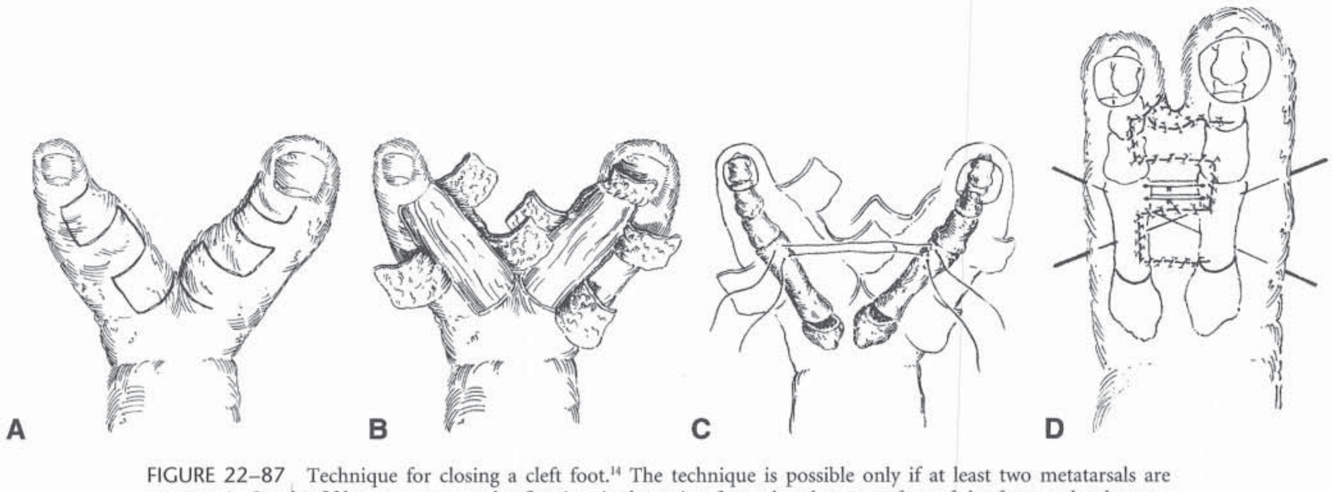

FIGURE 22–87 Technique for closing a cleft foot.[14] The technique is possible only if at least two metatarsals are present: **A,** On the fifth ray, a rectangular flap is raised starting from the plantar surface of the foot to the dorsum. Opposite this flap on the first ray, a rectangular flap is raised starting on the dorsum to the plantar surface. **B,** The flaps are raised until the skin of the entire cleft is removed. At the distal tip of the toe with the greatest length, a flap is raised to suture to the adjacent toe to make a wide toe web. **C,** If the toes spring apart, a closing wedge osteotomy is done at the base of each metatarsal to centralize the bones. An attempt is made to create an intermetacarpal ligament. **D,** K-wires are inserted until the bones heal, usually in 6 weeks. (From Wood VE, Peppers TA, Shook J: Cleft-foot closure: a simplified technique and review of the literature. J Pediatr Orthop 1997;17[4]:502.)

necessary. The intermetatarsal ligament is reconstructed with local ligamentous tissue, joint capsule, or tendon from the cleft, or from autografted plantaris or fascia lata. The closure is stabilized with K-wires. The authors recommended that the procedure be performed at about 6 months of age and before 1 year of age.

An alternative technique has been reported by Sumiya and Onizuka.[11] In their procedure, the defect is closed and a third toe is created with double pedicle flaps from the cleft area. Subsequent procedures divide the third and fourth toes to create five toes, using free skin grafts from the skin defects. The toes are reconstructed for cosmetic reasons. Subsequent grafting procedures are usually necessary for managing retraction of the toes. The authors recommend that surgery be performed before age 1 year.

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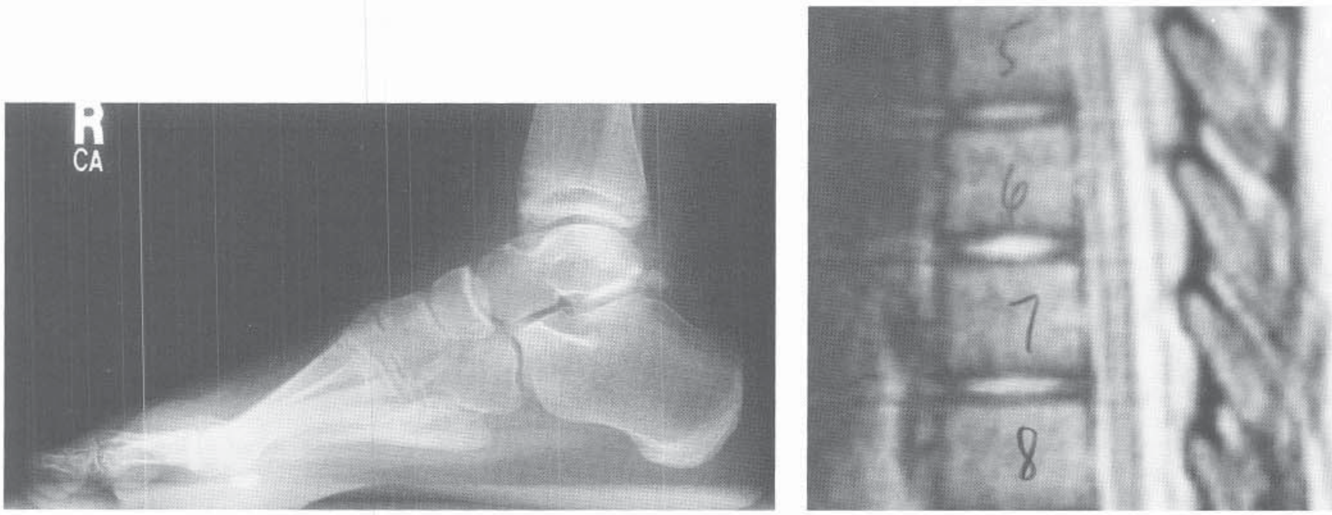
Neurogenic Abnormalities

CAVUS FOOT

Pes cavus is defined as an abnormal elevation of the longitudinal arch of the foot. The deformity is complex, consisting of forefoot equinus and varus or calcaneus of the hindfoot.

Etiology. The etiology of pes cavus is usually neurologic.[8,22,45] Such conditions as cerebral palsy,[7] poliomyelitis, Friedreich's ataxia, and myelomeningocele are frequently associated with pes cavus. Spinal cord pathology such as tethered cord, lipomeningocele, and diastematomyelia commonly result in cavovarus deformities (Fig. 22–88).[28,64] Patients with Charcot-Marie-Tooth disease and other peripheral neuropathies may initially present for treatment of cavus.[34] Pes cavus has also been described in patients with tumors or injuries of the sciatic nerve.[4,6]

The common thread among all these neurologic conditions is the presence of muscle imbalance. Cavus feet are produced by weakness and contracture of intrinsic foot musculature, with preservation of strength in other muscles. For example, in Charcot-Marie-Tooth disease, the posterior tibialis and peroneus longus remain strong, serving to invert the hindfoot and depress the first metatarsal head. The tibialis anterior and peroneus brevis are weak and therefore cannot dorsiflex the ankle, elevate the first metatarsal, or evert the foot. The result of this pattern of weakness is hindfoot varus and forefoot equinus and pronation.[25] The intrinsic muscles of the sole of the foot are weak and become contracted, leading to elevation of the longitudinal arch.[43] Clawing of the toes occurs as the first metatarsal head is depressed, leading to extension of the MTP joint. The toe extensors are recruited to help dorsiflex the ankle, adding



A B

FIGURE 22–88 **A** and **B**, Pes cavus in a 13-year-old boy with thoracic syringomyelia.

to MTP extension, and the long toe flexors are stretched tight, resulting in flexion of the PIP and DIP joints.

Another cause of pes cavus is residual deformity from a clubfoot. Cavus is often present in infants undergoing surgical release, and the recurrent clubfoot frequently has cavus as a component of the deformity.

There are posttraumatic causes for pes cavus, too. Patients who have compartment syndrome of the leg or foot following severe trauma may develop equinocavus deformities. Also, cavus has been seen in children with sciatic nerve palsies from intramuscular injections in the area of the nerve.[6] Children who sustain lacerations of either the peroneus brevis or peroneus longus have been found to develop cavus feet.[9,12]

Lastly, there is a significantly large group of patients in whom a complete neurologic evaluation fails to disclose the cause of the cavus deformities. A small subset of these children present with cavus as babies, in which case the deformities are termed congenital cavus deformities.[3,61]

Clinical Presentation. Cavus deformities of the feet can be divided into those limited to cavus without hindfoot deformity, those associated with hindfoot varus (cavovarus), and those that have hindfoot calcaneus (calcaneocavus). Simple pes cavus is characterized by plantar flexion of the forefoot that is balanced across the medial and lateral aspects of the foot, with even distribution of weight on the first and fifth metatarsal heads. The heel is in neutral position or a few degrees of valgus (Fig. 22–89). Cavovarus is much more common that calcaneocavus. Cavovarus results from elevation of the longitudinal arch with plantar flexion of the first metatarsal and, to a lesser degree, the second metatarsal. The first metatarsal is pronated. The depressed first metatarsal head acts as one limb of a tripod. When the metatarsal head strikes the floor, it results in inversion of the heel, thus leading to varus (Figs. 22–90 and 22–91). Calcaneocavus usually occurs in flaccid paralysis, such as myelomeningocele or poliomyelitis, and is caused by paralysis of the gastrocsoleus. The hindfoot is in fixed calcaneus position and the forefoot is plantar flexed.

Patients may present for treatment of cavovarus with complaints of frequent ankle sprains. The deformity of the foot creates an unstable base for weightbearing, and the ankle twists during activities. Ankle instability in those patients who have a neurologic cause for their cavovarus deformities is compounded by the muscular weakness in the calf. Other patients present with complaints of pain and callosities on the soles of the feet. The varus deformity of the hindfoot leads to calluses along the lateral border of the foot, especially in the area of the base of the fifth metatarsal (Fig. 22–92). Plantar flexion and equinus of the metatarsal heads in the presence of hyperextended MTP joints due to claw-toe deformities leads to abnormal pressure under the metatarsal heads and metatarsalgia. Lastly, those patients with clawing of the toes may have calluses or blisters over the dorsum of the flexed PIP joints of the toes which can rub with shoe wear.

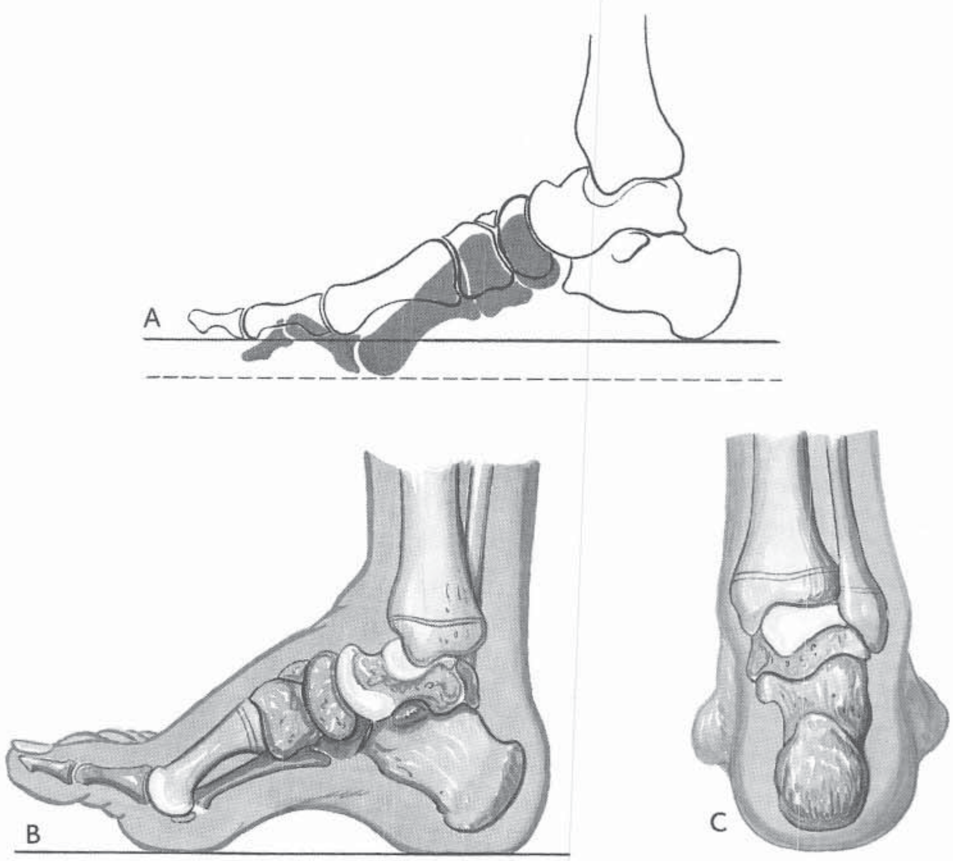


FIGURE 22–89 Cavus deformity of the foot. **A,** Pes cavus. There is fixed equinus deformity of the forefoot on the hindfoot. **B** and **C,** Simple pes cavus. The plantar flexion deformity of the forefoot is equal in its medial and lateral columns and the heel is in neutral position.

FIGURE 22–90 **A** and **B**, Pes cavovarus. Note the plantar flexion of the medial column of the forefoot and the inversion of the heel.

Clinical examination first should focus on documenting the components of the foot deformity.[26] Young patients less than 10 years old may have elevation of the longitudinal arch in the sitting position, but the arch may flatten somewhat when the child is standing if the deformity is flexible. The plantar fascia feels like a tight band in the sole of the foot as the forefoot is dorsiflexed. The tight structures include the plantar fascia, the abductor hallucis, the flexor hallucis brevis, the flexor digitorum brevis, the abductor digiti quinti, the interossei, the posterior tibialis, and the plantar aspects of the capsules of the joints of the midfoot. The foot appears shorter than the contralateral side because the midfoot is drawn up as the forefoot plantar flexes (Fig. 22–93).

Equinus of the forefoot should be evaluated, and prominence of the metatarsal heads noted. Some patients have prominence of only the first metatarsal head on the plantar aspect of the foot, while others have equinus of all of the metatarsals, leading to prominence of all the metatarsal heads. The hindfoot should be inspected for varus or valgus, and the presence of equinus or calcaneus determined. True equinus of the hindfoot is very rare in patients with pes cavus. Although the patient may walk on the toes, usually the hindfoot is already maximally dorsiflexed to compensate for the forefoot equinus. The hindfoot simply cannot dorsiflex any more to obtain a plantigrade position (Fig. 22–94).

Coleman described a "block test" to clinically evaluate the varus component of a cavovarus foot. The patient stands on a wooden block, such as one used to measure limb length discrepancy, or a phone book, which is readily available in any office. The heel and lateral border of the foot are placed on the block and the heads of the first through third metatarsals are allowed to drop off the block medially. This allows plantar flexion of the first ray relative to the rest of the foot. If the heel varus is flexible and secondary to the plantar flexion of the first metatarsal, the calcaneus will evert to neutral or valgus with this maneuver (Fig. 22–95). If the varus is fixed, the heel remains in varus despite allowing the first metatarsal to plantar flex (Fig. 22–96). This is clinically important when the surgeon is deciding whether bony hindfoot surgery is necessary in the reconstruction of the foot. Price and Price described having the patient lie prone, then manually examining the hindfoot for flexibility with the knee flexed.[42] Others recommend looking at the position of the hindfoot with the patient kneeling.

A thorough neurologic examination is crucial in the evaluation of a patient with pes cavus. Sensory disturbances, especially disturbances in light touch sensation, vibration, and proprioception, are commonly present in a stocking-glove distribution in patients with peripheral neuropathies such as Charcot-Marie-Tooth disease. Deep tendon reflexes

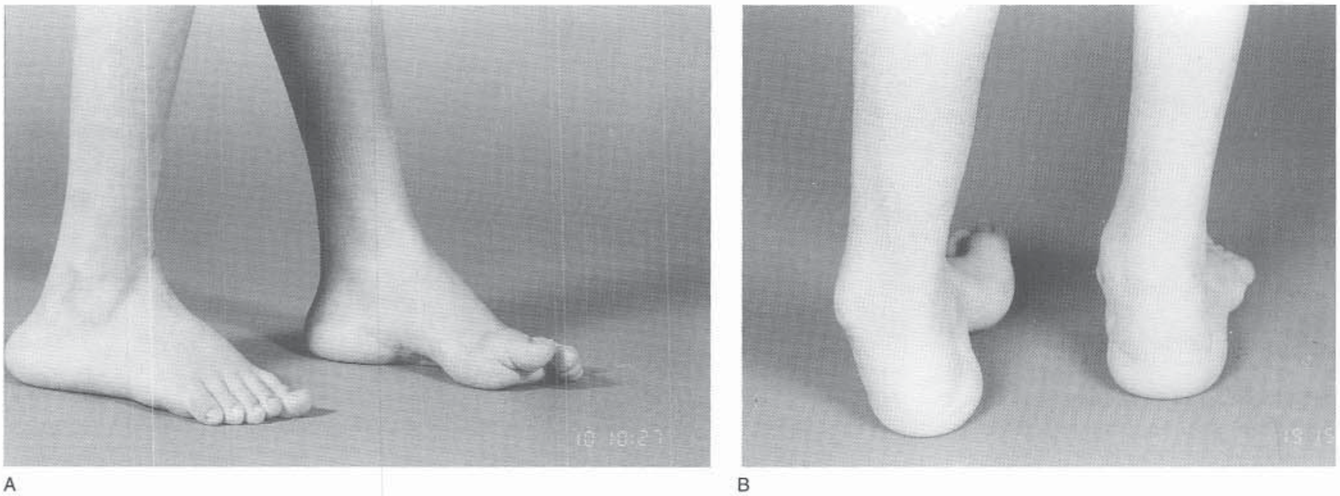


FIGURE 22–91 **A,** Pes cavovarus in a 13-year-old boy with a tethered cord. The first metatarsal is depressed, and there is clawing of the great toe of the left foot. **B,** The heel has a fixed varus deformity.

FIGURE 22–92 Plantar surface of a 13-year-old boy with unilateral pes cavovarus. The fifth metatarsal base is very prominent and has developed a callus.

are diminished in patients with peripheral neuropathy and Friedreich's ataxia, with the ankle jerk decreased more so than the patellar reflex. Ataxia and dysarthria are also seen in Friedreich's ataxia. Clonus is present in children with cerebral palsy and myelopathy due to spinal cord pathology. The motor examination is very important in pes cavus. Muscles that are weak should be identified, and those that remain strong may be useful in tendon transfer to rebalance the forces across the foot.

The legs should be inspected for evidence of atrophy. Patients with asymmetric foot deformities often have relative hemiatrophy of the calf musculature on the side of the cavus foot (Fig. 22–97). Leg length discrepancy is also frequently present, with an ipsilateral short leg relative to the contralateral side.

The spine should be examined in all patients with cavus deformities. A pearl to remember is to examine the bare feet of all children who present for evaluation of scoliosis. The presence of pes cavus in such a patient may be the

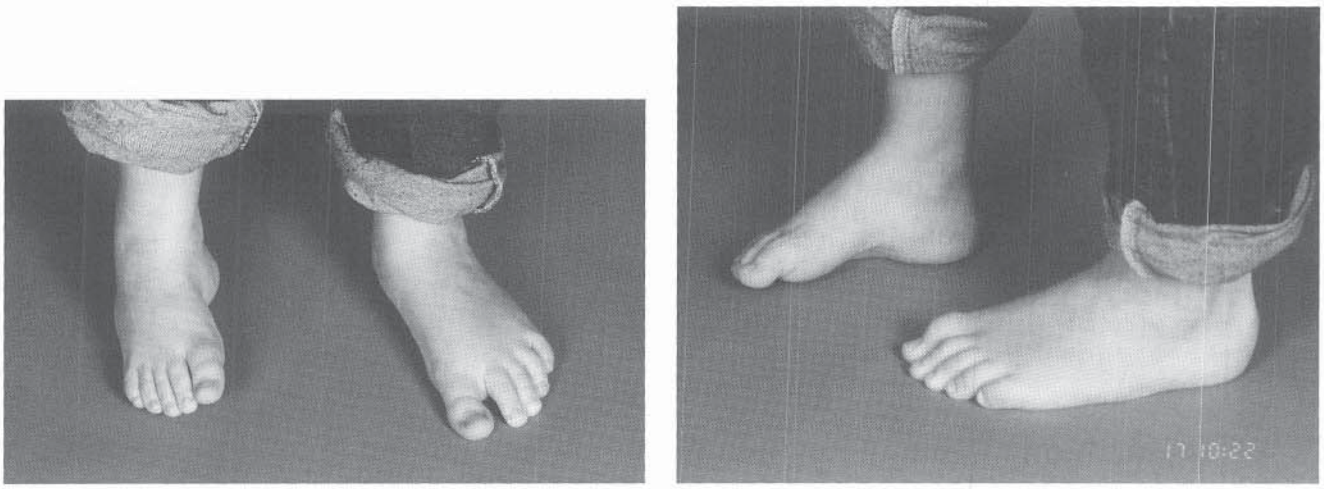


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FIGURE 22–93 **A** and **B,** Ten-year-old boy with a cavus right foot. The foot appears smaller than the contralateral foot due to elevation of the arch and forefoot equinus.

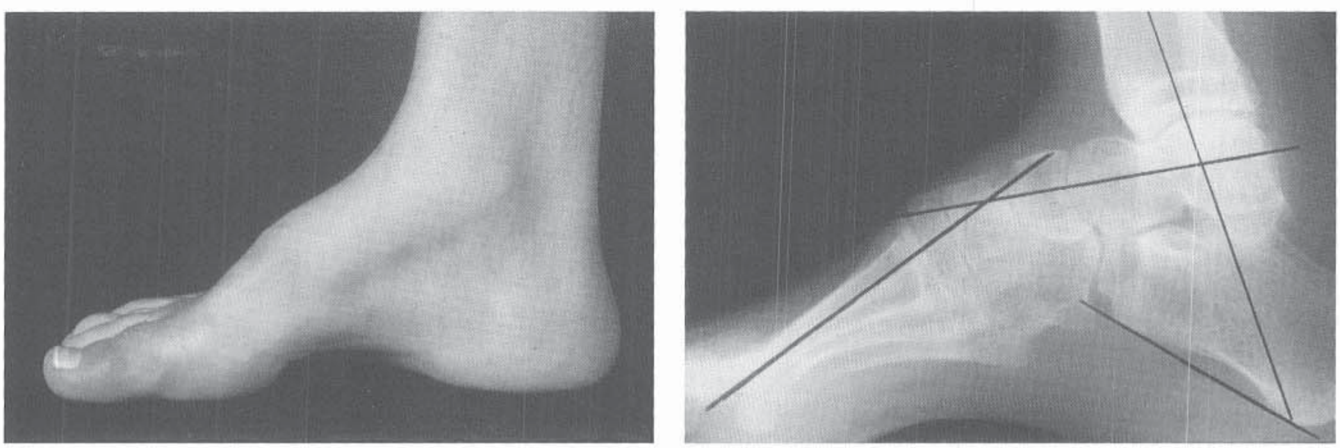

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FIGURE 22–94 **A,** Clinical photograph of a cavovarus foot. **B,** Even though the patient walks on his toes, the calcaneus is maximally dorsiflexed. The apparent equinus is the result of the mid- and forefoot plantar flexion.

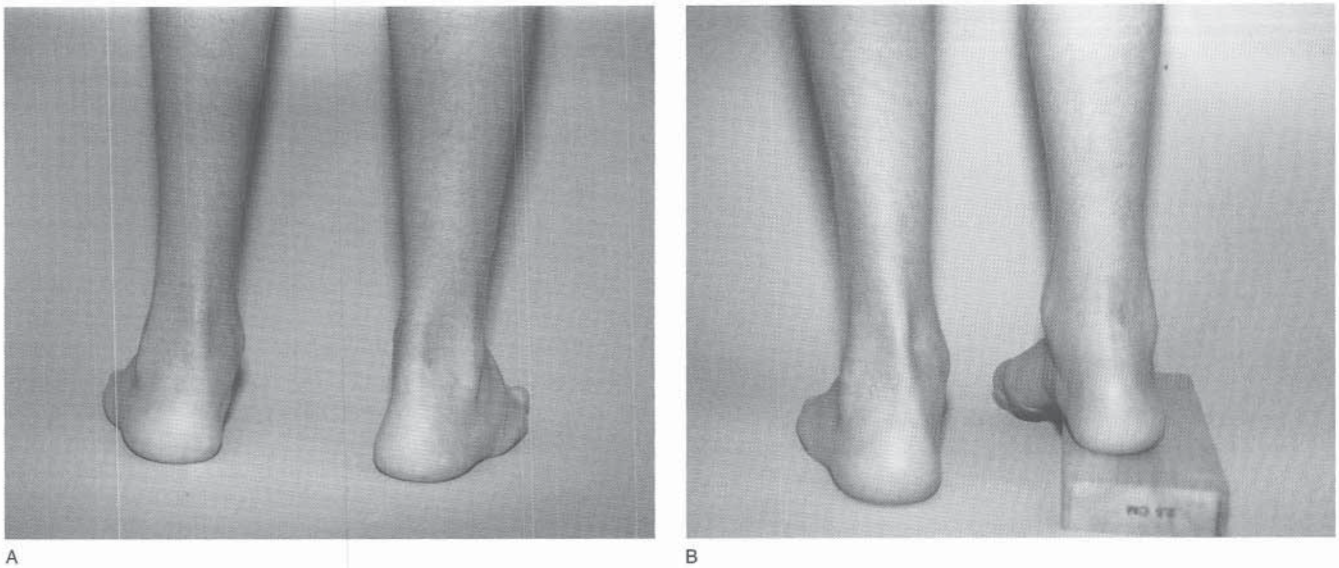

FIGURE 22–95 **A,** Pes cavovarus of the right foot in an otherwise normal male. **B,** The patient is instructed to stand on a block with his first metatarsal unsupported to allow it to plantar flex. The hindfoot assumes a valgus position when the deformity is flexible, as in the child's foot.

first clue to an underlying spinal cord pathology. Signs of dysraphism should be sought. A hairy patch on the back, an overlying hemangioma, or a deep dimple in the sacral region may alert the surgeon to the presence of a tethered cord or lipomeningocele.

