Clubfoot:

Biomechanics and practical principles-based application of the Ponseti method

CONTENTS

Vincent S. Mosca, MD

Global HELP Organization

This is a new Global-HELP Organization-sponsored publication.

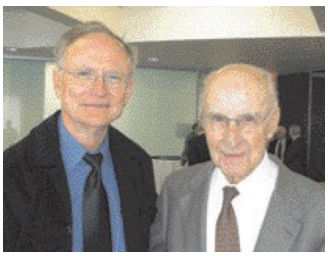
Between 2002 and 2009, Dr. Lynn Staheli, along with Dr. Ignacio Ponseti and several other collaborators, published the first three editions of a brief "how to" publication entitled Clubfoot: Ponseti Management. They were one- of-a kind publications intended to reach an international audience of health care providers who treat clubfeet. They were available in print and PDF format (global-HELP.org), translated into 35 languages, and distributed in over 200 countries.

This newly created monograph is based upon that monograph. More than an update or next edition, however, it is a completely reconsidered, single authored interpretation and presentation of the Ponseti method from a pediatric orthopedic foot and ankle surgeon with over 40 years' experience (including residency training) studying and treating clubfeet.

One major aim of this work is to provide the most current and in-depth explanation of the biomechanics of clubfoot deformity and deformity correction, including the most up-to-date terminology. It is the belief of this author that a true and in-depth understanding of the biomechanics by practitioners is critical to the success of Ponseti clubfoot management.

Lessons learned from over 35 years of personal experience in an academic clinical practice managing a large volume of clubfoot deformities is blended with the standard information about the Ponseti method that can be found elsewhere. I feel fortunate that, due to my age, I have a perspective on clubfoot management that few practicing orthopedic surgeons share. Having trained in the traditional operative approach to clubfoot management, I practiced that approach for 10 years. When the Ponseti method emerged from Iowa almost cataclysmically around 25 years ago, I was one of the first early adopters. I quickly perfected my technique and have practiced Dr. Ponseti's method, essentially without variation or major modification, for the last 25+ years. It is my early experiences with the unintended and undesirable consequences of clubfoot surgery that have kept me devoted to Dr. Ponseti's method. I hope that publications like this will encourage young orthopedic surgeons to stay true to the method and not, for apparent ease or expediency or ignorance of the past, return to the operative approach to clubfoot management.

I dedicate this monograph to the brilliant work of Dr. Ignacio Ponseti (1914-2009) and to my decades-long colleague, mentor, role model, and friend, Dr. Lynn Staheli (1933-2021), founder of Global- HELP. May they both rest in peace.



Drs. Staheli and Ponseti

Drs. Mosca and Ponseti

Contributor

Ignacio V. Ponseti, MD

Dr. Ponseti developed his method of clubfoot management more than 70 years ago and, since then, treated thousands of infants using his method. Dr. Ponseti passed away due to natural causes in 2009 at the age of 95. As Professor Emeritus in the Department of Orthopedics at the University of Iowa, he used his non-operative method to treat infant clubfeet up until the time of his death. He provided guidance throughout the production of the first three editions of the global-HELP.org publication Clubfoot: Ponseti Management and wrote the section on the Scientific Basis of Understanding and Management of Clubfoot. That section has been incorporated in this monograph.

CHAPTER 1

Clubfoot – the evolution of the Ponseti method

Circa 3000 – 2000 BC The earliest documentation of clubfoot was by the Ancient Egyptians in paintings on the walls of ancient tombs and pyramids.

1341-1323 BC King Tutankhamun (King Tut) lived only 18 years and was Pharaoh from age 8 years until his death. A statue of a diastrophic dwarf with clubfoot was found among thousands of artifacts in the multilayered, multimillion-dollar sarcophagus of the "Boy King". There is conflicting evidence that King Tut himself had a left clubfoot [Figure 1].

Circa 400 BC In ancient Greece, Hippocrates (the "Father of Medicine") was the 1st to describe clubfoot and its management. According to one translation of his writings: "Start treatment early, persevere unremittingly…as if moulding a wax model, you must bring to their natural position the parts which were abnormally displaced and contracted together, so rectifying them with your hands, and with the bandaging in like manner, as to bring them into their position, not by force, but gently; and the bandages are to be stitched so as to suit the position in which the limb is to be placed, for different modes of the deformity require different positions…the parts are to be secured with soft bandages in sufficient quantity, but not applied too tight; and the turns of the bandages should be in the same direction as the rectifying of the foot with the hands, so that the foot may appear to incline a little outwards." [Figure 2].

14th – 16th centuries The Mexican Aztecs used splints made from cactus leaves to correct clubfoot deformities.

17th century Pare and others corrected clubfoot deformities with repeated stretching using a turnbuckle apparatus.

18th century Cheseldon recommended correcting clubfoot deformities with repeated stretching and bandaging with linen dipped in egg whites and flour.

18th century Lorenz Heister (Frankfort) was the 1st to describe the subcutaneous tendo-Achilles tenotomy (subcut-TAT).

Early 19th century Antonio Scarpa used an apparatus with springs to reduce the "twisting of the scaphoid, os calcis, and cuboid around the astragalus" [Figure 3].

1823 Delpech (France) used the subcut-TAT for acquired clubfoot

1831 Louis Stromeyer (Germany) performed the 1st subcut-TAT in a deformed foot, presumably a clubfoot. He subsequently performed the procedure on WJ Little, from England, who had a tendo-Achilles contracture and foot deformity caused by poliomyelitis.

1837 WJ Little (England) is best known as being the 1st to identify spastic diplegia or so-called "Little's disease". He wrote his doctoral dissertation on tentotomy and popularized the procedure for use in children with CP and those with clubfoot.

1838 Guerin first to use plaster of Paris for clubfoot treatment.

1840s The operative era for clubfoot treatment began with the introduction of effective and relatively safe anesthesia. This was enhanced in 1867 by Lister (London) who introduced aseptic surgical technique, and further enhanced by the introduction of the Esmarch tourniquet in 1873.

1857 Solly described the first bone operation for clubfoot – partial cuboid resection.

1872 Lund described the talectomy for clubfoot.

Late 19th century Hugh Owen Thomas, a bone setter from Liverpool, England,

Figure 1. A. King Tutankhamun's gold burial mask. B. King Tut mummy.

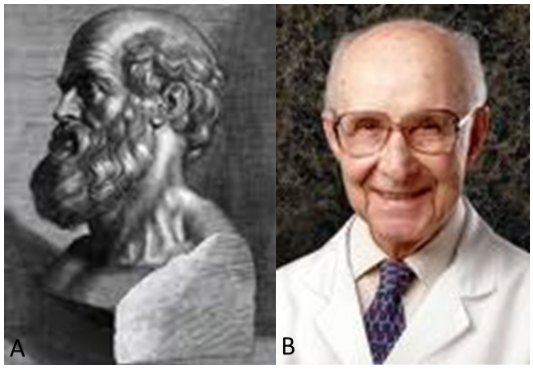


Figure 2. Remarkably similar approaches to clubfoot management by A. Hippocrates. B. Ignacio Ponseti.

Figure 3. Antonio Scarpa – originator of the acetabulum pedis concept (see page 10).

reversed course from the increasingly operative approach to clubfoot and introduced his eponymous wrench. A quick painful twist and no more clubfoot deformity. Fortunately, it didn't catch on [Figure 4].

1890 – 1920 More operations for clubfoot were proposed; primarily talar neck osteotomies.

1930 Hiram Kite (Atlanta) reported a very high success rate correcting clubfoot deformities with his method of serial manipulation and plaster cast immobilization. Most physicians and surgeons who attempted his technique were unsuccessful and resorted to surgery.

1948 Ignacio Ponseti (Iowa City) developed his method of serial manipulation and plaster cast immobilization for clubfoot correction that was significantly different from Kite's method. He published his first of many articles on the technique in 1963. Despite its clear, concise, detailed, and accurate description that has, without alteration, stood the test of time, it too did not catch on for reasons that are unclear to this day.

1969 – 1988 Bigger and more extensive clubfoot operations were proposed by Goldner, Turco, Carroll, McKay, Simons, and Ilizarov. Although the reported short-term results of all the surgical procedures were good to very good, the intermediate and long-term results were dismal in a high percentage of cases. Pain, stiffness, and the need for 1 or more additional surgical procedures were common with each subsequent operation increasing the likelihood of more pain and stiffness. Secondary iatrogenic deformities were, in some cases, worse than the natural history of the untreated clubfoot. Complications included recurrent/residual deformity, overcorrected talonavicular joint, overcorrected subtalar joint, dorsal bunion, posterior distal tibial growth arrest, wound necrosis, neurovascular injury resulting in partial or complete foot amputation.

1977 Masse originated the French functional physical therapy method for clubfoot correction. It consists of daily manipulations of the newborn clubfoot by a specialized physical therapist, stimulation of the muscles around the foot, and temporary immobilization of the foot with elastic and non-elastic adhesive taping. Physiotherapy is optimized by early triceps surae lengthening. Sequences of plaster can also be used. If conservative treatment is no longer effective, surgery is considered. Mini-invasive surgery is a complementary procedure to nonoperative treatment (surgery 'a` la carte'). The French method reduces, but does not eliminate, the need for mini-invasive surgical procedures. Equinus is the most difficult deformity to treat; posterior release is sometimes necessary in a severe foot.

1990s Dimeglio and Bensahel perfected and promoted the French method, including developing a classification system that is still in use.

1995 Cooper and Dietz, in *Treatment of idiopathic clubfoot: a thirty-year follow-up note. J Bone Joint Surg Am*, reported on 45 patients with 71 clubfeet who had been treated by Ponseti an average 34 yrs (range 25-42 yrs) previously. Using strict pain, function, pedobarographic, electrogoniometric, and radiographic assessment criteria, they reported 78% excellent/good outcomes in patients vs. 85% of controls,

1996 Publication of the clubfoot "bible": Ponseti IV. **Congenital Clubfoot. Oxford University Press**.

From that point onward, the Ponseti method has become the international gold standard for clubfoot management. Published studies from around the world have confirmed the unmatched efficacy and safety of the method. This monograph will elaborate on and update the Ponseti method from the perspective of a devotee.

2008 Richards et al., from the Texas Scottish Rite Hospital in Dallas, preformed a prospective study comparing the Ponseti method and the French functional method in which they found superiority of the Ponseti method.

The present and future risk for children with clubfoot deformities is reversion to clubfoot surgery because of the exacting techniques required for successful Ponseti management and the increasing unfamiliarity with the lessons from the past regarding clubfoot surgery (see Addendum: The Ponseti Method – Staying true to it.)

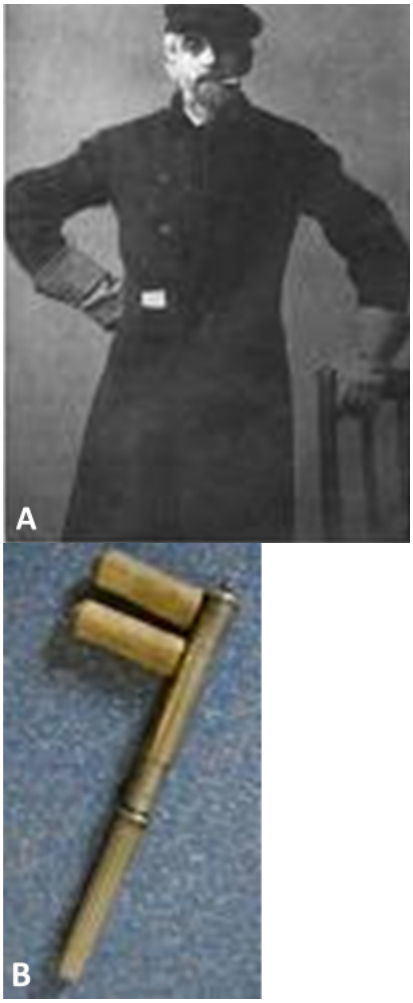


Figure 4. A. Hugh Owen Thomas. B. The Thomas wrench.

CHAPTER 2

Etiology, biomechanics, and terminology of clubfoot

Typical clubfoot is referred to as an idiopathic deformity, literally meaning that the cause is unknown. Genetics clearly plays a role. Research has identified several genes that are linked to the development of clubfoot deformity and researchers believe there are even more. The exact messaging is not worked out. Multi-factorial inheritance is suggested by its incidence of 1:1000 (typical for multifactorial conditions) and by evidence that there is an increased risk for clubfoot in babies born to mothers who smoked during the pregnancy. When one parent is affected with clubfoot, there is a 3% to 4% chance that the offspring will also be affected. However, when both parents are affected, the offspring have a 30% chance of developing clubfoot.

Scientific Basis of Understanding and Management of Clubfoot – by Dr. Ignacio Ponseti

The following section was written by Dr. Ignacio Ponseti in 2008 for the 3rd edition of the global-HELP.org publication **Clubfoot: Ponseti Management.** It has been excerpted, with permission, to serve as the introduction to this monograph.

Our treatment of clubfoot is based on the biology of the deformity and of the functional anatomy of the foot.

Biology

Clubfoot is not an embryonic malformation. A normally developing foot turns into a clubfoot during the second trimester of pregnancy. Clubfoot is rarely detected with ultrasonography before the 16th week of gestation. Therefore, like developmental hip dysplasia and idiopathic scoliosis, clubfoot is a developmental deformation.

A 17-week-old male fetus with bilateral clubfoot, more severe on the left, is shown [Figure 5].

A section in the frontal plane through the malleoli of the right clubfoot [Figure 6] shows the deltoid, tibionavicular ligament, and the tibialis posterior tendon to be very thick and to merge with the short plantar calcaneonavicular ligament. The interosseous talocalcaneal ligament is normal.

A photomicrograph of the tibionavicular ligament [Figure 7] shows the collagen fibers to be wavy and densely packed. The cells are very abundant, and many have spherical nuclei.

The shape of the tarsal joints is altered relative to the altered positions of the tarsal bones. The forefoot is in some pronation, causing the plantar arch to be more concave (cavus). Increasing flexion of the metatarsal bones is present in a lateromedial direction.

In the clubfoot, there appears to be excessive pull of the tibialis posterior abetted by the gastrosoleus and the long toe flexors. These muscles are smaller in size and shorter than in the normal foot. In the distal end of the gastrosoleus, there is an increase of connective tissue rich in collagen, which tends to spread into the tendo-Achilles and the deep fasciae.

Figure 5. 17-week-old stillborn fetus with bilateral clubfoot. [From: Ponseti IV. Congenital Clubfoot: Fundamentals of Treatment. Oxford: Oxford University Press, Figure 2, pg. 9, 1996]

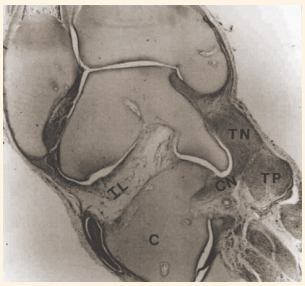


Figure 6. Histologic section in the frontal plane through the malleoli of the right clubfoot shows the deltoid ligament, tibionavicular (TN) ligament, and the tibialis posterior (TP) tendon to be very thick and to merge with the short plantar calcaneonavicular (CN) ligament. The interosseous talocalcaneal ligament (IL) is normal. [From: Ponseti IV. Congenital Clubfoot: Fundamentals of Treatment. Oxford: Oxford University Press, Figure 3C, pg. 11, 1996]

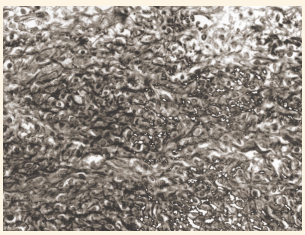


Figure 7. Photomicrograph of the tibionavicular ligament shows the collagen fibers to be wavy and densely packed (original magnification, x475). [From: Ponseti IV. Congenital Clubfoot: Fundamentals of Treatment. Oxford: Oxford University Press, Figure 17A, pg. 33, 1996]

In the clubfoot, the ligaments of the posterior and medial aspect of the ankle and **2** tarsal joints are very thick and taut, thereby severely restraining the foot in equinus and the navicular and calcaneus in adduction and inversion. The size of the leg muscles correlates inversely with the severity of the deformity. In the most severe clubfoot, the gastrosoleus is seen as a muscle of small size in the upper third of the calf. Excessive collagen synthesis in the ligaments, tendons, and muscles may persist until the child is 3 or 4 years of age and might be a cause of relapses.

Under the microscope, the bundles of collagen fibers display a wavy appearance known as crimp. This crimp allows the ligaments to be stretched. Gentle stretching of the ligaments in the infant causes no harm. The crimp reappears a few days later, allowing for further stretching. That is why manual correction of the deformity is feasible.

Kinematics

The clubfoot deformity occurs mostly in the tarsus. The tarsal bones, which are mostly made of cartilage, are in the most extreme positions of flexion, adduction, and inversion at birth. The talus is in severe plantar flexion, its neck is medially and plantarly deflected, and its head is wedge-shaped. The navicular is severely medially displaced, close to the medial malleolus, and articulates with the medial surface of the head of the talus. The calcaneus is adducted and inverted under the talus.

As shown in a 3-day-old infant [Figure 8], the navicular is medially displaced and articulates only with the medial aspect of the head of the talus. The cuneiforms are seen to the right of the navicular, and the cuboid is underneath it. The calcaneocuboid joint is directed postero-medially. The anterior two-thirds of the calcaneus is seen underneath the talus. The tendons of the tibialis anterior, extensor hallucis longus, and extensor digitorum longus are medially displaced.

No single axis of motion (like a mitered hinge) exists on which to rotate the tarsus, whether in a normal or a clubfoot. The tarsal joints are functionally interdependent. The movement of each tarsal bone involves simultaneous shifts in the adjacent bones. Joint motions are determined by the curvature of the joint surfaces and by the orientation and structure of the binding ligaments. Each joint has its own specific motion pattern. Therefore, correction of the extreme medial displacement and inversion of the tarsal bones in the clubfoot necessitates a simultaneous gradual lateral shift of the navicular, cuboid, and calcaneus before they can be everted into a neutral position. These displacements are feasible because the taut tarsal ligaments can be gradually stretched.

The correction of the severe displacements of the tarsal bones in clubfoot requires a clear understanding of the functional anatomy of the tarsus. Unfortunately, most orthopaedists treating clubfoot act on the wrong assumption that the subtalar and Chopart joints have a fixed axis of rotation that runs obliquely from anteromedial superior to posterolateral inferior, passing through the sinus tarsi. They believe that by pronating the foot on this axis, the heel varus and foot supination can be corrected. This is not so.

Pronating the clubfoot on this imaginary fixed axis tilts the forefoot into further pronation, thereby increasing the cavus and pressing the adducted calcaneus against the talus. The result is a breach in the hindfoot, leaving the heel varus uncorrected.

