RELAPSING CLUBFOOT: CAUSES, PREVENTION, AND TREATMENT[1]

Ignacio V. Ponseti

INTRODUCTION

Regardless of the mode of treatment, the clubfoot has a strong tendency to relapse. Stiff, severe clubfeet and small calf sizes are more prone to relapse than less severe feet. Clubfeet in children with very loose ligaments tend not to relapse. Relapses are rare after four years of age.

Not all components of the clubfoot tend to relapse to the same degree. In most of our cases, forefoot correction is permanent without metatarsus adductus. The relapse of the cavus deformity is rare and usually mild. The most important relapses occur in the hindfoot, first in the equinus, and then in the heel varus. In some relapsed clubfeet, the heel varus is very severe, while in others it is mild. Rarely, the heel in equinus may go into valgus resulting in a calcaneovalgus deformity. This is a frequent occurrence in surgically treated clubfeet.

In our experience, most relapses develop gradually and may be difficult to recognize in the early stages. A relapse is detected when there is an appearance of a slight equinus and varus deformity of the heel, most often without increased adduction and cavus in the forefoot. When walking, the child tends to put more weight on the outside of the sole of the foot.

CAUSES

It is wrongly assumed that relapses occur because the deformity has not been completely corrected. Actually, relapses are caused by the same pathology that initiated the deformity. Therefore, when we understand the pathogenesis of the clubfoot, the causes of the relapse will become clear.

The clubfoot in otherwise normal children is a developmental anomaly originating after the third month of intrauterine life. It is induced by an unknown dysfunction in the posterior and medial aspects of the lower leg, ankle and foot. There is a slight decrease in size of the muscles, and an excess of collagen synthesis with retracting fibrosis in the medial and posterior tarsal ligaments, in the deep fascia, the tendo Achilles, and the posterior tibial tendon. These changes induce severe equinus, medial displacement of the navicular, heel varus and foot adduction.

The period of dysfunction causing the deformity starting in the middle third of pregnancy lasts to the third or fourth year of life. In mild cases, it may start in late fetal life, and remain active for only a few months after birth. In all cases, the resulting fibrosis is most pronounced from a few weeks preceding birth, to a few months after birth. This is the period when collagen accretion is greatest in tendons and ligaments of normal mammals and presumably also of man. The speed of growth of the foot decreases after the first year of life, diminishing greatly after five years.

Relapses appear to be related to the intensity of collagen synthesis as the foot grows. Thus, relapses occur swiftly in premature infants and more slowly in older infants. Relapses are less common and less severe in mild club feet with little fibrosis and in children with loose ligaments. They occur because the factors inducing the deformity are still active. Relapses are rare after four years of age, regardless of whether the deformity is fully or partially corrected.

The clubfoot is no different from other non-embryonic human deformities such as torticollis, scoliosis, or Dupuytren's contracture, in that it develops in normal individuals, and progresses for a limited time before becoming inactive. Torticollis usually develops within days after birth and increases for a few weeks. Idiopathic scoliosis starts in late childhood and increases throughout adolescence. Dupuytren's contracture develops at maturity and may be active for a few years. Clubfoot develops in the middle of pregnancy and is active during the first to fourth years of life. In torticollis, Dupuytren's contracture, and presumably in clubfoot, a localized temporary increase of collagen synthesis is a common pathologic feature.

With our technique, most congenital clubfeet in infants are corrected within four to six weeks. However, splinting for several months or years is indispensable to help prevent relapses. Since the main corrective force

Department of Orthopaedic Surgery
University of Iowa
Iowa City, Iowa, U.S.A.

of the varus and adduction of the clubfoot is abduction (external rotation) of the foot under the talus, a splint is needed to maintain the foot in the same degree of abduction as it was in the last plaster cast. This is best accomplished with the feet in well-fitted, open-toed high-top shoes with a well-molded heel attached in 70 degrees external rotation to a bar of about the length between the baby's shoulders. Unless the feet are splinted in firm external rotation, the pull of the retracting fibrosis in the ligaments of the medial aspect of the ankle and of the tibialis posterior and toe flexors is strong enough to cause a recurrence of the deformity in most feet.