Finally, the examiner should question the parents regarding any foot deformity they may have. Charcot-Marie-Tooth disease is transmitted as an autosomal dominant trait, but the phenotype varies among family members. A moderately elevated arch in a parent supports the diagnosis of a hereditary motor and sensory neuropathy such as Charcot-Marie-Tooth disease. If parents report foot deformities, their feet should be examined as well.

Radiographic Evaluation. Standing AP and lateral radiographs can help clarify the deformity present. A standing lateral view of the foot to include the ankle joint is inspected for the position of the calcaneus. The angle of the inferior border of the calcaneus with the tibia should be measured. If the angle exceeds 30 degrees, the ankle is dorsiflexed, and the tendency for the patient to walk on the toes is specifically *not* due to ankle equinus (Fig. 22–98). Some patients do have ankle equinus, such as those who have residual clubfoot deformity, and this can be seen on this film. Dorsiflexion of the calcaneus greater than 40 degrees is excessive.[45]

Méary's angle is the angle between the longitudinal axis of the talus and the first metatarsal shaft on a standing lateral radiograph.[35] The normal value for Méary's angle is zero—that is, the axes of the first metatarsal and talus should be parallel. Plantar flexion of the first ray is seen as an increase in Méary's angle (Fig. 22–98). Hibbs's angle is defined as the angle between a line drawn along the longitudinal axis of the calcaneus and a line drawn down the shaft of the first metatarsal. This angle should be greater than 150 degrees in the normal foot (Fig. 22–99).[23]

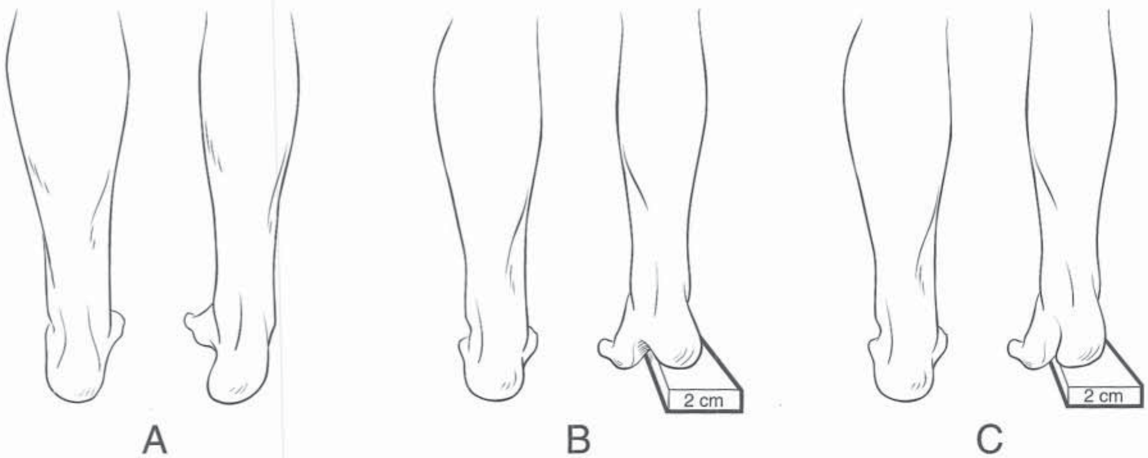

FIGURE 22–96 **A** to **C,** The Coleman block test. The heel of the foot and lateral border are placed on a wooden block, allowing the head of the first metatarsal to drop into plantar flexion. If the hindfoot varus is secondary to the tripod effect of the plantar-flexed first ray, the hindfoot will correct to neutral or valgus alignment.

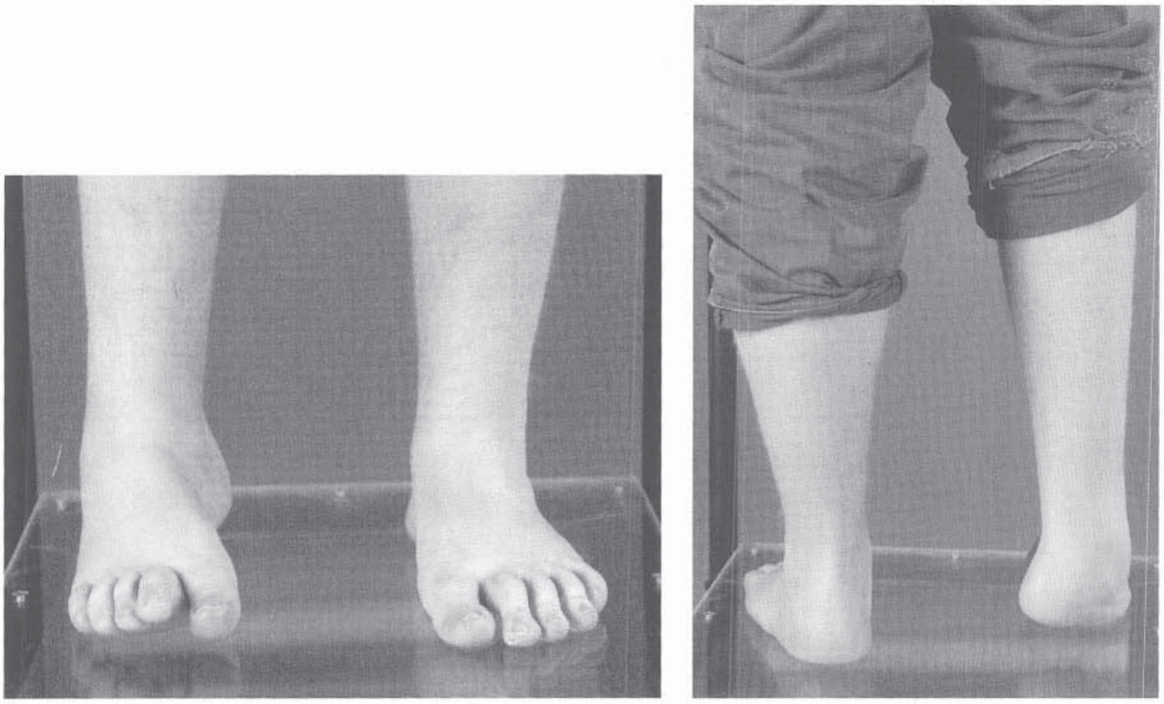

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FIGURE 22–97 **A** and **B**, Clinical appearance of a 16-year-old boy with right pes cavovarus due to probable neuropathy. Note atrophy of the right calf compared to the left.

Forefoot equinus is apparent on lateral weightbearing radiographs in patients with pes cavovarus. The apex of the deformity is often at the level of the cuneiforms or the cuneiform-metatarsal joints. The precise location of the apex of the cavus deformity is important to identify when planning surgical correction.

The subtalar joint should also be inspected on the standing lateral radiograph. Usually there is some overlap of the joint in the normal foot. In cavovarus, the subtalar joint is seen en fosse, so that one can see through it (Fig. 22–98). The calcaneus will also appear shortened in length due to the malrotation of the bone. The lateral talocalcaneal angle is decreased, with the two bones appearing relatively parallel on a standing lateral view of a cavovarus foot.

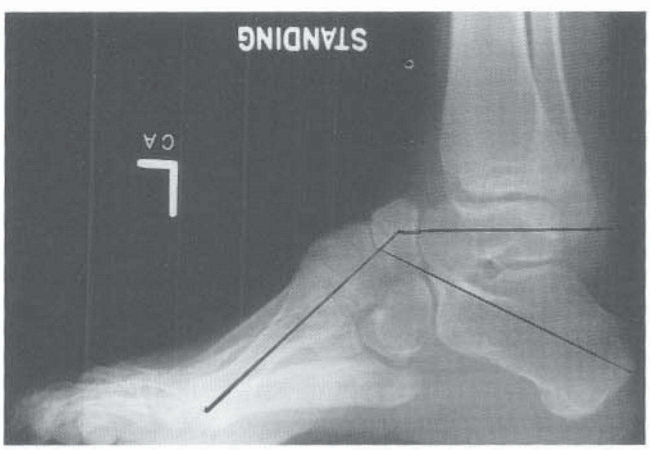


FIGURE 22–98 Radiograph of the foot of a 16-year-old girl with a chromosomal abnormality. The patient walks on her toes because of the cavus deformity. The calcaneus, however, is not in equinus. The first metatarsal is plantar flexed. Meary's angle, which is the angle between the axis of the talus and the shaft of the first metatarsal, measures 45 degrees (normal is 0 degrees). Hibbs's angle, which is the angle between the axis of the calcaneus and the first metatarsal, measures 110 degrees (normal is >150 degrees).

Further Diagnostic Evaluation. When the cause of the varus deformity is not readily apparent, a pediatric neurologist should be consulted. MRI of the brain and spinal cord is performed to rule out cerebral palsy or cord abnormalities. Electrodiagnostic studies are usually performed. Nerve conduction velocities are significantly slowed in patients with type I Charcot-Marie-Tooth disease, one of the most common causes of pes cavovarus. When the deformity is due to denervation, EMG will show a neuropathic pattern with fibrillation.

With recent discoveries in the field of molecular genetics, DNA studies are frequently done to look for mutations associated with peripheral neuropathies and Friedreich's ataxia.

Treatment

CONSERVATIVE TREATMENT. There is little role for conservative treatment in feet with cavus deformities.[41] Stretching the contracted plantar structures has no proven benefit in these children. Shoe inserts designed to elevate the metatarsal heads may relieve symptoms of metatarsalgia initially. AFOs may be prescribed to improve a drop foot gait seen in conjunction with pes cavus in children with peripheral neuropathies.

SURGICAL TREATMENT. Surgical decision making in pes cavus is determined by the following ten factors, described by Tachdjian:[54] (1) the apex of the deformity, (2) the type of pes cavus (i.e., cavovarus versus calcaneocavus), (3) the

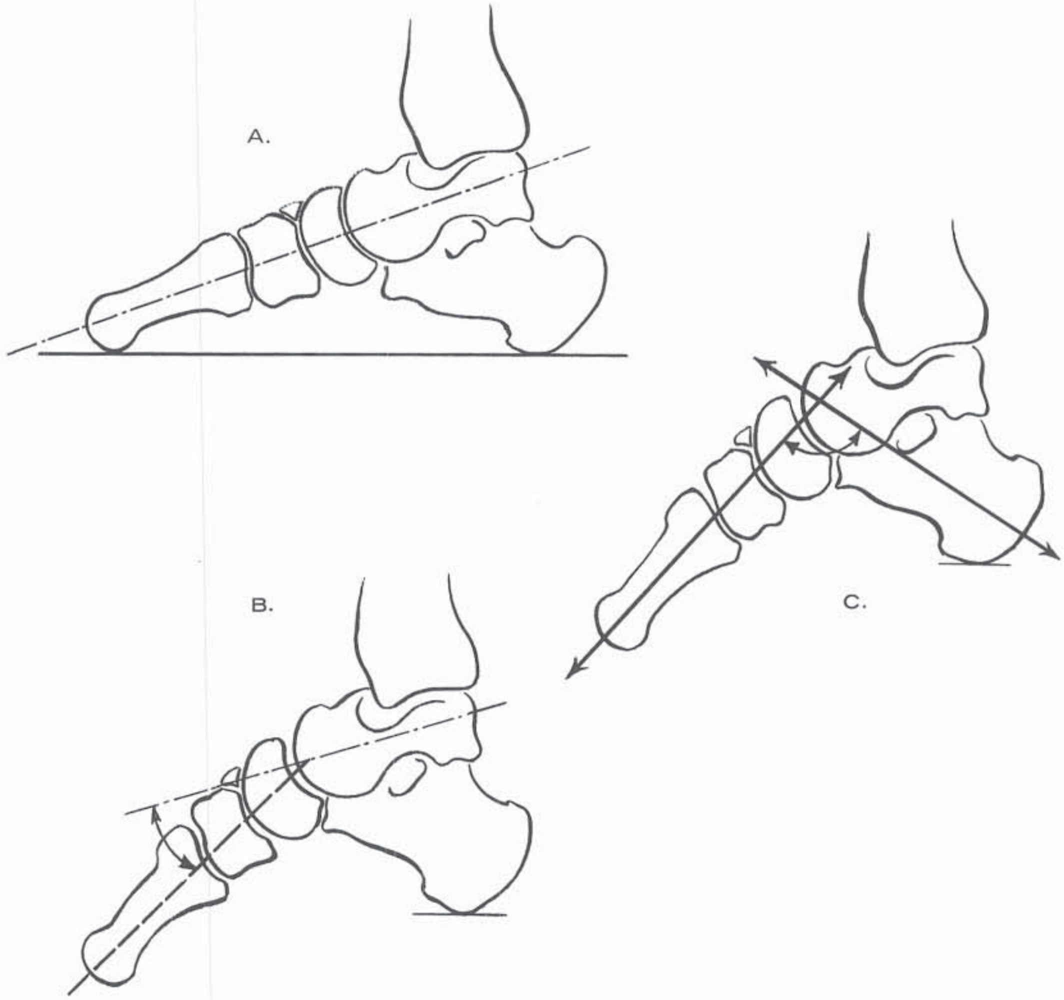


FIGURE 22–99 Methods of measuring the degree of pes cavus in the standing lateral radiograph of the foot. **A,** In a normal foot, the longitudinal axis of the talus is parallel to the longitudinal axis of the first metatarsal. **B,** Méary measures the angle formed between lines drawn through the centers of the longitudinal axes of the talus and the first metatarsal. **C,** Hibbs measures the angle formed between two lines drawn through the centers of the longitudinal axes of the calcaneus and the first metatarsal.

position of the hindfoot, (4) the presence of claw-toe deformity, (5) the presence of skin changes on the sole of the foot, (6) abnormal shoe wear, (7) the rigidity of the deformity, (8) the strength of the muscles, (9) the stability of the neurologic disease, and (10) the age of the patient and the skeletal maturity of the foot.

Surgical treatment for pes cavus can be divided into soft tissue surgery, osteotomies, and triple arthrodesis. Soft tissue surgery is always part of the surgical reconstruction of the cavus foot. Since the deformity is driven by muscular imbalance, contractures must be released and the forces exerted by the tendons rebalanced. In young patients with flexible deformities, soft tissue surgery is adequate to address the deformity. In older patients, flexibility is lost as adaptive bony changes occur, and an osteotomy will be necessary to restore a plantigrade foot.

The decision to surgically correct the hindfoot deformity in a cavovarus foot should be made prior to surgery. The Coleman block test, described earlier, establishes whether or not the hindfoot varus is fixed or is simply compensatory for the plantar-flexed first ray. When fixed deformity is present, soft tissue surgery with or without metatarsal osteotomies will be insufficient to fully correct the foot. Conversely, when the Coleman block test indicates flexibility of the hindfoot, surgical correction of the forefoot equinus by plantar release, usually combined with first metatarsal osteotomy, will allow the hindfoot to assume a more normal neutral to valgus posture.

The preoperative physical examination is also critical when selecting tendon transfers in a cavovarus foot. In patients with progressive peripheral neuropathies, such as Charcot-Marie-Tooth disease, weakness or paralysis of certain muscles rules out their use as potential transfer candidates. Similarly, in such patients there is often a clear lack of ability to dorsiflex the ankle, and in these children, anterior transfer of the posterior tibialis tendon to the dorsum of the foot may be indicated to restore dorsiflexion power.

Soft Tissue Surgery. Plantar release: Plantar release is always performed during surgery to correct a cavus foot. In mild flexible deformities in young children, a simple plantar fasciotomy may be adequate to allow the forefoot to dorsiflex and for the longitudinal arch to flatten. Although percutaneous techniques have been embraced by some, most still prefer an open approach to the plantar fascia.[56] Most often, a more aggressive plantar release is necessary. In this approach the plantar fascia is transected and the abductor hallucis is divided from its origin. Care must be taken to expose the neurovascular bundle medially, and to trace the tibial nerve out to its division into medial and lateral plantar nerves. The nerves may be inadvertently injured during the release if they are not protected (Plate 22–2).

Steindler described his plantar release for the treatment of pes cavus in 1920. Through a longitudinal medial incision, the plantar fascia is first divided. Then the flexor digitorum brevis, abductor digiti quinti, and abductor hallucis brevis are extraperiosteally released from their origins on the undersurface of the calcaneus and stripped distally.[52] Short-leg casts are then applied to stretch the forefoot out of equinus and lengthen the arch. The casts are changed at intervals as the foot is progressively corrected.[48] Additional release of the calcaneonavicular spring ligament, the knot of Henry, and the calcaneonavicular component of the bifurcate ligament has also been described (Fig. 22–100).[41] Paulos, Coleman, and Samuelson even recommended a medial release of the subtalar and talonavicular joints in some patients with cavovarus feet with inflexible heel varus, a procedure they found successful in 85 percent of patients so treated.[41]

Sherman and Westin studied 191 feet in 148 patients who had undergone plantar release for the correction of pes cavus associated with clubfeet and poliomyelitis.[48] They emphasized the need for serial stretching casts to be applied following the release. They found that 83 percent of feet treated by a complete plantar release alone had satisfactory results. The worst results occurred in patients with calcaneocavus from polio with paralysis of the gastrocsoleus. In these patients, Sherman and Westin thought that serial casting was ineffective, owing to the ability to excessively dorsiflex the calcaneus.[48]

We have used a medial incision when performing plantar releases and prefer it, as it permits excellent exposure of the neurovascular bundle. Thometz and Gould are proponents of a plantar incision centered over the sole of the foot.[56] They feel the scar is less problematic, and find that this incision allows them excellent visualization of the plantar structures. Lateral incisions have been also described, but we do not recommend them as the neurovascular structures cannot be seen easily and are prone to injury.

A pearl to remember is to defer lengthening of the Achilles tendon in patients who have a radiographically proven equinus component to their deformity until a later date.[41] When stretching the arch following plantar release, the Achilles tendon provides "something to push against." If a concomitant Achilles tendon lengthening procedure were performed, the tendon would be susceptible to overlengthening, and the stretch of the plantar release would be limited (Fig. 22–101).[34] Luckily, most patients with cavus feet do not have equinus at the ankle, but if this is the case, staging the Achilles tendon lengthening procedure is recommended.

Peroneus longus to brevis transfer: One transfer that has been used as part of the surgical reconstruction of the cavus foot is the peroneus longus to peroneus brevis transfer. The action of the peroneus longus is to depress the first metatarsal head. In patients with cavovarus, the peroneus longus overpowers the anterior tibialis, with excessive plantar flexion of the first ray.[58] Removing the deforming force and transferring the tendon to the peroneus brevis tendon, whose purpose is to evert the foot via its insertion at the base of the fifth metatarsal, can be helpful in maintaining correction in some children.[5,19]

The tendons are exposed laterally beneath the lateral malleolus. The peroneus longus is sutured to the peroneus brevis, and the longus tendon is transected distally. The repair is then protected in a short-leg cast for approximately 6 weeks.

Anterior transfer of the posterior tibialis tendon: Patients with cavovarus feet due to peripheral neuropathy or myopathies such as Duchenne's muscular dystrophy may benefit from anterior transfer of the posterior tibialis tendon (see Chapter 28, Muscle Diseases).[20,37] Candidates for this transfer have weak or paralyzed anterior tibialis muscles, and therefore cannot dorsiflex the ankle during gait. The strong posterior tibialis serves to invert the foot and promotes hindfoot varus. By transferring the posterior tibialis tendon through the interosseous membrane to the dorsum of the foot, the surgeon may be able to convert the deforming force leading to varus and inversion to an active dorsiflexor of the ankle. This transfer is not helpful in patients with cavovarus and cerebral palsy, as it may lead to a reverse deformity of calcaneovalgus over time.

The posterior tibialis tendon transfer is not effective in correcting the cavus foot deformity in and of itself, but it may be useful in augmenting dorsiflexion of the ankle and improving varus of the hindfoot in flexible deformities. The tendon transfer is performed in conjunction with other procedures that improve the cavus deformity.

The surgical technique is described in detail in Plate 28–1 (see Chapter 28, Muscle Diseases). In brief, the tendon is detached from its insertion, retracted up proximal to the medial malleolus, and then brought anterior to the ankle by being passed through a generous window in the interosseous membrane. The tendon is then inserted through a bony tunnel in the midline on the dorsum of the midfoot, and its suture is tied over a button on the sole of the foot. A short-leg cast is worn for 6 weeks.

Transfer of the toe extensors to the metatarsal heads: Patients with pes cavus often also have claw toes, owing to recruitment of the toe extensors to augment weak ankle dorsiflexors and to plantar flexion of the metatarsal heads. The MTP joints of the toes are extended, while the PIP and DIP joints become flexed (Fig. 22–102). The deformity may be flexible or rigid. Blisters and calluses may develop on the dorsum of the IP joints of the toes from rubbing up against the shoes (Fig. 22–103). When severe, the toes are suspended dorsally and do not make contact with the floor, while the metatarsal heads are prominent on the plantar aspect of the foot and develop calluses.

Cole described a procedure in which the extensor tendons are transected at their insertions and then transferred into the cuneiforms.[11] The toe extensors then augment the anterior tibialis in dorsiflexing the ankle.

Chuinard and Baskin recommended transfer of the extensor hallucis longus and extensor digitorum longus proximally into the corresponding metatarsal necks.[10] The IP joint of the great toe and the PIP joints of the lesser toes are then fused to prevent a flexion deformity from developing.[13] This procedure, commonly referred to as a Jones transfer,[30] was

Plantar Fasciotomy

A, A 1- to 2-cm incision is made over the medial aspect of the plantar fascia, which is easily palpable in the sole of the foot.

B, The plantar fascia can be seen within the wound.

C, The fascia is isolated on its dorsal and plantar surfaces, thus protecting the plantar divisions of the tibial nerve. The fascia is then divided using scissors across the sole of the foot.

PLATE 22–2. Plantar Fasciotomy

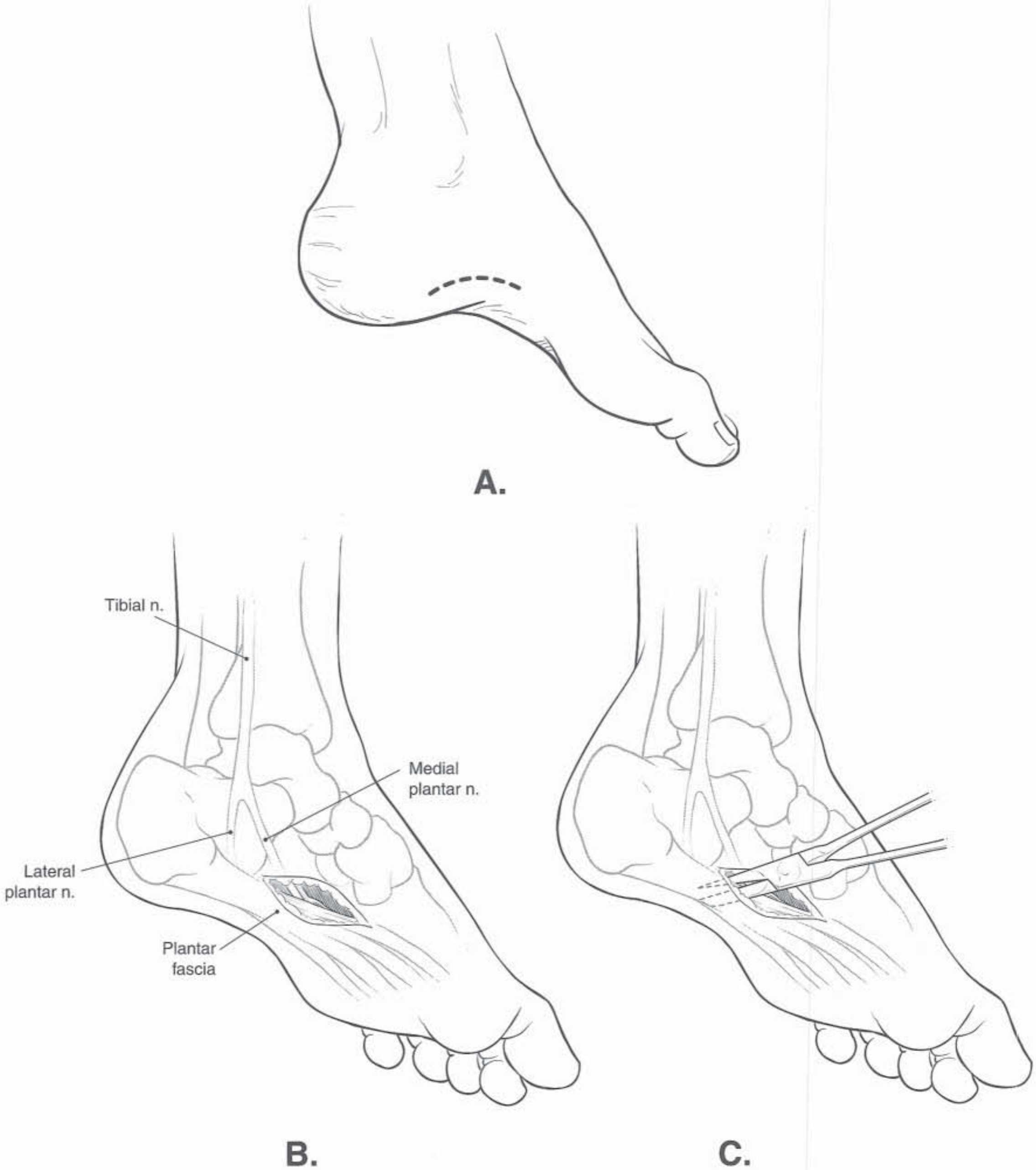


FIGURE 22–100 **A,** Lateral radiograph of a 4-year-old boy with severe cavus deformity and ankle equinus. **B,** Radiograph obtained immediately after surgery, which consisted of complete plantar release (including midfoot capsulotomies), posterior release, Achilles tendon lengthening, and first metatarsal osteotomy. **C,** Standing radiograph obtained 2 months after surgery showing marked improvement in foot position.

described by Hibbs in 1919 (Plate 22–3).[23] The transferred tendons assist in ankle dorsiflexion, and better yet may allow dorsiflexion of the metatarsal heads with improvement in the forefoot equinus component of the cavus deformity.[39,57] The Jones transfer is indicated only when the cavus deformity is being concurrently corrected or when the cavus is flexible. It is ineffective in cases of rigid cavus.

