SURGICAL CONSIDERATIONS FOR THE CORRECTION OF FLATFOOT DEFORMITY[1]

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The author states that three things must be considered in treating flatfoot: 1) the need of a more accurate method of defining and locating the deformity and treating it accordingly; 2) the need to apply or develop procedures that not only will yield a "good looking arch," but also a functional asymptomatic foot; and 3) the need to determine the etiologic factors causing the deformity. Are they neuromuscular imbalance, are they congenital, or are they compensatory, producing the subtalar and midtarsal joint pathology?

Various approaches to the treatment of flatfoot deformity are discussed.

As podiatrists, we are confronted with a variety of foot deformities which require our attention. Perhaps the most perplexing deformity we see is flatfoot which is perplexing, not only from the point of view of diagnosis, but the treatment is also perplexing. In the past, podiatry, as a profession, tended to treat this deformity in the child and adult on a conservative basis with a myriad of orthotic devices, and with a relative degree of success. The recent advancement of the profession in the area of biomechanics and surgery has placed an emphasis on surgical intervention which hereto did not exist for the treatment of the flatfoot. Our awareness, as a profession, of the serious consequences of inadequate, inappropriate, or lack of treatment of this deformity has stimulated our interest in the area of surgical treatment of this condition.

In the past the medical profession took a relatively unsophisticated approach to the surgical treatment of flatfoot. Little attention, if any, was paid to the pathomechanics of the deformity, and attention was given only to restoring the "normal" contour of the longitudinal arch. Paralysis of muscle groups, loss of ligaments, and structural faults were all given as primary causes of the deformity. A biomechanical approach diagnosing and treating the deformity could be found only infrequently.

Clinical Appearance and Classification of Flatfoot

The literature is filled with clinical descriptions of flatfoot, most of which are somewhat vague and nonspecific. Terms such as calcaneovalgus, convex pes planus, pes planus, congenital vertical talus and rocker bottom flatfoot have been used. All describe a foot which lacks a longitudinal arch, is convexed on its medial side and concave on its lateral side. In essence, we are referring to a subluxatory change occurring at the subtalar and midtarsal joints. The components of a flatfoot deformity are: 1) valgus rotated calcaneus; 2) equinus calcaneus (tight heel cord and low calcaneal inclination angle); 3) anterior and plantar displacement of the talus (obliteration of the sinus tarsi on lateral x-ray and increase of more than 30 degrees in the talar declination angle); 4) adduction of the talus (increase in Kite's angle of more than 35 degrees on the dorsoplantar x-ray); 5) subluxation at Chopart's joint (midtarsal joint) —abduction and dorsiflexion of the forefoot (the navicular may articulate with the dorsal aspect of the talus) (1).

The flatfoot may be flexible or rigid, it may contain all or some of the components of the deformity in varying degrees and this will determine the nature and severity of the deformity. The foot is in a position of excessive pronation of the subtalar joint (even to a point of subluxation), and the forefoot may be extremely hypermobile as a result of unlocking of the subtalar joint (2). For example, the calcaneovalgus foot is relatively flexible with the foot being dorsiflexed on the leg. There are soft tissue contractures, but the tendo achillis is not tight. The calcaneus is in mild valgus (but not equinus) and the talus is only moderately adducted and plantarflexed as opposed to the "convex pes planus foot" in which the deformity is of greater severity. There is marked equinus and valgus of the calcaneus, the tendo achillis is tight, and there is a marked degree of talar adduction and plantarflexion (vertical talus and subluxation of the forefoot on the rear foot at Chopart's joint in a dorsiflexion abduction direction).

Thus, we see that the nature of deformity is dictated primarily by the degree of subluxation of

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the subtalar and midtarsal joints. These two joints, as pointed out by Elftman (2), do not have independent function. It is the relative position of the subtalar joint in stance that dictates the function of the midtarsal joint and the forefoot. When the subtalar joint is pronated (in flatfeet it may be pronated beyond its normal range), the axes of the midtarsal joint, namely the oblique axis of the talonavicular joint and the longitudinal axis of the calcaneocuboid joint, are more parallel to each other, allowing for greater range of motion of the forefoot in an abductory inversion direction. This results in severe subluxation of the midtarsal joint in some flatfeet. When the subtalar joint is in a neutral or supinated position, these axes are more oblique to one another and thus Chopart's joint and the forefoot are stable.

It is a well accepted premise that the foot must function as both a mobile adaptor and a rigid lever in the stance phase of gait to dissipate shock and translational rotations of the lower extremity and support the body structure in propulsion. This is accomplished by pronation and supination of the subtalar joint and the locking and unlocking of the midtarsal joint. In the foot, the formation of the so-called arch is controlled by supination of the subtalar joint and locking of the midtarsal joint after the first 20% of the stance phase of gait. In the flatfoot, this usually does not occur as the subtalar joint is pronated and the midtarsal joint is unlocked throughout the stance phase of gait. The forefoot is hypermobile and the foot cannot function as a rigid lever. The midtarsal joint may be partially or completely subluxed. Clinically, the patient has an apropulsive gait, functions with an excessively flexed knee, and may develop forefoot and rear foot symptomatology such as hallux abducto valgus, plantar keratomas, and subtalar and midtarsal joint arthritis.

In essence, from a clinical and functional point of view, flatfoot pathology is a subtalar joint "lesion" (3). Its clinical appearance and function are centered around this joint. Although Miller, Giannestras, Jack, Hoke, and others have reported that a sagittal plane sag or breach at the navicular cuneiform or cuneiform first metatarsal joints are sites of a primary lesion pathology (4), this has not proved to be true in most cases.