In the clubfoot [Figure 9A], the anterior portion of the

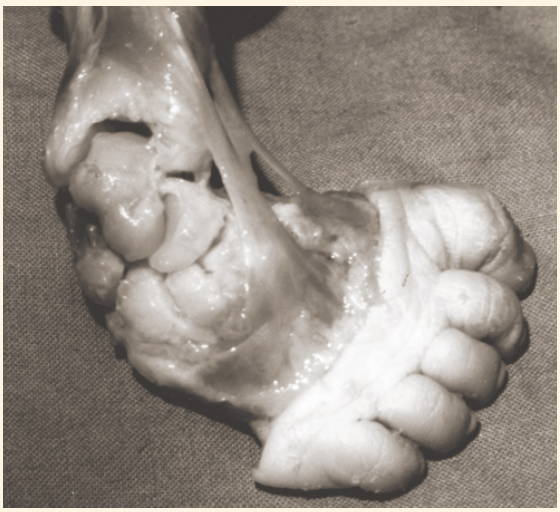


Figure 8. 3-day-old infant. The navicular is medially displaced and articulates only with the medial aspect of the head of the talus. The cuneiforms are seen to the right of the navicular, and the cuboid is underneath it. The calcaneocuboid joint is directed postero-medially. The anterior two-thirds of the calcaneus is seen underneath the talus. The tendons of the tibialis anterior, extensor hallucis longus, and extensor digitorum longus are medially displaced. [From: Ponseti IV. Congenital Clubfoot: Fundamentals of Treatment. Oxford: Oxford University Press, Figure 9A, pg. 18, 1996]

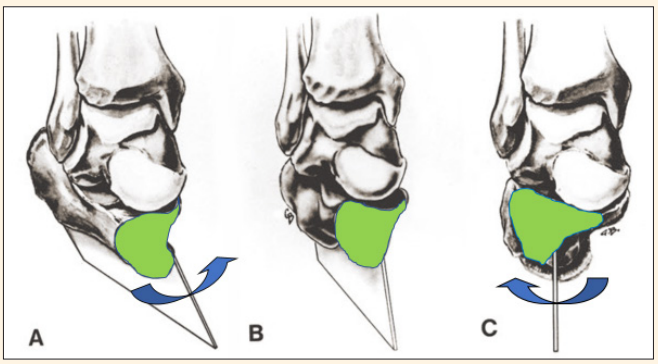


Figure 9. Clubfoot sketches. A. Original hindfoot deformity. The anterior portion of the calcaneus lies beneath the head of the talus. This position causes varus and equinus deformity of the heel. The articular cartilage surface of the anterior end of the calcaneus faces plantar and medial ("down and in"). B. Inversion deformity is partially corrected. The articular cartilage surface of the anterior end of the calcaneus faces more straight ahead. C. Final corrected position of the hindfoot deformity. The anterior portion of the calcaneus now lies lateral/plantar lateral to the head of the talus. This position creates physiologic valgus of the heel. The articular cartilage surface of the anterior end of the calcaneus faces dorsal and lateral ("up and out"). [From: Ponseti IV. Congenital Clubfoot: Fundamentals of Treatment. Oxford: Oxford University Press, Figure 19, pg. 38, 1996] These sketches (color and arrows added) and the legend have been modified by Dr. Vincent Mosca.

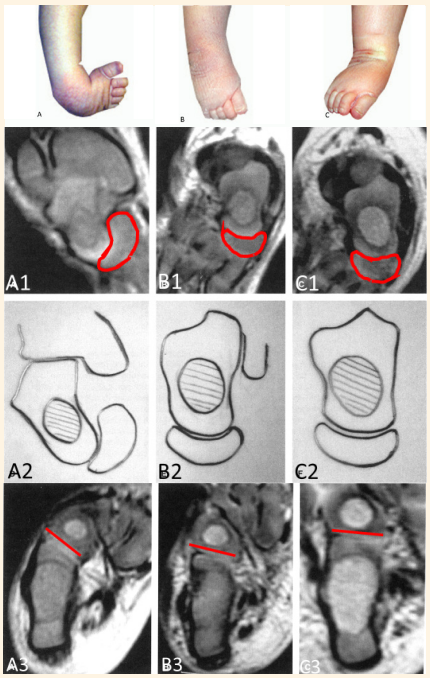


Figure 10. Temporally matched clinical photos of a clubfoot and MRI images from Pirani's study before (A, A1, A2, A3), during (B, B1, B2, B3), and at the end of Ponseti cast treatment (C, C1, C2, C3). Note the changes in the talonavicular joint and calcaneocuboid joint. Before treatment, the navicular (red outline in A1) is displaced to the medial side of the head of the talus. [The following was written by Dr. Mosca] Note how this relationship normalizes during cast treatment as the navicular and the rest of the subtalar joint rotate laterally around the talar head (A1, B1, C1 and A2, B2, C2). The calcaneocuboid joint (red line) is in varus alignment initially (A3). The calcaneocuboid joint is a stable joint, unlike the mobile talonavicular joint. The change in alignment at the calcaneocuboid joint is due to remodeling of the adjacent cartilaginous anlagen. The varus orientation of the calcaneocuboid joint changes to neutral (perpendicular to the calcaneus) according to the Heuter-Volkmann property of growing cartilage (A3, B3, C3). Finally, note the remarkable similarities between images A, A1, A2, and A3 with Dr. Ponseti's stillborn dissection in Figure 4. [From: Pirani S, Zeznik L, Hodges D. Magnetic resonance imaging study of the congenital clubfoot treated with the Ponseti method. J Pediatr Orthop. 2001;21:719-726, Figures 5 and 8]

calcaneus lies beneath the head of the talus. This position causes varus and equinus deformity of the heel. Attempts to push the calcaneus into eversion without abducting it [Figure 9B] will press the calcaneus against the talus and will not correct the heel varus. Lateral displacement (abduction) of the calcaneus to its normal relationship with the talus [Figure 9C] will correct the heel varus deformity of the clubfoot.

Correction of clubfoot is accomplished by abducting the foot in supination while counterpressure is applied over the lateral aspect of the head of the talus to prevent rotation of the talus in the ankle. A well-molded plaster cast maintains the foot in an improved position. The ligaments should never be stretched beyond their natural amount of give. After 5 days, the ligaments can be stretched again to further improve the degree of correction of the deformity.

The bones and joints remodel with each cast change because of the inherent properties of young connective tissue, cartilage, and bone, which respond to the changes in the direction of mechanical stimuli. This has been beautifully demonstrated by Pirani, comparing the clinical and magnetic resonance imaging appearance before, during, and at the end of cast treatment [Figure 10].

Before applying the last plaster cast, the tendo-Achilles may have to be percutaneously sectioned to achieve complete correction of the equinus. The tendo-Achilles, unlike the tarsal ligaments that are stretchable, is made of non-stretchable, thick, tight collagen bundles with few cells. The last cast is left in place for 3 weeks while the severed heel-cord tendon regenerates in the proper length with minimal scarring. At that point, the tarsal joints have remodeled in the corrected positions.

In summary, most cases of clubfoot are corrected after five to six cast changes and, in many cases, a tendo-Achilles tenotomy. This technique results in feet that are strong, flexible, and plantigrade. Maintenance of function without pain has been demonstrated in a 35-year follow-up study.

The following section and the rest of this monograph were written by Dr. Vincent Mosca.

Terminology/pathoanatomy/biomechanics

I'd like to reinforce and clarify Dr. Ponseti's statement that clubfoot is a developmental deformation and not an embryonic malformation. Malformation means "made wrong". Simplistic terms for the five categories of malformations are too large, too small, too many, too few, and failed to separate (1). Deformation, or deformity, means that the bones and joints are normally formed but they are malaligned. When skeletally immature, especially very immature, bones and joints are malaligned for a protracted period, they change shape according to the Heuter-Volkmann property of growing cartilage. The changes in shape are, therefore, secondary to the deformities and do not reclassify them as malformations. They weren't made wrong. Deformations can develop any time after joints are created in the 7th to 9th weeks of gestation. In the case of clubfoot, the malalignment occurs in the second trimester of pregnancy.

Malformations can never be made normal. Deformations also cannot technically be made normal, but they can approach normal with effective and timely treatment. Normal feet can be expected to have long term comfort, function, and appearance. Dr. Ponseti showed that his method can convert clubfeet into feet that are comfortable, functional, and "normal" in appearance for decades. The purpose of this monograph is to help others replicate his excellent documented results by ensuring that his method is fully understood and practiced without omissions or modifications, because strict adherence to all aspects of the method is mandatory. This starts with a full and complete understanding of the biomechanics of the clubfoot deformity and deformity correction.

Dr. Ponseti identified the clubfoot deformities as pronation of the forefoot (producing Cavus), adduction of the midfoot (Adductus), inversion of the subtalar joint/hindfoot (Varus), and plantar flexion of the talus in the ankle (Equinus), thereby creating the acronym CAVE [Figure 11].

And he listed the orderly correction of those deformities as first supinating the pronated forefoot and then abducting the adducted midfoot. But then he used terms that, based on current generally accepted terminology and understanding of biomechanics, are confusing and inaccurate.

He stressed that the subtalar joint inversion should be corrected by "Lateral displacement (abduction) of the calcaneus to its normal relationship with the talus" (2). However, lateral displacement and abduction are not synonymous terms. And, more importantly, neither term applies to the subtalar joint. "Lateral displacement" indicates shifting or translating the calcaneus in relationship to the talus. That is not physically possible. It would require joint subluxation or dislocation. "Abduction" is the term for dynamic rotation in a ball-and-socket joint in the coronal plane. The subtalar joint is not a ball-and-socket joint. Furthermore, there is no adduction or abduction in that joint. There are only inversion and eversion. And Dr. Ponseti strongly and specifically cautioned against everting the subtalar joint that he acknowledged was inverted.

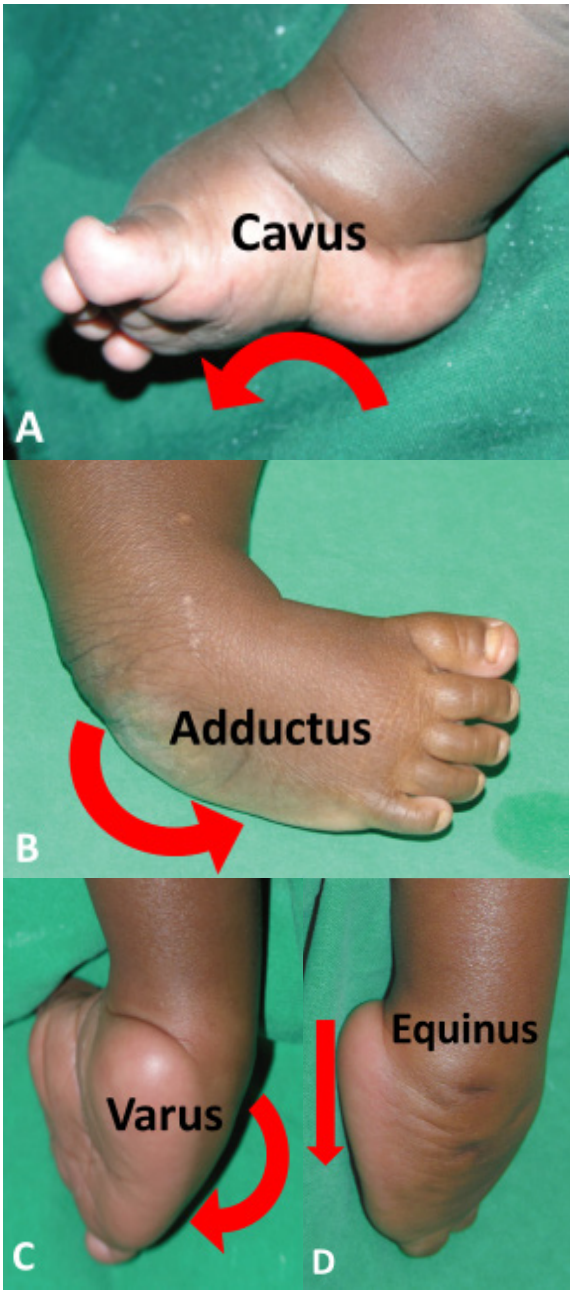


Figure 11. A. Cavus. B. Adductus. C. Varus. D. Equinus.

The terms "abducted" and "adducted" refer to static angular joint alignments or *positions* in the coronal plane, such as an abducted shoulder or an adducted hip, or in the transverse plane, as in metatarsus adductus. "Abduction" and "adduction" refer to *motions* of the ball-and-socket joints in the hands and long bones in the coronal plane (i.e. metacarpo-phalangeal, hip, and shoulder joints) and of the forefoot in the transverse plane (metatarso-phalangeal joints).

The term abduction, rather than lateral displacement, has been used most consistently by Dr. Ponseti to describe the correction of the hindfoot deformity in clubfoot. Abduction, if applied to the subtalar joint, would suggest that the entire calcaneus rotates medially and laterally under the talus in the coronal plane around an axis in the sagittal plane. That would require the subtalar joint to be a true ball-and-socket joint (like the hip or shoulder) or a coronal plane hinge joint (of which

none exist – all hinge joints, such as the knee, elbow, and ankle, are in the sagittal plane), and it is neither of those. Furthermore, abduction would result in the anterior articular surface of the calcaneus remaining in the coronal plane perpendicular to the long axis of the talus as it rotates laterally. That is not the case. Careful assessment of Figure 9 A, B, and C shows gradual "eversion" of the STJ, not "abduction" as Dr. Ponseti characterized it. One can clearly see that the articular surface of the anterior end of the calcaneus moves from/rotates facing down-and-in (inversion) to up-and-out (eversion).

The axis of the subtalar joint is not in any of the three orthogonal planes – coronal, sagittal, and transverse/axial. The coordinates of the axis of the subtalar joint are 23 degrees internal and 41 degrees dorsiflexed, i.e. oblique to the 3 standard orthogonal, or perpendicular, planes [Figure 12].

The subtalar joint complex rotates around the talocalcaneal interosseous ligament, a central point of rotation located between the middle and posterior facets along the oblique axis of the joint. One might best describe the subtalar joint complex as an "**oblique axis central rotatory joint**". As the anterior end of the calcaneus dorsiflexes and externally rotates (up-and-out) during eversion, the posterior end plantar flexes and internally rotates (down-and-in – internal/inward *in relationship to* the more anterior axis of rotation). Conversely, as the anterior end of the calcaneus plantar flexes and internally rotates (down-and-in) during inversion, the posterior end dorsiflexes and externally rotates (up-and-out – external/outward *in relationship to* the more anterior axis of rotation) [Figures 12 and 13].

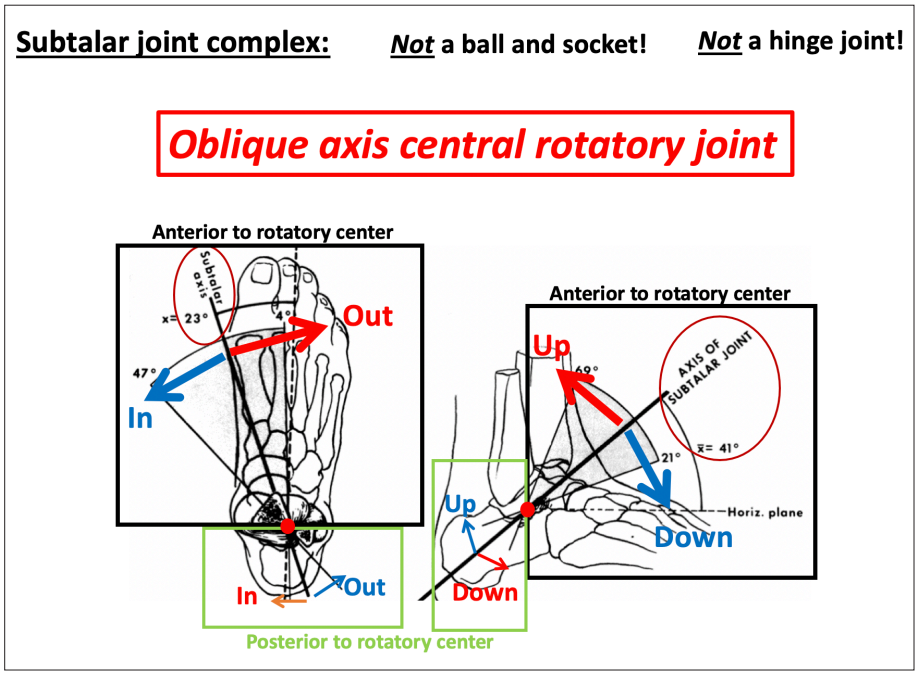


*Figure 12. The subtalar joint is not a ball and socket nor a hinge joint. Its axis of motion is not in any of the 3 orthogonal planes of the body, i.e. coronal, sagittal, or transverse/axial. The coordinates of its axis are 23 degrees internal and 41 degrees dorsiflexed. The talocalcaneal interosseous ligament (red dot), between the middle and posterior facets, is the central point of rotation along the axis of motion around which the subtalar joint complex rotates. The motions **anterior** to the interosseous ligament (in the black boxes) are eversion = dorsiflexion/up and external rotation/out (red terms and arrows), and inversion = plantar flexion/down and internal rotation/in (blue terms and arrows) of the subtalar joint complex. The simultaneous motions **posterior** to the interosseous ligament rotation point (in the green boxes) are eversion = plantar flexion/down and internal rotation/in (red terms and arrows), and inversion = dorsiflexion/up and external rotation/out (blue terms and arrows) of the subtalar joint complex. The terms internal and external rotation of the posterior calcaneus seem contrary because they are in relationship to the axis of rotation (the interosseus ligament) that is anterior to the posterior calcaneus.*

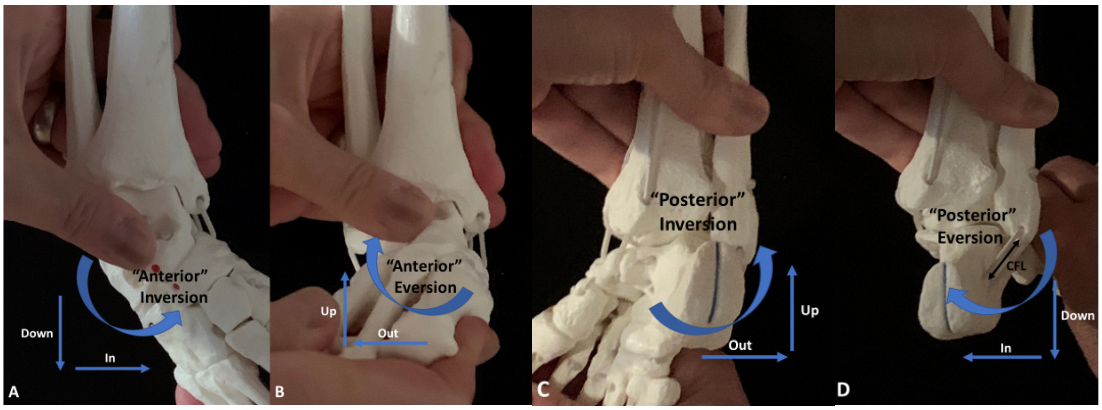


*Figure 13. Clubfoot model demonstrating equal and opposite inversion and eversion rotational motions of the anterior and posterior segments of the subtalar joint around the talocalcaneal interosseous ligament. A. Inversion of the subtalar joint (blue curved arrow) in the anterior segment of the subtalar joint – plantar flexion/down (blue straight arrow) and internal rotation/in (blue straight arrow). B. Eversion of the subtalar joint (blue curved arrow) in the anterior segment of the subtalar joint – dorsiflexion/up (blue straight arrow) and external rotation/out (blue straight arrow). C. Inversion of the subtalar joint (blue curved arrow) in the posterior segment of the subtalar joint – dorsiflexion/up (blue straight arrow) and external rotation/out (blue straight arrow) **in relationship** to the talocalcaneal interosseous ligament. D. Eversion of the subtalar joint (blue curved arrow) in the posterior segment of the subtalar joint – plantar flexion/down (blue straight arrow) and internal rotation/in (blue straight arrow) **in relationship** to the talocalcaneal interosseous ligament. Also, note the elongated CFL - calcaneo-fibular ligament (double headed black arrow).*

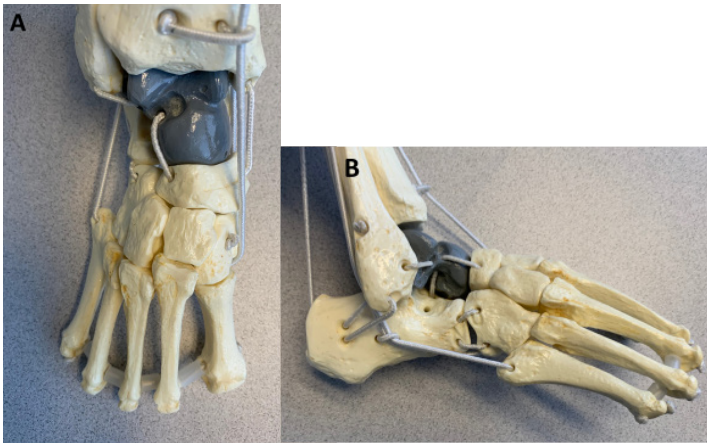


Figure 14. The calcaneo-pedal unit (CPU) is the term for all bones of the foot (white in color in this model made by John Mitchell and available through MD Orthopaedics, Inc.) except the talus (gray in color). The CPU and its innermost component, the acetabulum pedis, inverts and everts around the talus through the subtalar joint complex. A. Frontal view of foot model. B. Lateral view of foot model.