The surgical procedure entails exposure of the distal insertion of the long toe extensors. The tendons are transected, the tendon is tagged with suture, and each metatarsal neck is exposed. A trephine or drill is used to create a bony tunnel for passage of the extensor tendon. The tendon is then passed on its suture through the tunnel in the metatarsal neck and sutured to itself under tension. If there is an extension contracture of the MTP joint, the dorsal capsule should be released. The IP joint of the hallux or the PIP joint of the lesser toes is exposed, and all articular cartilage is resected from the joint. A Steinmann pin is used to fix the joint, and the foot is placed in a cast for 6 weeks to allow fusion. At 6 weeks, usually the IP and PIP joints are sufficiently healed to permit the pins to be removed.

The procedure, while not particularly difficult, can be rather lengthy, particularly when done bilaterally. For this reason, we usually stage these tendon transfers to follow the surgical correction of the cavus deformity—the plantar release and concomitant osteotomies.

Another tendon transfer recommended for use in clawtoe deformity is the Girdlestone-Taylor procedure, which entails transfer of the long toe flexors to the extensor mechanism.[55] This procedure has less predictable results than the Jones transfer, and for that reason it is not usually selected.[56]

Bony Surgery. Metatarsal osteotomies: In patients who have flexible hindfoot varus on the Coleman block test, plantar

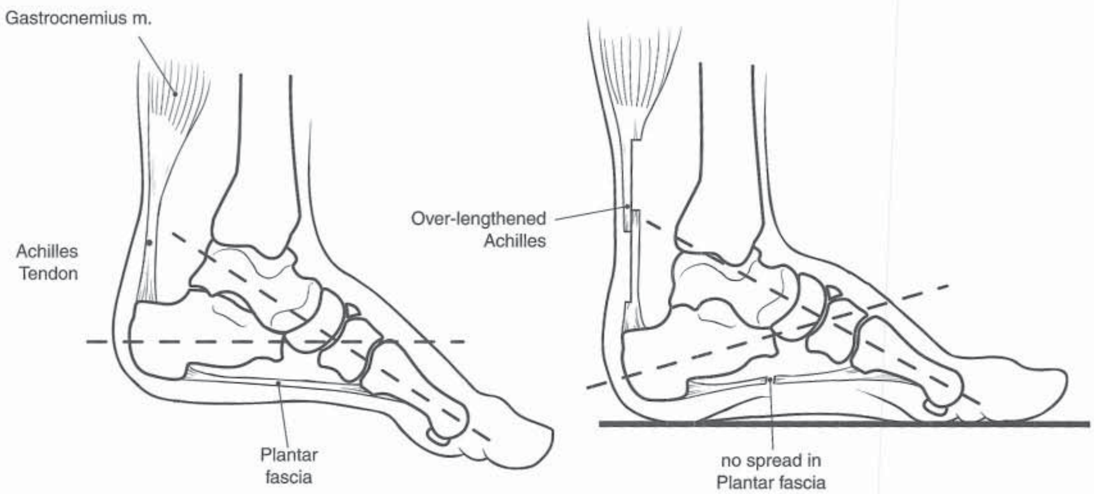


FIGURE 22–101 Equinus coexisting with cavus. The plantar fasciotomy should be performed first. If the Achilles tendon is lengthened simultaneously, the divided plantar fascia cannot be stretched and the Achilles tendon will be overlengthened, resulting in calcaneocavus.

flexion of the medial forefoot may be correctable by first metatarsal osteotomy.[2,34] The osteotomy is performed proximally, so care should be taken to avoid damaging the physis of the first metatarsal. A dorsal closing wedge osteotomy is done, in conjunction with a plantar release, and the forefoot is dorsiflexed. A short-leg cast is then worn for 6 weeks.

The advantage of a metatarsal osteotomy is that it allows correction of the plantar-flexed medial forefoot by bony realignment rather than through stretching casts, which may promote breakdown beneath the first metatarsal head in patients with poor sensation due to neurologic disease. The osteotomy is usually fixed with K-wires or a Steinmann pin, which can be removed 6 weeks following surgery.

Some patients have a more global equinus of the forefoot, with the medial and lateral aspects of the forefoot equally plantar flexed. In such patients, multiple metatarsal osteotomies have been performed to elevate the forefoot and correct the cavus (Fig. 22–104).[59] Greenstick dorsal closing wedge osteotomies of all the metatarsals was described by Swanson and colleagues[53] and later recommended by Gould.[19] Watanabe described performing oblique osteotomies of all five metatarsals to correct pes cavus, with excellent or good results in 84 percent of feet so treated. Complications consisted of persistent metatarsalgia, delayed healing, and residual varus.[60] A rocker-bottom deformity has been described, however, resulting from the prominence of the proximal metatarsals in the sole of the foot.[45] Other possible complications of multiple metatarsal osteotomies include cross-union, nonunion, and delayed union, particularly for osteotomies performed at the base of the fifth metatarsal.[56]

Calcaneal osteotomy: Calcaneal osteotomy is indicated in children with inflexible hindfoot varus on the Coleman block test. The osteotomy is made posterior to the posterior facet of the subtalar joint and extended obliquely and distally to the plantar surface of the calcaneus. Correction is achieved either by sliding the inferior fragment laterally or by creating

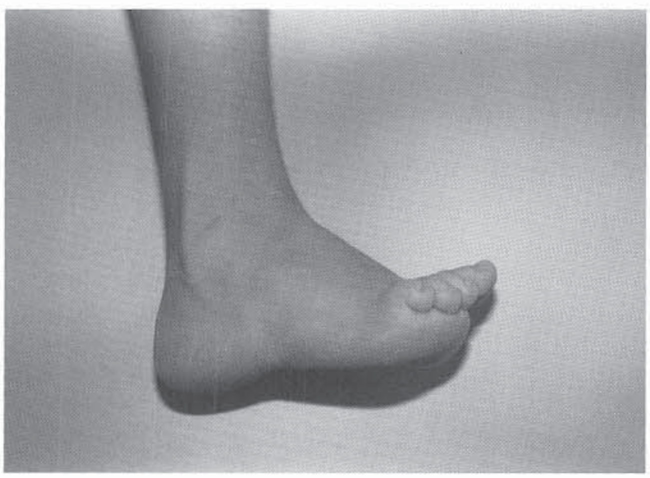


FIGURE 22–102 Clawing of the toes results from weakness of the anterior tibialis muscles. As the patient tries to dorsiflex the ankle, the toe extensors fire. Over time the MTP joints become dorsally subluxed and the PIP and DIP joints of the toes become flexed.

FIGURE 22–103 Teenage girl with Charcot-Marie-Tooth disease and symptomatic clawing of the lesser toes. The patient complained of rubbing of the dorsum of her toes against her shoes.

Transfer of Long Toe Extensors to Heads of Metatarsals (Jones Transfer)

A, A longitudinal incision is made on the dorsomedial aspect of the first metatarsal extending from the base of the proximal phalanx to the proximal one-fourth of the metatarsal shaft. The incision should be placed medial to the extensor hallucis longus tendon, toward the second metatarsal. The subcutaneous tissue is divided and the wound flaps are retracted with 0 silk sutures. The digital nerves and vessels should not be injured.

B, The extensor hallucis longus and brevis tendons are identified and sectioned at the base of the proximal phalanx. An alternative technique is to leave the insertion of the extensor hallucis brevis tendon intact; the stump of the extensor hallucis longus tendon is sutured to the intact brevis tendon. (This latter method is faster and is used by the author when the long toe extensors of all five toes are to be transferred to the heads of the metatarsals.)

C, Silk whip sutures (00) are inserted into the ends of the long and short toe extensors. The long toe extensor is dissected free, and with a sharp scalpel its sheath is thoroughly excised as far proximally as possible.

D, The epiphyseal plate of the first metatarsal is proximal, whereas that of the lateral four metatarsals is distal in location. The extensor hallucis longus tendon is transferred to the head of the first metatarsal. The long toe extensors of the lesser toes are transferred to the distal one-third of the metatarsal shafts, with care taken not to disturb the growth plate. When the patient is more than age 10 to 12 years, the tendons are transferred to the heads of the metatarsals, as by then growth of the foot is almost complete.

With small Chandler elevator retractors, the soft tissues are retracted. The periosteum is not stripped. Through a stab wound in the periosteum, a hole is drilled in the center of the first metatarsal head and is enlarged to receive the tendon. The extensor hallucis longus tendon is passed through the hole in the first metatarsal in a medial to lateral direction and sutured to itself, with the forefoot in maximal dorsiflexion.

E, The extensor hallucis brevis tendon is then sutured to the stump of the long toe extensor, holding the toe in neutral extension or in 10 degrees of dorsiflexion.

A similar technique is employed to transfer the long extensor tendons of the lesser toes. Longitudinal incisions are made between the second and third metatarsals and between the fourth and fifth metatarsals. The extensor brevis tendon of the little toe is either absent or not of adequate size to transfer to the stump of the longus.

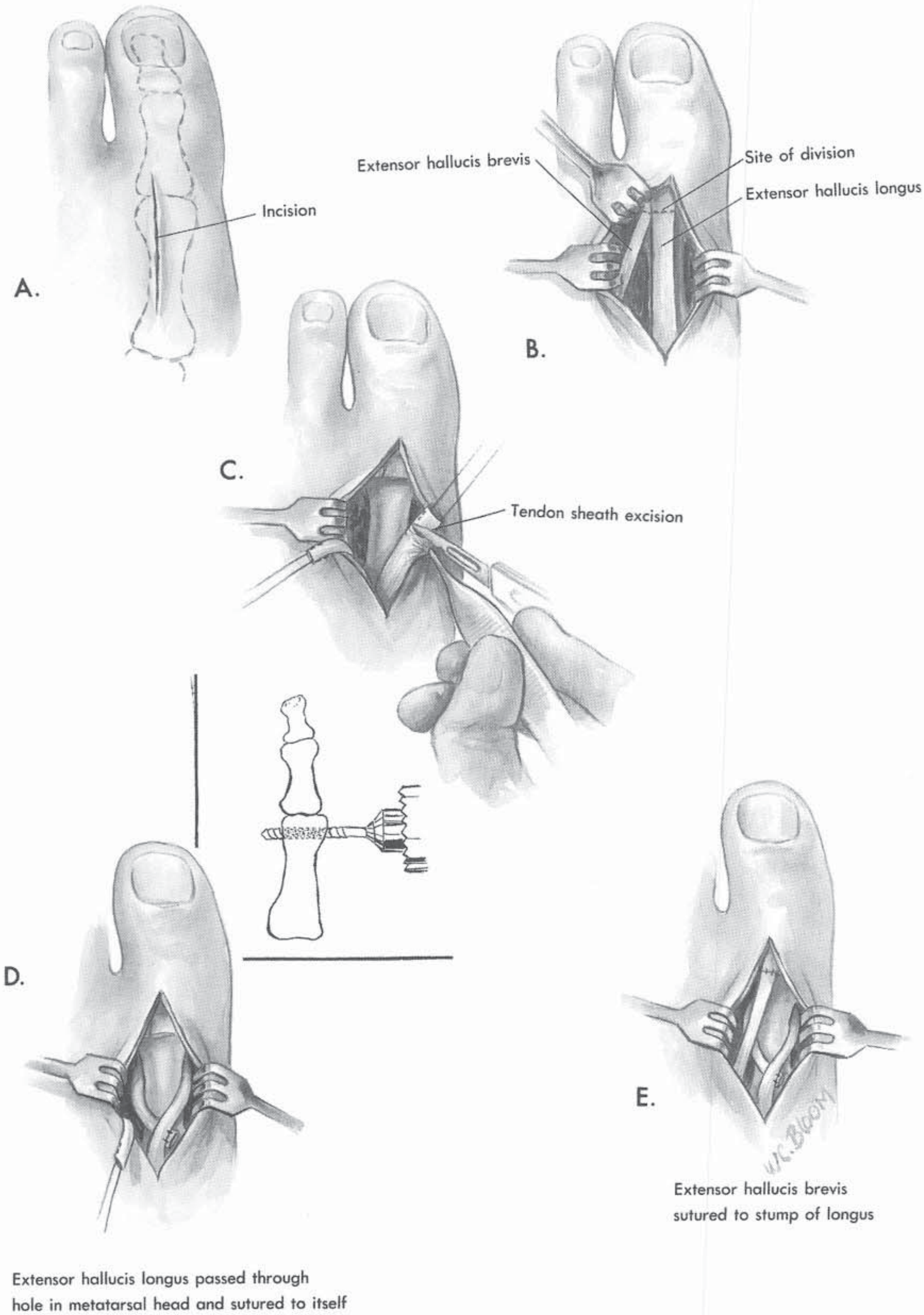
The tourniquet is released, and complete hemostasis is obtained. The wounds are closed with interrupted sutures.

POSTOPERATIVE CARE

A below-knee walking cast is applied, to be worn for 4 to 6 weeks. A sturdy, well-padded toe plate is made in the cast. The plantar aspect of the metatarsals should be well padded.

Special muscle training for the transferred tendons is not required, as the transfer is in phase.

PLATE 22–3. Transfer of Long Toe Extensors to Heads of Metatarsals (Jones Transfer)



Extensor hallucis longus passed through hole in metatarsal head and sutured to itself

Extensor hallucis brevis sutured to stump of longus

FIGURE 22–104 **A** and **B,** Preoperative radiographs in a 16-year-old girl with pes cavus secondary to Charcot-Marie-Tooth disease. **C,** Dorsal closing wedge osteotomies of the base of each metatarsal were performed. **D** and **E,** Postoperatively, alignment was improved.

a laterally based closing wedge, as described by Dwyer (Plate 22–4).[15–17,51] A concomitant plantar release must be done to correct the forefoot equinus and contracture of the plantar fascia. The calcaneal osteotomy is fixed with either a staple or threaded Steinmann pin, and a short-leg cast is applied (Fig. 22–105).

Dekel and Weissman in 1973 reported their results with the Dwyer osteotomy to treat cavovarus feet.[14] Slightly more than half of their patients had lasting satisfactory results following plantar release and calcaneal osteotomy; the rest had either incomplete correction or recurrence of deformity. Overcorrection into valgus is very rarely a problem. The worst results occurred in patients who had polio, the best in patients who had undergone clubfoot reconstructions. The common theme of less predictable results in patients with neurologic causes of their cavus was apparent in the results.[14,16] They concluded that calcaneal osteotomy was of benefit in patients less than 12 years old with inflexible varus of the hindfoot.

Mitchell described another calcaneal osteotomy for use in the surgical correction of calcaneocavus deformities.[38] The osteotomy is performed through a lateral approach, similar to the Dwyer osteotomy, but instead of the osteotomy being translated laterally, the distal fragment is slid superiorly and posteriorly. This adds to the length of the calcaneus and corrects the excessive vertical calcaneal pitch (Fig. 22–106). Samilson described a similar procedure in which the shape of the osteotomy is crescentic rather than linear to allow easier displacement.[44,45] Regardless of the shape of the osteotomy, a plantar release must be performed to permit translation of the osteotomy and correction of the arch of the foot. Often, tendon transfers into the calcaneus are also performed to augment the weak or paralyzed gastrocsoleus. This procedure gained popularity prior to the decline in poliomyelitis, but it remains an option in treating calcaneocavus due to other neurologic diseases.

Midfoot osteotomies: Several osteotomies of the midfoot have been proposed for the surgical reconstruction of the cavus foot. These osteotomies all involve removing a dorsally based V-shaped wedge of bone from the midfoot, at or just proximal to the apex of the cavus deformity. Plantar soft tissue release is generally performed either before or in addition to the osteotomy of the midfoot.

Cole described a dorsal closing wedge osteotomy of the midfoot in 1940.[11] The proximal cut is made through the navicular and cuboid bones and the distal cut is made at an appropriate level to allow adequate dorsal bony resection to correct the cavus deformity (Plate 22–5). Cole felt that preservation of hindfoot motion was important, and that this osteotomy allowed cosmetic improvement of the foot and improvement in painful symptoms.[11]

Jahss proposed a tarsometatarsal dorsal wedge resection osteotomy for the correction of cavus deformity.[26,27] The apex of the resected wedge is slightly more distal than that described by Cole, and the osteotomy described by Jahss does remove a small amount of plantar bone in the area of the TMT joints as well, to allow easier closing of the osteotomy. More bone is resected from the TMT joints of the second and third rays than from the first or the lateral rays. Jahss stated that the amount of forefoot equinus that can be corrected with this osteotomy should not exceed 20 to 25 degrees. He felt that greater correction led to the development of a rocker deformity in the sole of the foot and persistent symptoms, and recommended triple arthrodesis for feet with severe deformities. Additionally, he warned that if a callus is present on the sole of the foot preoperatively, the osteotomy must be performed proximal to the callus for successful redistribution of pressure across the foot. This holds true for calluses in the area of the cuboid, which will not improve following the Jahss osteotomy.

Japas described yet another midfoot osteotomy to be used in pes cavus (Plate 22–6).[29] In this case the osteotomy does not include resection of a wedge. A V-shaped osteotomy is made dorsally, with the apex of the V at the navicular and the limbs extending distally to just proximal to the cuboid-fifth metatarsal joint laterally and proximal to the medial cuneiform-first metatarsal joint medially. The forefoot is dorsiflexed through the osteotomy by depressing the base of the osteotomy and prying the distal foot dorsally and fixing it with Steinmann pins. The advantage of this osteotomy is that the foot is not shortened further by bony resection, yet the joints are all left mobile in the midfoot and hindfoot. The limitation of the osteotomy is that it cannot correct severe cavus, since a wedge is not resected. Additionally, rigid hindfoot varus is not addressed by the Japas osteotomy. Because the amount of correction achievable is limited and the osteotomy is technically challenging, we currently do not use it in our practice.

Wilcox and Weiner in 1985 described a midtarsal dome osteotomy for use in patients with cavus feet.[63] This osteotomy is a modification of the Japas osteotomy. A dome osteotomy with bony resection is made through the base of the fifth metatarsal, the cuboid, and the three cuneiforms. Joint fusions are not performed. The shape of the osteotomy allows greater ability to correct deformity, as the apex is gently curved and more mobile. The Akron experience with this osteotomy was that 64 percent of patients had satisfactory results. Since nearly all the failures in this series occurred in children less than 8 years old, the authors recommended that the osteotomy be performed only in patients older than 8 years.

Finally, as in most complex deformities, correction of severe pes cavovarus or calcaneocavus has been performed using osteotomy and gradual distraction and correction with such external fixation as the Ilizarov device.[40] The application of complex foot-fixation frames will not be described in detail here. The reader is referred to Chapter 23, Limb Length Discrepancy, which describes the Ilizarov technique, for further information.

Triple arthrodesis: When the amount of deformity present in a foot with pes cavus cannot be corrected fully by soft tissue release and osteotomy, a triple arthrodesis may be necessary to obtain a plantigrade foot (Fig. 22–107).[2,26] This procedure should be the last resort for correction of pes cavus, particularly when the etiology of the deformity is neuropathic and the sensation to the foot is disturbed.[45] The lack of protective sensation in such cases (for example, in myelomeningocele) leads to destruction and Charcot arthropathy in neighboring mobile joints and to possible pressure ulcerations if the foot is not perfectly positioned.[36,65]

The technique recommended for the triple arthrodesis has been debated. In all the techniques, correction of the deformity is achieved by resecting appropriate bony wedges. The more severe the deformity, the larger the wedges that

Text continued on page 1010

Dwyer Lateral Wedge Resection of Calcaneus for Pes Cavus

Forefoot equinus deformity is corrected first, either by plantar soft-tissue release or by dorsal wedge tarsal resection, depending upon the age of the patient and the severity of the deformity. Close-up lateral wedge resection of the os calcis is designed to correct the varus deformity of the hindfoot in which the heel is of adequate height and size.

OPERATIVE TECHNIQUE

A, A 5-cm-long oblique incision is made on the lateral aspect of the calcaneus parallel to, but 1.5 cm posterior and inferior to, the peroneus longus tendon. The subcutaneous tissue is divided and the wound flaps are retracted.

B and **C,** The peroneal tendons are identified and retracted dorsally and distally. The calcaneofibular ligament is sectioned, and the periosteum is incised. The lateral surface of the calcaneus is subperiosteally exposed; with Chandler elevator retractors, the superior and inferior aspects of the calcaneus are partially exposed. With a pair of osteotomes of adequate width, a wedge of the os calcis with its base directed laterally is excised. The site of osteotomy is immediately inferior and posterior to the peroneus longus tendon. The medial cortex should be left intact. The width of the base of the wedge depends on the severity of the varus deformity of the heel.

D, Next, a Steinmann pin is inserted transversely across the posterior segment of the calcaneus. The forefoot is dorsiflexed, putting tension on the Achilles tendon, and, with the Steinmann pin serving as a lever, the bone gap is closed. The heel should be in 5 degrees of valgus. The wound is closed and an above-knee cast is applied, the pin being incorporated in the cast. The knee is in 45 degrees of flexion.

POSTOPERATIVE CARE

The cast, pin, and sutures are removed in 4 weeks. Then a below-knee walking cast is applied for an additional 2 weeks, by which time the osteotomy should be healed.

PLATE 22–4. Dwyer Lateral Wedge Resection of Calcaneus for Pes Cavus

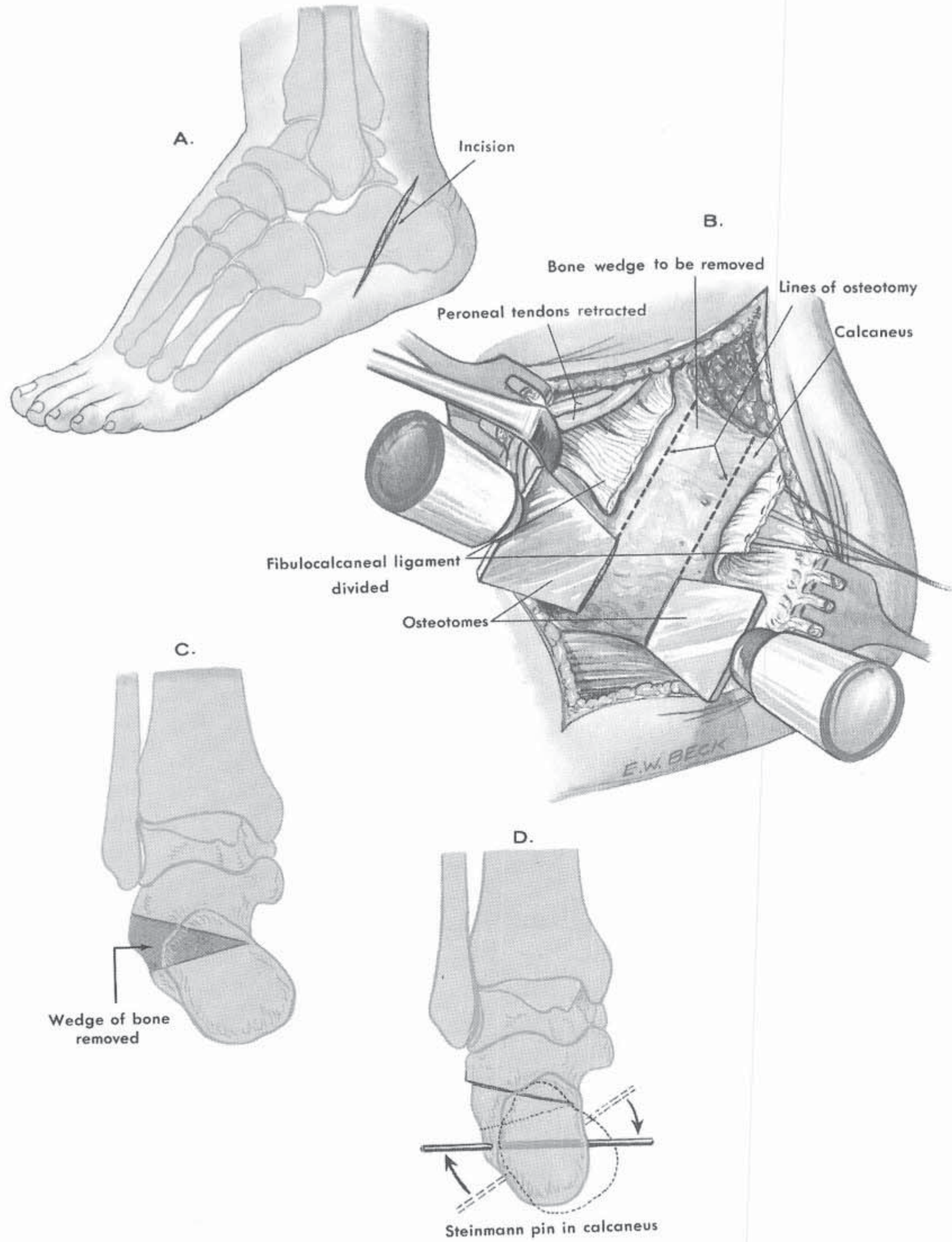


FIGURE 22–105 **A** and **B,** Preoperative radiographs in a 12-year-old girl with rigid pes cavovarus secondary to Charcot-Marie-Tooth disease. **C** and **D,** Surgical correction consisted of plantar fascia release, posterior tibial tendon transfer, first metatarsal osteotomy, and calcaneal osteotomy.