In describing the deformity and classifying it, it is important to identify the location and nature of the deformity. Is the deformity primarily 1) made up of a plantarflexed talus associated with dorsal subluxation of Chopart's joint? 2) made up of an everted (valgus) calcaneus associated with equinus of the calcaneus? 3) made up of adduction of the talus associated with abduction and subluxation of the forefoot, and is the deformity 4) flexible—completely reducible? semiflexible—partially reducible? or rigid—nonreducible? (associated with subluxatory changes of the subtalar and midtarsal joints).

Terms such as calcaneovalgus and convex pes planus usually are unsophisticated attempts at describing the clinical appearance of the deformity and frequently fall short of defining it. They tend to force the clinician into grouping the deformity, rather than evaluating it on its individual characteristics. If we are going to treat the deformity, we must accurately define it.

Etiology

The etiology of flatfoot may be divided into three categories; 1) neuromuscular imbalance, 2) congenital and 3) compensatory.

A. Neuromuscular. Muscle imbalance is created by spasticity or atrophy. Either can cause a flatfoot deformity. The most common neuromuscular disease associated with flatfoot deformity is poliomyelitis. Although Grice believed that atrophy of the anterior tibial and posterior tibial muscles would cause such a deformity, he found that spasticity of the peroneals and gastrocnemius would cause an equino valgus flatfoot (5).

As with any deformity associated with neuromuscular imbalance, disregard for the underlying problem will result in recurrence. Spastic muscles should be weakened so as not to create further deformity, or phasic transfers should be performed. Atrophic muscles should be assisted or replaced by muscle transfers. In phase transfers are preferable, but out of phase transfers have also been successful (6).

B. Congenital. Stiendler and others (7) have alluded to the fact that flatfoot deformities may be associated with abnormal intrauterine position or abnormal tilt of the sustentaculum tali causing inherent instability of the joint. Harris and Beath (8) noted that the anterior facet on the calcaneus was absent on those flatfeet that they dissected and saw it as a possible congenital cause of the deformity.

C. Compensatory. States of muscle contracture associated with neuromuscular disease may produce abnormal joint position, which in time may produce structural changes in the bones of the foot. Patients may also have congenital defects in the foot structure which may produce the deformity. However, the most overlooked cause of the flatfoot deformity is probably the functional compensation which occurs in the foot and leads to the deformity. We are aware of the fact that anything which causes the foot to function maximally pronated past the vertical throughout the stance phase of gait, whether it comes from above the foot (antetorsion, genu valgum, retroversion) or in the foot (gastrocnemius-soleus equinus, forefoot varus), will cause the bony structure of the foot to

adapt in a "flatfoot" attitude, especially in the young patient. The most common cause of compensatory flatfoot noted by Root (9) and others is gastrocnemius-soleus equinus—a state in which, because of inherent shortness of the gastrocnemius-soleus complex, the foot cannot get the needed 10 degrees of dorsiflexion in relationship to the leg in the mid-stance phase of gait. As a result, the subtalar joint can become pronated and the midtarsal joint unlocked at a time when the subtalar joint should be supinating and the midtarsal joint locked. The additional dorsiflexion may then be gained through subluxatory changes at the midtarsal joint and produce the so-called rocker bottom flatfoot.

Although a "tight" gastrocnemius-soleus is a common finding associated with a flatfoot deformity, it may not be the etiologic entity. As a result of the relative equinus and valgus attitudes in the flatfoot, an adaptive shortening of the tendon may occur. Thus, it is only the congenital short tendo achillis that causes compensatory flatfoot. The "tight" tendo achillis is an associated finding. This fact has its obvious implications in the treatment of the flatfoot (10).

Many operative procedures are designed to correct flatfoot deformities, but one must be completely aware of the possible functional compensatory etiology. If the factors causing excessive pronation, as in gastrocnemius-soleus equinus, are not controlled or eliminated, the procedure will be doomed to eventual failure.

Note: The peroneal spastic flatfoot resulting from tarsal coalitions or subtalar arthritis is a distinct entity and thus the author feels that its discussion is not within the realm of this paper.

III. Treatment

After determining the location and degree of the deformity, it is necessary to decide how and when to treat the deformity. One should keep in mind that the goal of treatment is not necessarily to obtain a foot with a "good arch," but one that functions efficiently and asymptomatically. In the young patient, one should attempt to stop the progression of deformity by preventing bony adaptive changes from occurring.

In the severely "flattened foot" dislocations of the subtalar and midtarsal joints are present. As the foot grows, adaptive changes of the bone occur making the deformity more difficult to treat in later years. In the young patient one should attempt to reduce the dislocation when present and, thus, through readaptation, give the foot a better chance of normal development.

Casting. Giannestras advocates casting to correct the components of the deformity in the infant (11). First the valgus heel and subluxations at Chopart's joint are corrected by casting. The foot is casted in maximum equinus, heel varus and forefoot adductus. Once the calcaneal deformity and midtarsal deformity have been corrected, an attempt is made to correct the equinus deformity. The casting period is approximately 9 weeks. Casting has a high degree of success in extremely flexible deformities. The patients are followed up by orthotic control therapy. (See Giannestras' text for the specific casting procedure.) The more rigid and severe deformities (such as the so-called "convex pes planus" or "vertical talus") do not respond nearly as well to casting. This does not mean that the procedure should be abandoned in these cases. Frequently, contractures of the skin and extensor and peroneal tendons may be relieved with casting and a degree of reduction of deformity may be obtained, making further treatment less involved and easier to accomplish.

Open Reduction of the Deformity. In feet that are resistant to casting, whether flexible or rigid, open reduction is advocated by many clinicians (12). We use a procedure described by Tachdjian (13) titled "plication and reefing of the talonavicular joint capsule and shortening and transplantation of the posterior tibial tendon and pinning of the talonavicular joint."