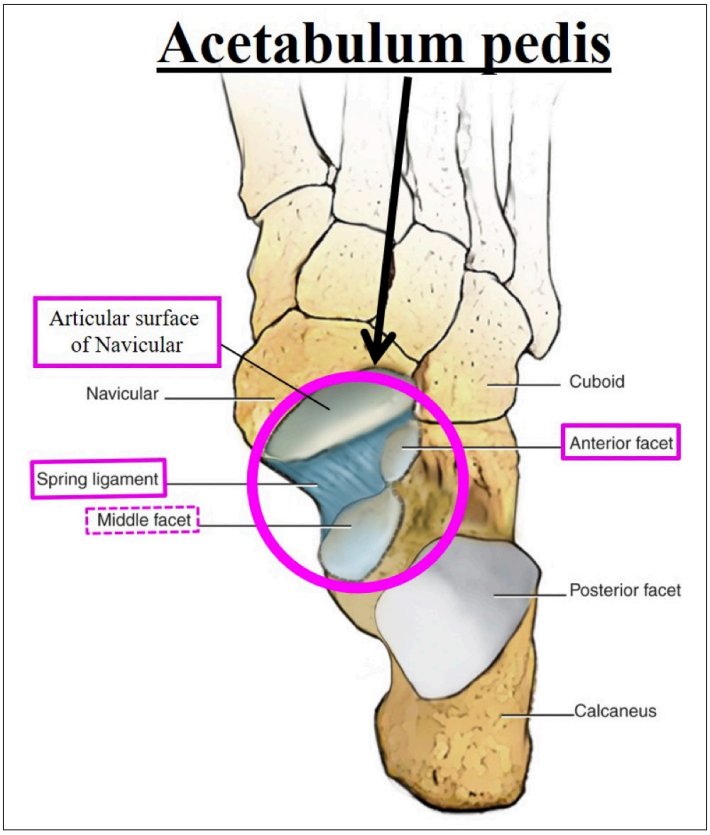


Figure 15. The acetabulum pedis (AP). [From: Mosca VS. Principles and Management of Pediatric Foot and Ankle Deformities and Malformations. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins, pg. 8, Figure 2-5, 2014]

Current generally accepted terminology for foot deformities and the biomechanics of foot motions are based on the concept of the calcaneo-pedal unit (CPU) (3). I believe it is important to translate Dr. Ponseti's terminology into the new, modern terminology to make it relevant and consistent with that used for all other foot deformities. This translation does not in any way change or diminish Dr. Ponseti's correct concepts. It merely changes some of the words used. The CPU refers to all bones of the foot, except the talus (and excluding the toes) [Figure 14].

Whereas static angular and rotational deformities may exist between the bones within the CPU, there is very little movement between those bones. The major motions within the foot occur between the CPU and the talus. The smaller and most important segmental unit within the CPU is the acetabulum pedis (AP), a term coined by Scarpa in 1818 (4). It consists of the bones and ligaments of the CPU immediately adjacent to the talus, i.e. the proximal articular surface of the navicular, the spring ligament, and the anterior/middle facets of the calcaneus [Figure 15].

The "joint" between the CPU/AP and the talus is referred to as the subtalar joint complex (STJC). The subtalar joint, strictly speaking, is the talo-calcaneal joint (sub = under the talus). But the calcaneus cannot move relative to the talus without the navicular, cuboid, and the rest of the CPU moving with it, because the CPU is an extension of the calcaneus. The STJC inverts and everts under the talus around the centrally located talocalcaneal interosseus ligament pivot point along the oblique axis of the subtalar joint. Inversion is a combination of plantar flexion (down), internal rotation (in), and supination of the CPU/AP around the talus. Eversion is a combination of dorsiflexion (up), external rotation (out), and pronation between those two parts. Because the center of rotation is the talocalcaneal interosseous ligament located between the middle and posterior facets of the subtalar joint, those described motions occur *anterior* to the interosseous ligament. *Posterior* to the interosseous ligament, the motions between the talus and calcaneus are opposite in direction, i.e. dorsiflexion (up) and external rotation (out) during inversion, and plantar flexion (down) and internal rotation (in) during eversion [Figure 12]. The CPU/AP can only rotate around the centrally located interosseous ligament along the fixed oblique axis of the STJC which is the same in normal feet and in those with hindfoot deformities. Individual components of inversion and eversion cannot be isolated. The STJC is in an exaggerated and fixed inverted position in clubfoot and cavovarus foot deformities. It is in an exaggerated everted position in flatfoot and skewfoot deformities. NOTE: the term "peritalar subluxation", often used when describing and discussing adult acquired flatfoot deformity, is a misnomer and should never be applied to pediatric foot deformities! The peritalar, or subtalar, joint never subluxates, i.e. loses articular contact. It simply rotates minimally or maximally. [Figure 16].

Ponseti was correct when he stated that one should not attempt to correct the hindfoot varus deformity until the forefoot

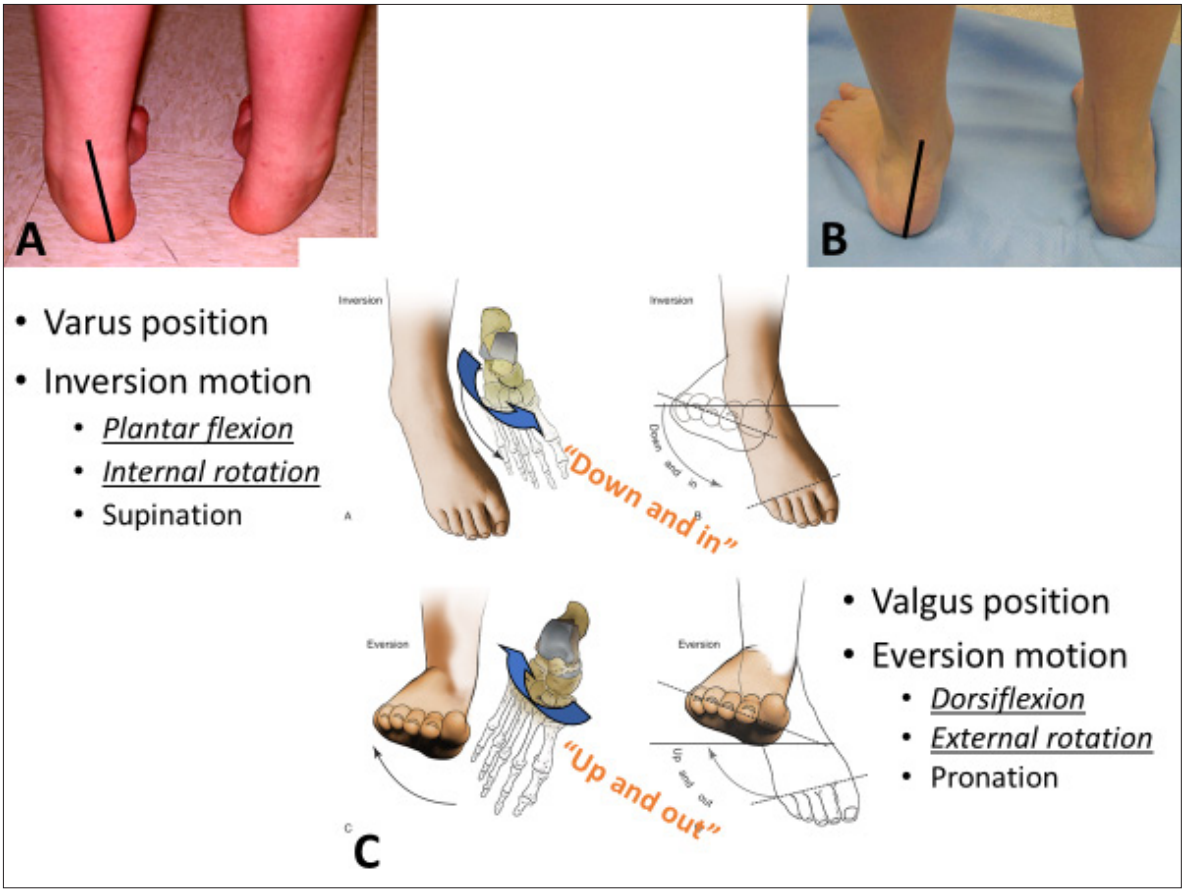


Figure 16. Subtalar joint complex (STJC) positions and motions. A. Hindfoot varus position as seen in clubfoot and cavovarus foot deformities. Also known as an inverted hindfoot. B. Hindfoot valgus position as seen in flatfoot and skewfoot deformities. Also known as an everted hindfoot. C. The subtalar joint assumes a static position of varus/inverted by inverting, a motion that consists of plantar flexion (down), internal rotation (in), and supination of the subtalar joint complex/ acetabulum pedis/calcaneopedal unit under the talus. The subtalar joint assumes a static position of valgus/everted by everting, a motion that consists of dorsiflexion (up), external rotation (out), and pronation of the STJC/AP/CPU under the talus. These motions are relative to, and anterior to, the talocalcaneal interosseous ligament located between the middle and posterior facets of the subtalar joint, the rotational pivot point [Figure 8]. [Modified from: Mosca VS. Principles and Management of Pediatric Foot and Ankle Deformities and Malformations. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins, Figure 2-7, pg. 9, 2014]

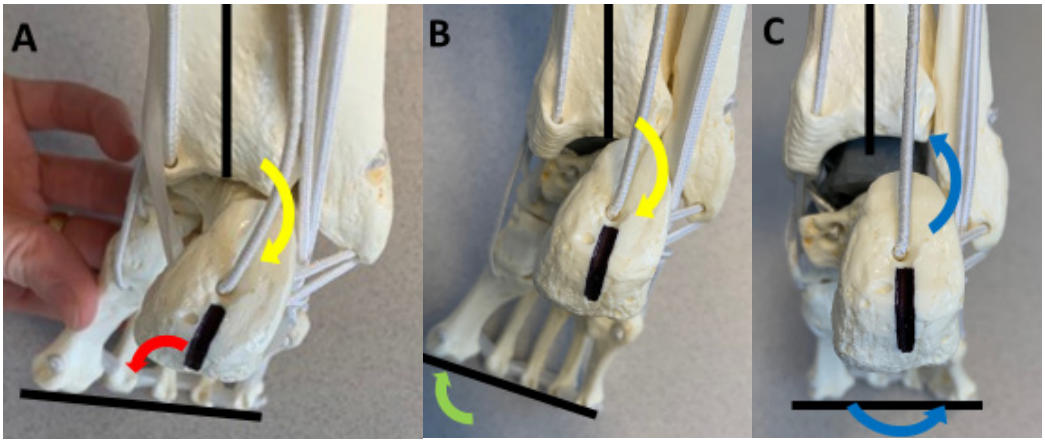


Figure 17. Clubfoot model. A. The forefoot is pronated in relation to the hindfoot. Pronation is an interosseous deformity between the bones in the CPU. The transverse plane of the metatarsal heads (black line under the MT heads) is pronated (red curved arrow) in relationship to the sagittal plane of the calcaneus (short black line). Hindfoot varus is the static alignment between the calcaneus and the talus/tibia due to inversion of the subtalar joint (yellow curved arrow). B. Supination molding of the forefoot (green curved arrow) corrects the pronation deformity within the CPU resulting in the normal perpendicular relationship between the transverse plane of the metatarsal heads (black line under the MT heads) and the sagittal plane of the calcaneus (short black line). This correction is usually achieved after the 1st or 2nd cast. Then, the only residual deformity in the clubfoot (not including the equinus in the ankle joint) is inversion/varus deformity of the subtalar joint (yellow curved arrow). C. Eversion molding of all the bones of the CPU as a single unit (blue curved arrows) under and around the talus in the subtalar joint complex completes correction of all deformities within the clubfoot - leaving only ankle equinus deformity which, in most cases, requires a tendo-Achilles tenotomy. Note that the relationship between the transverse plane of the metatarsal heads (black line under the MT heads) and the sagittal plane of the calcaneus (short black line) within the CPU does not change, i.e. the bones and joints within the CPU remain anatomically aligned in this last stage of deformity correction. Plantar flexion/internal rotation (inversion) deformity of the CPU corrects to dorsiflexion/external rotation (eversion).

pronation and midfoot adduction deformities are corrected. Kite's method of cast-correction of clubfoot deformities involved pronating the already pronated forefoot (5) and it was not successful. The reason might relate to the postmortem fetal clubfoot dissection findings of Epeldegui (6,7). He found that the calcaneo-navicular spring ligament was contracted in clubfeet, thereby resulting in a smaller than normal acetabulum pedis, although this is my interpretation of his findings and not his stated conclusion. Perhaps that small AP cannot rotate around the larger head of the talus. And perhaps supinating the forefoot and abducting the midfoot with serial casting allow the spring ligament to stretch because it is at the central core of those more superficial deformities and contractures. Stretching the spring ligament would enlarge the AP enabling it to rotate around the head of the talus when counter-pressure is applied to the dorso-lateral aspect of the head of the talus.

Forefoot pronation and hindfoot varus are rotationally opposite direction deformities, as if the foot is wrung out like a towel (1). Forefoot pronation is a static rotational deformity within the CPU, i.e. the forefoot is pronated relative to the sagittal plane of the calcaneus [Figure 17A]. Hindfoot varus is a static angular deformity of the calcaneus in relation to the tibia that is due to inversion of the subtalar joint. And inversion is a dynamic rotational and angular deformity between the CPU and the talus in the STJC. Correcting forefoot pronation (a static rotational deformity within the CPU) and midfoot adduction (a static angular deformity within the CPU) in the first one or two casts creates normal interosseous alignment between all the bones and joints within the CPU. This is manifest by a perpendicular relationship between the transverse plane of the metatarsal heads, as a proxy for the anterior CPU, and the sagittal plane of the calcaneus [Figure 17B]. Then the only residual deformity, other than equinus, is inward rotation/plantar flexion (inversion) of the CPU under the talus. With stretching of the spring ligament and enlargement of the AP, the subtalar joint can be safely everted, not abducted, around the talus. The "apparent" forefoot supination deformity that is intentionally created by casting reverts to neutral as the subtalar joint is everted. When the subtalar joint is fully everted, the forefoot is in neutral rotational alignment relative to the tibia and the hindfoot, neither pronated nor supinated [Figure 13C]. The point is that eversion is not a dirty word. It's the current universally accepted term for correction of subtalar joint inversion deformity regardless of etiology, whether congenital or acquired.

The central structural axis of rotation of the STJC is the talocalcaneal interosseous ligament that is located between the middle and posterior facets (1). The clubfoot manipulations, as described by Ponseti, align the anterior aspect of the STJC by rotating the navicular dorso-laterally around the head of the talus [Figure 18A]. At the same time, the calcaneo-fibular ligament stretches to enable the posterior aspect of the STJC to passively rotate plantar-medially away from the fibula based on the central position of the interosseous ligament and the oblique axis of the subtalar joint [Figure 18A]. And the hand positions that Dr. Ponseti so clearly and accurately described allow that to occur exactly as he proved decades ago (2). The subtalar joint can only move around its axis of rotation and in no other way unless its motions are inappropriately restricted externally. That happens if counter pressure is applied to the medial side of the calcaneus rather than the medial malleolus, a point stressed by Dr. Ponseti. Counter pressure on the medial side of the calcaneus prevents stretching of the contracted calcaneo-fibular ligament and rotation of the calcaneus away from the fibula. The center of rotation then shifts from the talocalcaneal interosseus ligament to the calcaneo-fibular ligament thereby creating false correction of the STJC deformity [Figure 18B].

The talonavicular joint will usually align whether the postero-medial point of counter pressure is the medial malleolus or the medial side of the calcaneus. But the CPU will only externally rotate on the interosseous ligament to the normal anatomic position under the talus if the posterior point of counter pressure is the medial malleolus. This is analogous to the McKay postero-medial-lateral clubfoot surgical release (PMLR) in which he stressed the importance of releasing the contracted calcaneo-fibular ligament (8). A positive, or outward, thigh-foot angle is achieved with the Ponseti non-operative and the McKay operative techniques [Figure 18A]. Think of this as untwisting a cap on a bottle where the center of rotation is the center of the neck of the bottle. Alternatively, counter pressure on the medial side of the calcaneus will result in persistent inward rotation of the CPU despite alignment of the talonavicular joint because the center of rotation becomes the calcaneo-fibular ligament. This is analogous to the Turco postero-medial surgical clubfoot release (PMR) in which he failed to address the postero-lateral corner (9). A negative, or inward, thigh-foot angle results from the Kite non-operative and the Turco operative techniques [Figure 18B]. Think of this as changing rotation of the subtalar joint on a central axis to the opening of a book in which the calcaneo-fibular and talocalcaneal interosseous ligaments are the spine of the book. The Kite non-operative and Turco operative techniques effectively create a coronal plane hinge joint that rotates around a sagittal plane axis thereby enabling non-physiologic "abduction" of the hindfoot. I only mention these surgical procedures to help explain the anatomy and pathoanatomy and not to promote the surgical management of clubfeet.

More biology

I would also like to take this opportunity to provide some additional understanding and explanation of the biologic changes in collagen and cartilage that take place during deformity correction with the Ponseti method.