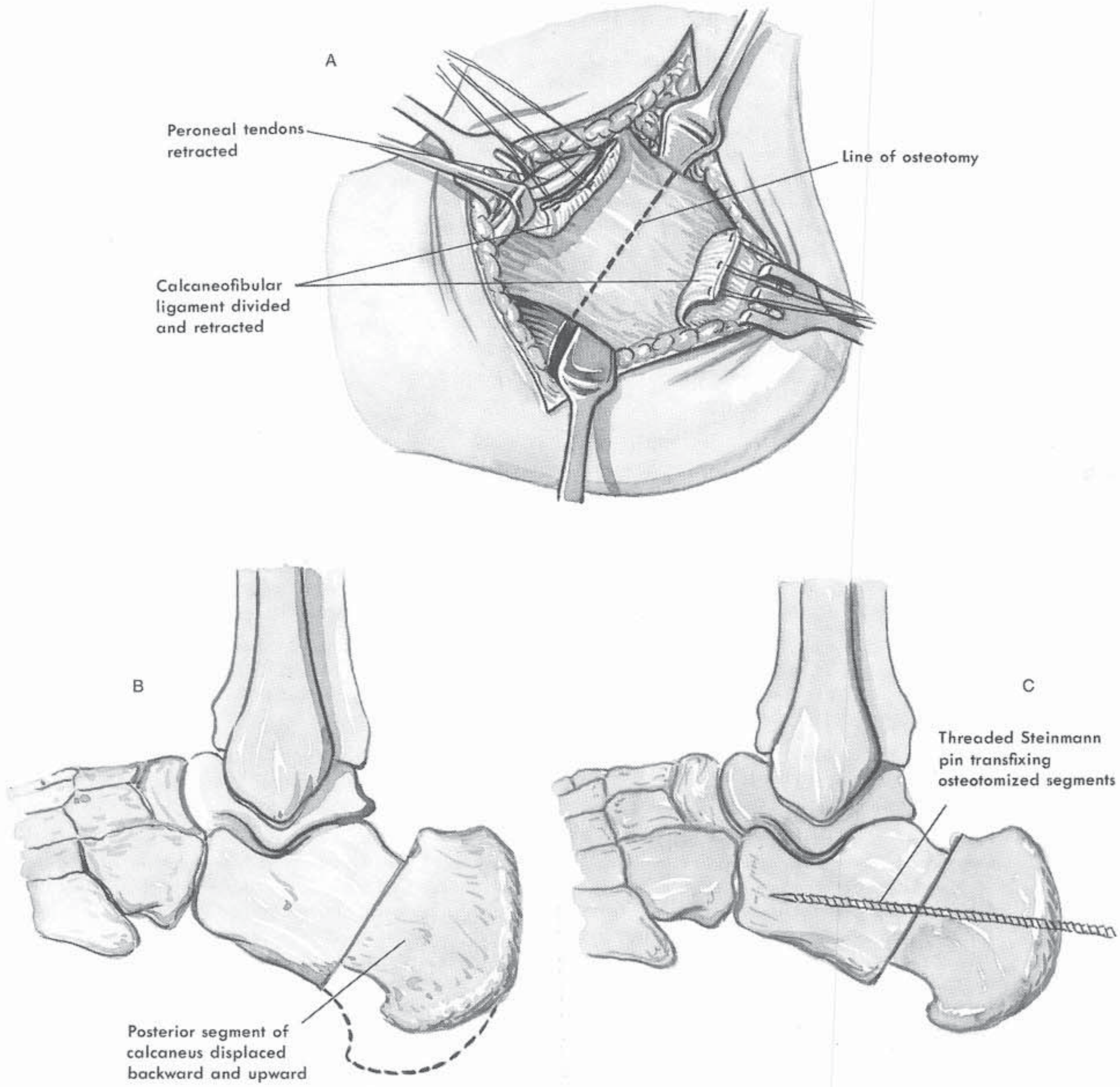


FIGURE 22–106 **A,** In patients with calcaneocavus deformity an oblique osteotomy of the calcaneus can be performed through a lateral oblique incision. The line of osteotomy from the superior part of the calcaneus inclines plantarward and distally. **B,** The posterior fragment is displaced superiorly in a maneuver that requires a plantar release. **C,** Fixation with a threaded Steinmann pin is used to maintain the displacement. A short-leg cast is then applied.

Dorsal Wedge Resection for Pes Cavus

The dorsal aspect of the tarsal bones may be exposed by several means. Cole and Japas make a single dorsal longitudinal incision approximately 6 to 8 cm long in the midline of the foot and centered over the midtarsal arch (naviculocuneiform junction). Subcutaneous tissue is divided, and the long toe extensors are identified and separated. The plane between the long extensor tendons of the second and third toes is developed, and the extensor digitorum brevis muscle is identified, elevated, and retracted laterally with the peroneus brevis tendon. The anterior tibial tendon and the long extensor tendons of the second and big toes are retracted medially. The periosteum is incised, longitudinally elevated, and retracted medially and laterally.[27,82]

Méary makes two longitudinal incisions, each about 5 to 6 cm in length, on the dorsum of the foot. The medial incision is parallel to the longitudinal axis of the second metatarsal and is centered over the intermediate cuneiform bone. The extensor hallucis longus tendon, dorsalis pedis vessels, and the anterior tibial tendon are identified, dissected free, and retracted medially. The lateral incision is about 3 cm long and is centered over the cuboid bone. The peroneus brevis is identified and retracted laterally.

This author uses two longitudinal incisions, one dorsolateral and the other medial.

OPERATIVE TECHNIQUE

A and **B**, Two longitudinal skin incisions are made. The medial incision, about 5 cm long, is over the medial aspect of the navicular and first cuneiform bones in the interval between the anterior tibial and posterior tibial tendons. The subcutaneous tissue is divided. The anterior tibial tendon is retracted dorsally; the posterior tibial tendon is partially detached from the tuberosity of the navicular and is retracted plantarward to expose the medial and dorsal aspects of the navicular and first cuneiform bones. The dorsolateral incision, about 4 cm long, is centered over the cuboid bone. The extensor brevis muscle is identified, elevated, and retracted distally and laterally with the peroneus brevis tendon. The long toe extensors are retracted medially.

C, Next, through the medial wound, the capsule and periosteum of the navicular and first cuneiform bones are incised and elevated. The soft tissues are retracted dorsally and plantarward with Chandler elevator retractors. The capsule of the talonavicular joint should not be disturbed. If in doubt, the surgeon should obtain radiographs to identify the tarsal bones with certainty.

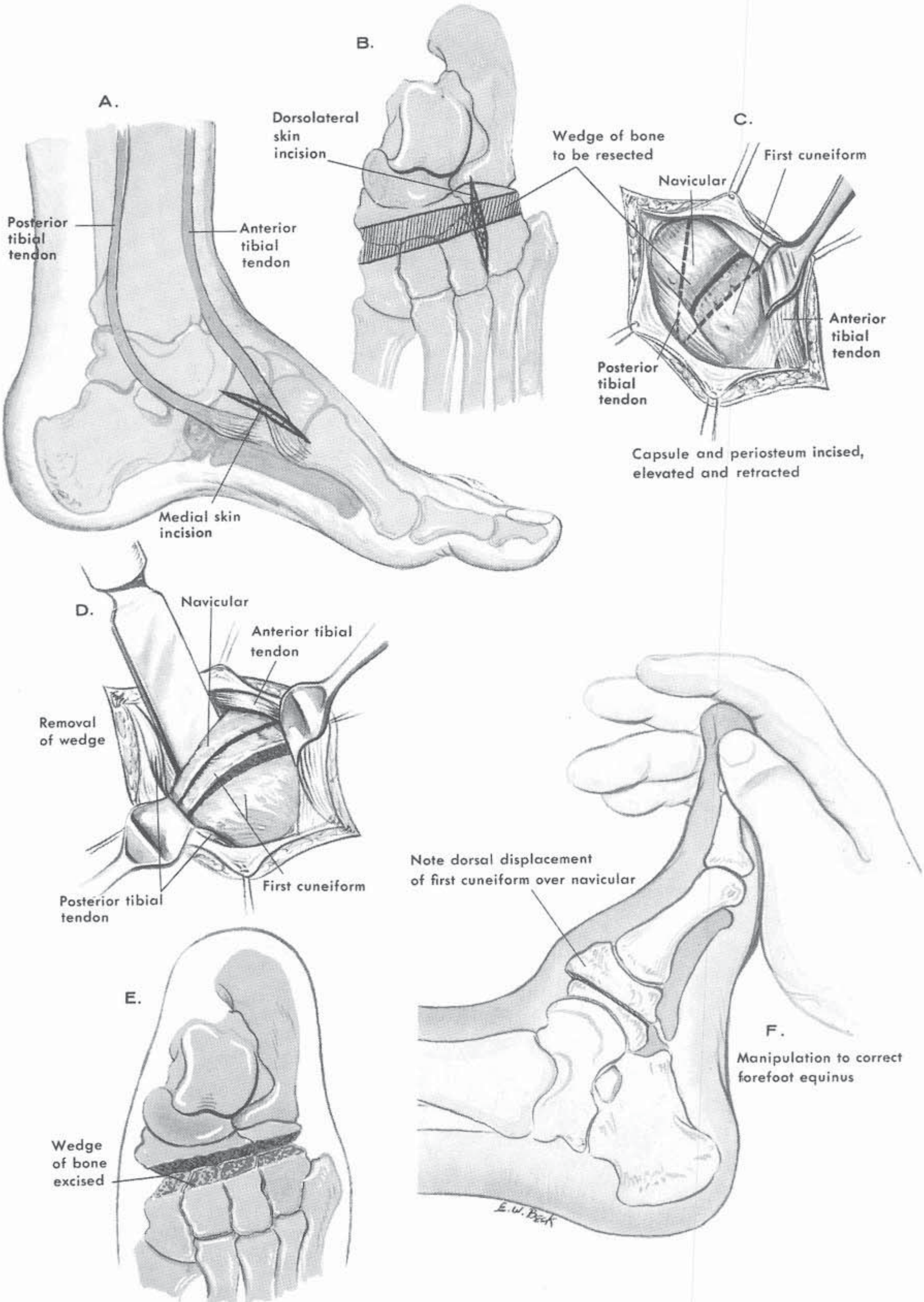
D and **E**, With osteotomes, a wedge of bone is excised, including the naviculocuneiform articulation. The base of the wedge is dorsal, its width depending on the severity of the forefoot equinus deformity to be corrected. The wedge osteotomy of the cuboid is completed through the dorsolateral incision.

F, The forefoot is then manipulated into dorsiflexion. If the plantar fascia is contracted, a plantar fasciotomy is performed. In severe cases the short plantar muscles are also sectioned. The first cuneiform bone should be dorsally displaced over the navicular bone. Two Steinmann pins are inserted to transfix the tarsal osteotomy. The medial pin is inserted into the shaft of the first metatarsal, directed posteriorly through the first cuneiform, across the osteotomy site, and into the navicular and the head of the talus. The lateral pin is started posteriorly along the longitudinal axis of the calcaneus and directed across the calcaneocuboid joint and into the cuboid and the base of the fifth metatarsal. (Méary uses staples to maintain the position of the osteotomy.) Radiographs are obtained to verify the position of the pins and maintenance of correction of forefoot equinus deformity. The tourniquet is released, and complete hemostasis is obtained. The incisions are closed. The pins are cut subcutaneously, and a below-knee cast is applied.

POSTOPERATIVE CARE

The foot and leg are immobilized for 6 weeks, at which time the cast, pins, and sutures are removed. A new below-knee walking cast is applied, to be worn for another 2 to 4 weeks.

PLATE 22–5. **Dorsal Wedge Resection for Pes Cavus**



Japas V-Osteotomy of the Tarsus

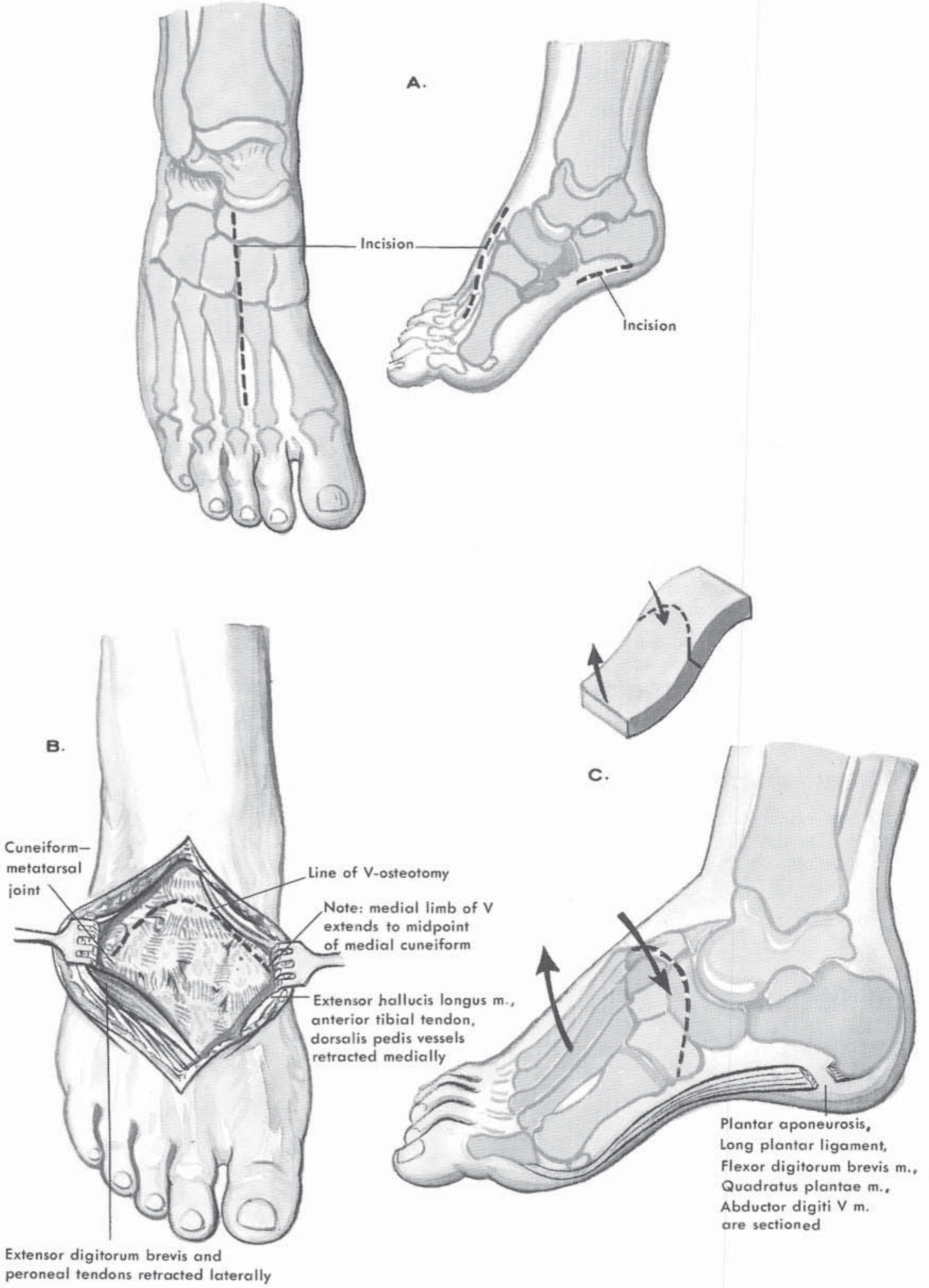
OPERATIVE TECHNIQUE

A, The dorsal aspect of the tarsal bones is exposed through a longitudinal incision 6 to 8 cm long in the midline of the foot (i.e., between the second and third rays) and centered over the midtarsal area at the naviculocuneiform junction.

B and **C,** The subcutaneous tissue is divided. The superficial nerves are isolated and protected. The long toe extensor tendons are identified and separated, and the plane between those of the second and third toes is developed. The extensor digitorum brevis muscle is identified, elevated extraperiosteally, and retracted laterally with the peroneal tendons. The extensor hallucis longus tendon, dorsalis pedis vessels, and anterior tibial tendon are identified, dissected free, and retracted medially. The osteotomy site is exposed extraperiosteally.

The talonavicular joint is identified next. Caution! Do not injure the midtarsal joint and compromise its function. If bony landmarks are distorted, radiographs are obtained for proper orientation. Inadvertent partial ostectomy of the head of the talus will result in aseptic necrosis and traumatic arthritis. The V line of the osteotomy is marked. Its apex is in the midline of the foot at the height of the arch of the cavus deformity; its medial limb extends to the middle of the medial cuneiform, exiting proximal to the cuneiform–first metatarsal joint; and its lateral limb extends to the middle of the cuboid, emerging proximal to the cuboid–fifth metatarsal joint. Often the V is shallow, shaped more like a dome.

PLATE 22–6. Japas V-Osteotomy of the Tarsus



Japas V-Osteotomy of the Tarsus *Continued*

D and **E,** The osteotomy is begun with an oscillating bone saw and completed with an osteotome. Splintering of the ends of the medial and lateral limbs should be avoided. Next, a curved periosteal elevator is inserted into the osteotomy site, manual traction is applied on the forefoot, and, with the elevator used as a lever, the base of the distal fragment is depressed plantarward. This maneuver corrects the cavus deformity and lengthens the concave plantar surface of the foot. The foot is not shortened, as it would be by resection of a bone wedge, and any abduction or adduction deformity can be corrected if necessary.

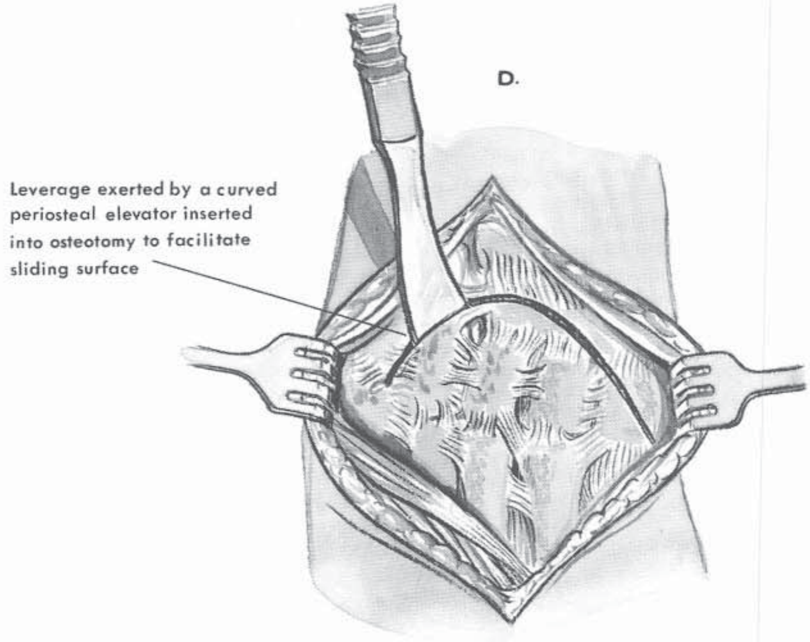
F, Once desired alignment is achieved, a single Steinmann pin is inserted through the distal part of the first metatarsal and directed posteriorly and laterally to terminate in the lateral part of the calcaneus or the cuboid. Radiographs are obtained to verify the completeness of correction. Then the tourniquet is removed, hemostasis is achieved, and the wound is closed with interrupted sutures. The pin is cut subcutaneously, and a below-knee cast is applied.

POSTOPERATIVE CARE

Two weeks after surgery a walking heel is placed posteriorly—under the long axis of the tibia—and gradual partial weightbearing is permitted with crutches. In 6 weeks the cast, sutures, and Steinmann pin are removed. Radiographs are obtained. If healing is not adequate, another below-knee cast is applied for an additional 2 to 4 weeks.

PLATE 22–6. **Japas V-Osteotomy of the Tarsus**

D.



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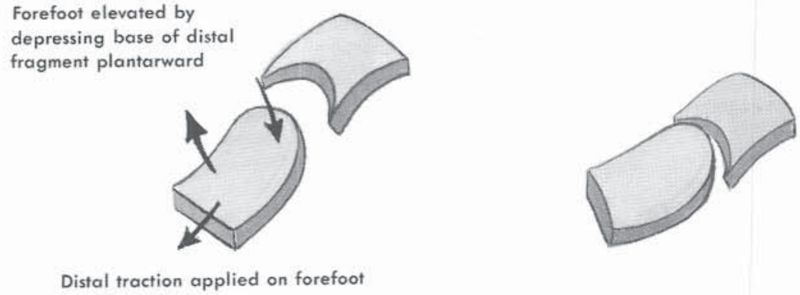

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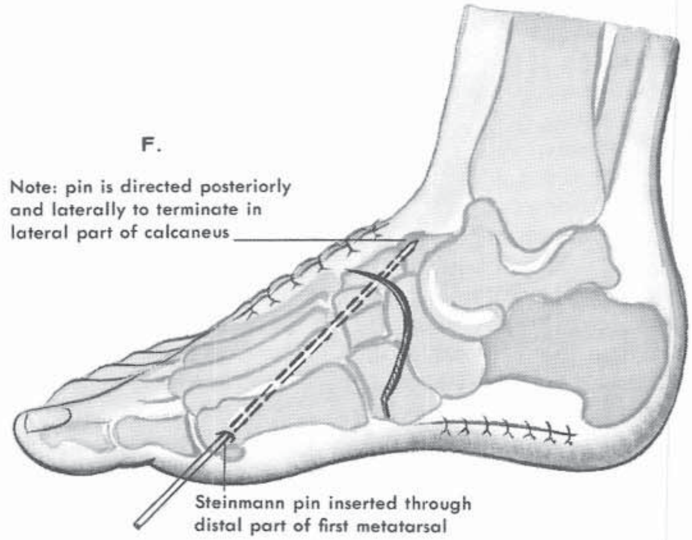

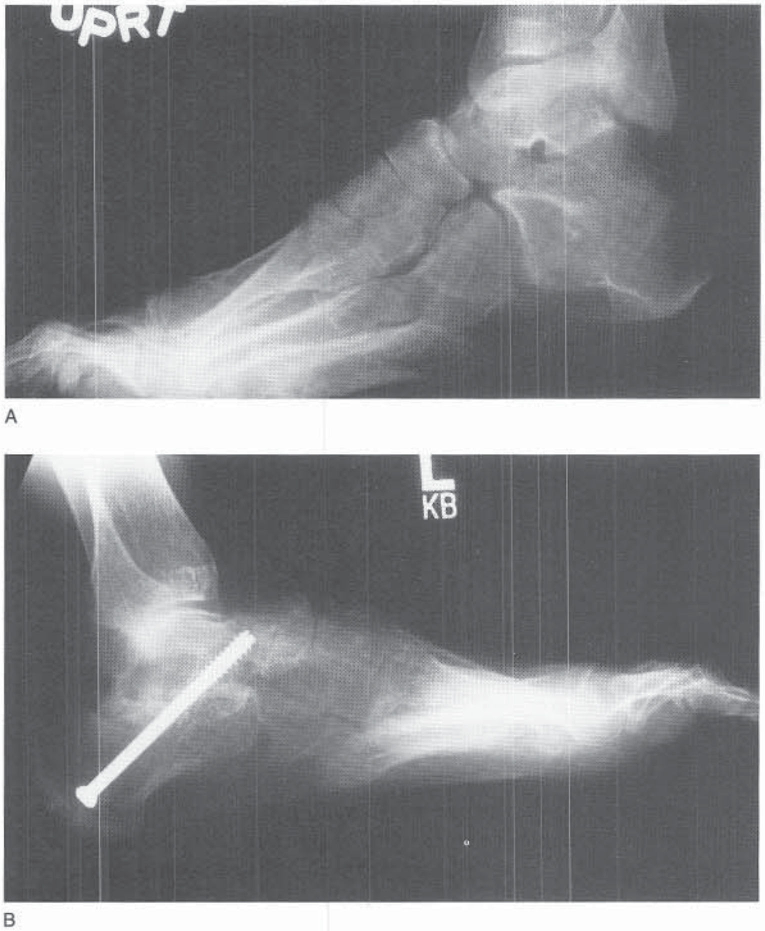

FIGURE 22–107 **A,** Preoperative radiographic appearance in a 17-year-old male patient with cavus foot secondary to Charcot-Marie-Tooth disease. The deformity recurred following plantar fascia release and first metatarsal osteotomy, posterior tibialis tenotomy, and the Jones procedure. **B,** Triple arthrodesis resulted in a plantigrade foot.

must be removed. This leads to noticeable shortening of the foot. Similarly, removal of the joint surfaces of the hindfoot tarsals leads to growth inhibition of these bones and further shortening. For this reason, triple arthrodesis is reserved for patients 10 to 12 years of age and older.[18]

Siffert and associates described a technique for triple arthrodesis that they felt would best correct the cavus foot, called the "beak" triple arthrodesis.[49,50] In this procedure, a dorsal wedge resection is carried out through the talonavicular and calcaneocuboid joints, but a small shelf of dorsal talar head is left intact (Fig. 22–108). The navicular (with the attached forefoot) is then placed beneath this protrusion of the talar head, resulting in less overall shortening of the foot. The vascular supply to the talus is better preserved with this approach, but a dorsal prominence from the anterior beak of the talus may be obvious and bothersome.

The Lambrinudi or Hoke triple arthrodeses are more commonly used in patients with cavus feet.[21,24,31] The cavus deformity is corrected by taking a larger wedge from the talonavicular and calcaneocuboid joints dorsally than from the plantar surface of the joint. Hindfoot varus is addressed by resecting a laterally based wedge from the subtalar joint. Fixation with screws or staples is used to maintain the position of the foot postoperatively, and a nonweightbearing cast is worn.