Procedure. A curvilinear incision approximately 8.0 cm. in length is made on the medial aspect of the foot. The incision commences approximately 0.5 cm. below the distal tip of the tibial malleolus and runs anteriorly, terminating at the level of the first cuneiform. The small vessels are clamped and ligated. Exposure to the posterior tibial tendon is obtained. The sheath is incised with an incision paralleling the skin incision. The posterior tibial tendon is dissected free and reflected out of the surgical site. A T-incision is made into the talonavicular joint with the vertical portion of the T in the joint space and the horizontal portion of the T bisecting the talus neck and head. The incision creates a dorsal and plantar flap which are undermined and reflected off the talus. The forefoot is forcibly abducted and the talar head is delivered into the surgical site. A Kirschner wire is drilled through the center of the head of the talus and exits from the foot at the posterior aspect medial to the tendo achillis. The pin is drilled retrograde through the talus and the navicular, and exit is through the dorsal aspect of the foot at the level of the first metatarsal base. The pin is drilled retrograde after the deformity has been reduced. The talonavicular joint capsule is sutured in the following manner: The inferior flap is sutured dorsally, the superior flap is sutured inferiorly, and the flap on the navicular is sutured over the two previous flaps producing a strong plication and reefing of the talonavicular joint capsule. The posterior tibial

tendon is reinserted and sewed into the periosteum at the medial inferior aspect of the first cuneiform.

The spring ligament which has also been incised is now reinforced into its insertion into the sustentaculum tali with several simple interrupted sutures of 2-0 Dexon (Davis and Geck, Pearl River, N. Y.) All capsular closure and tendon transplant work was done with 2-0 Dexon. The sheath of the posterior tibial tendon is placed in apposition and approximated with several simple interrupted sutures of 3-0 Dexon, the subcutaneous tissue is placed in apposition and maintained with several interrupted sutures of 3-0 Dexon, and the skin is placed in apposition and maintained with several interrupted sutures of 5-0 nylon.

Xylocaine (Astra Pharmaceutical Products, Inc., Worcester, Mass.) and Decadron (Merck Sharp & Dohme, West Point, Pa.) are instilled into the surgical sites and a dry sterile dressing is applied along with an above-the-knee cast. The above-the-knee cast is changed at the end of 6 weeks during which time no weightbearing is allowed. The sutures are removed at this time along with the Kirschner wire. A below-the-knee cast is then applied for an additional 6 weeks. After removal of the cast, guarded walking is allowed and the patient is followed-up with orthotic control therapy.

The procedure to date has been performed on two patients, four feet. Postoperative results immediately show excellent reduction of the deformity. This reduction persists for 6 to 8 months, and in each case the patient experienced a popping and then swelling of the medial side of the foot, followed by 75% loss of correction on the follow-up roentgenograms. Both patients were 4½ years of age, and although good correction was obtained postoperatively, it was not lasting. The reason for failure, we believe, is the inability of the capsuloplasty to maintain the reduction of the deformity. We feel that the procedure should be performed in patients under the age of 3 years, because the growth and weight of the patient are less, giving a better prognosis for the procedure.

Open Reduction with Tendon Transfers. Many authors have advocated tendon transfers to supplement open reduction of the talonavicular joint and subtalar joints. Osmond-Clarke (14) supported the idea of transferring the peroneals or lateral toe extensors into the head of the talus to maintain reduction. The author has had no experience with this procedure, but feels that it should not be performed. Tendon transfers should be performed only as associated procedures when a neuromuscular problem exists in which atrophy and spasticity are present. Then the principles outlined previously should be applied for tendon transfer or replacement. If no neuromuscular problem exists, it is feared that we are creating states of muscle imbalance which will probably intercede to cause further deformity as the foot matures.

Calcaneal Osteotomies for Flatfeet. Gleich was the first to utilize calcaneal osteotomies for flatfeet. The procedure has recently been popularized by Silver and his associates (15). The intent of the procedure is to realign the valgus attitude of the calcaneus and change the weight stress from inside the foot to the medial tubercle or outside the foot, correcting the excessive pronation without the necessity of arthrodesis and thus maintaining function. In the flatfoot the subtalar joint usually functions in a position of maximum pronation. There is no evidence to support the fact that changing the weight stress through the calcaneus will cause the subtalar joint to function differently. The foot, however, is a more stable unit. Unlocking of the midtarsal joint is less likely to occur and subluxatory changes of the foot which may have progressed with the calcaneus everted have been prevented.

Some other considerations must be weighed. The function of the subtalar joint is still in maximum pronation which may result in the onset of arthritic degeneration. Inadvertent overcorrection can lead to an inappropriate weight-bearing foot, with the patient unable to get the medial side of his foot to the ground (uncompensated subtalar varus). Also, he may have a tendency toward lateral ankle sprains. The procedure does have merit, however, and offers a viable alternative to the consequences of severe flatfoot deformity.

Opening Varusing Osteotomy of the Calcaneus with Tibial Bone Graft. We use the procedure under limited circumstances—only when the patient has a "true" calcaneovalgus deformity, i.e., when the posterior tubercle of the calcaneus is in a valgus rotation in relationship to the posterior facet of the calcaneus (Fig. 1).

A curvilinear incision approximately 8.0 cm. in length is made on the lateral aspect of the heel. The incision commences approximately 2.0 cm. above the fibular malleolus and posterior to the peroneal tendons and curves around the inferior aspect of the fibular malleolus and terminates at the anterolateral aspect of the calcaneus. The incision is deepened by blunt and sharp dissection, exposing the sural nerve and short saphenous vein. The above structures are reflected plantarly out of the surgical site, exposing the calcaneofibular ligament which is incised with an incision paralleling the skin incision. The calcaneofibular ligament and the periosteum of the calcaneus are elevated and reflected both dorsally and inferiorly exposing the lateral aspect of the calcaneus.