Creep is the term for the viscoelastic property of collagen that enables it to elongate when it is subjected to a constant load (10). The 30-60 seconds of manipulation and stretching immediately before each cast application take advantage of that property. The crimping of the collagen fibers is diminished. The collagen is longer and the deformities are improved at the end of the manipulation. There is a finite, yet unpredictable, amount of stretch/elongation of collagen that can occur at any point in time. The goal is to stretch the collagen and not tear or rupture it. That's the reason Dr. Ponseti stressed that the child should not cry during manipulation of the foot. Of course,

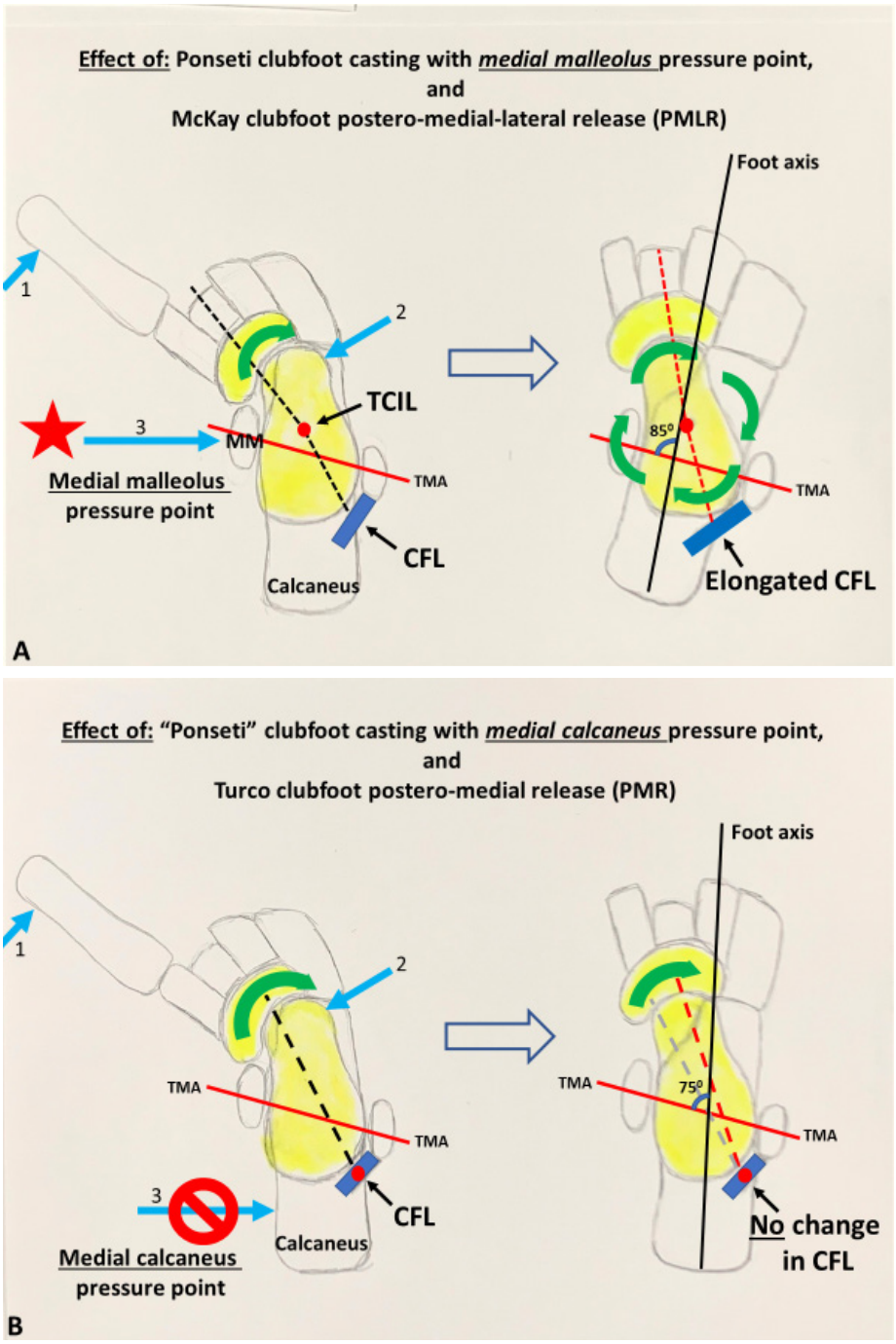


Figure 18. Clubfoot deformity correction. A. The central point of rotation along the axis of the subtalar joint around which the STJC rotates is the talocalcaneal interosseous ligament (TCIL - red dot). It is located between the middle and posterior facets of the subtalar joint. According to Ponseti, the 3 pressure points (blue arrows) for manipulations and casting are the plantar-medial side of the 1st metatarsal head (1), the dorso-lateral head of the talus (2), and the medial malleolus (3). Alignment of the anterior aspect of the subtalar joint complex is produced by everting (green curved arrow) the CPU around the head of the talus after the pronation and adduction deformities within the CPU have been corrected. Due to the relative size of the segments, the manipulations are, by necessity, anterior to the interosseous ligament rotation point. The medial malleolus pressure point allows the calcaneo-fibular ligament (CFL) to stretch and the posterior aspect of the STJC to rotate plantar-medially away from the fibula. The black dotted lines convert to the red dashed lines following deformity correction by means of an external rotation spin (green curved arrows) around that central axis of the subtalar joint. The transmalleolar axis (TMA) is drawn as a reference line to show correction of the internal rotation deformity in the subtalar joint complex to a normal transmalleolar angle of 85 degrees (blue curved arc between the Foot axis and the TMA). The McKay postero-medial-lateral surgical clubfoot release (PMLR), that includes release of the CFL, accomplishes the same goal, but at the cost of stiffness and eventual pain. B. Pressure on the medial aspect of the calcaneus (blue arrow covered with "no symbol"), rather than the medial malleolus, prevents the calcaneo-fibular ligament from stretching and the calcaneus from rotating away from the fibula. With this pressure point on the medial calcaneus, the center of rotation of the subtalar joint complex shifts to the CFL (red dot on CFL), thereby changing spin of the joint around the central interosseus ligament to rotating the joint on the CFL with all rotation occurring anterior to that point. The CFL center of rotation prevents the natural spin of the subtalar joint. The talocalcaneal interosseous ligament then either stretches inappropriately or the cartilage anlage of the talus and calcaneus on the lateral side of the talocalcaneal joint compress according to the Heuter-Volkmann property of growing cartilage. The dashed red line represents an iatrogenically created axis in the near-sagittal plane around which the calcaneus can rotate in the coronal plane, effectively creating true (though undesired) "abduction" of the subtalar joint. Note the persistent inward rotation of the foot in relation to the TMA – the transmalleolar angle (blue curved arc) between the Foot axis and the TMA is 75 degrees, 10 degrees less than would have occurred with appropriate hand positioning. The Turco postero-medial surgical clubfoot release (PMR), by ignoring and failing to release the postero-lateral corner, results in the same false correction as the improper casting technique. Like the McKay procedure, the Turco procedure creates stiffness and eventual pain, but without even truly correcting the deformity.

babies cry for many reasons, but the goal is to stretch the collagen gently so as not to cause pain-inducing tissue damage with its associated bleeding and subsequent scar formation.

Stress relaxation is the term for the viscoelastic property of collagen that enables it to gradually elongate after a static maximum load has been applied (10). The collagen relaxes/elongates over time, thereby decreasing the effective load that was initially applied. A clubfoot cast is applied after the manipulation (creep) has achieved maximum collagen elongation at that moment in time. At the time of cast removal several days later, the collagen is longer and the deformities are improved compared with their appearance at the time of cast application. That's stress relaxation. It is not known exactly how long it takes for the collagen to reach its maximum passive elongation. Most pediatric orthopedic surgeons, like myself, change the casts weekly, based on our clinic schedules rather than the clubfoot biology. The efficacy and safety of shorter intervals between casts have been reported by some authors. At the time of the cast change, the collagen in the tendons, ligaments, and muscles is longer than it was. Unlike a rubber band that returns to its original length even after being held in an elongated position for a protracted period, the collagen with its elastic fibers is longer – a longer "rubber band". The longer rubber band is then serially subjected to creep and stress relaxation from a new starting length at each cast change. Of course, without the application of a cast after manipulation, the collagen resumes its former length. And after full deformity correction has been achieved, full time and then part time brace wear are necessary to maintain maximum stretch on the collagen and prevent recurrent deformity. This is particularly important during the first 3-4 years of life when excessive collagen synthesis in the ligaments, tendons, and muscles persists and is a known cause of relapses.

There is one other biologic change that takes place during Ponseti clubfoot management and it relates to the shapes of the bones and orientation of the joints. Pirani (11) showed this beautifully in his serial MRI monitoring of clubfeet undergoing Ponseti treatment. The mild misshapenness of the navicular and talus correct to normal as the navicular is rotated to its anatomic alignment on the head of the talus. The varus/adducted mal-orientation of the calcaneo-cuboid joint corrects to anatomically normal orientation with reshaping of the adjacent bone ends [Figure 10]. This is due to the Heuter-Volkmann property of growing cartilage.

The tendo-Achilles, unlike the tarsal ligaments and the other plantar-medial tendons that are stretchable, is made of non-stretchable, thick, tight collagen bundles with few cells. It rarely stretches completely even with skillful Ponseti casting, most often requiring a tenotomy to complete correction of the equinus deformity (2). Care must be exercised to avoid over-stretching the plantar-medial soft tissues and creating a rocker-bottom foot deformity when attempting to fully stretch the unyielding tendo-Achilles. Percutaneous tendo-Achilles tenotomy is needed in almost all clubfoot deformities following the 4-8 casts that are required to correct the other deformities. The last cast is left in place for 3 weeks while the severed heel-cord tendon regenerates filling the gap between the tendon ends at the proper length and with minimal scarring.

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CHAPTER 3

Ponseti clubfoot management - FAQ

How does Ponseti clubfoot management compare with other reported methods of clubfoot treatment?

Over the past 25 years, Ponseti management has become accepted throughout the world as the most effective and least expensive treatment of clubfoot. It is the gold standard. Although Dr. Ponseti developed his method 70-75 years ago, it was not generally accepted outside of Iowa until the publication of the landmark long-term follow-up study on Dr. Ponseti's patients by Cooper and Dietz in 1995 and the publication of Dr. Ponseti's classic book in 1996.

Can clubfoot deformity be diagnosed on fetal ultra-sound?

If present, most clubfoot deformities can be seen on the 20-week fetal ultrasound, though often not prior to that gestational age [Figure 19].

Inexperienced ultra-sonographers and those who rarely perform fetal ultra-sounds might not appreciate the deformity even if it is present. Identification of clubfoot on fetal ultra-sound, in and of itself, is not an indication for amniocentesis. Most clubfoot deformities are isolated, idiopathic deformities. If there are no other abnormalities seen on the ultra-sound and if all routine pregnancy blood tests are normal, the possibility of finding an associated underlying syndrome or neurogenic condition in the fetus is so low that the risks of amniocentesis are rarely justified.

Is there a role for prenatal consultation when clubfoot is identified on fetal ultra-sound?

Studies have shown that prenatal consultation regarding clubfoot is very well accepted and appreciated by prospective parents. The term clubfoot is frightening to most pregnant couples who imagine that their as-of-yet unborn child will have life-long pain and disability. The opportunity to learn of the relatively easy and fast non-operative Ponseti treatment method with its excellent prognosis for comfort, appearance, and function enables them to relax somewhat for the remainder of the pregnancy. My experience, performing 1-2 prenatal consultations per month, consistently bears out these comments. The parents who have been counseled prenatally arrive with their newborn ready to begin the casting, excited about the opportunity to get started, and enthusiastic about the future. Those who have not been counseled, either because they were unaware their child had clubfoot or did not seek prenatal consultation, appear stunned and apprehensive. They are often tearful and distressed not appearing to take in the educational information that is so well absorbed during prenatal consultation visits.

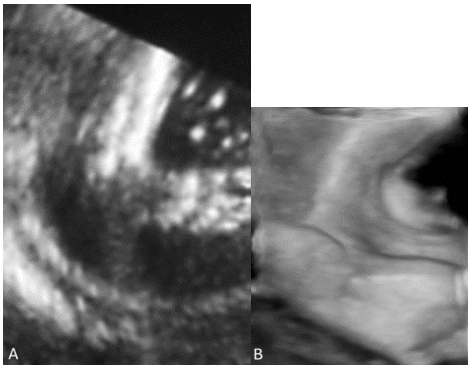

Figure 19. Fetal ultrasound clubfoot. A. Routine. B. 3D.

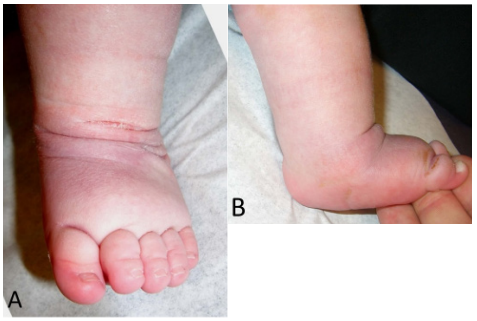

Figure 20. Fat clubfoot, even in a child who is not fat. A. Front view. Note irritated fissure in the redundant skin over the antero-lateral ankle. B. Side view. Note rocker bottom appearance of foot. These feet are often more challenging to correct than a typical thin clubfoot. More casts than average are generally required. The casts tend to slip down or off, sometimes causing pressure ulcers.

When should treatment with Ponseti management be undertaken?

When possible, treatment should start soon after birth (7 to 10 days). However, most idiopathic clubfoot deformities can be corrected, or at least significantly improved, using Dr. Ponseti' s management method even if it is initiated later in childhood. Starting early in the child's life ensures the best outcomes with the shortest duration of active treatment.

When treatment is started early, how many cast changes are usually required?

Most idiopathic clubfoot deformities can be corrected in approximately 6 weeks by weekly manipulations and long leg cast applications. Reasons for protracted treatment, i.e. more than approximately 6 casts, are: non-idiopathic syndromic or neurogenic clubfoot, congenital or acquired atypical or complex clubfoot, and the so-called "fat" clubfoot (which is poorly defined, but obvious when experienced) [Figure 20].

How late can treatment be started and still be helpful?

Initiating Ponseti cast treatment even into teenage years can diminish, and in some cases correct, the deformities, thereby decreasing the extent of, or necessity for, clubfoot surgery and its attendant risks and complications. In some cases, operative correction will be required, but the magnitude of the procedure may be less than would have been necessary without preliminary Ponseti casting.

What is the expected outcome for the infant with clubfoot treated by the Ponseti method?

A clubfoot treated by the Ponseti method will have very good short-term and long-term appearance, comfort, and function [Figure 21].

It will not be normal, because it is a genetic deformity and cannot, therefore, be made normal. Nevertheless, the achievable goals are laudable. In all patients with unilateral clubfoot, the affected foot is slightly shorter and narrower than the normal foot, and the affected limb is shorter and the circumference of the lower leg smaller than the normal limb. These differences at maturity are an average 1.3 cm or 1 shoe size, 0,4 cm narrower foot, 1-2 cm shorter limb (in which normal leg length discrepancy for any adult is up to 2 cm), and 2.3 cm smaller calf circumference. In bilateral cases, the same differences from normal will be present for both limbs, so they'll be symmetric and therefore not noticeable. A Ponseti treated clubfoot should be strong, flexible, and pain free. These features are expected to persist throughout the person's lifetime. This provides the opportunity for normal function during childhood and a pain-free and mobile foot during adult life.

How do the outcomes of surgery and Ponseti management compare?

Surgery improves the initial appearance of the foot but makes it stiff, weak, and often painful as early as adolescence and certainly in adult life. Furthermore, surgery does not prevent recurrence of deformity. A high percentage of operated clubfeet require at least one additional operation that creates more stiffness and more eventual pain.

How often does Ponseti management fail and operative correction become necessary?

The success rate in idiopathic clubfoot depends on several factors, including the age at which treatment is initiated, the degree of stiffness of the foot, the experience of the practitioner, and the reliability of the family. In most cases, the success rate can be expected to exceed 95% if treatment is initiated within the first few weeks of life. Failure is more likely if the foot is extremely stiff with a deep crease on the sole of the foot and another behind the ankle, there is severe cavus, the foot is fat, and/or the gastrocnemius is small with fibrosis of its lower half. As with all medical conditions, 100% success with a specific intervention is a stretch goal that is often approached, but rarely achieved. This is due to biologic variability. It is acknowledged that 1) there is a range in the number of casts required, 2) most, but not all, feet need a

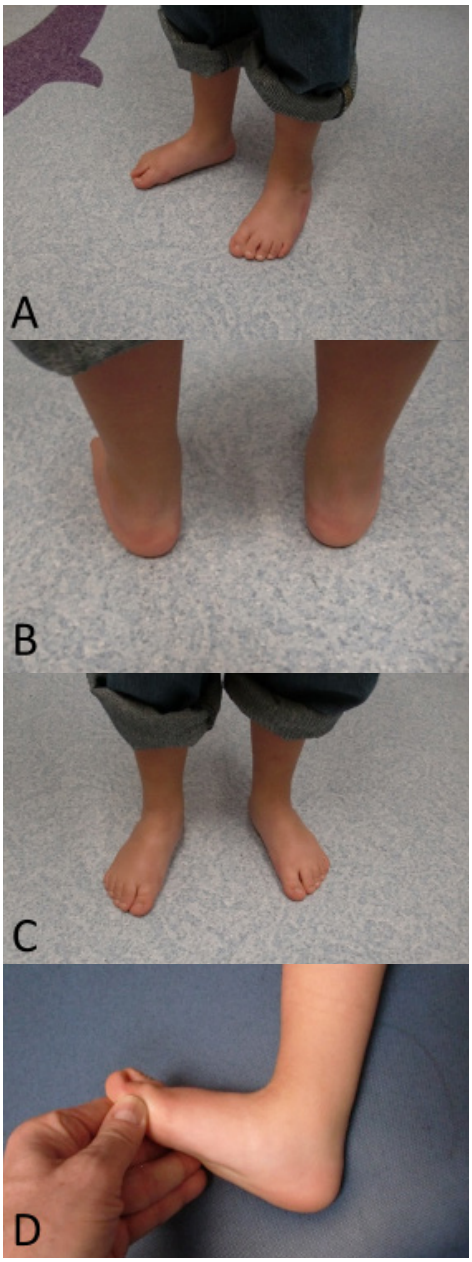

Figure 21. Ponseti-corrected clubfeet at age 7 years.

tendo-Achilles tenotomy, and 3) a significant number of feet need a tibialis anterior tendon transfer. It follows logically that some feet, admittedly small in number, do not respond to the Ponseti method and require a la carte clubfoot surgery regardless of the experience of the practitioner. Finally, family non-compliance with all aspects of the treatment method is directly related to the success or failure of the method.

Is Ponseti management useful for clubfoot in infants with other musculoskeletal problems?

Ponseti management is appropriate for use in children with idiopathic and non-idiopathic clubfoot, such as those with arthrogryposis, myelomeningocele, Larsen and other syndromes [Figure 22].

Treatment is more difficult, as cast correction takes longer and there is a higher likelihood that an a la carte surgical release will be necessary. Additionally, special care must be exercised in infants with foot sensation problems, such as those with myelomeningocele, to prevent creating skin ulcers. As with idiopathic neglected or recurrent clubfoot, using the Ponseti method for non-idiopathic clubfoot is beneficial as it may either eliminate the need for extensive surgery or at least decrease the extent of surgery and its attendant risks and complications.

Is Ponseti management useful for clubfoot previously treated by other methods?

Ponseti management is also successful when applied to feet that have been manipulated and casted by other practitioners who are not yet skilled in this very exacting management. And the method should be applied to recurrent clubfoot deformities that have previously undergone surgical treatment. Decreasing or eliminating the need for another operation is vital to prevent the additional stiffness and eventual pain that more surgery would create.

What are the usual steps of clubfoot management?

Most clubfeet can be corrected by serial brief manipulations followed by casting in the position of maximum correction that was achieved after each manipulation. After approximately 5-6 castings, the cavus, adductus and varus are corrected. A percutaneous tendo-Achilles tenotomy is performed in nearly all feet to complete the correction of the equinus. The cast applied immediately following the tenotomy is worn for 3 weeks. This correction is then maintained by 3 months of full-time (23 hours per day) bracing followed by 4-5 years of night bracing using a foot abduction brace. Maintaining deformity correction with night bracing is equal in importance to achieving deformity correction with the castings and tenotomy, yet it is the most challenging part of the treatment for the child and family. Between 30 and 55% of clubfeet have relative weakness of the peroneal muscles compared with the tibialis anterior and require transfer of the tibialis anterior to the lateral (3rd) cuneiform usually after age 3 years.

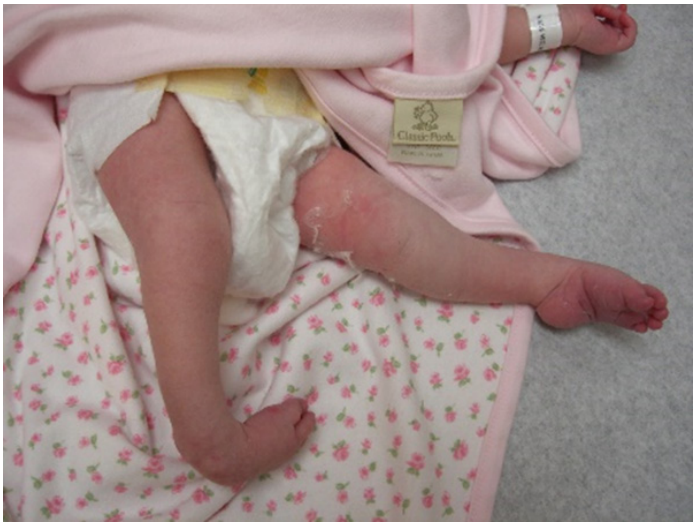

Figure 22. Infant with arthrogryposis, right severe and rigid clubfoot, left equino-cavus foot deformity, and bilateral knee extension contractures. Ponseti management was utilized on the right clubfoot with partial correction leading to a limited a la carte surgical release.