Although a solid fusion should maintain correction, it has been found that if muscle imbalance persists, loss of correction can occur over time in the cavus foot that is driven by a neurologic disease. Tendon transfers may be necessary to balance the forces across the foot.[32] These transfers can be staged to follow the arthrodesis, but the decision to include a tendon transfer in the surgical reconstruction must be made prior to the triple arthrodesis, as it is impossible to test the motor power of many of the muscles to the foot following solid hindfoot fusion.[27]

Studies of teens who underwent triple arthrodeses tend to show deterioration of function with the passage of time.[25] This is particularly true in the Charcot-Marie-Tooth population, where long-term studies 21 years following surgery find that almost one-half of the patients have poor results.[62,65] Symptoms on follow-up studies result from failure to achieve or maintain a plantigrade foot in the face of progressive neuropathy. Pseudarthroses of the talonavicular joint may occur[36,46] but are usually not painful.[33,47] Revision surgery to address pseudarthrosis or suboptimal position of fusion is complicated, but possible. Patients with paralytic deformities, such as poliomyelitis, tend to have better results following triple arthrodesis than those with peripheral neuropathy or spina bifida. Adelaar and associates found good or excellent results in 68 percent of feet at 20 years of follow-up, but the majority of their patients had polio.[1]

We recommend triple arthrodesis in patients in whom previous attempts at surgical correction of cavus have failed and in patients with severe, rigid deformities not amenable to other less aggressive procedures (see Fig. 27–8). We prefer calcaneal osteotomy for hindfoot varus when the cavus is

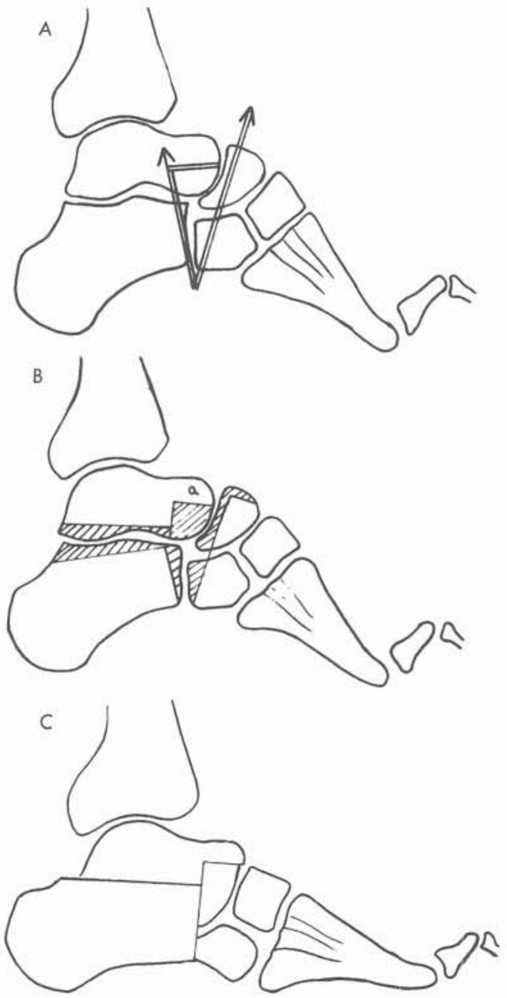

FIGURE 22–108 Technique of "beak" triple arthrodesis for correction of severe pes cavus deformity. Medial and lateral incisions are employed for exposure of the subtalar, talonavicular, and calcaneocuboid joints. With the exception of the head of the talus, all joint surfaces are denuded of hyaline cartilage, as in an ordinary triple arthrodesis. A dorsally based wedge is removed from the calcaneocuboid joint and navicular bone. The plantar half or one-third of the talar head-neck is resected to form a beak. Care is taken not to disturb the soft tissues in the superior aspect of the talus and anterior part of the ankle joint. **A,** The lines of the osteotomy. **B,** The area of bone to be resected (*hatched areas*). **C,** Final result, demonstrating correction of the cavus deformity. Note that the forefoot is displaced plantarward and locked under the talar beak. (From Siffert RS, Forster RL, Nachamie B: "Beak" triple arthrodesis for correction of severe cavus deformity. Clin Orthop 1966;45:102.)

correctable with plantar release, and midfoot osteotomy when the cavus is rigid but the hindfoot is not severely deformed. When inflexible hindfoot varus is present with a very stiff cavus deformity, triple arthrodesis offers the only possible solution, despite long-term studies showing deterioration in results. Unfortunately, in these children, less aggressive surgery via osteotomy cannot fully correct the deformity, and all studies of osteotomies include a percentage of patients who ultimately require triple arthrodeses following the initial procedure. Great care must be taken in obtaining the most plantigrade position possible, for residual deformity will accelerate the appearance of symptoms following triple arthrodesis.

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Toe Deformities

HALLUX VALGUS

Children with hallux valgus deformities, commonly known as bunions, are frequently seen in a busy pediatric orthopaedic practice. But, just as in the adult population, there is no consensus on how treatment should proceed in these patients. The deformity itself consists of lateral deviation of the great toe, with the apex of the deformity at the first MTP joint. Hallux valgus is usually bilateral (Fig. 22–109).

There is a female preponderance, with up to 88 percent of adolescent patients being girls. Usually there is a strong family history, with most patients inheriting the disorder from their mothers.[12,37,42,77,107] A positive family history was elicited in 72 percent of 43 patients in a series reported by Coughlin.[21] Multigenerational family histories positive for bunions in female ancestors suggests a probable sex-linked dominant trait with variable penetrance, while bunions in males may be due to an autosomal dominant trait or a more complex inheritance.

Symptoms. Patients with hallux valgus usually present with pain or cosmetic concerns. The pain is located over the prominent head of the first metatarsal or over the medial aspect of the first MTP joint. Rarely, a patient may complain of pain over the plantar sesamoids. Redness, swelling, and an inflamed bursa may be noted over the MTP joint prominence. Stiffness is usually not present.

Shoe wear tends to exacerbate the symptoms, and many patients complain of being unable to wear the fashionable shoes that are valued by their peers. Others complain about the appearance of their feet. In the absence of pain, caution should be taken prior to operating on these patients, as the cosmetic appearance of the corrected foot still is not normal and expectations may be unrealistically high.

The clinician should examine the whole patient, looking for an underlying cause of the hallux valgus. Cerebral palsy and its spasticity lead to hallux valgus due to increased tone in the adductor hallucis and equinovalgus deformity (Fig. 22–110). Patients with connective tissue diseases such as Ehlers-Danlos syndrome and Marfan's syndrome are prone to develop bunions. There is controversy as to whether or not the presence of flexible flatfeet leads to hallux valgus.

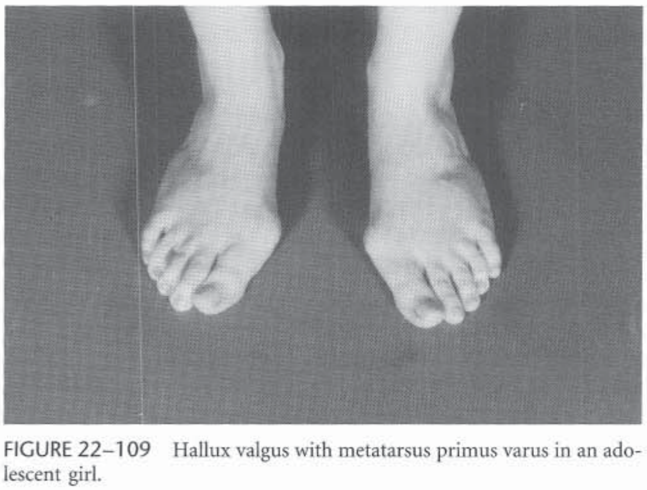


FIGURE 22–109 Hallux valgus with metatarsus primus varus in an adolescent girl.

FIGURE 22–110 Bunion deformity in a 16-year-old male patient with spastic diplegia. There is an increased intermetatarsal angle.

Several authors have implicated pes planus as a risk factor for hallux valgus and for recurrence of deformity following surgical correction.[52,60,107] Others, however, find that the incidence of bunion patients with pes planus does not differ from the general population.[21,68]

Anatomy. A complete understanding of the anatomy of the medial side of the forefoot is necessary before discussing the treatment of juvenile or adolescent hallux valgus. The first metatarsal articulates proximally with the medial cuneiform and, to a much lesser extent, with the second metatarsal. Although the metatarsocuneiform joint is usually transverse in direction, in some children with hallux valgus the joint is crescentic and sloped medially (Fig. 22–111). This directs the shaft of the first metatarsal medially.

There is also a common association with metatarsus primus varus, defined as an intermetatarsal angle between the first and second rays of 10 degrees or more on a weightbearing radiograph. Although it is widely accepted that many patients with hallux valgus have metatarsus primus varus, which condition is primary and which is compensatory remains controversial.[4,9,31,46,66]

The first metatarsal head articulates with the proximal phalanx of the great toe at the MTP joint. The joint is broader on its plantar surface, and narrower and therefore more unstable dorsally. The joint relies on soft tissue for stability, including the medial and lateral collateral ligaments, the adductor hallucis, and the abductor hallucis. The abductor inserts onto the plantar aspect of the proximal phalanx. The adductor hallucis inserts onto the lateral aspect of the proximal phalanx, and also anchors the lateral aspect of the plantar plate and the sesamoids. The plantar surface of the first metatarsal head has two grooves, in which rest the tendons of the flexor hallucis brevis enveloping the sesamoids. The flexor hallucis brevis inserts into the base of the proximal phalanx, while the flexor hallucis longus inserts into the distal phalanx.

With the development of hallux valgus, the adductor tendon becomes a deforming force, while the abductor is rendered relatively powerless owing to its plantar location.

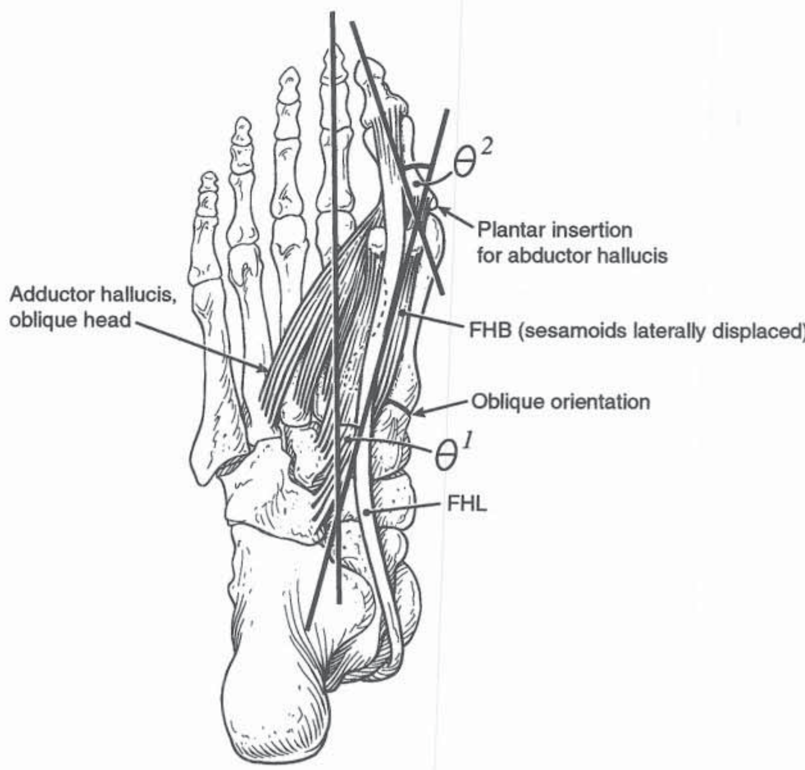


FIGURE 22–111 Pertinent anatomy in hallux valgus. The adductor hallucis is a deforming force pulling the great toe laterally. The insertion of the abductor hallucis is more plantar than normal, leading to muscle imbalance. The sesamoids are held within the flexor hallucis brevis and are displaced laterally. The flexor hallucis longus then migrates laterally across the first MTP joint. θ^1 = intermetatarsal angle, θ^2 = hallus valgus angle. Note the obliquity of the first metatarsocuneiform joint.

This imbalance leads to internal rotation of the great toe. The sesamoids translate laterally, leaving their grooves. The extensor hallucis longus and flexor hallucis longus tendons bowstring across the lateral side of the first MTP joint (Fig. 22–111).[117]

Radiographic Examination. Standing AP and lateral radiographs of the feet are necessary in order to critically assess the hallux valgus deformity. The hallux valgus angle is measured using the technique of Hardy and Clapham.[45] A line is drawn along the shaft of the proximal phalanx and another along the shaft of the first metatarsal; their intersection is the hallux valgus angle (Fig. 22–112). The normal hallux valgus angle is 16 degrees or less.[117]

The intermetatarsal angle between the first and second metatarsals is measured to assess the foot for the presence of metatarsus primus varus. The normal intermetatarsal angle is 9 degrees or less. When the intermetatarsal angle is elevated, the slope of the first metatarsal-medial cuneiform joint should be inspected. The metatarsocuneiform articulation is normally transverse. An oblique orientation of this joint predisposes to metatarsus primus varus, and then to hallux valgus (Fig. 22–112).[43]

The distal metatarsal articular angle (DMAA) is measured by drawing a line across the articular surface of the metatarsal head at the MTP joint and another line along the first metatarsal shaft. The intersection is the DMAA. The normal DMAA is 15 degrees or less.[117] It is common in juvenile and adolescent patients to find a varus angulation of the distal first metatarsal articulation. The first MTP joint in these

FIGURE 22–112 AP radiograph of the foot of a 15-year-old adolescent. The metatarsocuneiform joint is sloped medially. The intermetatarsal angle is increased, measuring 12 degrees. The hallux valgus angle measures 32 degrees. It is the angle formed by a line drawn along the axis of the proximal phalanx and a second line drawn along the shaft of the first metatarsal.

patients is congruous, which becomes clinically important if surgery is contemplated.[21] Likewise, the presence of an incongruous first MTP joint should be determined radiographically.

The length of the first metatarsal relative to the second metatarsal should be assessed. Coughlin described measuring the lengths with a reference line drawn from the proximal medial navicular to the proximal lateral cuboid. The first metatarsal may be relatively long, of similar length, or relatively short compared to the second metatarsal.

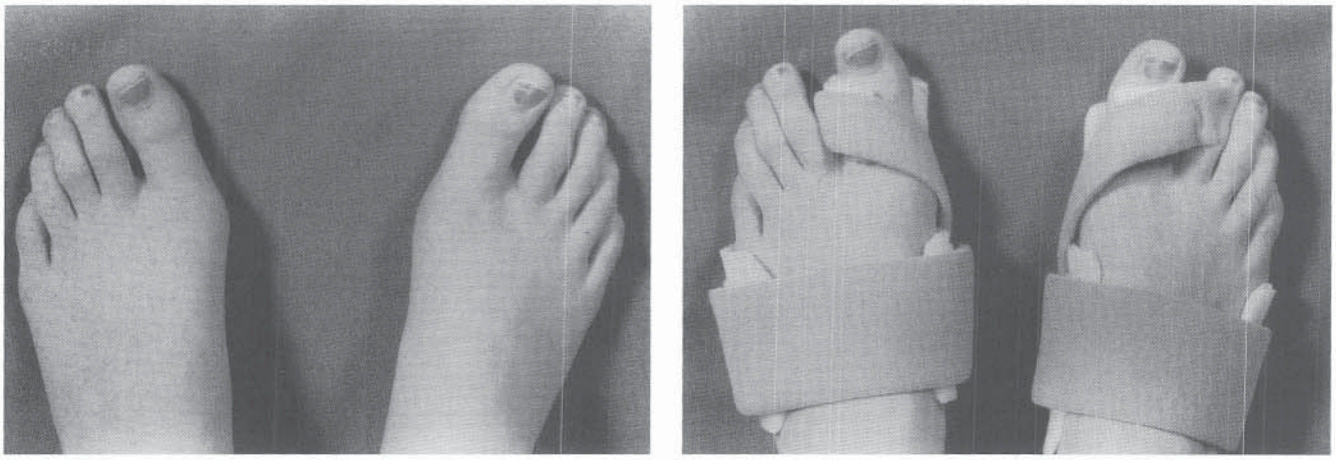
Finally, the physes of the proximal phalanx and of the first metatarsal should be viewed to see if they remain open or not. The first metatarsal physis is located proximally, unlike the physes of the second through fifth metatarsals. This may influence surgical treatment as well.

Classification. Hallux valgus may be classified as mild, moderate, or severe. In mild deformities the hallux valgus angle is less than 25 degrees. In moderate hallux valgus, the hallux valgus angle measures between 25 and 40 degrees. In severe deformity, the hallux valgus angle measures more than 40 degrees.[79]

Treatment. Pediatric patients with hallux valgus are initially encouraged to pursue conservative treatment.[69] First, shoes should be sufficiently wide in the toe box to not rub or place lateral pressure on the great toe. The use of high-heeled shoes with narrow toe boxes should be specifically discouraged, as this forces the toe up onto the narrow dorsal surface of the first metatarsal head and then pushes it into a valgus position. Toe spacers and other splints have been tried,[42] but most have found them not to be useful in improving hallux valgus (Fig. 22–113).[22,67] Patients with hallux valgus and pes planus may benefit from using an arch support.[86] Conservative treatment may be helpful in relieving pain but usually cannot correct the deformity.[117]

If the patient is skeletally immature, it is wisest to delay surgery until the first metatarsal physis has closed, if possible. The risk of recurrence is higher in younger patients following surgical correction.[9,107,110] Coughlin found that the risk of overcorrection into hallux varus was highest in the patients who had open physes in his series.[21] Prior to skeletal maturity, there is a theoretical risk of damage to the proximal first metatarsal physis during osteotomy, although problems resulting from physeal arrest have not been reported.

Surgical treatment can be divided into four components: distal soft tissue realignment (McBride procedure), distal metatarsal osteotomy (Mitchell and chevron osteotomies), basilar first metatarsal osteotomy, and metatarsal-cuneiform fusion. Which procedure is chosen should be based on the radiographic measurements made on the AP radiograph. First, is the MTP joint congruent or not? If the joint is incongruous, a distal soft tissue realignment must be included in the surgical procedure in order to restore a more normal joint alignment and decrease the risk of arthritis. If the joint is congruous, there may be no need to open the joint capsule, and correction can be achieved by nonarticular osteotomies. Second, is metatarsus primus varus present? If so, it is of utmost importance to restore a normal intermetatarsal angle between the first and second rays in order to decrease the risk of recurrence. Finally, how severe is the bunion? Certain procedures are indicated for mild hallux valgus and contraindicated in severe cases.



A B

FIGURE 22–113 **A** and **B,** Teenage girl with mild bilateral hallux valgus. Splints have been prescribed, yet bunions persist when the splints are removed.

There are a few caveats that must be kept in mind when operating on bunions in adolescents. First, the metatarsal head usually does not have a large protuberant medial eminence. It is generally not necessary to shave off the medial aspect of the metatarsal head, and when shaving is done, care should be taken not to remove too much bone, as this can lead to postoperative complications such as stiffness and hallux varus.[103] Second, do not violate the physis of the first metatarsal in a skeletally immature patient. Finally, internal fixation is generally advised to maintain alignment while osteotomies heal.

Soft tissue realignment should not be performed in isolation, but it is often part of the surgical reconstruction of hallux valgus. The subluxed MTP joint is realigned by advancing the medial capsule by a V-Y-plasty with the base of the V based distally. When the lateral soft tissues are addressed, the adductor tendon is released or transferred to the lateral aspect of the distal first metatarsal. McBride described suturing the first and second metatarsals through the web space to narrow the intermetatarsal space. We have not done this. He also advocated removal of the lateral sesamoid, which has fallen out of favor.[80,82] When performing soft tissue realignment, the surgeon should not address the medial and lateral aspects of the first metatarsal head concurrently, as the vascular supply to the metatarsal head could be injured, with resultant avascular necrosis.

When soft tissue realignment is performed without osteotomy, recurrence is frequent, occurring in 50 to 75 percent of adolescents.[21,48,106] Hallux varus is also known to occur following McBride's procedure.[80,105] Soft tissue realignment in isolation has been abandoned in the treatment of juvenile hallux valgus.

In most children with hallux valgus, the soft tissue realignment is performed in conjunction with an osteotomy of the first metatarsal. Distal osteotomies consist of the Mitchell osteotomy and the chevron osteotomy. In the Mitchell osteotomy, the distal first metatarsal is osteotomized transversely, and then a second osteotomy that originates on the medial cortex but does not penetrate the lateral cortex is made a few millimeters distal to the first cut (Fig. 22–114). This incomplete osteotomy should be located 2 cm proximal to the articular surface of the first metatarsal.[20] The distal fragment is then translated laterally with the step-cut hooked over the lateral edge of the proximal fragment. Mitchell originally described holding the osteotomy with suture through drill holes,[89] but current opinion favors internal fixation.[11] The metatarsal head should not be dorsiflexed, as this will unweight the first metatarsal and cause transfer metatarsalgia.

The Mitchell osteotomy is adequate for the treatment of mild to moderate hallux valgus. It is contraindicated in the presence of a short first metatarsal, as it produces further shortening through its step-cut. It is effective when used in conjunction with medial reefing of the capsule.

The results of the Mitchell osteotomy vary between series. Ball and Sullivan found recurrent hallux valgus in 11 of 17 feet (61 percent) following the Mitchell procedure.[6] They also saw significant MTP stiffness following the surgery, and concluded that the Mitchell osteotomy should not be used in the treatment of adolescent hallux valgus.[6] Canale and colleagues had somewhat better results, but 31 percent of a series of 51 feet still had fair or poor results following a Mitchell osteotomy.[12] They concluded that many of the problems the patients in their series experienced resulted from loss of fixation, and they advocated the use of internal fixation with Kirschner wires. They also found plantar calluses beneath the second metatarsal due to transfer lesions following shortening of the first metatarsal.[12] Metatarsal shortening has led to the development of second and third metatarsal stress fractures following distal osteotomy.[24]

More positive series have published up to 93 percent good and excellent results following the Mitchell osteotomy.[36,77,123] Weiner and colleagues used pin fixation and modified the shape of the step-off, making a trapezoidal cut, which they felt better preserved metatarsal length. They reported satisfactory results in 91 percent of 69 feet.[123] McDonald and Stevens performed a similar procedure, adding a plantar-based wedge to plantar flex the first metatarsal, and obtained satisfactory results in 81 percent of cases.[83] Geissele and Stanton reported 95 percent excellent results with Mitchell

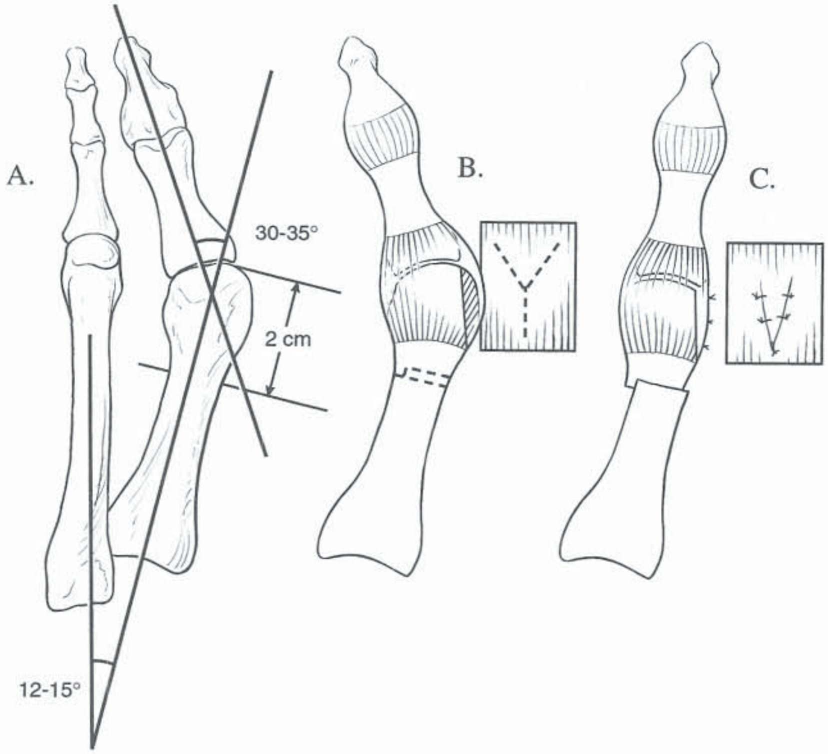


FIGURE 22–114 A to C, Mitchell's first metatarsal osteotomy. Two osteotomies are made distally in the first metatarsal, the more distal of which is incomplete laterally. The osteotomy is then displaced laterally and secured with screws or pins. The medial eminence is cautiously resected, and a V-Y-capsulorrhaphy is performed at the medial MTP joint.

osteotomies for the treatment of metatarsus primus varus, and linked postoperative recurrence with inadequate correction of the intermetatarsal angle.[36] It appears that if care is taken to preserve length, plantar flex the metatarsal head, and internally fix the fragments, good results can be obtained with the modified Mitchell osteotomy.

A second distal metatarsal osteotomy that has been popular for the treatment of hallux valgus is the chevron osteotomy.[5] A V-shaped osteotomy is performed with the apex of the V at the distal metatarsal. The two limbs are directed proximally to the dorsal and plantar surfaces of the distal first metatarsal. The distal fragment is then slid laterally to correct the metatarsus primus varus (Fig. 22–115). Good results have been published for this osteotomy, with poor results attributed to technical errors such as loss of position and overzealous resection of the medial eminence, producing a gouged metatarsal.[47] Zimmer and associates found that 85 percent of adolescents who had undergone chevron osteotomies for the treatment of bunions were satisfied at

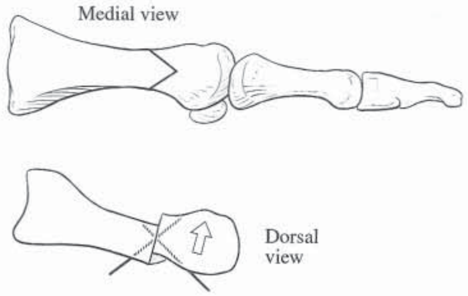


FIGURE 22–115 Distal chevron osteotomy of the first metatarsal. A V-shaped osteotomy based distally is performed. The distal fragment is then translated laterally and held with pins or screws.

follow-up, despite a recurrence rate of 20 percent.[125] They did not find that metatarsal shortening was problematic following this procedure.

Avascular necrosis can result from distal first metatarsal osteotomies, and has been described following both the Mitchell and and the chevron osteotomy.[36,58,100] Circulatory changes can result from disrupting the nutrient vessels that feed the metatarsal on the lateral side of the metatarsal neck. Combining a distal metatarsal osteotomy with a lateral capsular release places the first dorsal metatarsal artery at risk. Minimal periosteal stripping on the lateral side of the metatarsal should be allowed during the exposure for distal osteotomy. When AVN does occur, it commonly is asymptomatic.