An osteotomy is performed on the lateral aspect of the calcaneus paralleling the skin incision just inferior to the peroneal tendons, running in the same direction as the peroneal tendons. A bone graft is now secured. An incision approximately 6.0 cm. in length is made on the anteromedial aspect of the proximal one-third of the tibia of the leg. The incision is deepened by blunt and sharp

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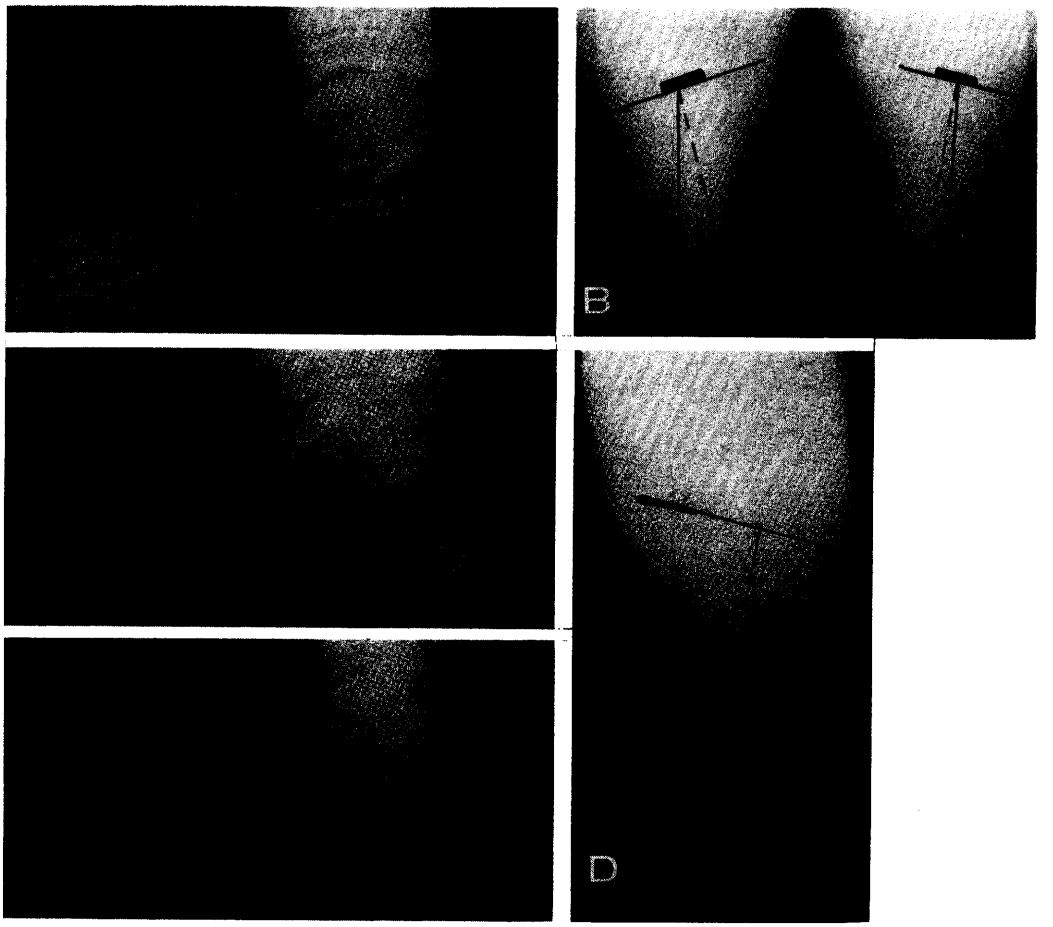


Figure 1. Varusing osteotomy of the calcaneus. A, preoperative lateral x-ray. B, preoperative tangential view of the calcaneus demonstrating a true valgus torque to the posterior tubercle of the calcaneus. C, postoperative x-ray—varusing osteotomy of the calcaneus. (Note bone graft in place.) D, postoperative tangenital view of varusing osteotomy of the calcaneus. E, postoperative lateral x-ray 7 months postoperatively.

dissection, exposing the periosteum of the proximal one-third of the tibia. The periosteum is incised with a linear incision paralleling the skin incision and reflected both medially and laterally, exposing the anterior medial aspect of the tibia. A section of bone approximately 4.0 cm. in length and 2.0 cm. in width is excised from the tibia at that level. The periosteum is closed with several interrupted sutures of 2-0 Dexon. The subcutaneous tissue is placed in apposition and maintained with several simple interrupted sutures of 2-0 Dexon. The skin is placed in apposition and maintained with several simple interrupted sutures of 4-0 nylon.

The bone graft is cut in half, when both feet are going to be operated upon, and one piece is placed aside for the osteotomy on the second foot. The bone graft should be placed in the calcaneus so that it makes contact with the cortical bone of the calcaneus and not deep within the soft cancellous bone of the heel at the osteotomy site. This will prevent closure of the osteotomy site because of impaction of the bone graft on the cortical bone. Placing the bone graft in the calcaneus causes the heel to be rotated in a varus direction. The position of the calcaneus should not be overcorrected.

The periosteum and calcaneofibular ligament are now closed over the osteotomy site, using several interrupted sutures of 2-0 Dexon. The subcutaneous tissue is closed by using several interrupted sutures of 2-0 Dexon. The skin is placed in apposition and maintained with several interrupted sutures of 4-0 nylon. Dry sterile dressings are placed on the wounds, Xylocaine and Decadron are instilled into the surgical sites and an above-the-knee cast is applied. The above-the-knee cast is changed at the end of 6-weeks, at which time all sutures are removed and a below-the-knee cast is applied. The patient is allowed supported ambulation at this particular point and the cast is worn for an additional 6 weeks. At the end of a 12-week period the cast is removed, and orthotic control therapy is utilized.