What is the prognosis for clubfeet treated with the Ponseti method?

Feet treated with the Ponseti method, despite not technically being normal, have been shown to be strong, flexible, and primarily pain free, allowing a normal life. This is not to say that they never hurt or that they enable all activities with the same functional mobility and strength as normal feet. In fact, the levels of comfort and function of Ponseti-treated clubfeet have been shown to be vastly higher than clubfeet treated by any other method, while being individually unpredictable within a desirable range. There are no shortcuts and every aspect of the method must be implemented exactly as described by Dr. Ponseti to achieve the same excellent reported outcomes. And because of the excellent reported outcomes of this multifaceted treatment method, only prospective randomized controlled trail studies that challenge one or more aspects of the method should be accepted.

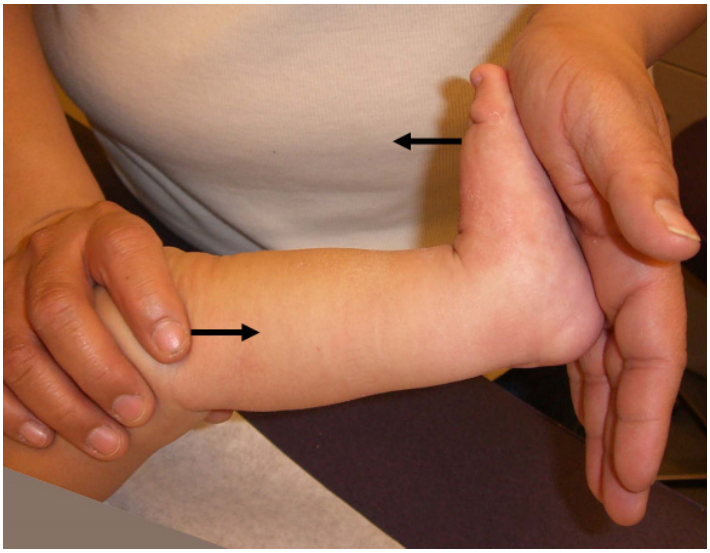

Figure 23. The parents are instructed to stretch the tendo-Achilles with an upward and outward pressure for 60 seconds – 30 seconds, relax, 30 seconds – at least 3 times per day. This begins immediately after initiation of bracing and continues until the child becomes a good walker. Right hand to right foot and left hand to left foot; toes at wrist and heel in palm.

Is physical therapy a component of the Ponseti method?

Formal physical therapy was not recommended by Dr. Ponseti. The French method for clubfoot correction is based on physical therapy and, although the French method has not been shown to be as effective as the Ponseti method, the concept of regular soft tissue stretching is sound and may be additive to the Ponseti method. Without data to support its efficacy but without any risk of harm, daily stretching of a clubfoot by parents makes biologic sense and may be helpful in maintaining deformity correction. The primary and tested method for maintaining deformity correction is the nighttime foot abduction brace. It is particularly good at maintaining maximum eversion stretch on the subtalar joint and abduction of the midfoot, while applying submaximal stretch on the tendo-Achilles. At least until the child begins to walk, I recommend that the parents stretch the ankle into maximum comfortable dorsiflexion for 60 seconds (30 seconds x 2) at least 3 times per day [Figure 23].

Is idiopathic clubfoot associated with other deformities?

Regardless of the method of treatment, a clubfoot is 1 to 2 shoe sizes smaller than the other foot in unilateral cases. The implication for bilateral clubfoot is that, although both feet are the same shoe size, they are 1 to 2 shoe sizes smaller than they would otherwise have been. In unilateral clubfoot, the muscles in the lower leg are smaller in circumference than those in the other leg. The implication for bilateral clubfoot is that, although the lower leg muscles are symmetric in circumference, they are smaller than they would otherwise have been. In unilateral cases, the limb with the clubfoot is typically 1-2 cm. shorter than the normal limb at skeletal maturity, whereas the lower extremities are of equal length in children with bilateral clubfoot. They are presumably 1-2 cm shorter than they would otherwise have been.

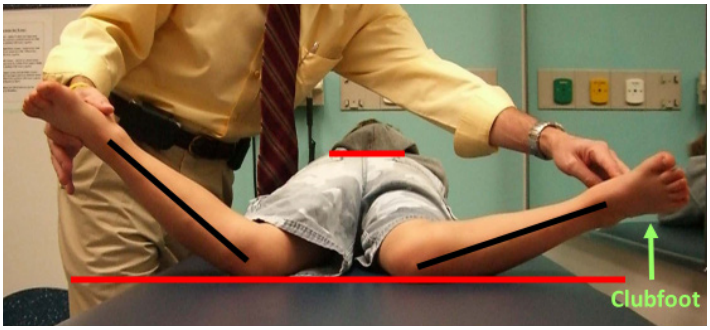

Figure 24. There are, on average, 10 degrees greater femoral anteversion in a limb with a clubfoot than in a limb without a clubfoot as seen here.

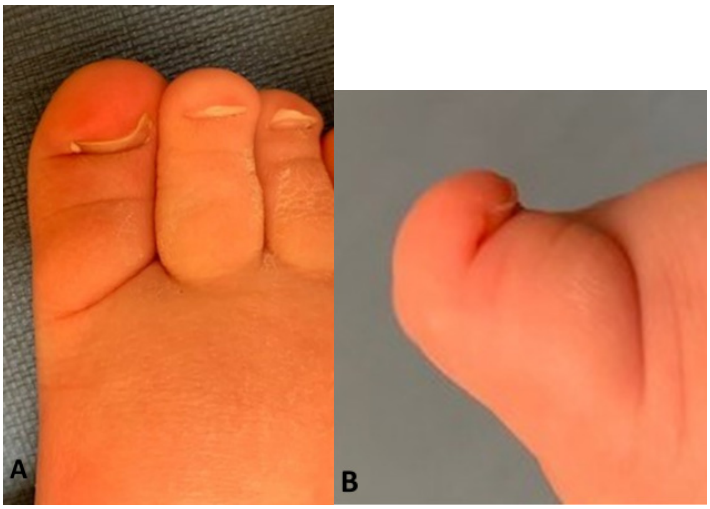

Figure 25. Fat feet are often accompanied by fat great toes. A. The abnormal fat distribution is on the dorsum of the foot and at the tip of the hallux. B. The toenail is hyper-dorsiflexed with a ski-jump appearance. Parents should be informed that the fat will resolve on the dorsum of the foot and at the tip of the hallux. The toenail will assume a normal appearance over time.

There are, on average, 10 degrees greater femoral anteversion in a limb with a clubfoot compared with the normal other limb in unilateral cases [Figure 24].

In bilateral cases, there are 10 degrees greater femoral anteversion than in sex- and age-matched normal limbs. The data on tibial torsion is controversial with some suggestion that there is greater than normal internal tibial torsion in limbs with clubfeet. The studies that I believe were well-done match my observations that tibial torsion is not associated with clubfoot. Apparent internal tibial torsion is usually residual internal rotation through the subtalar joint as a component of incompletely corrected inversion.

Fat clubfeet are often accompanied by fat great toes. The abnormal fat distribution is on the dorsum of the foot and at the tip of the hallux. The toenail is hyper-dorsiflexed with a ski-jump appearance. Parents should be informed that the fat will resolve on the dorsum of the foot and at the tip of the hallux. The toenail will assume a normal appearance over time [Figure 25].

CHAPTER 4

Clubfoot assessment, classification, and scoring

Screening

All primary healthcare providers in the US and abroad must screen all newborns and infants for foot deformities and other musculoskeletal problems, obviously after they are appropriately educated and trained. Referrals should be made to pediatric orthopedic or specific clubfoot clinics as soon as clubfoot deformities are suspected or identified. True clubfoot deformity does not correct spontaneously whatsoever. Early initiation of treatment is easier for the child and more successful than later initiation. For primary healthcare providers who are inexperienced with congenital foot deformities, metatarsus adductus and other positional foot deformities may be misdiagnosed as clubfoot. It is in the child's best interest for these children to also be referred and assessed early on by a pediatric orthopedist. Confirmed true clubfeet can then begin treatment right away and the parents of babies with other positional deformities can be reassured.

Confirming

The essential features of a clubfoot include cavus, adductus, varus, and equinus (CAVE). Increased resistance to correction increases in that order. The severity and rigidity of each segmental deformity should be assessed and documented using one of the published classification systems. For the practitioner who does not plan to evaluate and publish his/her results of treatment, it is best to use a simple system that he/she is comfortable with. The recorded features and angles do not have to be exact, but they should be reasonably reproducible by the practitioner. This will help with monitoring and assessment of progressive deformity correction and identification when no further correction is occurring. It is important to document muscle activity the best one can in an infant with a rigid foot deformity. Active dorsiflexion of the great toe following plantar-lateral foot stimulation is a good indication that it is a typical and not paralytic clubfoot.

The diagnosis of clubfoot is clinical. There is no indication for x-rays of a suspected neonatal clubfoot deformity, unless it is atypical is some way.

A complete musculoskeletal examination is strongly recommended during the first evaluation of a child with clubfoot. Although most clubfeet are isolated deformities, some are one manifestation of an underlying condition, including musculoskeletal (arthrogryposis, tibial hemimelia), neuromuscular (myelomeningocele, lipomeningocele, diastomatomyelia, caudal regression syndrome), chromosome abnormality.

There is no association of clubfoot and developmental dysplasia/dislocation of the hip (DDH). The risk for clubfoot is 1:1,000 and that for DDH is 1:1,000, therefore the risk of having both conditions is 1:1.000,000. Certainly, every newborn, with or without clubfoot, should undergo a manual hip screening exam; but there is no scientific justification for routine hip imaging in a child with clubfoot unless there is also a family history for DDH, or the child was breech.

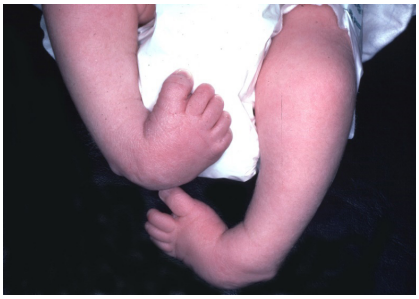

Figure 26. Bilateral idiopathic clubfoot deformities.

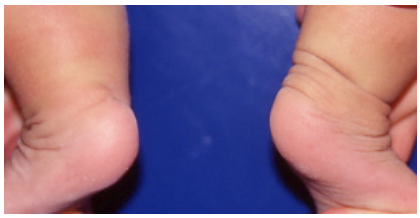

Figure 27. Left clubfoot with single posterior ankle skin crease. Normal right foot with multiple normal posterior skin creases.

Classifying the clubfoot according to type and/or etiology (not severity or rigidity)

Clubfoot is classified into etiologic categories. This classification is made to plan etiologic-specific management and establish etiologic-specific prognosis. The classification of a clubfoot may change with time depending on the effects of management.

Typical, or idiopathic, clubfoot

This is the classic and most common type of clubfoot. It is an isolated condition found in an otherwise normal infant. It is a genetically programmed deformity, but the affected child is usually the first in the family to manifest the deformity. He or she may not be the last. The 4 segmental deformities (Cavus, Adductus, Varus, and Equinus) are present and not passively correctable, although there will be varying degrees of mild to moderate flexibility of some components on initial evaluation [Figure 26].

There is a single, deep posterior ankle skin crease in the typical severe, rigid clubfoot and, perhaps, 2-3 deeper than normal posterior ankle skin creases in less rigid deformities [Figure 27].

The child can actively dorsiflex the great toe when the foot is stimulated. Management of this deformity is the focus of this monograph.

Positional clubfoot

The 4 segmental deformities are present, but completely or almost completely passively correctable. There are multiple normal posterior ankle skin creases. All muscles appear to be strong. This deformity is flexible and is thought to be due to intrauterine crowding rather than the result of genetic coding. Correction often occurs spontaneously within the first few weeks of life, but some benefit from one or two castings. Bracing is rarely indicated.

Delayed treated clubfoot

If treatment of idiopathic clubfoot is delayed beyond 6 months of age either because of delayed diagnosis or poor access to care, management becomes progressively more difficult and prolonged. With application of Ponseti principles and techniques, full correction is still possible even into late childhood. If correction is incomplete and residual deformity is unacceptable, soft tissue and/or bony surgery is required to complete the correction.

Recurrent, or relapsed, typical clubfoot

This may occur whether the original treatment was with the Ponseti method or other non-operative or operative method. Relapse after initial Ponseti management is less common than after other forms of treatment and, when observed, is usually due to poor compliance with bracing, premature discontinuation of bracing, and/or muscle imbalance. Equinus is the last segmental deformity to correct and the first to recur. This is followed by supination/inversion of the hindfoot. The deformities are first dynamic but then become fixed over time. Management of relapses is discussed in Chapter 9.

Alternatively treated typical clubfoot

These include feet that were treated with surgery or non-Ponseti casting.

Atypical, or complex, clubfoot

This deformity is characterized by severe equinus, a deep transverse soft tissue crease across the entire plantar midfoot associated with exaggerated plantar flexion of all metatarsals, and hyperextension/clawing of the great toe [Figure 28].

The term "atypical" refers to those that are present at birth. The term "complex" refers to those that develop during serial casting, often after the first 1-3 casts. The atypical or complex clubfoot appears taller at the instep than it is long from heel to toes. The adductus and varus are usually already corrected or over-corrected when the atypical clubfoot develops. The etiology is unknown, but they are often seen in especially fat clubfeet and those that slip out of their casts. They are often associated with swelling, redness, hypersensitivity, and warm shiny skin - all features of reflex sympathetic dystrophy. They require early identification and temporary discontinuation of casting. Within 1-2 weeks, the swelling, redness, hypersensitivity, and warm shiny skin resolve. It is then appropriate to reinitiate casting with the so-called "double thumbs technique" described by Ponseti [Figure 29].

These casts tend to slip off due to the abundant soft tissue envelope in which there is almost equal length-to-thickness of the foot. Dr. Ponseti recommended applying the above-knee portion of the cast in 110 degrees flexion to prevent slippage. An alternative method that avoids popliteal fossa skin crease

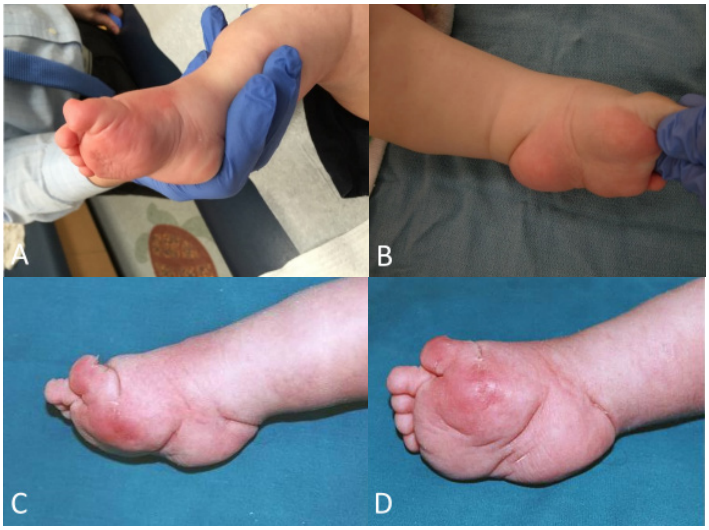

Figure 28. Atypical, or complex, clubfeet. There are equinus and trans-tarsal cavus with a deep plantar crease across the entire midfoot and, typically, clawing of the great toe. Note the iatrogenic flattened heel pad in C and D.

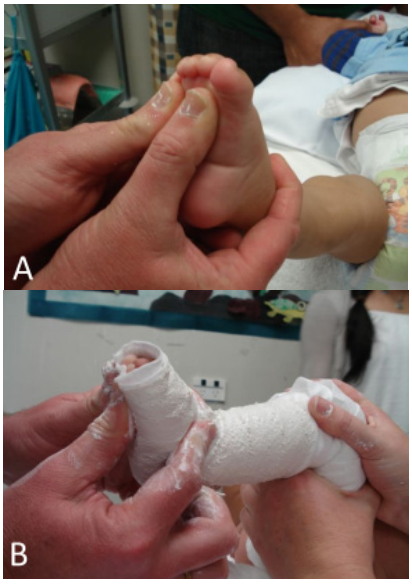

Figure 29. Double thumbs casting technique for atypical, or complex, clubfoot. All the metatarsals are plantar flexed, i.e. trans-tarsal cavus. One thumb pushes upwards under the 1st metatarsal head and one under the 5th metatarsal head to dorsiflex the entire forefoot in relation to the hindfoot against the resistance of the contracted tendo-Achilles. Since the adductus and varus/inversion are typically already corrected, avoid abduction/eversion molding beyond neutral. The deformities are usually just equinus and cavus, i.e. deformities in-series in the sagittal plane.

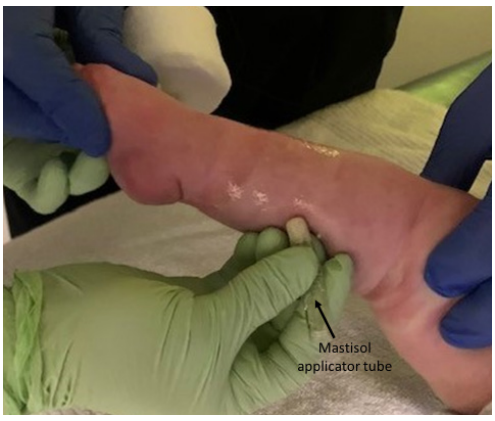

Figure 30. Mastisol liquid adhesive being applied to the skin to prevent slipping of the cast off the short, fat leg.

irritation is the use of Mastisol, or other skin adhesive, on the skin [Figure 30].

A tendo-Achilles tenotomy is necessary in most cases. Do not perform the TAT until the trans-tarsal cavus is corrected, or you might convert equino-cavus to calcaneo-cavus. The tendo-Achilles contracture acts as a post against which to stretch the extremely tight plantar soft tissues. Even in this scenario, it is sometimes necessary to change casts at weekly intervals after the tenotomy to gain more ankle dorsiflexion if sufficient dorsiflexion is not achieved immediately after the tenotomy.

The thigh-foot angle should be no greater than 30 degrees external during casting and after the TAT. 30 degrees of external rotation is also recommended in the foot abduction brace. The follow-up management remains the same.

These feet take more casts to correct and have higher rates of recurrence than typical clubfeet.

Syndromic clubfoot

Other congenital abnormalities are present. The clubfoot is part of a syndrome, such as dystrophic dysplasia, Möbius syndrome, Larsen syndrome, Wiedemann-Beckwith syndrome, and Pierre Robin syndrome. Ponseti management remains the standard of initial care, but it may be more difficult to achieve correction as rapidly or successfully as in typical clubfoot and recurrence is more likely. The long-term outcome usually depends more on the underlying condition than the clubfoot itself.

Teratologic clubfoot – congenital synchondrosis, arthrogrypotic

These include "clubfeet" with congenital tarsal synchondroses and arthrogrypotic clubfeet.

Those with a ***talocalcaneal tarsal synchondrosis*** are seen in limbs with *tibial hemimelia* and occasionally those with *hemi-atrophy*. They will not respond to casting, because there is no subtalar joint. They are often best treated with a Syme amputation [Figure 31].

Arthrogrypotic clubfeet are either very stiff or extremely stiff and all are resistant to casting. They typically develop in the first trimester and do not move throughout gestation, thereby being quite stiff at birth [Figure 32].