Proximal metatarsal osteotomy is currently used frequently in the surgical correction of hallux valgus in adolescents. It allows good correction of metatarsus primus varus and can be combined with distal soft tissue realignment without undue risk of AVN. The osteotomy may be an opening wedge based medially, a closing wedge with the base lateral, or most commonly a crescentic osteotomy without bony resection. The choice of an opening or closing wedge osteotomy is determined by the preoperative length of the first metatarsal relative to the second metatarsal. When a proximal osteotomy is performed, the physis of the first metatarsal should be left undisturbed in younger patients. Fixation with either a Steinmann pin, Kirschner wire, or screw is advised (Fig. 22–116).

As in all bunion surgeries, the major complication following proximal metatarsal osteotomy is recurrence. Scranton and Zuckerman found fewer recurrences when closing wedge osteotomies were performed than when opening wedge osteotomies were performed.[107] The opening wedge increases the length of the first metatarsal and increases tissue tension across the capsule of the MTP joint. Closing

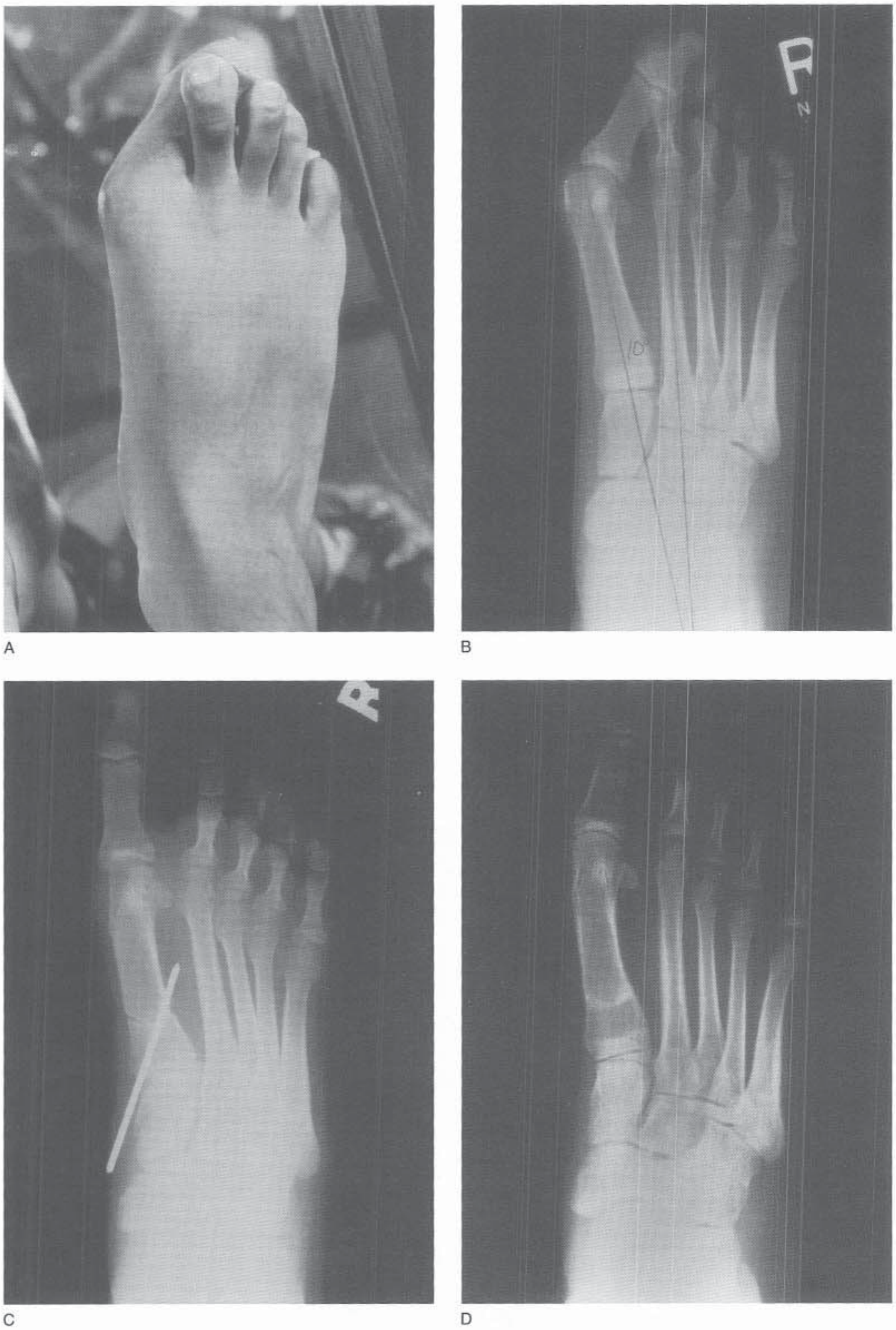


FIGURE 22–116 **A,** Severe hallux valgus in a 16-year-old girl. **B,** Radiograph showing increased intermetatarsal angle, lateral translation of the sesamoids, and an increased hallux valgus angle. The first metatarsocuneiform joint is normal. **C,** A crescentic proximal first metatarsal osteotomy with pin fixation and distal soft tissue realignment was performed. Resection of the medial eminence was slightly excessive. **D,** Follow-up radiograph shows healing, in good alignment.

wedge osteotomies shorten the metatarsal, and may therefore predispose toward lateral metatarsalgia, although some surgeons have found this not to be a problem.[108] Success has been achieved with a proximal opening wedge osteotomy with bone grafting, either using the exostosis or iliac crest.[75] As most adolescents do not have large exostoses, it is questionable whether it is best to choose a procedure in which an iliac crest graft must be taken if alternatives exist. Metatarsal length is usually preserved with a crescentic osteotomy of the base of the first metatarsal, as described by Mann and colleagues. They recommend basing the concavity of the crescent proximally and using a crescentic saw blade to make the cut. When this technique was used, 95 percent of the patients were satisfied with the results.[81] Loss of correction of the intermetatarsal angle following proximal crescentic first metatarsal osteotomy is rare.[30]

Peterson and Newman have described yet another metatarsal osteotomy for the treatment of adolescent bunions.[101] In patients with a congruous but medially directed MTP joint, they recommend a closing medially based osteotomy of the distal metatarsal to direct the joint surface more laterally, and a proximal opening wedge osteotomy to close down the metatarsus primus varus. They use the distal wedge as bone graft in the proximal osteotomy. Fixation is achieved with a longitudinal intramedullary pin. The length of the first metatarsal is preserved, and since the entire procedure is extracapsular, stiffness of the first MTP joint is not a problem. They recommend this procedure only for severe hallux valgus deformities, but achieved very good results with one hallux varus out of 15 procedures.[101]

Another way to realign the first metatarsal and narrow the intermetatarsal distance between the first and second rays is to fuse the base of the first metatarsal to the medial cuneiform, a procedure popularized by Lapidus.[73] This procedure is proposed as treatment of hallux valgus in the presence of a hypermobile first ray in patients who are skeletally mature. Often this group of patients will have generalized ligamentous laxity and flatfeet. Modifications of the original procedure include the use of internal fixation and the abandonment of resecting bony wedges, which led to relative shortening of the first ray. The first metatarsal should also be slightly plantar flexed to share in the weightbearing forces with the lateral metatarsals. A distal soft tissue realignment of the first MTP joint is usually done concomitantly. Good results following the modified Lapidus procedure have been found in 77 to 91 percent of patients.[17,40,92]

The Akin procedure consists of a medial closing wedge proximal phalangeal osteotomy.[2] While it may align the toe, it does not address the increased intermetatarsal angle, and therefore it is not recommended in cases of adolescent hallux valgus.[38] The Keller resection arthroplasty is also not recommended for use in adolescents.

Lastly, fusion of the first MTP joint may be performed in patients with hallux valgus. Those patients who are candidates for MTP fusion are those who have underlying neurologic conditions such as cerebral palsy, and those with inflammatory arthritis with joint degeneration, such as teenagers with rheumatoid arthritis (Fig. 22–117).[35,39,50] MTP fusion is not selected as treatment for idiopathic hallux valgus. MTP fusion is described in further detail in the cerebral palsy section of Chapter 24, Disorders of the Brain.

Our procedure of choice for most patients with adolescent bunions has been a basilar first metatarsal osteotomy

A B

FIGURE 22–117 **A,** Symptomatic bunion deformity in an adolescent boy with juvenile polyarticular arthritis. **B,** First MTP fusion with pin fixation led to relief of symptoms. Concomitant hindfoot and ankle valgus were also treated.

with distal soft tissue realignment. Good correction of the intermetatarsal angle can usually be obtained. We stabilize the osteotomy either with threaded Steinmann pins or with a screw. We prefer the crescentic osteotomy to the opening or closing wedge techniques as it preserves metatarsal length. We have performed metatarsocuneiform fusion in selected patients with hypermobility, with satisfactory outcome. We do not have experience with the double osteotomy of the first metatarsal described by Peterson, but we recognize that it may be useful in those children who have congruent but maldirected MTP joints preoperatively.

HALLUX VALGUS INTERPHALANGEUS

Hallux valgus interphalangeus is a rare deformity in which the great toe deviates laterally, with the apex of deformity located at the IP joint (Fig. 22–118). It is not associated with metatarsus primus varus. Symptoms include difficult shoe wear and pain located over the medial prominence of the great toe.

Treatment is by osteotomy of the proximal phalanx.[38,108] A closing wedge based medially is preferred. Internal fixation or fixation with threaded Steinmann pins is performed.

HALLUX VARUS

Hallux varus is described as a medial deviation of the great toe at the MTP joint. In children the condition can be either congenital or acquired. In children acquired hallux varus is rare; in adults it is usually acquired as a complication of hallux valgus surgery.[103]

Congenital hallux varus can be divided into three types. First, hallux varus may occur in isolation, with a normal first metatarsal. In this form, a taut fibrous band runs from the medial side of the great toe to the base of the first metatarsal, leading to medial deviation of the hallux. Second, hallux varus may coexist with other malformations of the foot, such as a longitudinal bracket epiphysis of the first metatarsal or preaxial polydactyly (Fig. 22–119).[54] Third, hallux varus is uncommonly part of an underlying skeletal dysplasia, such as diastrophic dwarfism (Chapter 29, Skeletal Dysplasias).

Symptoms from hallux varus are both cosmetic and related to the ability to wear shoes. Shoe wear is nearly impossible with the deviation of the great toe. Additionally, the deformity is believed to worsen with age.[59] For this reason, surgical correction is proposed in infancy.

Surgery for congenital hallux varus consists of release of the tight tissues on the medial side of the toe, including the abductor hallucis, and the medial capsule of the MTP joint. The great toe is usually syndactylized to the second toe to maintain correction.[63] The lateral MTP joint may be reefed, and extensor hallucis brevis rerouting has been described.[33,84] When present, accessory ossicles or bones should be excised. Temporary fixation of the MTP joint with a Kirschner wire should be done to maintain position while the tissues heal. Mills and Menelaus reported satisfactory results at 12.7 years in 12 of 17 feet that had undergone surgical correction of congenital hallux varus using the Farmer and McElvenny procedures described above.[87] Arthrodesis of the first MTP joint is reserved for those in whom primary reconstruction fails and painful arthritis develops.[87]

Acquired hallux varus generally occurs in adults. Causes include overcorrection from bunion surgeries such as the McBride procedure, trauma, and systemic arthritis. Many authors have reported on surgical correction of this deformity following hallux valgus surgery.[29,56,93,111,119]

HALLUX RIGIDUS

Hallux rigidus is defined as a condition in which there is restriction of motion at the first MTP joint.[109] This condition is far more common in older adults, but it can rarely occur in adolescents. Girls are affected more often than boys, and the disease is frequently bilateral. There may be a positive family history. There are several theories regarding the etiol-

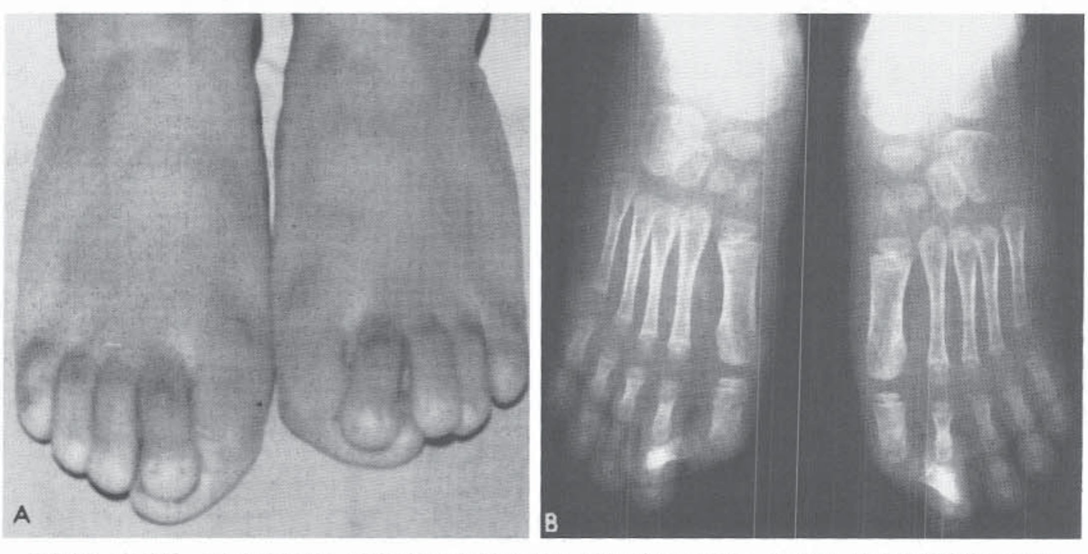


FIGURE 22–118 Bilateral hallux valgus interphalangeus. **A,** Clinical appearance. Note the lateral deviation of the distal phalanx of the great toe. **B,** AP radiograph of both feet showing lateral subluxation of the IP joint of the hallux.

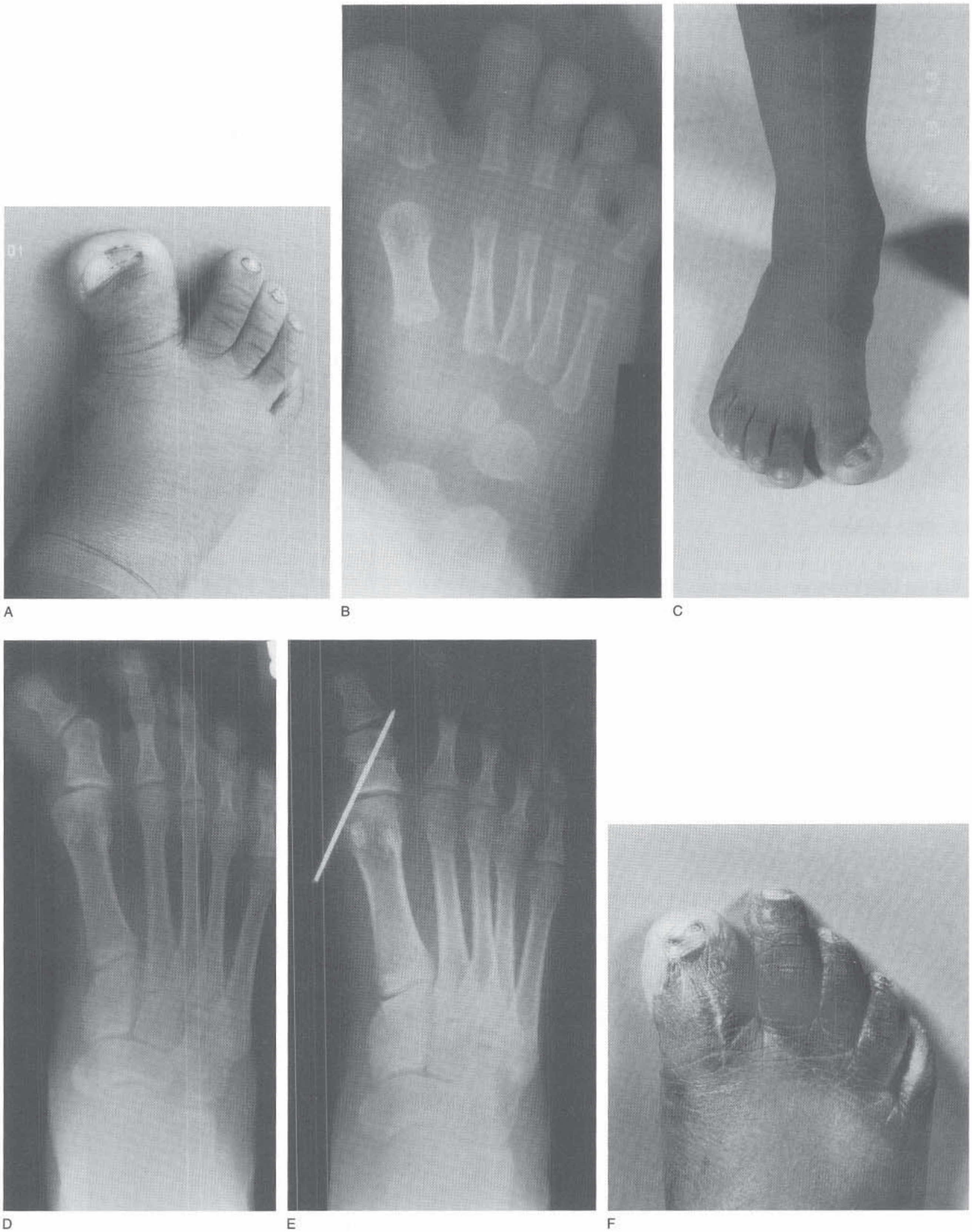


FIGURE 22–119 **A** and **B**, Preoperative clinical and radiographic appearance of a 1-year-old girl's foot with complex preaxial polydactyly and hallux varus. **C** and **D**, Appearance at age 14. Hallux varus has recurred through the IP joint. **E**, A lateral closing wedge proximal phalangeal osteotomy was performed. **F**, Clinical appearance of the foot following surgery.

ogy of hallux rigidus, including repetitive trauma, a hypermobile long first ray, osteochondritis dissecans, and plantar contracture.[112]

Presenting symptoms include pain during gait. Discomfort in the MTP joint is greatest during heel rise, as the great toe normally dorsiflexes at this time. Physical examination reveals painful limitation in dorsiflexion of the first MTP joint. Dorsiflexion is lost before plantar flexion is. There is often a palpable osteophyte on the dorsum of the joint, and swelling may be present. The base of the metatarsal appears more plantar than normal, and the metatarsal head is elevated. Observation of gait shows that the patients walk on the lateral border of the feet, to avoid rolling over the great toe.

Radiographs show narrowing of the joint (Fig. 22–120). Lateral plain films can demonstrate the dorsal osteophytes at the base of the proximal phalanx and metatarsal head. The exostoses may become quite large and obscure visualization of the joint itself. Osteochondritic lesions of the metatarsal head can be seen in young patients.[116] The first metatarsal may be relatively long.[65]

Treatment is initially directed toward symptomatic relief, and nonsteroidal medications are prescribed.[109] The patient should be restricted from participating in sports. Shoe modifications that have been recommended include a high toe box to relieve rubbing of the dorsum of the joint, a rigid shank within the sole of the shoe to limit motion of the MTP joint at heel rise, a stiff insert that extends past the MTP joint medially, again to limit motion, and a rocker-bottom shoe to assist in push-off.

Surgical treatment for early hallux rigidus is cheilectomy, with resection of the prominent osteophytes, debridement of the joint, and synovectomy.[78] If cheilectomy is unsuccessful, a dorsal closing wedge osteotomy of the proximal phalanx may be helpful.[65] In advanced cases, arthrodesis of the MTP joint is recommended in young, active patients.[76,109]

POLYDACTYLY

Polydactyly is the most common congenital toe deformity, with an incidence of 1.7 per 1,000 live births.[34] Polydactyly is seen more frequently in black babies, with an incidence of 13.9 per 1,000 live births in blacks and 1.3 per 1,000 live births in whites.[124] A positive family history is present in 30 percent of patients.[102] Polydactyly is bilateral in approximately 50 percent of patients, but bilateral cases need not be symmetric.[102] Most series report a slight predominance of males.[14,102]

The fifth toe is most frequently duplicated, a condition termed postaxial polydactyly (Fig. 22–121). Postaxial polydactyly accounts for 80 percent of polydactyly in the foot.[102] Preaxial polydactyly is duplication of the great toe (Fig. 22–122). The central toes are infrequently duplicated. Polydactyly is usually an isolated malformation, but there are certain syndromes in which polydactyly is a feature, among them Ellis-Van Creveld syndrome, trisomy 13, tibial hemimelia, and Down syndrome.[13,15] Postaxial duplications are associated with other malformations in only 11.8 percent of cases, and preaxial polydactyly is seen as part of a syndrome in 20 percent of patients.[15,23,62] Polydactyly of the hand is seen in 34 percent of children with polydactyly of the foot.[102]

Polydactyly of the foot can be further divided into two groups, type A being a well-formed articulated digit and type B a rudimentary digit (Fig. 22–123).[102] Venn-Watson classified the metatarsal abnormalities in polydactyly in 1976. The metatarsal may be normal with a duplicated distal phalanx of the toe. It may be a block metatarsal, which is a widened and often shortened metatarsal. The metatarsal can be Y-shaped, giving rise to two separate articulations with the toes, or T-shaped, with a less distinct division between the articular surfaces. The metatarsal head may be widened and may articulate with both proximal phalanges

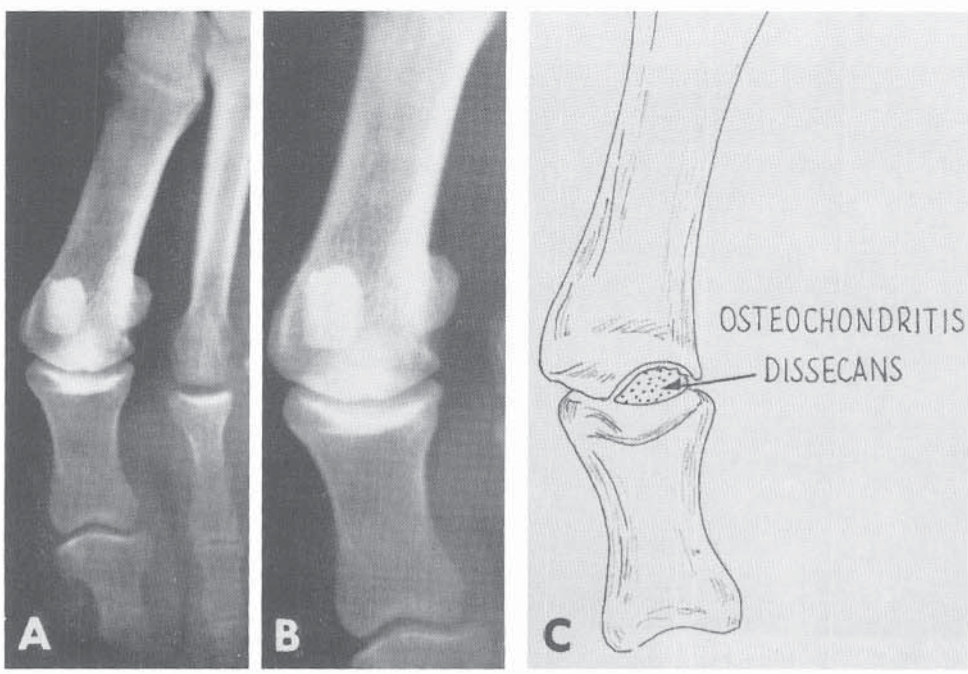


FIGURE 22–120 A to C, Osteochondritis dissecans of the first metatarsal head. This condition is an occasional cause of hallux rigidus. (From Kelikian H: Hallux Valgus, Allied Deformities of the Forefoot and Metatarsalgia, p 273. Philadelphia, WB Saunders Co, 1965.)

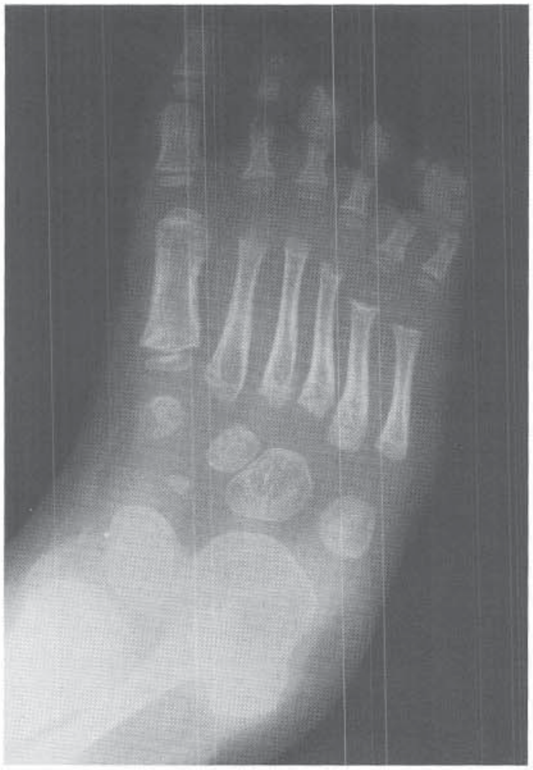


FIGURE 22–121 Postaxial polydactyly with duplication of all phalanges of the small toe, the fifth metatarsal, and the cuboid.