Pronation Blocking Procedures (Chambers and

LeLievre). The author has no experience with these procedures, but believes the concept behind them to be excellent. Chambers and LeLievre both desire to establish a normal joint relationship while maintaining subtalar joint function, and thus avoid arthrodesing procedures in the flatfoot. Chambers elevated a wedge of bone from the sinus tarsi in front of the posterior facet of the calcaneus. The osteotomy included part of the posterior facet. This prevented anterior displacement of the talus on the calcaneus and prevented pronation (16). LeLievre used a bone graft inserted into the sinus tarsi to prevent its closing with pronation and thus limited motion in this direction (17).

Both procedures seemed to be aimed at preserving function. They seem reasonable provided, as with any procedure designed to reduce the deformity at the subtalar joint, the deformity is primarily one of subtalar joint instability, or that any forces causing excessive pronation (gastrocnemius-soleus equinus) can be controlled. If the deformity is not of primary subtalar joint instability, and the excessive pronatory forces cannot be controlled (as in an ankle equinus), then this, as well as any other procedure, will fail to maintain the reduction of the deformity.

We have used a modification of LeLievre's procedure, substituting a Silastic (Dow Corning Co., Midland, Mich.) implant for a bone graft. The lateral arthroereisis is performed as follows: A curvilinear incision, approximately 5.0 cm. in length, is made on the dorsolateral aspect of the foot. The incision commences at the lateral border of the extensor digitorum tendons and terminates at the medial border of the peroneal tendons and is located over the sinus tarsi of the foot. The incision is deepened by blunt and sharp dissection, exposing the fibrofatty pad that occupies the sinus tarsi. The fibrofatty pad is reflected distally, exposing the floor of the sinus tarsi. The floor of the sinus tarsi is evacuated of its contents and a Silastic-rubber implant prepared from a Silastic block is inserted into the sinus tarsi. The implant is cone-shaped, approximately 2.5 cm. in length and 1.0 cm. in diameter. The implant is placed in the sinus tarsi with the apex of the implant pointing medially and the base pointing laterally. With the implant inserted, it is noticed that excessive abnormal pronation is prevented. The fibrofatty plug is sutured over the implant, utilizing several interrupted sutures of 3-0 Dexon. The skin is placed in apposition and maintained with several simple interrupted sutures of 5-0 nylon. Xylocaine and Decadron are instilled in the surgical site and a below-the-knee cast is applied. The below-the-knee cast is removed at the end of 3 weeks. All sutures are removed and guarded ambulation is allowed. The patient is followed-up with orthotic control therapy.

We believe that in the growing foot realigning the articular surfaces of the subtalar joint and midtarsal joint will give inherent stability to the foot as a result of the readaptive changes occurring at these joints (provided, of course, that if excessive pronatory forces are present, they are eliminated or controlled). The use of the implant may then be unnecessary and it can be removed once this stability is gained with growth.

It has been noted by Tachdjian (18) that limiting subtalar motion with the above procedure and the procedures of Chambers and LeLievre may cause arthritic degeneration of the subtalar joint, pain and/or peroneal spasm. We believe that this is a possibility only if the foot does not have the ability to readapt to the realignment of the subtalar and midtarsal joints. That is, the patient should be below the bone age of 10 years, when full growth of the bony structures of the foot has not occurred. This foot has the optimal chance for readaptation. The deformity, of course, must be flexible in nature. Reduction of the deformity must be performed off weightbearing.

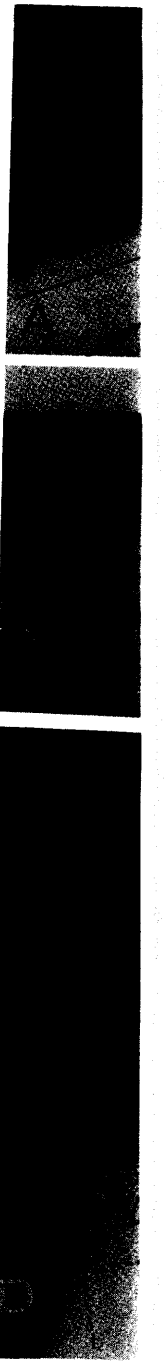
To date, we have performed this procedure on two patients, along with a tendo Achillis lengthening to correct congenital gastrocnemius-soleus equinus. Both patients were between the age of 5 and 7 years. The first procedure was performed 1 year ago. We hope to remove the implant 2 to 3 years postoperatively (Fig. 2).

The procedure described above is a relatively innocuous procedure. No bone flaps or grafts are necessary. Inadvertent subtalar fusion that has occurred with the Chambers or LeLievre procedures is avoided. The occurrence of subtalar arthritis from the creation of joint incongruity by elevating the posterior facet of the calcaneus in the Chambers procedure is also avoided. As a result, the opportunity for optimum functional outcome is good because of the factors mentioned and the limited necessity for surgical exposure and trauma.

We believe the procedure can be utilized even in situations in which subtalar joint pathology is not primary but is secondary. In congenital gastrocnemius-soleus equinus, if secondary changes of the subtalar joint and midtarsal joint are present, in addition to performing a tendo achillis lengthening, it may be necessary to maintain and reduce these subluxations until readaptation can occur once the pronatory force has been eliminated.

Sustentaculum Tali Procedure. This procedure as described by Selakovich (19) has as its basic premise that the flatfoot deformity which the procedure is designed to correct is one in which there is a congenital anomaly of the sustentaculum tali. As a result of this, there is no support for the talar head which falls in adduction and plantarflexion. The sustentaculum tali is levered forward and upward by means of a bone graft placed behind the medial facet of the subtalar joint on the calcaneus. Selakovich performed the procedure on those feet that are flexible and demonstrated this anomaly.