Nevertheless, Ponseti management should be used initially. Expect more of these to require a la carte surgery and a high recurrence rate, even with day and nighttime bracing. The magnitude of the surgery will be less as a result of the preliminary Ponseti casting. Minimally invasive percutaneous releases with ongoing serial casting should be used in those feet that respond so minimally to casting that even a talectomy might result in the inability to suture the wound edges together at the end of the procedure [Figures 33 and 34].

Neurogenic clubfoot

These are associated with a neurological disorder such as meningomyelocele, lipomeningocele, caudal regression syndrome, and diastomatomyelia [Figure 35].

Insensate skin increases the risk of cast pressure-induced ulcers in these typically severe and rigid deformities. Apply more padding than usual and avoid excessive pressure in molding. If the feet are very fat, cast slipping will result in painless deep ulceration. Use Mastisol on the skin to prevent slippage [Figure 30]. If little improvement is occurring, it

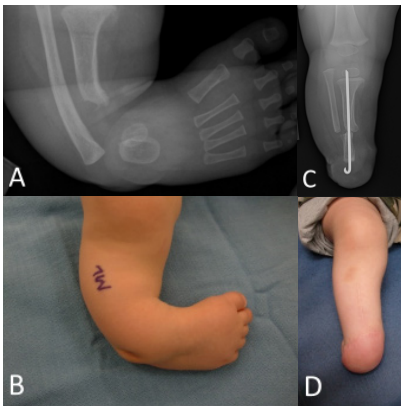

Figure 31. Incomplete tibial hemimelia with club-type foot deformity. A. X-ray. B. Clinical appearance. C. X-ray after Syme amputation of the foot and tibia-fibula synostosis. D. Clinical appearance postoperatively. The hindfoot was sectioned in the coronal plane. There was a complete subtalar synchondrosis.

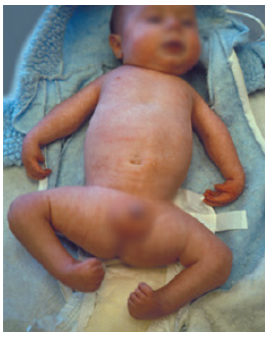

Figure 32. Clubfeet in baby with arthrogryposis.

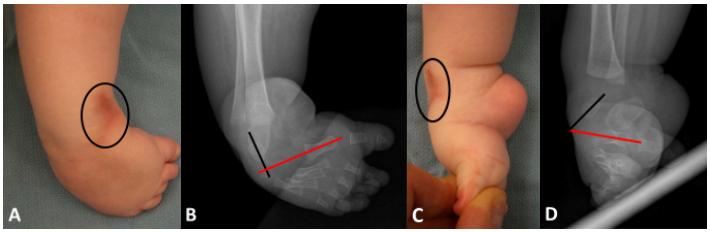

Figure 33. Severe, rigid, resistant arthrogrypotic clubfoot. Residual deformity after 14 long leg casts. A. Pre-op front view with simulated weightbearing. Healed recurrent pressure sore in black oval. B. Pre-op AP foot x-ray with simulated weightbearing. Axis of talus (black line) and axis of 1st metatarsal (red line) reveal the severe residual deformity. C. Pre-op medial view with maximum dorsiflexion force. Healed recurrent pressure sore in black oval. D Pre-op AP foot x-ray with simulated weightbearing. Axis of talus (black line) and axis of 1st metatarsal (red line) reveal the severe residual deformity.

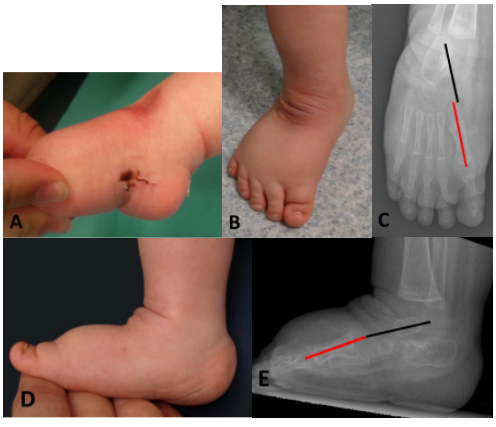

Figure 34. A. One week after percutaneous tenotomies of tendo-Achilles and long toe flexors, as well as mini-open plantar fasciotomy and tibialis posterior tenotomy in the foot in Figure 29. B to E after 4 more casts: B. Simulated standing top photo. C. Simulated standing AP x-ray. Axis of talus (black line) and axis of 1st metatarsal (red line) reveal full correction of deformities. D. Medial photo with maximum dorsiflexion. E. Lateral x-ray with maximum dorsiflexion. Axis of talus (black line) and axis of 1st metatarsal (red line) reveal full correction of deformities.

may be best to wait several months until the foot is longer and thinner before reinitiating casting. Surgery on short, fat, severely deformed clubfeet may result in full deformity correction before wound closure, but the thick layer of fat on the dorsal foot and anterior lower leg may impinge on each other making maintenance of deformity correction with bracing unsuccessful. Then, after the fat naturally diminishes, there will be recurrent/residual deformity and surgical scar tissue that will make subsequent deformity correction more difficult. In these rare situations, it might be best to brace in the best position achieved after initial casting and early TAT. Then resume Ponseti management later. The ultimate outcome will be better. Talectomy is not a panacea. It is a destructive operation that does not always achieve a plantigrade foot, and recurrent deformity is possible-to-likely. There is no next operation after a talectomy other than "ankle" fusion or amputation.

Acquired clubfoot

A good example is the clubfoot associated with Streeter dysplasia, a.k.a. amniotic band syndrome, when one or more deep fibrous bands encircle the lower leg [Figure 36].

These feet may appear to be typical clubfeet with rigid CAVE deformities and a single posterior ankle crease; but sometimes they have normal posterior ankle creases yet rigid CAVE deformities. The first group probably represents a typical genetic clubfoot with an unrelated acquired banding. The latter group likely represents a genetically normal foot that developed the clubfoot deformities secondary to constriction from the amniotic band later in gestation. Ponseti management should be employed in both scenarios. These limbs typically require surgical release of the deep band prior to casting. Without preliminary band release, even the minor swelling that accompanies serial casting can result in venous and lymphatic congestion distal to the band. That creates exaggerated pain and swelling and could result in foot necrosis [Figure 37].

Ponseti casting can usually begin safely a few weeks after the released band has healed [Figure 38].

Good correction of deformity can usually be achieved, but some may have permanent weakness of some or all muscles due to the constriction/compression. These may require permanent day and nighttime bracing (with an AFO) and occasionally non-typical tendon transfers.

In cases in which the foot distal to an amniotic band is malformed and unrecognizable as a foot, a Syme amputation is recommended [Figure 39].

These malformations likely resulted from amniotic banding early in gestation. Preliminary amniotic band release is recommended in this scenario as well or else the post-Syme amputation swelling could cause distal vascular congestion and necrosis of the heel pad.

The clubfoot from Streeter dysplasia is acquired prenatally. Also potentially included in the category Acquired Clubfoot is a postnatally acquired "clubfoot", i.e. normal at birth but then develops cavus, adductus, varus, and equinus. These are more appropriately called equino-cavovarus. They result from underlying neuromuscular disorders, such as cerebral palsy, Charcot-Marie-Tooth disease, and tethered cord. They should be managed based on the disease-specific principles

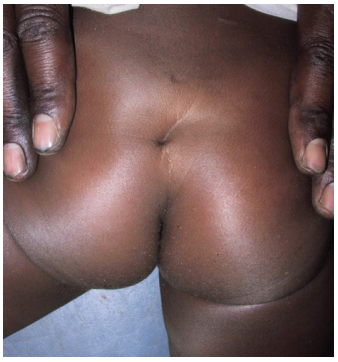

Figure 35. Myelomeningocele after closure

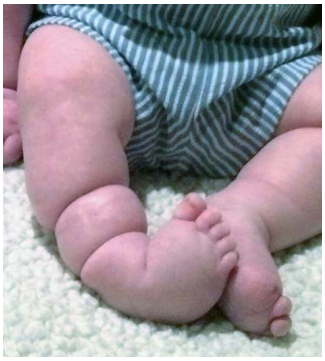

Figure 36. Right clubfoot distal to 2 deep Streeter/amniotic bands.

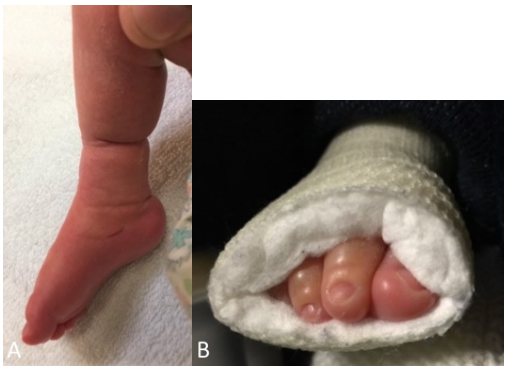

Figure 37. A. Deep Streeter band proximal to mild clubfoot. B. Swollen, shiny toes 2 days after Ponseti cast application. The cast was removed and reinitiated several weeks after surgical release of the amniotic band. The deformity corrected completely and without issues after 6 casts and a TAT.

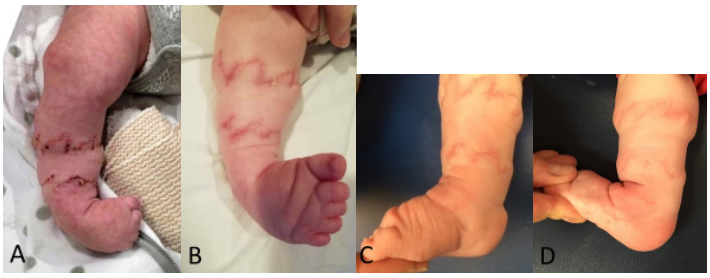

*Figure 38. A. 1 week after the 4-month-old in Figure 36 underwent simultaneous release of both Streeter/amniotic bands. B. 2 months after the releases, the scars were well healed and there was excellent vascularity of the limb. Ponseti casting was initiated. **NOTE**: The dark coloration of the foot is merely a shadow inadvertently created by the photographer. C. Ready for percutaneous tendo-Achilles tenotomy after 5 weekly casts. D. 2 years later. Excellent shape, flexibility, and muscle balance, although many such feet have muscle weakness and imbalance due to constriction/compression.*

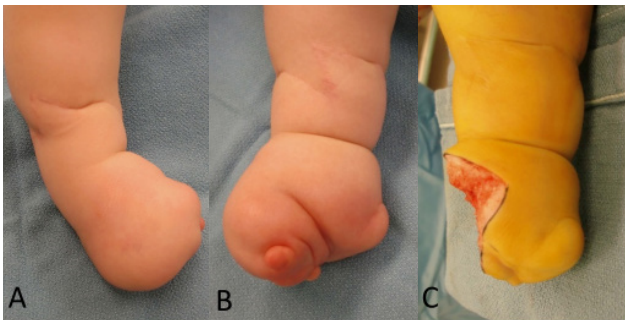

Figure 39. A. Frontal view of clubfoot-type malformation distal to an amniotic band that has already been released. B. Medial view. C. Intra-operative medial view during Syme amputation.

of management that might include preliminary Ponseti-type serial casting.

Clubfoot Scoring – classifying according to severity and/or rigidity

The need for clubfoot scoring is controversial. There are, and have been, proponents and detractors. Dr. Ponseti was one of the latter. There are scoring systems for many musculoskeletal conditions that have been developed to assess severity of a condition, determine prognosis, guide the choice of treatment options, and assess serial and/or final effects of treatment. There have been many proposed clubfoot scoring systems, the most accepted of which are the Pirani score and the Dimeglio score. They both attempt to objectively rate severity of clubfoot deformity and assess serial and final effects of treatment. Proponents find serial scoring useful in classifying the severity of the clubfoot, assessing progress and completion of treatment, and identifying signs of relapse. And, from an academic standpoint, scoring systems add some data to research studies that could otherwise not be objectively evaluated and compared.

However, neither score, nor any other, accurately determines prognosis in regard to duration of treatment, number of casts required (in regard to the Ponseti method), the need for tendo-Achilles tenotomy or anterior tibial tendon transfer, duration of bracing, or the risk for deformity relapse. Neither indicates the need for a different treatment option. Those practitioners who use the Ponseti method use it for all comers just as those who use the French method use it for all comers.

The Pirani score has a ceiling effect, but it's fast and easy to use and it is reasonably reliable. The Dimeglio score is more granular, but fairly subjective when attempting to objectively assess multiple angles in short, fat clubfeet. In addition, it is difficult to understand the proper way to measure certain angles.

It is, no doubt, valuable for all practitioners to assess the severity of clubfoot deformities and follow their progress in deformity correction using one of the established scoring systems or their own. Obviously, using any other than one of the two systems named here will not result in publishable outcomes data, but that's OK for most practitioners. It's also OK for a practitioner to be personally consistent in the use of his/her system or interpretation of a recognized system even if not consistent with others who might assess their patients. This is to say that absolute values are less important than trends.

And scoring certainly has value in being able to provide parents with a relative frame of reference for the severity of their child's clubfoot and general expectations for duration of casting. Recall that the duration of casting cannot be accurately predicted by any of the published scoring systems. A simple and straightforward clubfoot scoring system to share with parents *could* be "his/her foot/feet is/are [*pick one* – mild/moderate/severe], so I expect he/she will require [*pick one* – 4/6/8 casts]. There is a 90% chance that a tendo-Achilles tenotomy will be required after [*pick one* – the 4th/6th/8th cast] and a 40% chance that an anterior tibial tendon transfer will be needed after age 3 years".

Pirani Score

The Pirani Clubfoot Score documents the severity of the deformity and sequential scores are an excellent way to monitor progress [Figure 40].

Method Use six clinical signs to quantify severity of each component of the deformity. Each component is scored as 0 (normal), 0.5 (mildly abnormal) or 1 (severely abnormal). Record each score and the sum of the scores at each clinic visit.

Progress assessment During Ponseti management, the record shows whether the deformity is correcting normally or whether there is a problem, and the degree of correction of each component of the clubfoot. The score also helps in deciding when to perform a tendo-Achilles tenotomy.

Dimeglio Score

The Dimeglio Clubfoot Score provides an additional method of assessing each component of the clubfoot deformity [Figure 41].

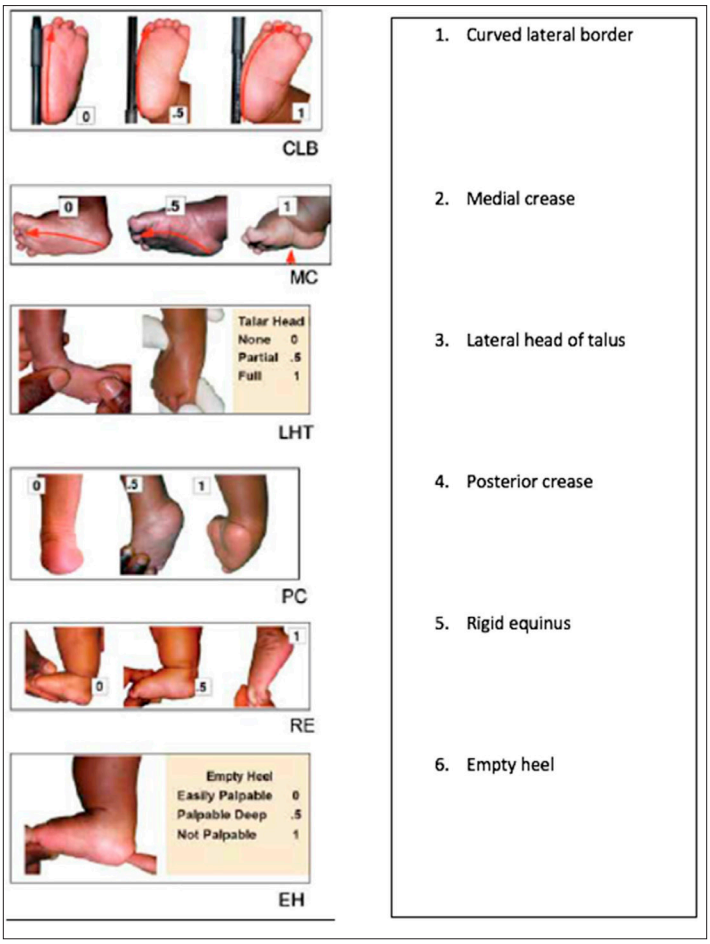

Figure 40. Pirani Score. 0-6 scale.

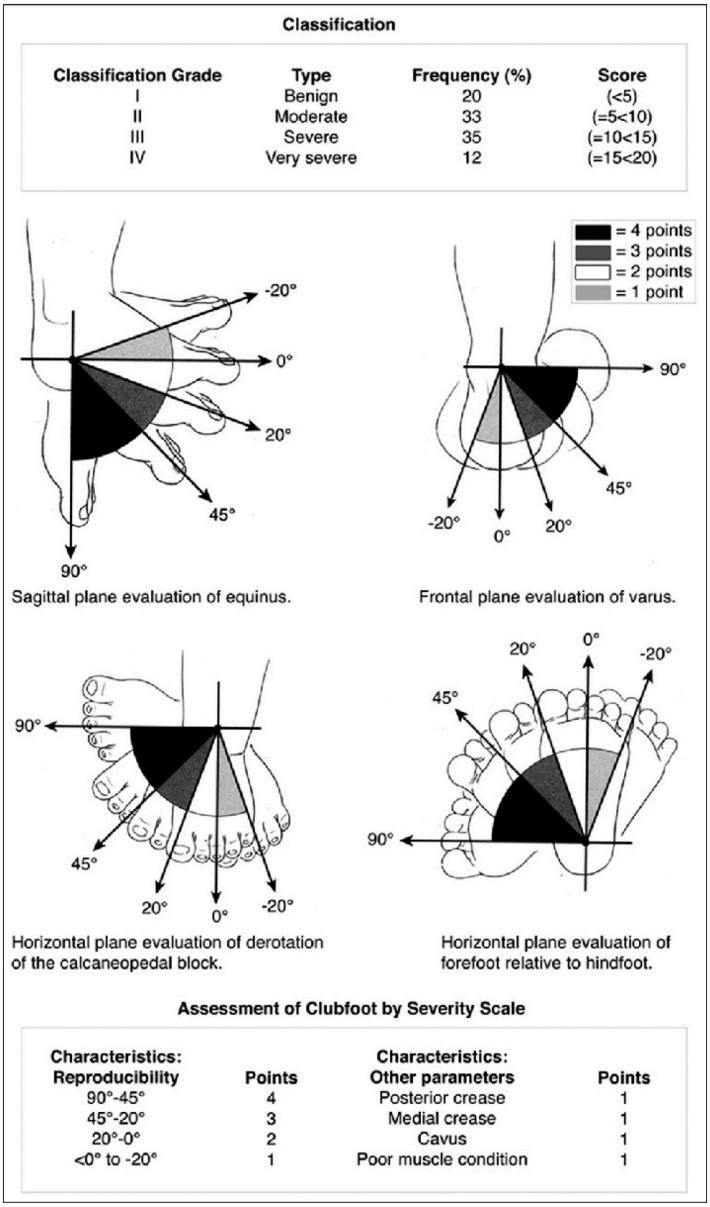

Figure 41. Dimeglio Score.

CHAPTER 5

Ponseti manipulation and cast correction

Setup

The child should be made to feel calm and comfortable by lowering the lights in the room, avoiding loud noises, and playing soft, soothing white noise or background music, typically on a cell phone or tablet. Start with a clean diaper. The child should be recently fed by breast or bottle, burped, and content. Some prefer to feed the child during casting, but I prefer using a 24% sucrose solution applied to the baby's pacifier as needed during the casting. It is an effective mild analgesic. Swaddling is also an effective calming technique for most infants.