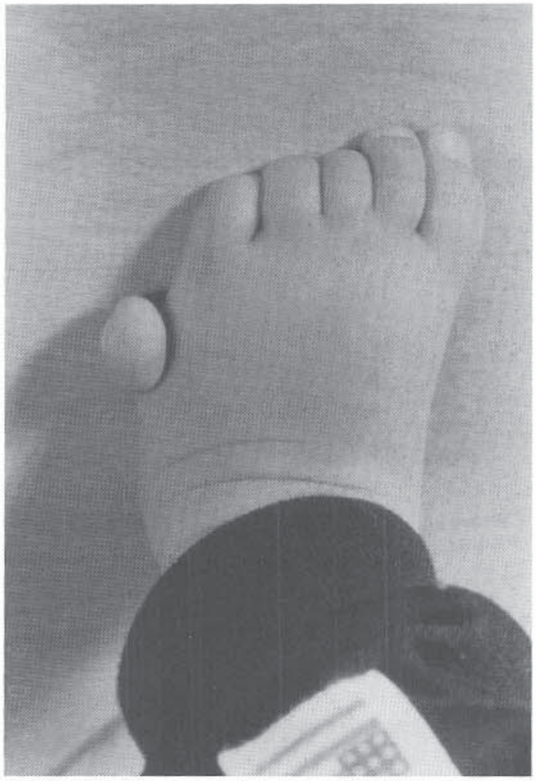


FIGURE 22–123 Rudimentary sixth toe in an 18-month-old boy.

of the duplicated toes. Lastly, the ray may be duplicated altogether, with a duplicated metatarsal articulating with the duplicated toe (Fig. 22–124).[122]

Treatment is surgical excision of the duplicated toes around 1 year of age.[97] Surgery improves cosmesis and facilitates normal shoe fitting. Preoperative radiographs should be obtained to define the anatomy of the metatarsal and to identify any additional duplications within the foot that will require excision. Generally speaking, the outer toes are

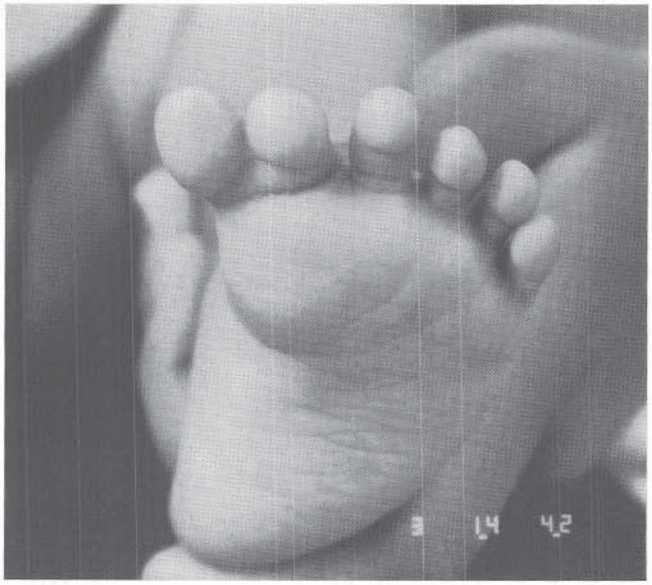


FIGURE 22–122 Preaxial polydactyly with duplication of the great toe.

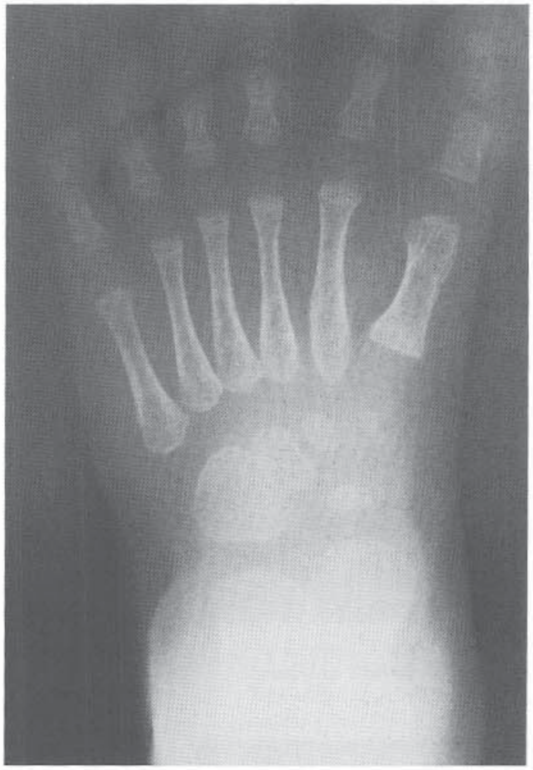
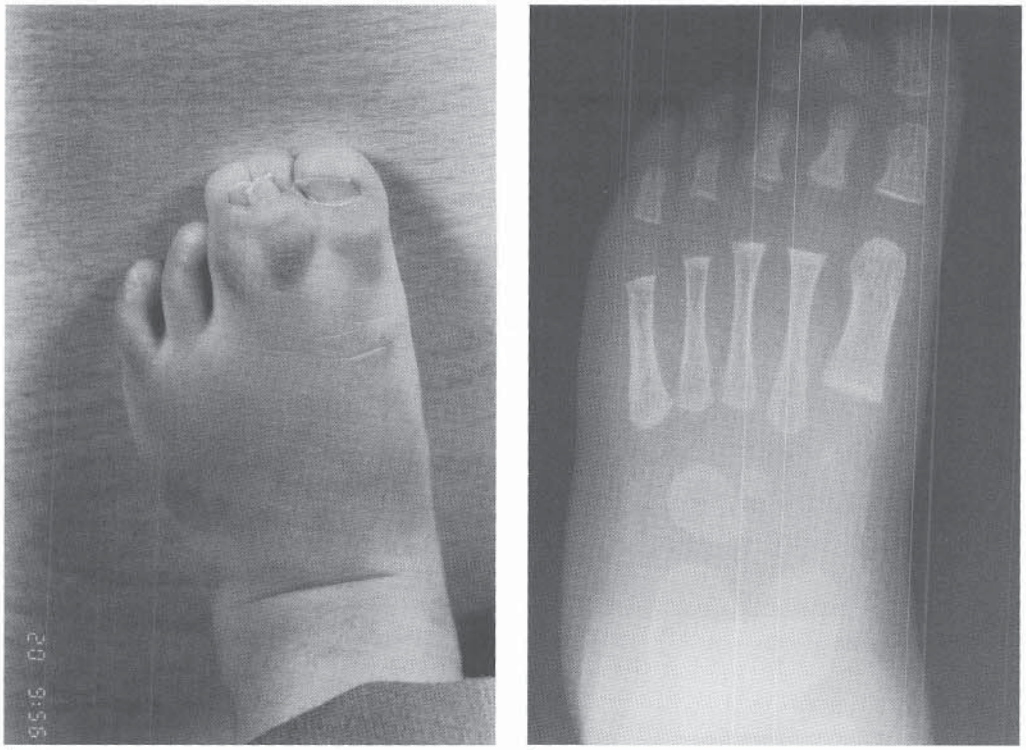


FIGURE 22–124 Polydactyly with duplicated metatarsal.



A B

FIGURE 22–125 **A** and **B**, Syndactyly of the first, second, and third toes in a 1-year-old child.

usually amputated in preaxial and polyaxial polydactyly, unless those toes are clearly better developed than the inner duplicated toes. When the inner toes are removed, capsular and intermetatarsal ligament repair is very important.

In postaxial polydactyly, amputation of the most lateral small toe is usually performed using a racquet incision. Excision of the lateral aspect of a Y- or T-shaped metatarsal should also be performed through a dorsolateral extension of the racquet incision. In cases of a widened metatarsal head without a separate articulation, the lateral prominence should be shaved down to reduce the bump over the fifth metatarsal head. Growth arrest of the distal metatarsal physis has not been a problem.[102]

Usually the most medial great toe is excised in preaxial polydactyly, except when the more lateral great toe is clearly underdeveloped. In those cases, the medial toe may be preserved and the lateral great toe amputated. Regardless of which toe is removed, careful soft tissue balancing of the adductor and abductor hallucis insertions should be performed, since hallux varus is a frequent complication of preaxial polydactyly surgery.[102] When preaxial polydactyly is seen with a block first metatarsal, transfer lesions to the lesser metatarsals can develop as a result of shortening of the first metatarsal relative to the rest of the foot.

Central polydactyly is usually treated with amputation of the duplicated central digit and repair of the intermetatarsal ligament. With growth, the foot may widen.[102]

SYNDACTYLY

Congenital webbing of the toes is a common malformation. The syndactyly may extend all the way to the tips of the toes (Fig. 22–125), but more commonly it is incomplete, with only proximal webbing. The most common site is between the second and third toes (Fig. 22–126), followed by syndactyly of the fourth and fifth toes.[16] The toenails may be confluent in more severe cases.

Syndactyly does not lead to functional problems. The webbing is simply a cosmetic issue and does not require treatment. Families usually readily agree to nontreatment once they are counseled regarding the need for skin grafts and the inevitable visible scars following release.

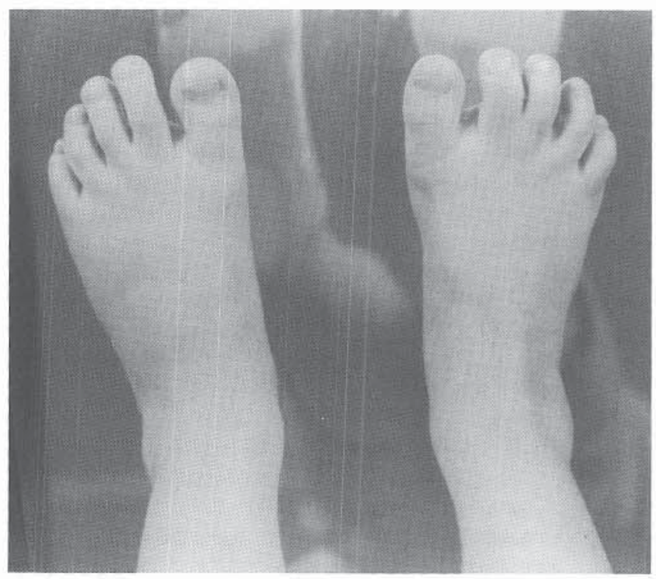


FIGURE 22–126 Subtle syndactyly of the second and third toes extending just to the PIP joint on the right.

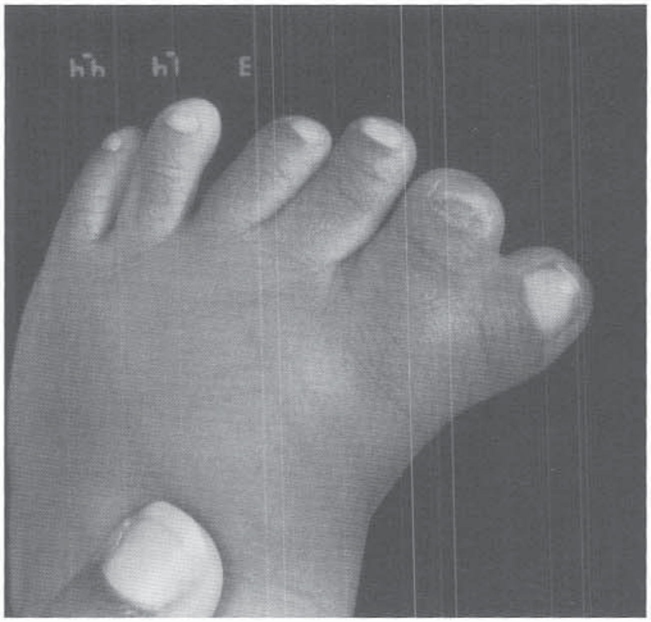


FIGURE 22–127 Preaxial polysyndactyly.

Syndactyly may be present in combination with polydactyly (Fig. 22–127).[15,97] In these cases it is advisable to resect the most peripheral toe with preservation of skin flaps. The skin from the amputated toe can then be used to cover the remaining toe.[3] Patients with syndactyly of the fourth and fifth toes often have coexisting postaxial polydactyly. Stevens recommends resection of the duplicated sixth toe without separation of the fourth and fifth toes, as spread between the fourth and fifth toes is likely to develop over time.[112]

Complex syndactyly of the toes is seen in patients with Apert's syndrome.[19,26] Treatment is quite difficult because of the significant deformities, but separation of the syndactylies of the toes again is not advised.

MACRODACTYLY

Macrodactyly is the term given to enlargement of one or more toes due to hyperplasia of the tissues of the toe.[1,27,49] The condition may be idiopathic, and it may involve the hands too.[61,64,71] Some patients have a positive family history.[88] Macrodactyly may be a manifestation of a generalized syndrome. Proteus syndrome, tuberous sclerosis, neurofibromatosis, and Klippel-Trénauney-Weber syndrome all may produce macrodactyly of the toe.[85,98,113,121] The hamartomatous tissue in the enlarged toe may be lymphatic, vascular, fibrofatty, or nerve-related, and the differentiation can be made with MRI.[10,25,28]

Two forms of macrodactyly exist. In the first, the toes are enlarged in proportion to a hypertrophied foot. Shoewear problems are limited to the possible need to buy two different-sized shoes. Treatment is usually not necessary.

In the second form the toe or toes are markedly enlarged relative to the rest of the foot (Fig. 22–128). This is termed localized gigantism. The phalanges and the associated metatarsal are enlarged, leading to deformity and inability to fit shoes because of the protruding digit. Surgical treatment is helpful. Reconstruction can be by epiphysiodesis with or without debulking, distal amputation of the phalanges,[70] or ray resection of the metatarsal and phalanges.[41] When epiphysiodesis is performed, the toe remains very big until the other ipsilateral toes and the contralateral foot undergo sufficient growth that the toe does not appear protuberant.[118] Another disadvantage to epiphysiodesis is that the excessive length may be corrected, but the abnormal width of the toe and foot will remain. When one toe is grotesquely enlarged, ray resection is recommended, as the cosmetic result is usually quite acceptable to the parents, multiple surgeries are not needed, and the foot can be fit into a shoe immediately following surgery.

Debulking of the hypertrophied toe at first sounds appealing, but a study of the complications following this procedure usually sways the orthopaedic surgeon from performing debulking. First, the hypertrophic tissue tends to regrow. Second, stiffness of the MTP and IP joints may develop. Third, wound healing is delayed, and fourth, bleeding from the surgical wounds is not uncommon.

In patients with Klippel-Trénaunay-Weber syndrome, macrodactyly can be very difficult to satisfactorily treat. In this condition, localized gigantism or hemihypertrophy, hemangioma, and abnormal venous varicosities are seen (Fig. 22–129).[85] The pathologic circulation to the toe leaves the child susceptive to wound dehiscence and infection following surgery.

Recurrence of tissue enlargement following resection may occur, and in some cases there has been proximal migration of hemihypertrophy. These cases are extremely difficult to treat.

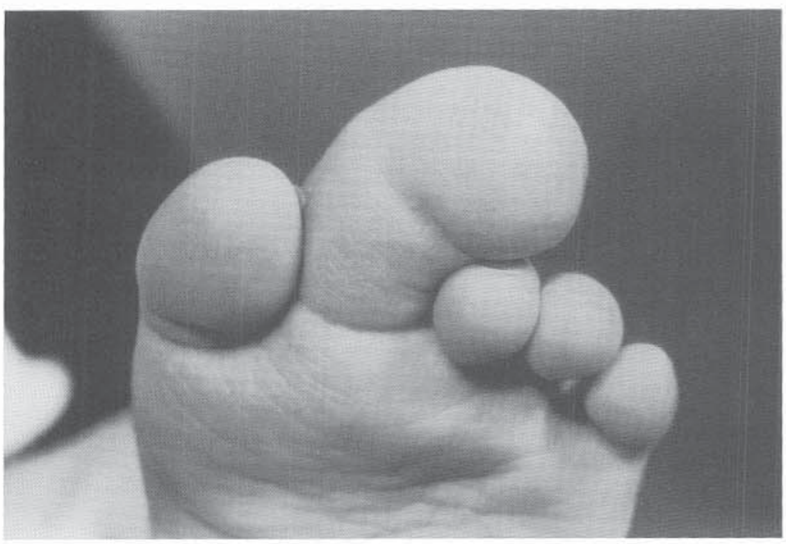
VARUS FIFTH TOE

This deformity, also known as congenital digitus minimus varus, is present at birth and may be genetic in etiology. The fifth toe is dorsiflexed and adducted and overlaps the fourth toe. The fifth toe rotates so that the toenail lies laterally (Fig. 22–130). Symptoms develop over time and consist of pain over the dorsum of the fifth toe from local irritation from wearing shoes. The deformity is usually bilateral.

Conservative measures such as buddy taping have not been efficacious. Treatment is surgical.[90,99] Soft tissue reconstruction consists of release of the contracted extensor tendon, dorsal capsulotomy of the MTP joint, and excision of the abnormal skin crease between the fourth and fifth toes.[51] Temporary fixation with a smooth K-wire is usually helpful. A dorsal incision should be avoided, as it may contract postoperatively and lead to recurrence. The Butler procedure includes release of the dorsal MTP joint capsule and tenotomy of the extensor digitorum longus tendon, but a circumferential incision about the base of the toe is used, with dorsal and plantar longitudinal extension (Fig. 22–131).[8,18,117] When this procedure is performed, care must be taken to identify and protect the neurovascular bundles. This incision may place the circulation to the toe at risk, particularly in older children, and should be used with care.

The McFarland operation consists of extensor tenotomy, dorsal and medial capsulotomy of the MTP joint, proximal phalangectomy, and syndactylization of the fourth and fifth toes. Tachdjian preferred this procedure,[115] but we do not have experience with it at our hospital (Plate 22–7).

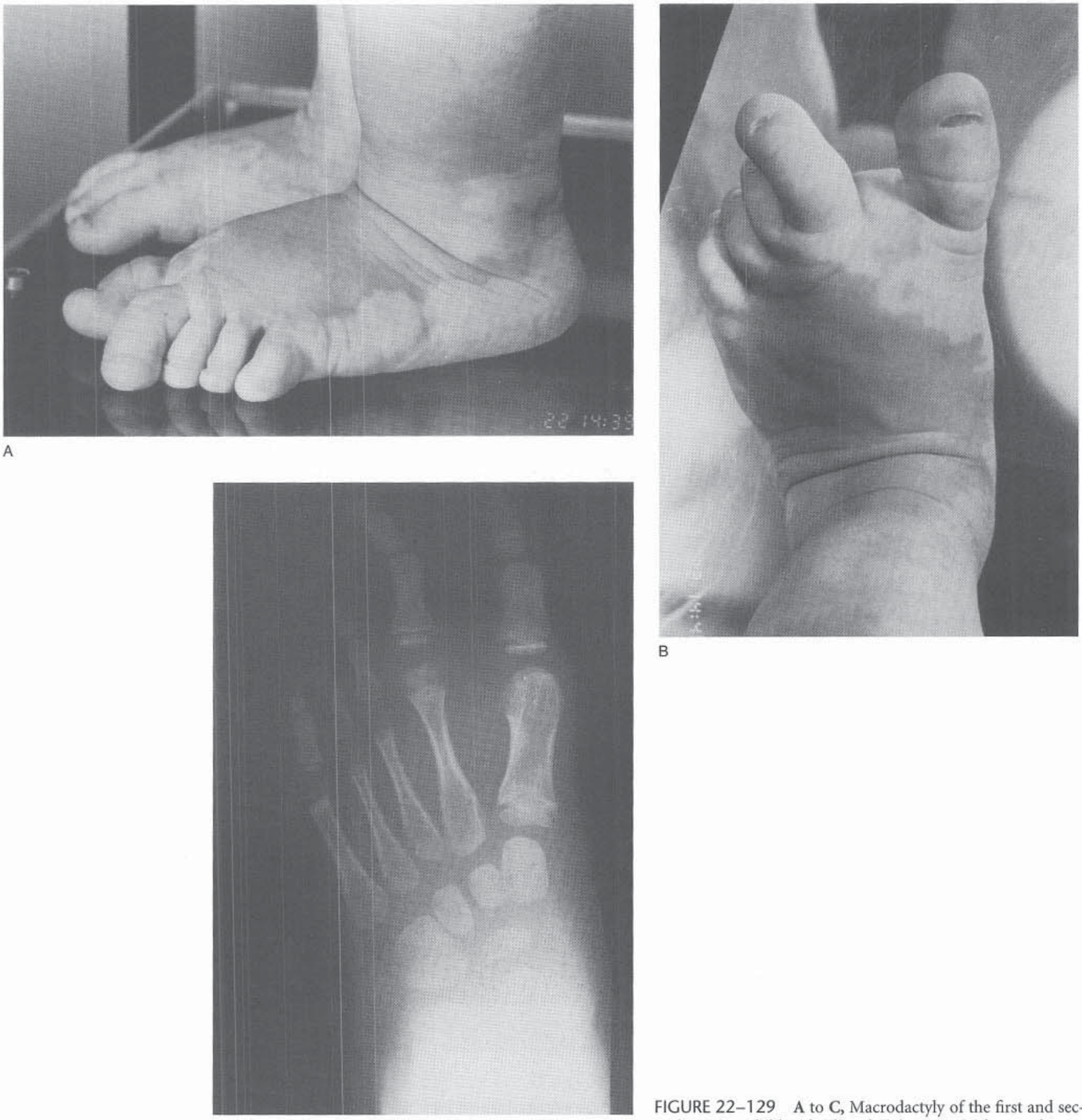
A



B

C

FIGURE 22–128 **A** to **C,** Macrodactyly of the second toe in a 2-year-old boy. A second ray resection restored the ability to wear shoes.



A

B

C

FIGURE 22–129 **A** to **C**, Macrodactyly of the first and second toes in a child with Klippel-Trénaunay-Weber syndrome.

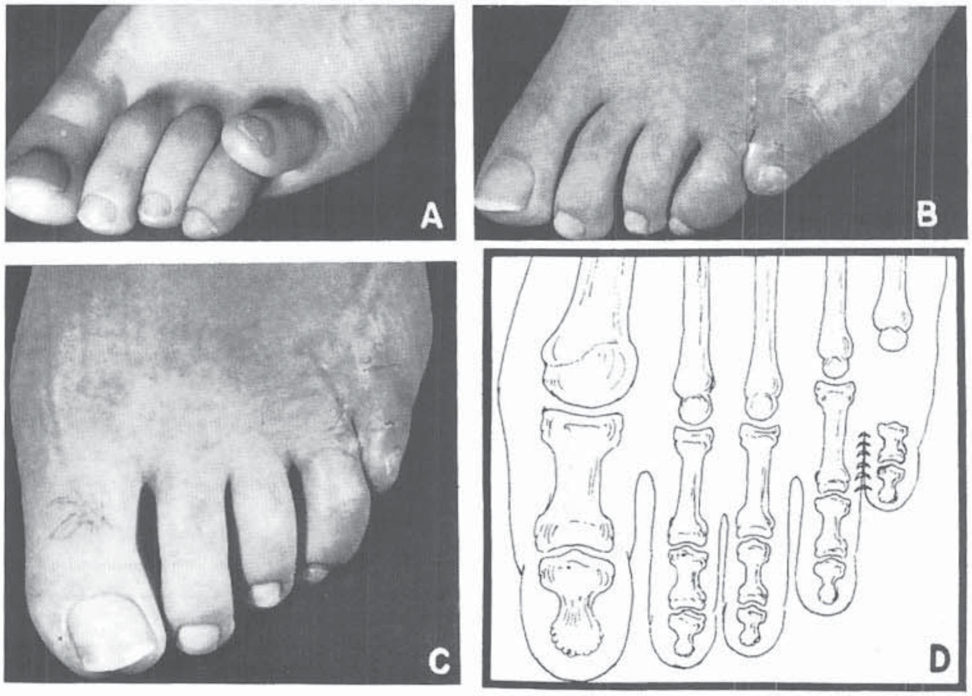


FIGURE 22–130 Varus fifth toe. The deformity was treated by excision of the proximal phalanx of the little toe, extensor tenotomy, dorsal capsulotomy of the fifth MTP joint, and surgical syndactyly of the fourth and fifth toes. **A,** Preoperative photograph. **B** and **C,** Postoperative photographs. **D,** Interpretive diagram. (From Kelikian H: Hallux Valgus, Allied Deformities of the Forefoot and Metatarsalgia, p 328. Philadelphia, WB Saunders Co, 1965.)

When there is recurrence following soft tissue release, proximal phalangectomy may be indicated. Amputation of the fifth toe is not recommended.

CURLY TOES

This common deformity is characterized by flexion and medial deviation of the PIP joint of the toe. The toe rotates laterally at the DIP joint and underlaps the adjacent normal toe. The cause of the curly toe deformity is congenital tightness of the flexor digitorum longus and brevis tendons.[117] Curly toes are usually present bilaterally and most commonly affect the third or fourth toes (Fig. 22–132). There often is a positive family history consistent with an autosomal dominant mode of transmission.

The natural history of curly toe is resolution in 24 percent in cases and persistence in the remaining 76 percent.[114] Taping may improve the toe position transiently, but the deformity usually returns once taping is stopped.[120] Symptoms result from abnormal pressure on the adjacent metatarsal heads, and pain in the underlapped toe itself, which may develop corns.

Surgery is recommended for children who have persistent deformity at age 6 years. Flexor to extensor tendon transfer led to satisfactory results in 37 of 43 children with curly toes.[7] Simple open flexor tenotomy led to relief of symptoms and did not lead to a hyperextended position of the toe in 95 percent of 62 patients treated.[104] In a study comparing the results of flexor to extensor transfer versus flexor tenotomy, similar results were obtained with either procedure.[44] Tenotomy of the long and short toe flexors without tendon transfer is currently recommended by most authors for the treatment of curly toes.[104,112,120] If a longitudinal skin incision is made, it should not cross the flexor creases, since this may predispose to recurrence. Extensor tenotomy of the toe that lies dorsally over the curly toe should not be performed.

HAMMER TOES

The hammer toe deformity consists of flexion of the PIP joint, with or without flexion of the DIP joint. It is similar to a curly toe except that the hammer toe is not malrotated. The MTP joint is usually hyperextended. When the MTP joint is passively plantar flexed, the PIP joint may be extended.[112] The most common toe affected is the second toe, and the second metatarsal may be longer than normal.[117] Although a hammer toe is usually asymptomatic in young patients, painful calluses may develop over the PIP joint over time.