This author has no experience with the proce-



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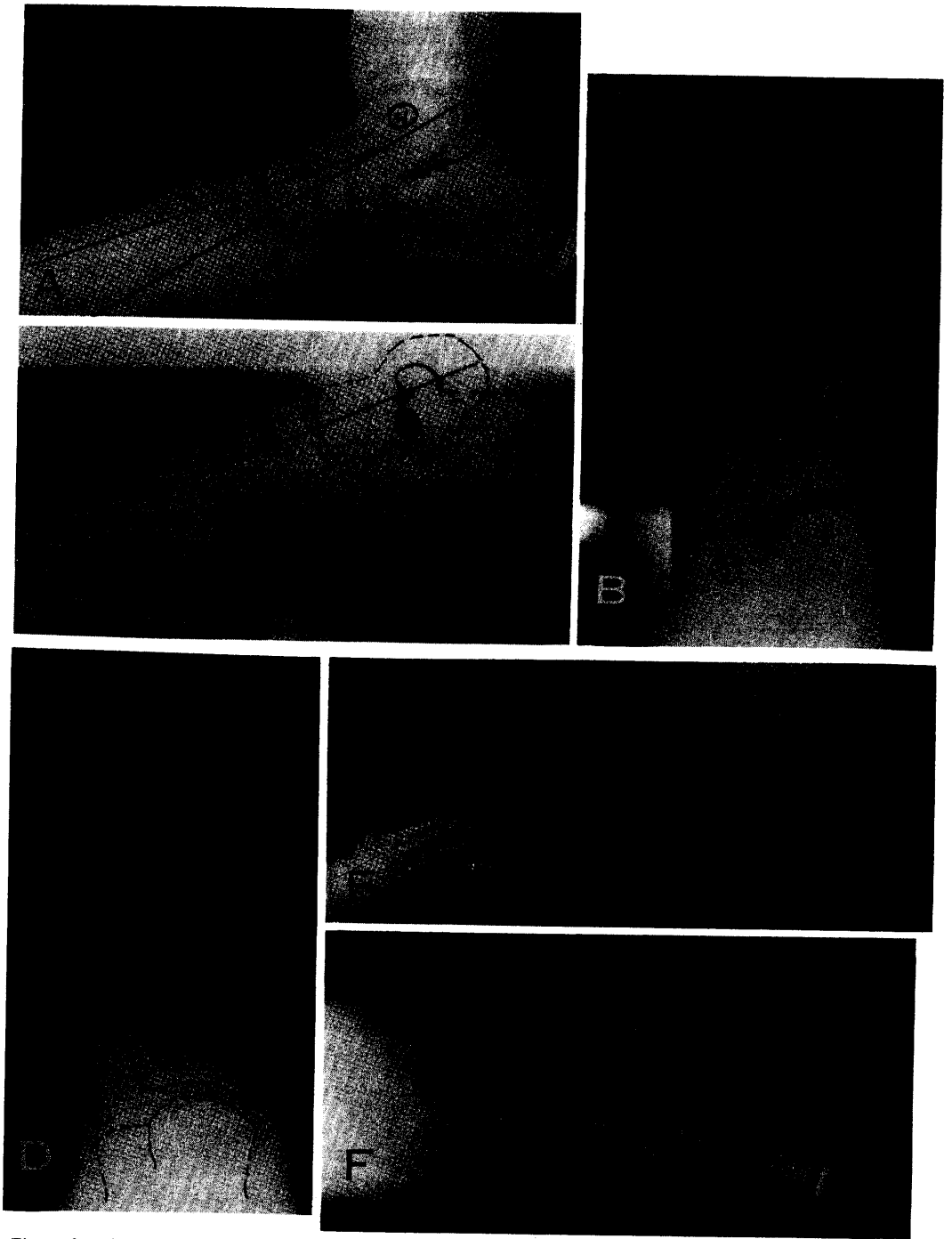

Figure 2A–C. Operative x-rays for lateral arthroereisis of the subtalar joint. A, preoperative lateral x-ray. (Note changes on the lateral x-ray: obliteration of the sinus tarsi, creation of a pseudo sinus tarsi, anterior broken cyma line, low calcaneal inclination angle, and increase in talar declination angle.) B, dorsoplantar preoperative roentgenogram. (Note break in the dorsal cyma line and subluxatory changes at Chopart's joint. Also note that less than one-half of the talus articulates with the navicular.) C, postoperative lateral arthroeresis of the subtalar joint. (Note realignment and reduction of the talar declination angle, opening of the sinus tarsi, increase in calcaneal inclination angle and placement of the position of the Silastic implant.)

Figure 2D–F. D, postoperative dorsal plantar x-ray. (Note realignment of the talus on the calcaneus—reduction of Kites angle. Also note that greater than 75% of the talus now articulates with the navicular. E and F, 1 year postoperatively. (Note maintenance of reduction of deformity on lateral and dorsoplantar views.)

dure, but believes that the basic premise of the procedure is a good one. Unfortunately, there are other important considerations which make this procedure unattractive. For one, the procedure, like the Lenox-Baker (20) and Chambers procedures, causes incongruity of the articular facets of the subtalar joints. This leads to the early development of subtalar arthritic degeneration and may, in some cases, lead to inadvertent fusion of the joint.

Miller, Hoke, Young Procedures. These procedures were principally designed to correct the so-called sagittal plane breech as described by Giannestras (4). Associated with some flatfoot deformities one may find a plantar sag at the navicular cuneiform or cuneiform first metatarsal joint. The above-named procedures attempt to plantarflex the forefoot on the rear foot at the location of the "sag" and create a longitudinal arch: the Miller procedure, by periosteal flap and navicular cuneiform fusion; the Hoke, by fusion of the navicular and all three cuneiform bones; and the Young, by tendinous sling of the anterior tibial to the navicular.

In considering these procedures, I must concur with Tachdjian (21) who points out that it is usually the pathology of the subtalar and midtarsal joints that produces functional impairment and symptomatology. When a sagittal plane breech occurs with a valgus heel, it appears not to be the primary pathology, and it is difficult to see how correction of the sagittal plane breech would correct the hindfoot pathology. In the author's experience, when the sagittal plane breech is located as an isolated entity with no hindfoot pathology, the foot deformity is not severe enough to warrant surgical intervention. As a result, I don't believe such procedures have a place in the surgical correction of the flatfoot.