When possible, have a trained assistant, such as a fellow, resident, physician assistant, nurse/nurse practitioner, orthopedic technician, or medical assistant. Sometimes it is necessary for the parent to assist, but this is not recommended.

The child may be on the parent's lap or on an exam table. I prefer the latter for logistical and access reasons. The child's feet are at the end of the table. The parent is next to the child's head for soothing and comforting, including application of the sucrose to the pacifier. [Figure 42].

Figure 42. Swaddled clubfoot infant on examination table in low lit room with cast technician at the end of the table and the health care provider on the lateral side of the affected foot. Mother is on the opposite side of the table at the baby's head soothing the baby with a pacifier, sucrose solution, and playing calming music on a cell phone or tablet.

Manipulation

Before manipulating and casting a clubfoot, it is important to study in detail the section of this monograph entitled "Terminology/pathoanatomy/biomechanics" on pages 8-14. The best chance for success is based on a complete understanding of the pathoanatomy of the deformity and the biomechanics of the deformity and deformity correction.

Precisely locate the head of the talus. This step is essential, because the dorso-laterally prominent talar head is the fulcrum for deformity correction. First, palpate the lateral malleolus with the thumb of your hand on the lateral side of the affected foot and ankle – your left thumb on the baby's right clubfoot and vice versa [Figure 43A]. Slowly slide your thumb from the lateral malleolus antero-distally. You may encounter 1, 2, or 3 bony prominences. In a severe clubfoot, the first bony prominence encountered is the lateral dome of the talus, that is exposed due to severe ankle equinus [Figure 43B]; the second bony prominence is the talar head, that is exposed due to plantar-medial displacement of the navicular as a component of inversion of the subtalar joint [Figure 43C]; and, in some cases, a third bony prominence can be palpated plantar to the talar head. It is the beak of the calcaneus, and it is exposed due to plantar-medial deviation of the calcaneo-cuboid joint [Figure 43D]. In mild-moderate clubfoot deformities, the first bony prominence distal to the lateral malleolus is the head of the talus [Figure 43C].

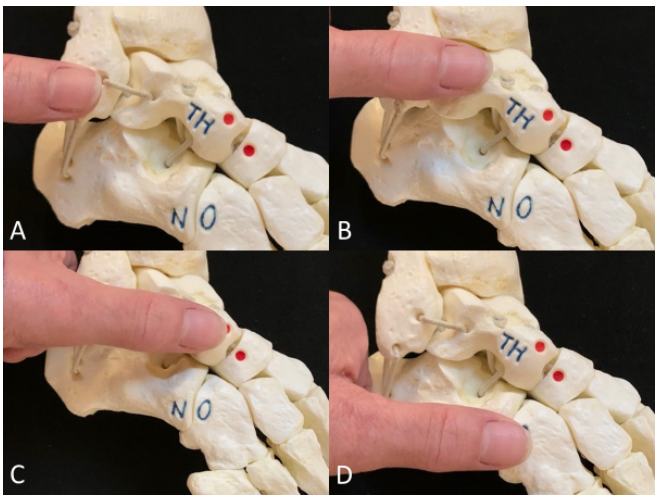

Figure 43. Thumb on 4 possible bony prominences on the lateral ankle and hind-midfoot. A. Lateral malleolus – the desired first bony prominence identified. B. Dome of talus – occasionally identified, particularly in those clubfeet with very severe equinus deformity. C. Head of talus – the thumb covers the letters "TH" (shorthand for thumb) that identify this as the desired fulcrum point for clubfoot deformity correction. D. Beak of calcaneus – the pressure point to AVOID when correcting clubfoot deformity, therefore the word "NO" at that location. This is the lateral pressure point for cast correction of isolated metatarsus adductus.

Place your ipsilateral index and/or long fingertips on the medial malleolus around the back of the ankle – NOT on the medial side of the calcaneus. Stabilize the forefoot with your other hand by holding the 1st and 2nd metatarsal heads with your thumb dorsal and your index and long fingertips plantar [Figure 44].

Manipulations should be done gently making all efforts to avoid creating pain, particularly if that pain represents soft tissue injury. Forceful manipulations will not only make the baby cry but will tear the collagen (rather than stretch it) creating soft tissue trauma with its associated inflammation, swelling, bleeding, and subsequent scar tissue formation. That tissue trauma is similar in kind, though perhaps not in magnitude, to the tissue trauma caused by extensive surgery.

The collagen will stretch maximally according to the viscoelastic property of creep within 30-60 seconds. Make sure your hands are warm. Watch the child's face. Apply the forces slowly and gently backing off a little if the baby starts to fuss or cry. Then reapply the forces more slowly and gently. Babies cry for many reasons including hunger, upset stomach, soiled diaper, fatigue, and pain. Control what you can and it will be the best possible experience for the baby and the family – and for you and your cast technician.

All components of clubfoot deformity, except for the ankle equinus, are corrected simultaneously, although the focus starts in the forefoot and moves to the midfoot and then the hindfoot in that order. The first step is correction of the cavus deformity by positioning the forefoot in proper alignment with the hindfoot. The forefoot and hindfoot are rotationally malaligned in opposite directions from each other. The cavus, manifest as a high medial arch, is due to pronation of the forefoot in relation to the hindfoot within the calcaneo-pedal unit (CPU). With your thumb on the dorso-lateral aspect of the talar head and ipsilateral index and/or long fingertips on the medial malleolus, your other hand grasps the forefoot and elevates the first ray thereby supinating it in relation to the hindfoot. Gentle counter-pressure on the talar head stabilizes the entire foot and enables this to occur [Figure 44].

The cavus deformity is the supplest of all 4 deformities and merely requires elevating/dorsiflexing the first ray of the forefoot to stretch the plantar fascia and achieve a normal longitudinal foot arch. It rarely takes more than 2 casts to correct this deformity. The forefoot is supinated to the extent that visual inspection of the plantar surface of the foot reveals a normal appearing arch - neither too high nor too flat. Alignment of the forefoot with the hindfoot to produce a normal arch is necessary before correcting the adductus and varus.

As the cavus is being corrected in the first 1-2 casts, adduction of the midfoot is simultaneously being corrected by abduction and increasing outward rotation forces against the talar head. Although the true fulcrum for midfoot adduction deformity correction is the lateral midfoot (calcaneocuboid joint), the subtalar joint rigidity enables the talar head to act as its proxy. This is fortunate because the talar head is the true fulcrum for the most significant deformity within the clubfoot, subtalar joint inversion. When the cavus and adductus deformities within the CPU no longer exist, all the bones in the

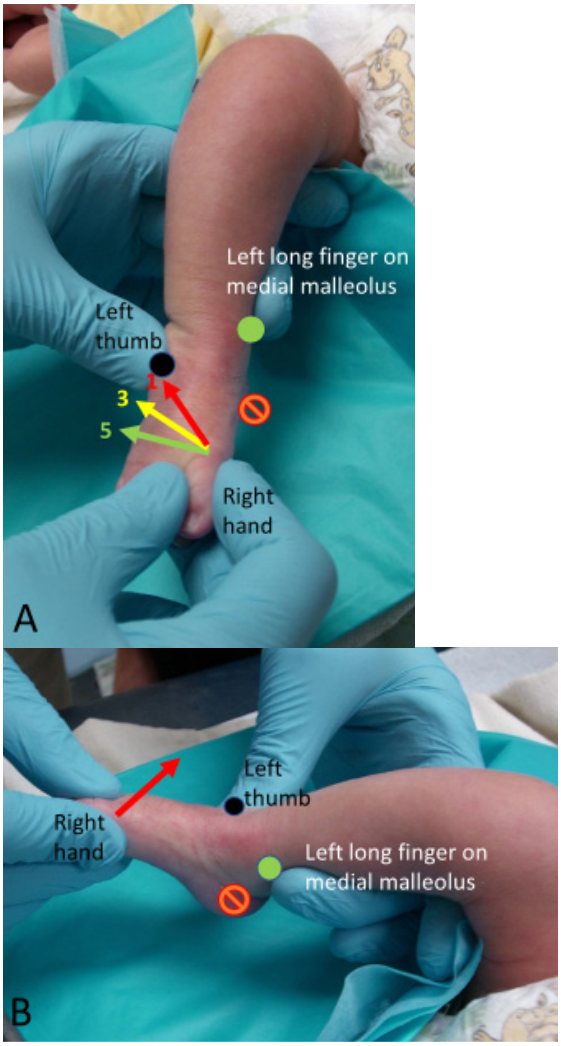

Figure 44. A. Left thumb on dorso-lateral head of right talus (black dot). Left index and/or long finger around the back of the ankle on the medial malleolus (green dot), never the medial side of the calcaneus (NO symbol). Right thumb, index, and long fingers stabilize the forefoot. Red arrow shows initial direction of supination/abduction/eversion force on the forefoot. The thumb on the talar head is the fulcrum against which the corrective forces are applied. Yellow arrow shows direction of force during 3rd manipulation as pronation/adduction/inversion deformities begin to correct. Green arrow shows direction of force during 5th manipulation as all deformities further correct. The navicular and the entire CPU rotate under and around the head of the talus. B. Medial view of foot with red arrow showing direction of right-hand supination/abduction/eversion force against left thumb on head of talus and with left long fingertip on medial malleolus, never the medial side of the calcaneus (NO symbol).

CPU can be considered as 1 bone that must be everted under the talus using the talar head as the fulcrum. Eversion will occur naturally following the oblique axis of the subtalar joint by externally rotating the forefoot against counterpressure on the head of the talus. Care must be exercised to avoid applying to much pressure to the skin over the talus and creating injury either with the manipulations or casts. Through all subsequent manipulations and casts, the CPU is maintained in slight but decreasing degrees of supination in relation to the leg. It is only when the subtalar joint is fully and slightly over-corrected and the talar head no longer palpable that the transverse plane of the metatarsal heads is perpendicular to the tibia [Figure 45].

Intentional and forced ankle dorsiflexion manipulation and

casting are to be avoided. The collagen in the tendo-Achilles is the most resistant to manipulation and casting of all the contracted soft tissues. And since the cavus is corrected to planus with the first 1-2 casts, there is high likelihood that the midfoot will over-correct to a breech rocker-bottom deformity with forceful attempted dorsiflexion of the ankle. Usually the ankle is in neutral (0 degrees) dorsiflexion after 6 casts that focused on and achieved full correction of cavus, adductus, and varus. That is the indication for tendo-Achilles tenotomy. The need for the tenotomy is not due to a failure of the method. It is expected to be needed in 90-95% of cases. Iatrogenic breech deformity created in an attempt to avoid the tenotomy is a failure in the application of the method.

Steps in cast application

Dr. Ponseti recommended the use of plaster of Paris because it is was the only casting material available for the first several decades of his clinical practice. When fiberglass, and in particular semi-rigid fiberglass casting tape, was invented in the late 1980's to early 1990's, Dr. Ponseti saw no reason to start using it. Plaster is less expensive, readily available around the world, and more precisely molded by practitioners inexperienced with its use. However, there are many advantages to the use of semi-rigid fiberglass casting tape. It is neat and clean to apply and remove. It does not generate heat when hardening and it is removed by unraveling rather than with a saw or knife. Therefore, the padding can be very thin thereby helping with precise molding. Skin injury from cast removal tools is avoided. Parents merely unravel the cast an hour before they leave home for their next appointment. No wasted time or inefficient room utilization in clinic soaking and/or cutting the cast off. Appointments take no longer than 15 minutes. Parents bathe the child quickly after removing the cast and before leaving home. Published studies as well as extensive personal experience using semi-rigid fiberglass casting tape indicate that, in experienced hands, it has equal efficacy and safety to plaster. The key for success with semi-rigid casting tape is using 1" cast padding and casting tape. The heel fat pad will be crushed and the molding will be inaccurate with 2" tape. Compared with plaster, it may take an extra minute for the tape to harden completely without losing its mold. Regardless of the cast material used, the cast must be applied in 2 sections, a short leg section up to the knee followed by extension to the hip. And once a well-molded cast is hard, the clubfoot has no idea what it's made of!

Preliminary manipulation - Before each cast is applied, the foot is manipulated for 30 to 60 seconds as described in detail above.

Holding the foot during casting – The surgeon can either hold the foot while the cast technician rolls the cast or vice versa. I prefer to hold the foot, having personally trained a few of the cast technicians in my department in the proper method to apply the casts. Holding the foot in the proper position is more important and harder to teach than is cast application. And cast application is what cast technicians do for a living.

Stand on the lateral side of the affected foot and side of the table with the cast technician at the end of the table. A parent should be on the opposite side of the table next to the baby's

Figure 45. A. Clubfoot cast models showing gradual and successive forefoot supination-to-neutral. B. Casted foot matching final clubfoot cast foot model just prior to tendo-Achilles tenotomy.

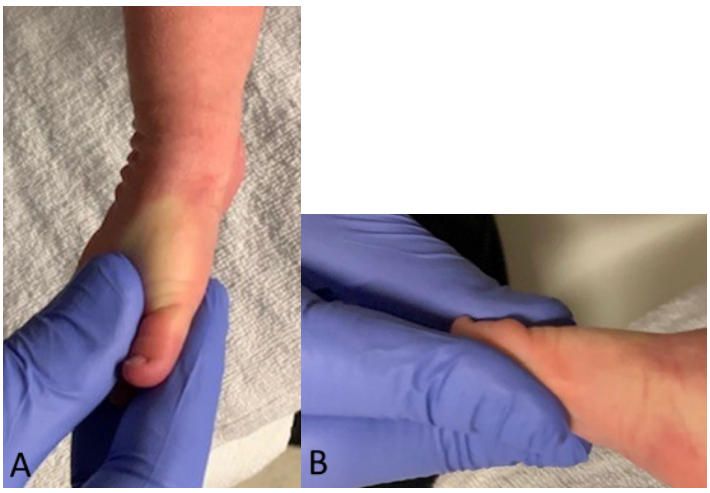

Figure 46. Holding the forefoot during cast application. A, B. Tips of thumb, index, and long fingers create a tripod at the metatarsal heads. Fingers and thumb are parallel, and fingers are slightly wider than metatarsal heads.

head. The parent or another individual might need to hold the other foot out of the way to prevent the baby from kicking the foot/leg being casted with the other foot [Figure 42].

The forefoot is held and stabilized with your right hand for a right clubfoot and vice versa. The tip of your index finger is placed under the distal end of the 1st metatarsal head, the tip of your long finger under the distal end of the 5th metatarsal head, and the tip of your thumb over the distal end of the 3rd metatarsal head, thereby making a triangle. The distal phalanges of your fingers and thumb should be almost parallel so as not to take up too much room under the casting materials. Do not hold the toes, but do not hold the forefoot so far proximally that there will be a large gap between the foot and the cast when your fingertips are removed. You merely need control of the forefoot. Your index and long fingers should be far enough apart to ensure that the padding and cast material are wrapped around them and do not compress the toes medial-lateral [Figure 46].

Flex the hip 90 degrees and hold the 90-degree flexed knee with your other hand (left hand for a right foot). Apply a medially directed hip adduction force with that hand. Simultaneously, externally rotate the leg at the knee while supinating the forefoot, abducting the midfoot, everting the hindfoot, and ever so slightly dorsiflexing the ankle with the hand that is holding the forefoot. These opposite direction forces at a distance away from each other simulate the forces applied during direct manipulation of

the foot. The medially directed force and external rotation at the knee are proxies for the pressure point over the head of the talus. These forces do not exactly match the soft tissue stretch achieved with both hands on the foot, but they enable near maximal eversion force in the subtalar joint against the tibia that is prevented from externally rotating further by the collateral ligaments at the knee.

This phenomenon can be explained by the work of Huson, who identified a kinematic opposite direction linkage between rotation in the tibia and the subtalar joint. External tibial torsion and inversion/internal rotation of the subtalar joint are associated, as are internal tibial torsion and eversion/external rotation of the subtalar joint. The tibia, in this situation, is gently slightly externally rotated in relation to the femur. The collateral ligaments of the flexed knee prevent the tibia from externally rotating beyond a certain point. When the subtalar joint is forcefully externally rotated/everted, the tibia does not externally rotate further at the knee, so it effectively acts as if it is internally rotated in relation to the subtalar joint, exactly as Huson indicated. It is this biomechanical principle that further justifies the need for the long leg cast and the marked external rotation that is gradually achieved with the long leg casts and maintained with the foot-abduction brace.

Holding the foot and leg in this relationship during cast application also helps avoid applying excess padding and casting materials over the talar head/sinus tarsi that might otherwise block full correction during molding and/or create a pressure sore there. The reason for not having a thumb over the talar head during cast application is Pauli's Exclusion Principle. It states that 2 objects cannot be in the same place at the same time. Removing and replacing your thumb with every turn of padding and casting material does not maintain maximum deformity correction as well as the maneuver described earlier in this paragraph. It is also awkward to do so. Once the materials are in place, your thumb can be applied to the talar head over the materials [Figure 47].

Applying the padding – Apply only a thin layer of cast padding to allow precise molding of the deformities. The padding must extend beyond the ends of the toes, particularly under their plantar surfaces. Use 1-inch padding, if available, or cut a larger roll down to this size. No more than 2-3 layers of padding are needed if semi-rigid fiberglass casting tape is used, because the material hardens without the heat of an exothermic reaction that accompanies the hardening of plaster. And, since the material is unraveled rather than cut off, there is no concern for accidental skin injury from a knife, scissors, or cast saw during removal.

First apply the cast padding (and the cast) up to the knee and then extend both to the upper thigh after the below-the-knee cast has hardened. The most important goal of casting is to apply precise three-point pressures on the foot and ankle. If one applies the whole long leg cast at once, it is likely that precise molding of the foot and ankle will be compromised in the attempt to simultaneously control the knee, particularly if the child is fussy and kicking. And if that occurs, bunching up of cast padding in the popliteal fossa may occur resulting in skin irritation and even necrosis [Figure 48].

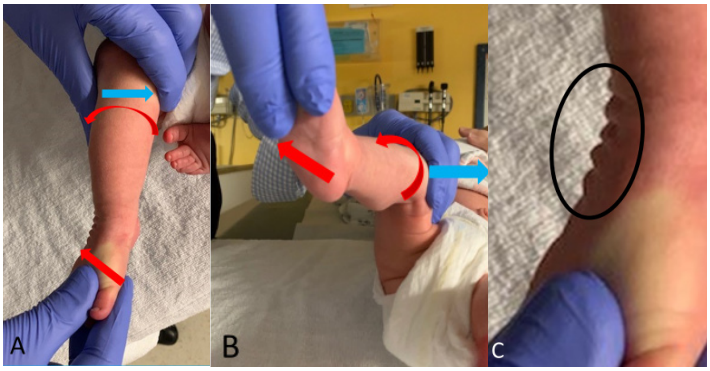

Figure 47. A. For a right clubfoot, hold the forefoot with the tips of your right thumb, index and long fingers. With the hip flexed 90 degrees, hold the 90-degree flexed knee with your left hand. Apply a medially directed force at the knee with your left hand (blue arrow). Simultaneously, externally rotate the lower leg at the knee (red curved arrow) while supinating/abducting/everting and slightly dorsiflexing the foot with the right hand (red straight arrow). These opposite direction forces at a distance away from each other simulate the forces applied during direct foot manipulation and enable applying the padding and cast materials without interference. Additionally, this external rotation of the subtalar joint against the tibia (which is prevented from rotating further by the knee collateral ligaments) helps maintain the eversion force applied at that location. B. Viewed from caudal to cephalad. C. The effectiveness of this maneuver is evidenced by the wrinkled and redundant skin over the lateral hindfoot/ankle (within black oval).