The splints are worn full time for two to three months, and thereafter at night and naps for two to four years. The splint should maintain the foot in 70 degrees of abduction to prevent relapse of the varus deformity of the heel, of the adduction of the foot and the in-toeing. The ankle should be in dorsiflexion to prevent relapse of the equinus. This is accomplished by bending the splint with the convexity of the bar distally directed. A splint or strapping that cannot firmly maintain the foot in marked abduction without pronation is ineffectual. The added advantage of shoes attached to a bar, as opposed to a fixed splint, is that it allows motion of the feet, ankles and knees. Most babies feel uncomfortable for the first two to three days when trying to kick their legs alternately. Parents can easily teach their babies to kick both legs simultaneously. The splints are then well accepted.

In the first 20 years of my practice, relapses occurred in about half of the patients at ages ranging from ten months to five years, averaging two-and-one-half years. Usually, relapses were observed from two to four months after the splints were prematurely discarded at the families' own initiative, believing that the correction was stable. More recently, relapses have been less frequent because, for one thing, I have further overcorrected the deformity in the last plaster cast, to be certain that the calcaneus is fully abducted and its anterior joint surface is well under the head of the talus. Secondly, there has been greater awareness on the part of the parents regarding the importance of maintaining the night splints after correction for three to four years.

In recent years, I have treated 90 patients—52 of them initially seen from birth to three months of age, and 38 from three months to one year of age. Seventy percent of the patients had plaster casts or physical therapy elsewhere. Forty patients had been previously indicated for surgery by the initial treating physician. To my surprise, it was possible to successfully correct all these feet with manipulations, and four or five plaster casts, changed every five days. I performed percu-

taneous Achilles tenotomy in 84 percent of the patients. Eighty-eight percent of the patients were compliant with the use of the foot abduction splint. There were 14 relapses. The rate of relapse was seven percent in compliant patients, compared to 78 percent in non-compliant patients. Relapses were unrelated to age at presentation or to the number of casts required for correction.

TREATMENT

In general, the original correction may be recovered in four to six weeks with manipulations and plaster casts, changed every 14 days, holding the foot in marked abduction and as much dorsiflexion as possible at the ankle in the last cast. This treatment is followed by lengthening the tendo Achilles when dorsiflexion of the ankle is less than 15 degrees. A percutaneous tenotomy can be performed until one year of age. The last plaster cast is left on for three to four weeks. When the cast is removed, shoes attached in external rotation to a bar are worn at night and with naps, until the child is about four years old.

To prevent further relapses, the tendon of the tibialis anterior muscle is transferred to the third cuneiform in children over two-and-one-half years of age, if this muscle tends to strongly supinate the foot. Often this supination takes place when the medial navicular displacement is not fully corrected and the AP talocalcaneal angle is under 20 degrees. Transfer of the tibialis anterior tendon averts further relapses, maintains the correction of the heel varus, improves the antero-posterior talocalcaneal angle, and thus greatly reduces the need for medial release operations. The tibialis anterior tendon transfer is an easy operation and much less damaging to the foot than the release of the tarsal joints. Joint releases are needed when the deformity recurs in spite of the tibialis anterior transfer. The tibialis anterior tendon should never be split, so as to not lose its eversion power, nor should it be transferred to the fifth metatarsal or to the cuboid, since this would excessively evert the foot, causing severe foot pronation and heel valgus.

CONCLUSION

Since I developed this method of treating clubfoot 50 years ago, only an occasional posterior release operation of the ankle and subtalar joints has been necessary. In the 90 patients I treated in recent years, four patients required surgery: one posterior release of the ankle, and three anterior tibialis tendon transfers to the third cuneiform combined with a lengthening of the tendo Achilles.