Conservative treatment with stretching and taping usually fails. The preferred surgical treatment is flexor tenotomy. Satisfactory results were found following flexor tenotomy in 95 percent of cases.[104] The Girdlestone procedure, in which the flexor tendon is transferred to the toe extensor, has similar success, but is a more extensive procedure and probably the transfer is unnecessary.[96] Complete or partial resection of the proximal phalanx or arthrodesis of the PIP joint may be necessary in older adolescents with fixed deformities (Plate 22–8).[55,94,112]

MALLET TOE

The mallet toe is a deformity consisting of flexion of the DIP joint due to congenital shortening of the flexor dig-

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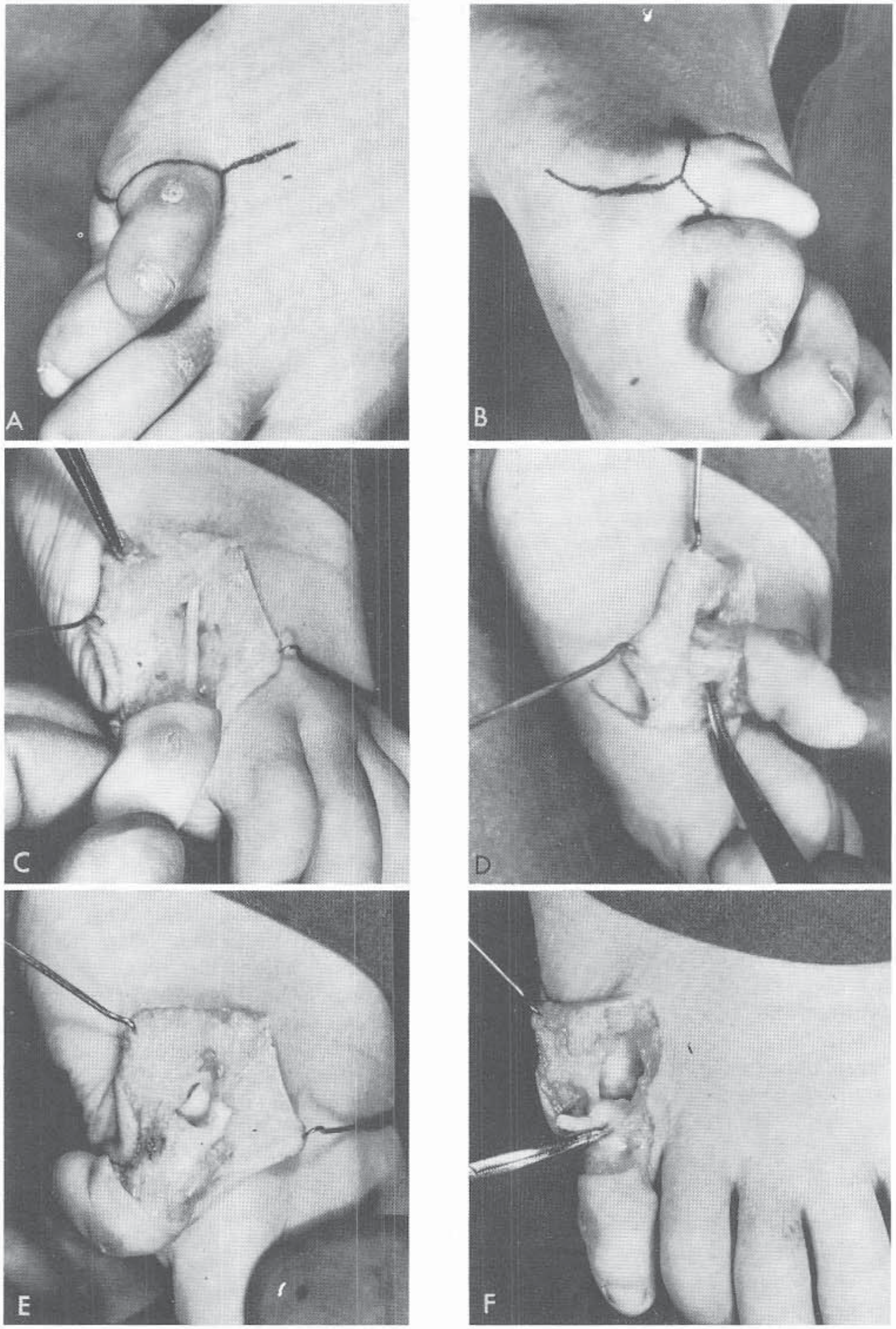


FIGURE 22–131 Butler's operation for an overriding fifth toe. **A** and **B,** A dorsal racquet incision is made, with a second handle added on the plantar aspect. The plantar handle is inclined laterally and is a little longer than the dorsal handle. **C** and **D,** The contracted extensor tendon to the fifth toe is exposed by elevating the skin flaps. The neurovascular bundles should be identified and carefully preserved. **E,** Sectioning of the extensor tendon and the dorsomedial part of the capsule of the MTP joint. **F,** In severe deformity, the articular surfaces of the MTP joints may be incongruous. This is due to plantar capsular adhesions.

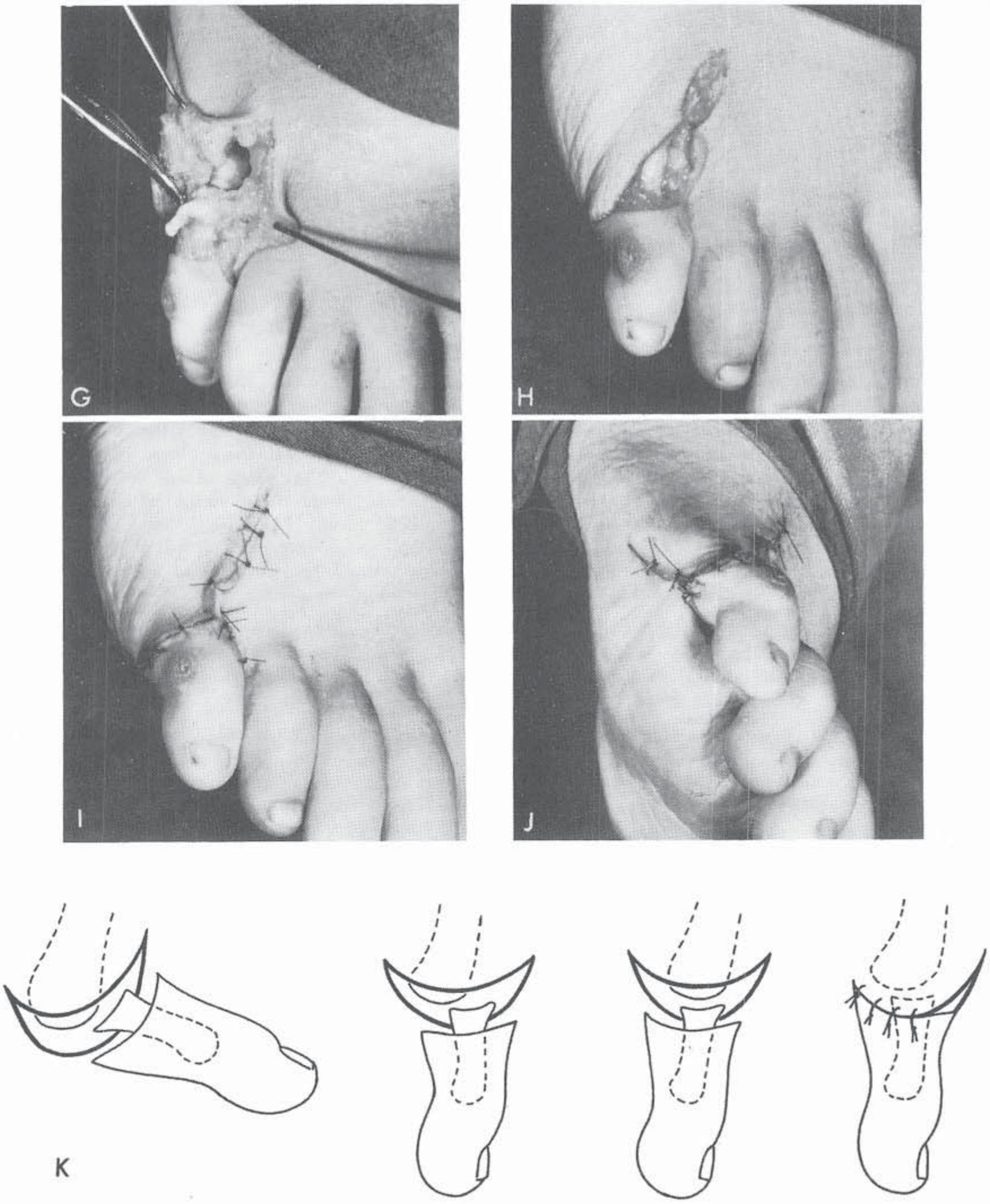


FIGURE 22–131 *Continued.* **G,** Adhesions on the plantar part of the capsule are freed by blunt dissection. The little toe now lies in the fully corrected position. **H,** Appearance of the toe before skin closure. It lies freely in normal alignment without tension. **I** and **J,** Closure of the wound. Skin sutures securely hold the toe in the correct position. **K,** Diagrammatic illustration of the mechanics of the operation. (From Cocklin J: Butler's operation for an overriding fifth toe. J Bone Joint Surg 1968;50-B:78.)

Correction of Digitus Minimus Varus (McFarland, Kelikian)

OPERATIVE TECHNIQUE

A, First a 00 silk whip suture is passed through the pulps of the fourth and fifth toes. The suture ends are clamped with hemostats and the toes are pulled apart, bringing the web space into full view.

B, Three sets of incisions are made: (1) A web-bisecting incision that starts on the dorsum of the forefoot in the groove between the metatarsal heads and extends distally to bisect the web, then passes plantarward to terminate at about the same point posteriorly on the plantar aspect of the forefoot as it does on the dorsum. (2) Two paradigital incisions, one for each toe, which begin at the point where the web-bisecting incision begins to dip plantarward and extend lengthwise along the adjacent side of each toe. The paradigital incision for the little toe ends on the side of the distal phalanx at a point plantar and just proximal to the base of the nail, whereas the incision for the fourth toe is the same length as that for the fifth. The paradigital incisions are placed slightly toward the plantar border of the toe to give a semblance of an interdigital groove after surgical syndactylism. (3) Two connecting oblique incisions extending from the terminal point of the paradigital incision on each side to the proximal end of the web-bisecting incision on the plantar aspect.

C, The triangular patches of skin between the paradigital and oblique connecting incisions are excised. In dissecting the subcutaneous tissue in this area, the surgeon must be careful not to injure the plexus of veins. The skin flaps are mobilized and retracted to their respective sides. The digital nerves and vessels should be identified and protected from injury.

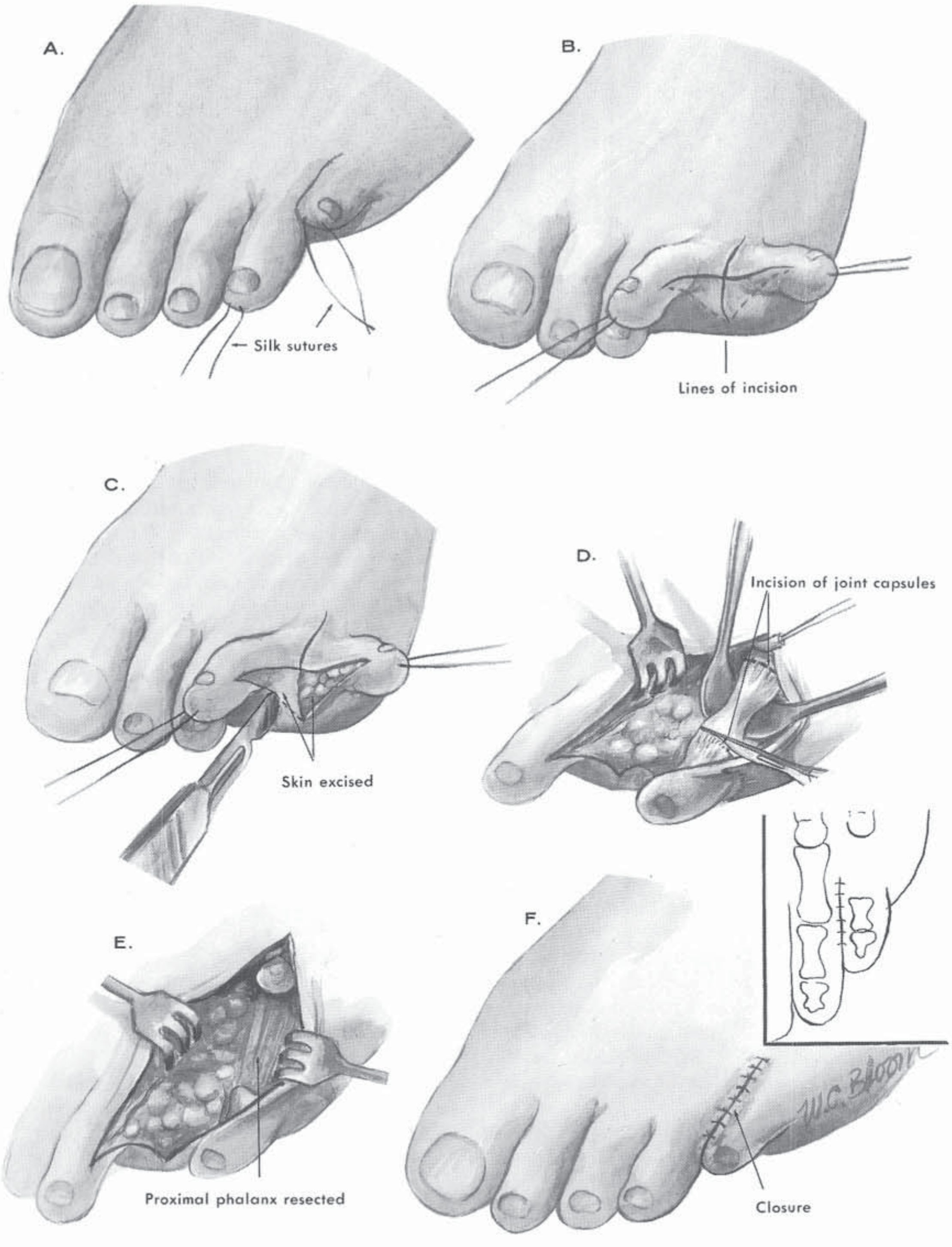
D and **E,** The long extensor tendon of the fifth toe is divided at its insertion; a 00 silk whip suture is applied to its distal end. (This end is later transferred to the fifth metatarsal head according to the technique for the Jones procedure described in Plate 22–3.) Next, the long flexor of the fifth toe is dissected free of the proximal phalanx. Small retractors are placed on the dorsal and plantar aspects of the bone to protect the soft tissues. The capsules of the metatarsophalangeal and proximal interphalangeal joints of the little toe are divided, and the proximal phalanx is excised. The long fifth toe extensor is transferred to the fifth metatarsal head. The wound is packed with moist gauze, the pneumatic tourniquet is deflated, and bleeding vessels are clamped and coagulated.

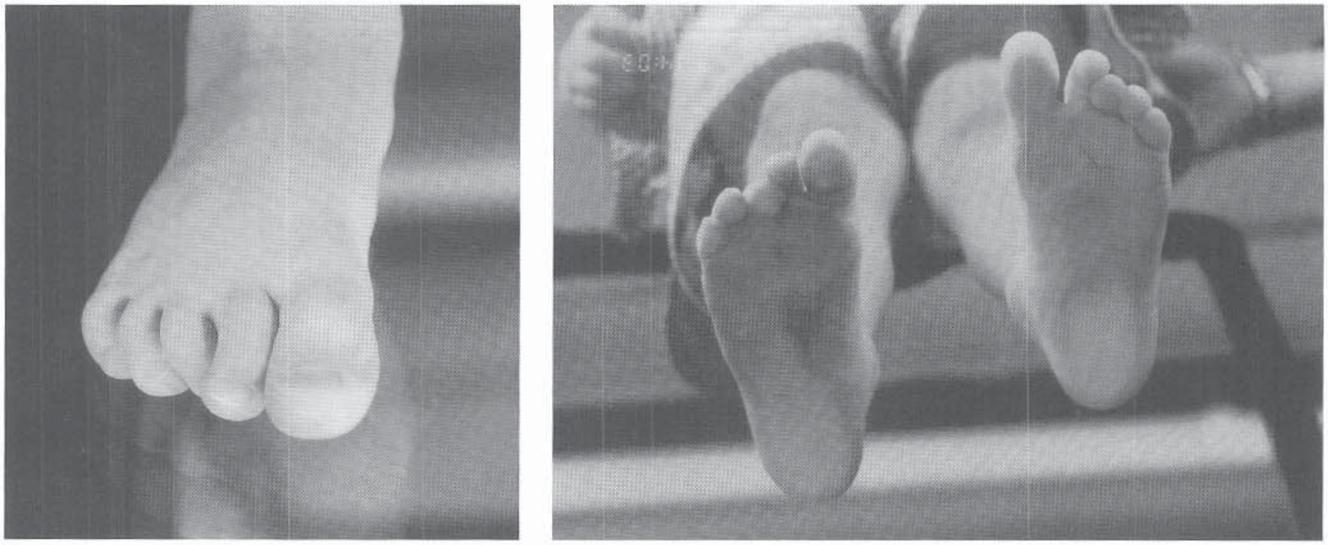
F, The terminal points of the paradigital incisions are sutured together with 0000 nylon, bringing the toes together. The alignment of the toes is inspected. Care is taken to avoid eversion or inversion of the toes; if necessary, the terminal suture is removed and reapplied. The dorsal wound is closed with 0000 nylon and the plantar skin edges with 0000 plain catgut.

POSTOPERATIVE CARE

A below-knee walking cast is applied. Three to four weeks following surgery, the cast and sutures are removed. The patient is allowed to bear weight and resume normal activities.

PLATE 22–7. Correction of Digitus Minimus Varus (McFarland, Kelikian)





A B

FIGURE 22–132 **A** and **B**, Curly toes of the third, fourth, and fifth toes.

itorum longus (Fig. 22–133). Similar to hammer toes, the second toe is most commonly involved, and the second ray may be long.[117]

Surgical treatment is most commonly tenotomy of the flexor digitorum longus. Resection of the head of the middle phalanx may be indicated in older teens with fixed deformities. Arthrodesis of the DIP joint has also been advocated in older patients.[112]

LONGITUDINAL EPIPHYSEAL BRACKET

A longitudinal epiphyseal bracket, also known as delta phalanx, is a congenital condition in which the epiphysis extends along the base of the phalanx, up along the side of the diaphysis, and across the distal surface of the phalanx in a U configuration. The deformity was first described and named delta phalanx by Jones in 1964.[57] This condition may also involve the first metatarsal or metacarpal, since they also have proximally located epiphyses.[91] Growth of the abnormal epiphysis leads to a shortened, wide, and usually triangular or trapezoidal phalanx. The condition may be present in the hands, feet, or both.[53] It may be familial (Fig. 22–134).[95] When the condition is present in the great toes, a hallux varus deformity develops.

In many cases, polydactyly, syndactyly, or tarsal coalition is present.[91] It has been postulated that the cause of a longitudinal epiphyseal bracket may be a failure of proper fetal formation of the primary ossification centers from the apical ectodermal ridge.[74,112] This condition may also be present in children with Apert's syndrome.

Treatment is surgical. Osteotomy alone leads to persistent abnormal growth of the aberrant epiphysis. Resection of the abnormal longitudinal section of the epiphysis and interposition with PMMA or fat has been described. The transverse extensions of the epiphysis are preserved both proximally and distally. Mubarak and colleagues demonstrated improved longitudinal growth in the first metatarsal following such a procedure.[91] They recommended performing this surgery at age 6 months for best results. At this age, the bracket will not be demonstrable on plain radiographs; however, it can be seen on MRI.[91] Loupe magnification during surgery may be helpful in these small toes.

SUBUNGUAL EXOSTOSIS

Subungual exostosis is a benign bone growth located on the dorsomedial surface of the distal phalanx of the toe, just beneath the toenail. It is the great toe that is most commonly affected (Fig. 22–135).[72] Patients typically present with this condition in adolescence. Girls are more often affected than boys. A posttraumatic etiology has been proposed.[32,72]

Symptoms consist of pain and toenail irritation. On physical examination, the lesion feels firm and is palpable beneath the toenail. The nail may be tented by the mass. Pressing on the toenail produces pain. Lateral radiographs will reveal the mass. Histology reveals mature trabecular bone capped by fibrocartilage.[112]

Treatment is surgical excision by partial nail removal, longitudinal incision of the nail bed, and nail bed repair. Recurrence is unusual, occurring in 11 percent in a series of 44 exostoses.[72]

GLOMUS TUMOR

The glomus tumor resembles a hemangioma, and is present in the distal phalanx. It is very rarely seen in children. Radiographically, a lytic lesion that is round and well-circumscribed is seen. Histologic examination reveals characteristic intraluminal glomus cells. Treatment consists of excision of the lesion.[112]

A. B.

FIGURE 22–133 Mallet toe. **A,** Severe. **B,** Mild.

A B

FIGURE 22–134 **A,** Bracket epiphysis in a girl age 2 years 9 months that involves the proximal phalanx of the great toe. **B,** Radiograph of her father's foot shows a similar deformity.

FIGURE 22–135 Subungual exostosis of the distal phalanx of the great toe.

Correction of Hammer Toe by Resection and Arthrodesis of Proximal Interphalangeal Joint

OPERATIVE TECHNIQUE

A, A 3- to 4-cm longitudinal incision is made over the dorsal aspect of the proximal interphalangeal (PIP) joint parallel to and at the lateral border of the extensor digitorum longus tendon. The subcutaneous tissue is divided and the skin flaps are retracted.

B, The long extensor tendon is split and retracted to expose the capsule of the PIP joint. The digital vessels and nerves are protected from injury. A transverse incision is made in the capsule and the joint surfaces are widely exposed.

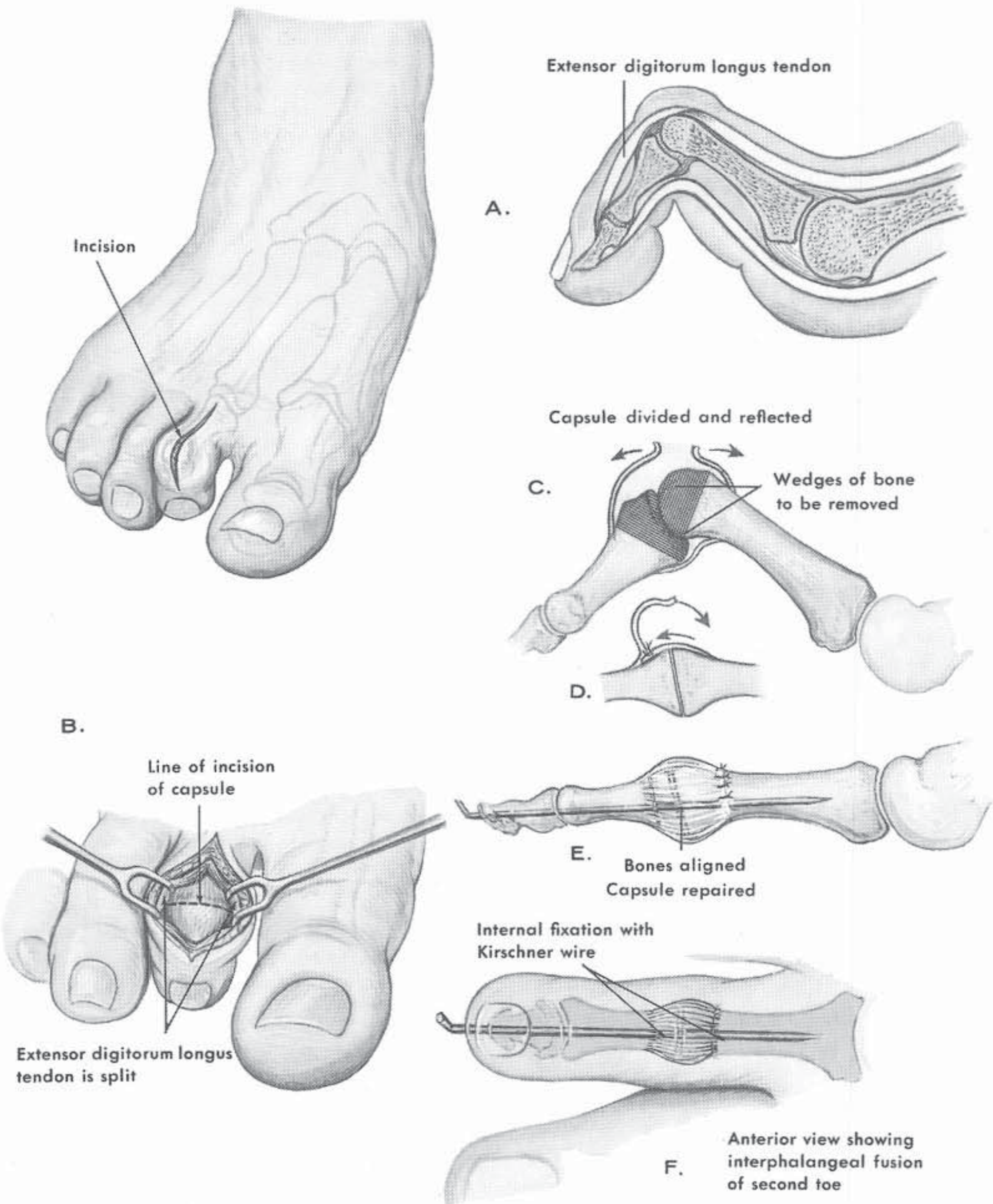
C and **D,** With a rongeur, wedges of bone based dorsally are resected from the head of the proximal phalanx and the base of the middle phalanx. Enough bone should be removed to allow correction of the deformity.

E and **F,** The proximal and middle phalanges are held together by internal fixation with a Kirschner wire that is inserted retrograde. The Kirschner wire should not cross the metatarsophalangeal joint. The cancellous bony surfaces of the middle and proximal phalanges should be apposed, and the rotational alignment should be correct. The capsule is resutured tightly by reefing. The wound is closed in routine manner. With a pair of nose pliers, the end of the Kirschner wire is bent 90 degrees and cut, leaving 0.5 cm protruding through the skin.

POSTOPERATIVE CARE

A below-knee walking cast is applied with a band of plaster of Paris protecting the toe. The wire and cast are removed in 6 weeks, when the radiographs show fusion of the interphalangeal joint.

PLATE 22–8. Correction of Hammer Toe by Resection and Arthrodesis of Proximal Interphalangeal Joint



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