Fusions

Grice Procedure—EASTA (Extra-Articular Subtalar Arthrodesis)

Grice designed this procedure principally for the correction of spastic flatfoot deformities. The procedure produces extra-articular fusion of the subtalar joint while permitting growth to occur because the articular surfaces are not removed to achieve fusion (22). Principally, the problem of triple arthrodesis in the growing foot was a decline in growth after triple arthrodesis.

It was the original intent of Grice to complete the triple arthrodesis of the foot later by fusing the midtarsal joints when the foot had reached skeletal maturity, the advantage being that less bone would have to be removed to obtain proper alignment of the foot because the deformity had been reduced with the extra-articular subtalar arthrodesis. Grice noted that solid union of the subtalar joint occurred and fusion at the midtarsal joint was unnecessary because the feet were asymptomatic. As a result, the procedure has been used in the treatment of non-neuropathic hypermobile flatfeet (4). We have used the procedure in the treatment of atrophic or spastic flatfeet where stability is desired and in severe cases of hypermobile flatfeet that have been resistant to other modes of therapy and are symptomatic.

Operative Technique. A curvilinear incision approximately 8.0 cm. in length is made over the sinus tarsi of the foot. The incision commences at the lateral border of the extensor tendons and terminates at the medial border of the peroneal tendons and crosses the sinus tarsi. The incision is deepened by blunt and sharp dissection, exposing the fibrofatty plug that occupies the sinus tarsi. The plug is excised *in toto*. The origin of the extensor brevis muscle is dissected off the floor of the calcaneus and reflected distally out of the sinus tarsi. The talocalcaneal ligament is identified and resected *in toto*. A trough approximately 1/4 inch by 3/16 inch is created on the inferior surface of the body of the talus. A similar trough is created on the superior surface of the calcaneus slightly anterior to the one created on the talus. The troughs are created to accept a fibular bone graft to be inserted into the sinus tarsi approximately perpendicular to the axis of motion of the subtalar joint.

A fibular bone graft is now secured. An incision is made on the lateral aspect of the leg over the bony prominence of the fibula. The incision, approximately 6.0 cm. in length, is made at the level of the junction of the distal and middle one-thirds of the fibula and is deepened by blunt and sharp dissection. The deep fascia is incised with an incision paralleling the skin incision. The muscle origin of the peroneus brevis is reflected off the fibula with its periosteum.

A screw is placed through the distal end of the fibula at the junction of the distal one-third and middle one-third of the fibula to stabilize the distal end of the fibula to the tibia. The screw transverses the fibula and is placed into the tibia. A bone graft, approximately 4 inches long, is removed from the fibula. The periosteum is closed over the defect in a tubular fashion to insure regeneration of the fibular defect. The deep fascia is closed with several simple interrupted sutures of 5-0 Dexon. The subcutaneous tissue is closed with a running mattress suture of 5-0 Dexon, and the skin is placed in apposition and maintained with several simple interrupted sutures of 5-0 nylon.

The fibular graft is cut transversely into two grafts approximately 2 inches long when the procedure is to be performed bilaterally. The foot is maximally inverted, opening the sinus tarsi. The fibular graft is placed in the troughs and

the foot is everted to lock the graft into position. The subcutaneous tissue is placed in apposition and maintained with several simple interrupted sutures of 5-0 Dexon. The skin is closed and maintained with several simple interrupted sutures of 5-0 nylon. Xylocaine and Decadron are instilled into the surgical site and a dry sterile dressing is applied. A non-ambulatory above-the-knee cast is applied and left on for a period of 6 weeks, at which time the cast is changed and all sutures removed and the wounds inspected. A below-the-knee cast is then applied which is removed in 6 weeks and the patient is allowed to walk with support. Orthotic control therapy is then instituted.

Care must be taken not to overcorrect the subtalar joint. One should have the heel vertical or in slight valgus. The graft should be placed perpendicular to the axis of motion at the subtalar joint to block its motion and prevent fatigue fractures of the graft. One must also note the presence of a valgus ankle mortise before surgery so as not to

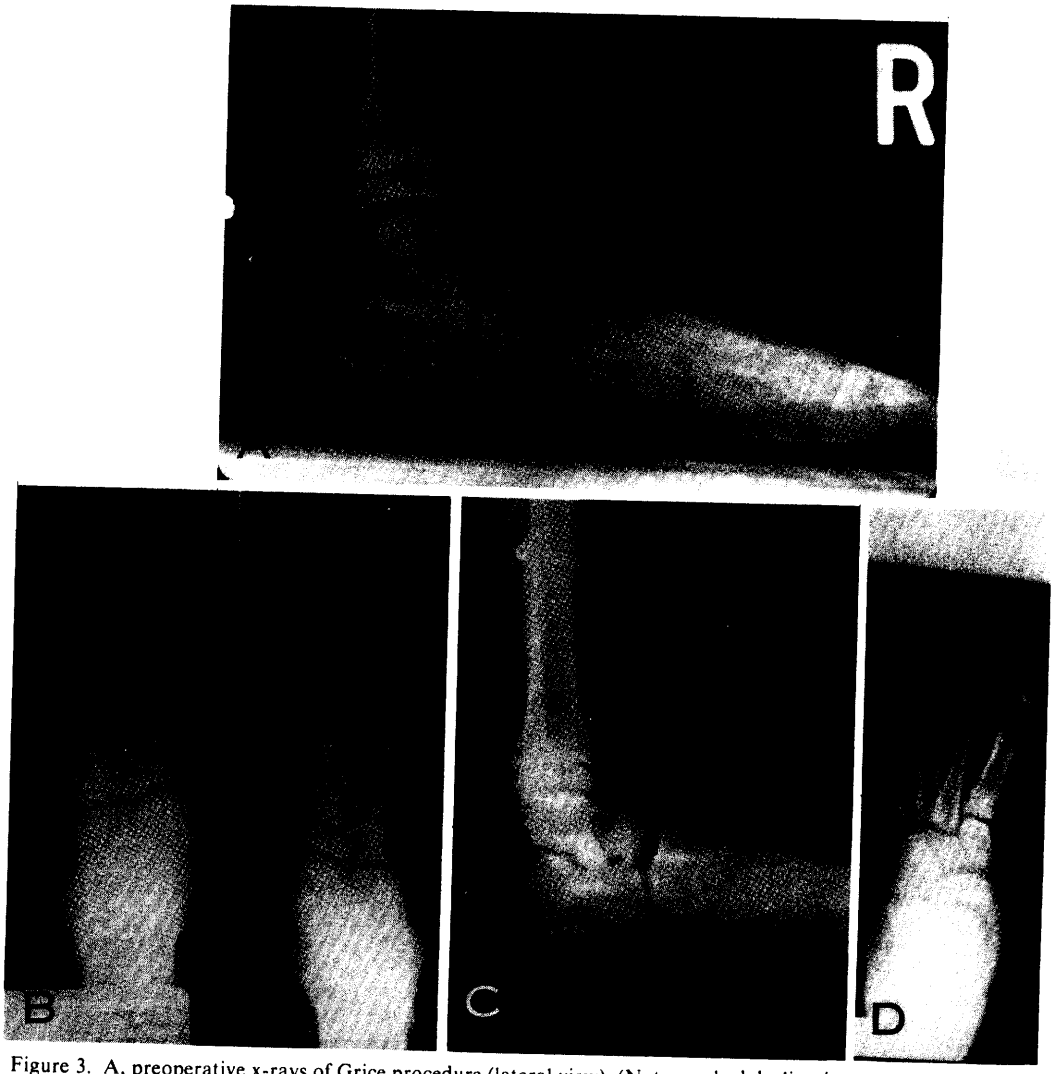

Figure 3. A, preoperative x-rays of Grice procedure (lateral view). (Note marked declination angle of the talus and subluxatory changes occurring at the subtalar and midtarsal joints.) This patient was previously treated with soft tissue procedures for the correction of flatfoot which had failed. B, preoperative dorsoplantar x-ray of the foot. (Note marked dislocation of Chopart's joint. Also note that less than 50% of the talus articulates with the navicular.) C, lateral x-ray after Grice procedure. (Note marked and pronounced reduction of the deformity with the extra-articular arthrodesis, placement of the bone graft approximately perpendicular to the axis of motion of the subtalar joint, and placement of the transfixing screw to stabilize the lower one-third of the fibula to tibia.) D, dorsoplantar view after Grice procedure. (Note placement of the graft on the dorsoplantar view and marked reduction of the dislocation at Chopart's joint. Also note that greater than 75% of the talus now articulates with the navicular.)

make the mistake of overcorrecting the heel to compensate for it. Above all, the foot must be relatively flexible and capable of manipulation into the corrected position before surgery. The interosseous ligament may have to be severed in order to achieve this at the time of surgery.

Expert reduction of the deformity is possible with this procedure, but its functional implications are significant. The foot may look good but it is stiff, and the principal criteria with the procedure, as with most fusions, is that pain and instability outweigh foot function. Future fusion of the midtarsal joint may still be necessary (Fig. 3).

The procedure may also be used in the adult foot as a means of accomplishing one-third of the triple arthrodesis in the symptomatic flatfoot (subtalar fusion).

Triple Arthrodesis—for the Flatfoot

The triple arthrodesis is a salvage procedure and should be used as a last resort for instability and pain. The procedure is used in the adolescent and adult symptomatic flatfoot which cannot passively be reduced and is fixed. We use a Hoke procedure with two incisions to accomplish triple arthrodesis because the talus is usually plantarflexed and adducted and is difficult to approach through a lateral incision.

Tendo Achilles Lengthening

A word here is in order because of the functional implications. As mentioned earlier, if a congenital short tendo achillis is the etiologic factor causing subtalar pathology, any attempt at correction of the subtalar pathology will fail unless the force causing excessive pronation is eliminated. Fortunately, or unfortunately, in performing surgery on the flatfoot, many surgeons routinely lengthen the tendo achillis. Some do not lengthen it at all. Obviously, the tendo achillis must be lengthened if it is grossly shortened and produces an equinus gait, but also when it can be implicated as a cause of subtalar joint pronatory changes. This occurs in the more subtle functional or congenital gastrocnemius-soleus equinus deformities where a lack of 10 degrees of dorsiflexion is present. We prefer to use a percutaneous lengthening of the tendo achillis when the shortage is not gross. For spastic equinus, open "Z-plasty" is recommended.

Sgarlato, Root and others have been able to reverse the subluxatory changes of the subtalar joint by tendo achillis lengthening when the subtalar joint pathology is secondary to functional or congenital gastrocnemius-soleus equinus (23). This author has been unable to demonstrate roentgenographically this reverse post-tendo achillis lengthening for functional or congenital gastrocnemius-soleus equinus, although elimination of the progression of the deformity and orthotic control therapy have been made possible.

Conclusion

In considering the dilemma in treating flatfoot, three things are apparent: 1) we need a more accurate method of defining and locating the deformity and directing our therapy accordingly; 2) we need to apply or develop procedures that will not only yield a "good looking arch" but also a functional asymptomatic foot, and 3) we must determine the etiologic factors causing the deformity—are they neuromuscular imbalances, are they congenital, or are they compensatory, producing the subtalar and midtarsal joint pathology?

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