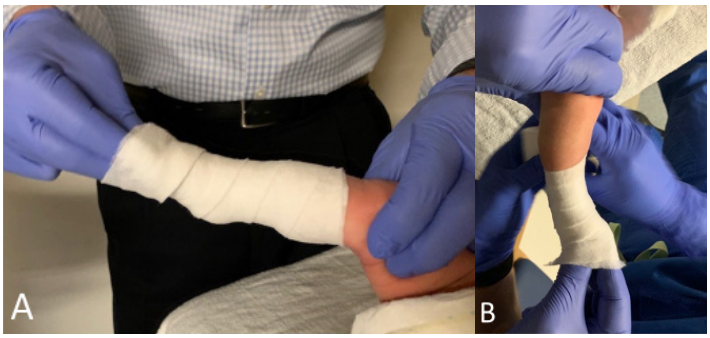

Figure 48. Below the knee cast padding extended beyond the toes.

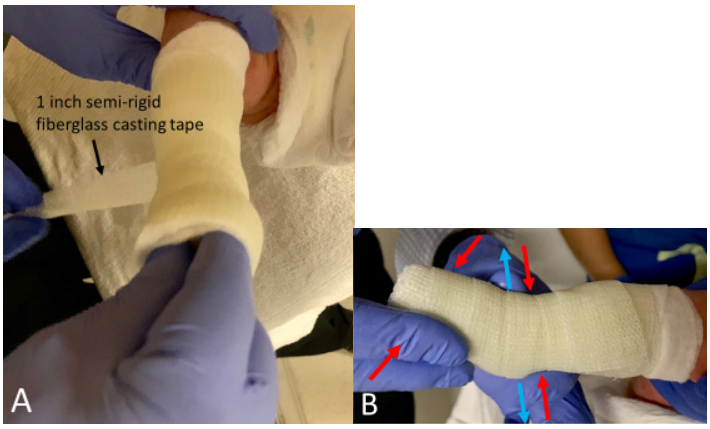

Figure 49. A. Apply the short leg cast first. Maintain finger positions on the forefoot as described in Figure 39. When using semi-rigid fiberglass casting tape, use 1" casting material to prevent inadvertent crushing of the soft tissues. Extend the cast padding and the cast material distal to the toes without compressing them medial-to-lateral. B. The casting material is wrapped snuggly around the forefoot (red arrows on forefoot), loosely around the heel (blue arrows), and snuggly around the ankle over the tendo-Achilles (red arrows at the ankle).

Applying the cast - The cast material, like the padding, must extend beyond the ends of the toes, particularly under their plantar surfaces. The reason is that the long flexor tendons of the toes must stretch farther than any of the other muscle/tendon units in the foot and ankle. They have the greatest length from origin to insertion. And, as mentioned above, keep your index and long fingers far enough apart on the forefoot to ensure that the cast material, like the padding, is wrapped around them and does not compress the toes medial-to-lateral. However, after cast application, it should look as though your fingers were never inside.

Begin with three to four turns around the toes. Then progress proximally with a few snug turns around the forefoot. Avoid wrapping snuggly around the back of the calcaneus so as not to crush and flatten the heel fat pad. Add a little tension to the turns of the casting material above the heel fat pad, i.e. over the tendo-Achilles. And then extend the cast up to the proximal lower leg.

When using plaster of Paris, use 2" soft, creamy plaster. Some plaster of Paris products are coarse and granular and do not roll well around small curved objects. When using semi-rigid fiberglass casting tape, you must start with 1" wide casting material. There is a risk of crushing the heel pad if 2" tape is used because it does not lay flat when turning corners on small objects unless excessive tension is applied [Figure 49].

When using semi-rigid fiberglass casting tape, underlap the beginning of each successive roll with the end of the previous roll. This will make cast removal easier [Figure 50].

Molding the cast – As soon as the final roll of casting material is applied, the surgeon takes control of the cast with both hands and the assistant takes manual control of the thigh. The hand positions on the foot are exactly those used during the manipulation stage. And the molding pressures applied are those necessary to match the bone and joint alignments that were achieved at the end of the manipulation stage. No additional force is applied because creep has already maximized the stretch of the collagen at that point in time.

Molding is a dynamic process in which the hands and fingers move continuously. The skin at greatest risk for excessive pressure is that over the talar head, yet that is the most important pressure point. Rather than pushing on the talar head with the tip of a thumb and creating a deep and potentially dangerous indentation, create a large concavity centered over the talar head with one's thenar eminence. The center of pressure application should be the same, but it is more evenly and gradually distributed over a greater surface area [Figure 51].

The index finger of the hand molding the hindfoot rotates medial-to-lateral behind the tendo-Achilles and above the heel fat pad. This mold helps keep the heel down to prevent the cast from slipping off. The index and long fingers of the other hand flatten the plantar surface of the forefoot while supinating/abducting/everting it against the pressure point on the talar head. Care is taken to avoid undesirable hyper-dorsiflexion of the forefoot/midfoot that could create a rocker bottom deformity [Figure 52].

With the thumb, index, and long fingers holding the forefoot as described, there will have been good control of the foot yet, after releasing the forefoot, it should look as though your fingers were never inside the cast. Those digits are finally used to flatten

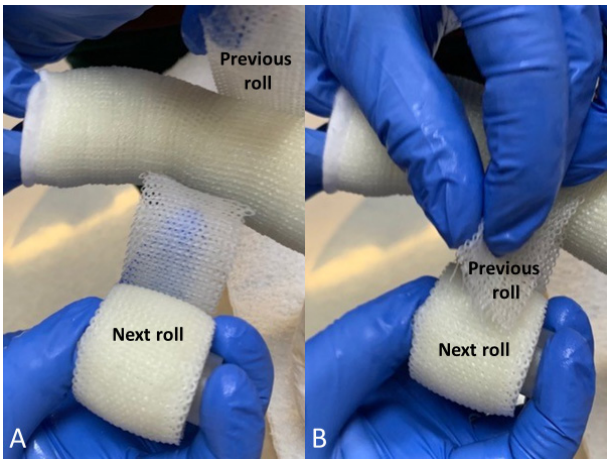

Figure 50. Start each successive roll of semi-rigid fiberglass casting tape by underlapping a short segment with the end of the previous roll.

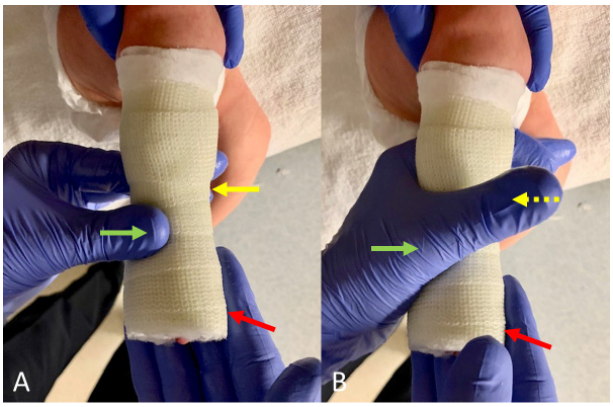

Figure 51. Hand positions for right clubfoot cast molding. A. Left thumb over talar head (green arrow). B. Left thenar eminence over talar head (green arrow) to avoid excessive potentially pressure sore-inducing force. Note that, in both images, the forefoot supination/abduction force (red arrow) and medial malleolus pressure point (yellow arrow) are the same.

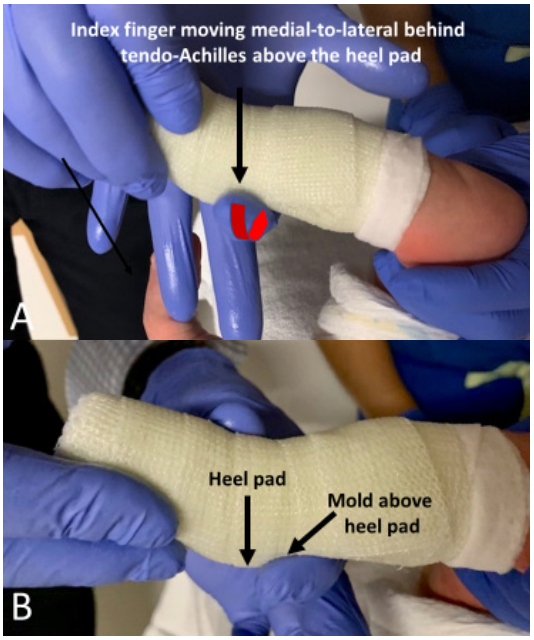

Figure 52. A. The index finger rotates medial-to-lateral behind the tendo-Achilles and proximal to the heel pad to avoid crushing the heel pad and to help provide an obstruction to cast slippage. B. The well contoured hindfoot can be seen. The index and long fingers of the other hand flatten the plantar surface while avoiding inadvertent dorsiflexion of the forefoot/midfoot.

the forefoot from dorsal to plantar [Figure 53].

The calcaneus is never touched during the manipulation or casting. Continue molding and smoothing the cast until the material is fully hardened. As discussed earlier, the forefoot supination gradually diminishes with successive casts after the first 1-2 as the subtalar joint everts [Figures 44 and 45].

Extend cast to thigh – There are two reasons to extend the cast above the knee. One is to prevent the cast from slipping off. The clubfoot deformities of inversion and adduction create an acute angle between the foot and the tibia that helps prevent the first few casts from slipping off the tiny foot. As the adduction and inversion deformities are correcting, the foot effectively becomes plantar flexed and in line with the tibia, thus making slippage of the cast more likely [Figure 45]. Until the ankle begins to dorsiflex, the flexed knee is the main deterrent to slippage. The mold over the tendo-Achilles above the heel pad is also an important deterrent to slippage. The second and more important reason for extending the cast above the knee is to correct the internal rotation deformity in the subtalar joint, i.e.inversion. Clubfeet are internally rotated at birth. The primary internal rotation is in the subtalar joint as a component of inversion. Midfoot adduction is an angular deformity that adds "apparent" internal rotation to the true internal rotation in the subtalar joint. Internal clubfoot rotation may create an internal thigh-foot angle of 70-80 degrees [Figures 8, 10, 11, 22]. Dr. Ponseti stated that the thigh-foot angle in the final clubfoot cast after the tendo-Achilles tenotomy should be 60-70 degrees externally rotated.

While it is true that there can be 70-80 degrees of internal rotation (combined inversion and adduction) in a clubfoot, 60-70 degrees of external rotation in the subtalar joint is the definition of a vertical talus. The maximum amount of safe external rotation in the subtalar joint of a cast-corrected clubfoot is perhaps 25-30 degrees, manifest radiographically by an AP talo-1st metatarsal angle of that degree. The remainder of the external rotation must, therefore, occur in the knee joint (personal opinion based upon in-depth study and understanding of the biomechanics of the child's foot). I discussed this point with Dr. Ponseti on several occasions and, although we agreed to disagree, he never offered an alternative explanation. There are no published reports on long term knee problems from the 60-70 degrees of external rotation achieved and then maintained with nighttime bracing in these infants.

Flex the knee to 90 degrees and gently externally rotate the short leg cast until resistance is met. Do not force it. [Figure 54].

The external rotation force of the leg at the knee must be gentle to avoid over-stretching the collateral ligaments at the knee before the subtalar joint is fully everted. Recall the findings of Huson (page 28) of the kinematic opposite direction linkage between rotation in the tibia and the subtalar joint. To reiterate this important biomechanical principle, external tibial torsion and inversion of the subtalar joint are associated, as are internal tibial torsion and eversion of the subtalar joint. The tibia, in this situation, is slightly externally rotated in relationship to the femur, but it is internally rotated in relationship to the forcefully externally rotated/everted subtalar joint.

Use a 2-inch stockinette on the thigh under the cast padding

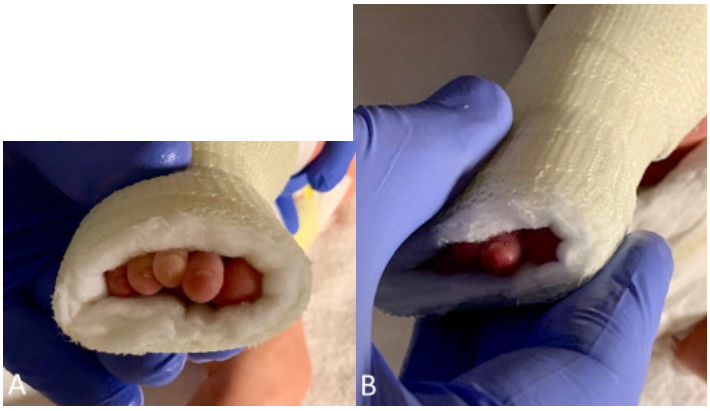

Figure 53. A. When thumb and fingers release the forefoot, it looks as though they were never in the cast. There is neither medial-to-lateral cramping of the toes nor excessive room around the forefoot. B. The final mold is dorsal-to-plantar to exactly match the shape of the forefoot. The cast material extends beyond the toes, particularly on their plantar surfaces. The muscle-tendon units with the longest distance from origin to insertion are the flexor hallucis longus and flexor digitorum longus. They must be supported and stretched.

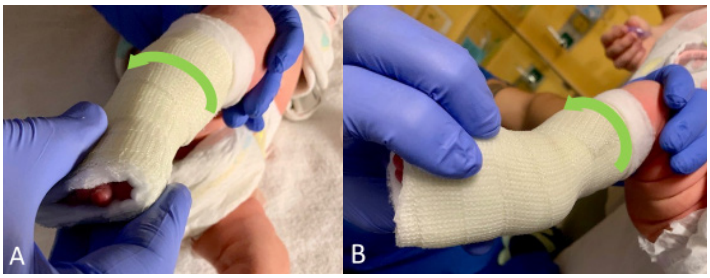

Figure 54. A. and B. Two views of gentle external rotation of the short leg cast (green curved arrow) with a 90-degree flexed knee in preparation for extending the cast above the knee. External rotation/eversion forces in the subtalar joint that are achieved in the short leg cast are maintained in the long leg cast extension. The subtalar joint can slowly revert to slight inversion over time in a short leg cast (or brace) due to the abundant soft tissue envelope surrounding a baby's leg and foot. Maximum, yet gentle, external rotation at the knee locks and prevents the tibia from externally rotating farther, thereby making it a fixed post against which the external rotation forces in the subtalar joint are maintained. The effect is one of relative internal rotation of the tibia against external rotation of the subtalar joint.

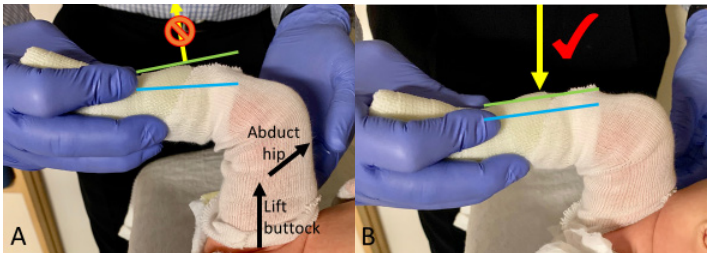

Figure 55. A. Use stockinette under upper portion of cast to create a clean, smooth, and well-padded proximal trim line. Abduct the hip and elevate the buttock to present more thigh for casting. This maneuver moves the thigh away from the abdomen. While externally rotating the short leg cast, avoid lifting up on the cast (yellow arrow covered by NO symbol). Lifting up will move the anterior shell of the cast (green line) away from the tibia (blue line), thereby plantar flexing the ankle within the cast. B. Instead, force the anterior shell of the short leg cast against the tibia (yellow arrow). This maneuver will maintain the degree of ankle dorsiflexion achieved so far.

to provide a clean and trim proximal edge of the cast. Hold the short leg cast with one hand and abduct the hip while externally rotating the short leg cast. Lift up under the ipsilateral buttock with the other hand. This latter maneuver will present more thigh to work with, so it will truly be a long leg cast and not a long short leg cast [Figure 55].

Apply cast padding only slightly overlapping the top of the short leg cast. Then apply the cast material. Ensure that there is no step off at the junction between the proximal anterior edge of the short leg cast and the extension of the cast to the hip. Such a step off indicates undesired ankle plantar flexion within the cast as the tibia moves away from the anterior surface of the cast, thereby compressing the posterior soft tissues against the posterior surface of the cast. This effective plantar flexion of the ankle eliminates the dorsiflexion stress that was applied at the time of short leg cast application [Figure 56].

Fold the stockinette back over the proximal edge of the cast for a neat and well-padded trim line [Figure 57].

Each successive cast application will result in additional unforced external rotation. The thigh-foot-angle in the cast applied immediately after the tenotomy is 60-70-degree external [Figure 58].

There are no reported differences in outcomes whether plaster of Paris or semi-rigid fiberglass casting tape is used [Figure 59].

Trim the cast – The cast must extend beyond the plantar surface of the toes. Dr. Ponseti recommended trimming the cast back dorsally to expose them. Covering them dorsally actually does no harm. The child cannot possibly actively dorsiflex the toes during cast corrections because of the mechanical disadvantage they are subjected to under dorsiflexion stretch. Just ensure that the toes are not compressed medial-to-lateral [Figure 53], [Figure 60].

Characteristics of adequate correction of cavus, adductus, and varus in preparation for tendo-Achilles tenotomy

Cavus must be converted to planus (flatfoot) without overcorrection to a rocker bottom deformity.

Adductus (convex lateral border) must be converted to a straight lateral border of the foot without overcorrection to abduction (concave lateral border) with a lateral midfoot crease.

Varus must be corrected to slight valgus without overcorrection to severe valgus. The head of the talus should no longer be palpable in the dorso-lateral midfoot/hindfoot in the sinus tarsi region.

Equinus should be corrected to approximately neutral, i.e. 0 degrees of dorsiflexion (foot is 90 degrees to the tibia). When all of these characteristics are achieved, any formal attempts to dorsiflex the foot risk creating a rocker bottom deformity. The collagen in the tendo-Achilles, unlike the collagen in the plantar soft tissues, is made of non-stretchable, thick, tight collagen bundles with few cells.

The outcome of casting

At the completion of casting, the foot appears to be slightly over-corrected with a flat longitudinal arch, straight lateral border, valgus hindfoot, slight supination of the forefoot, and a

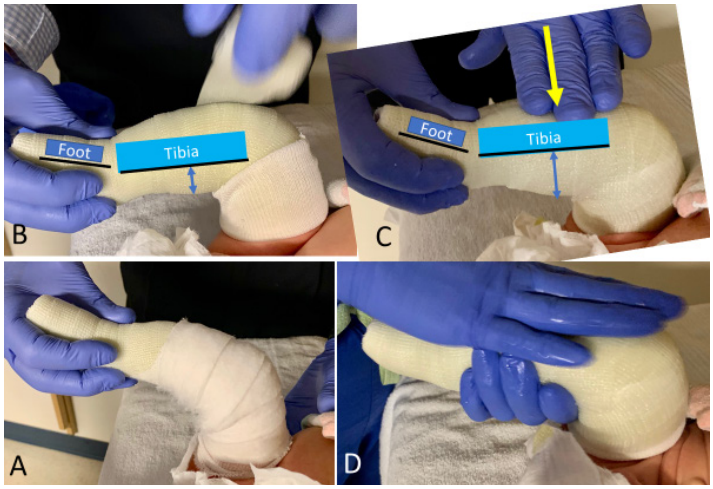

Figure 56. A. Apply cast padding that only slightly overlaps the top of the short leg cast. B. Then apply the cast material while trying to keep the anterior shell of the cast against the tibia. This is challenging until the long leg cast is being molded. Unless it's forced down, the posterior soft tissues of the calf enable the ankle to plantar flex within the cast and the tibia to become adjacent to the posterior shell of the cast (blue double headed arrow). C. Following application of all cast material, force the anterior shell of the cast against the tibia (yellow arrow). That will move the tibia away from the posterior shell of the cast (blue double headed arrow) and return the anterior shell of the cast to the anterior cortex of the tibia. This will regain the degree of ankle dorsiflexion achieved with pre-cast manipulation. In this case, appropriate molding regained 6 degrees of dorsiflexion that was "lost" when the cast was not properly positioned. D. Final molding.

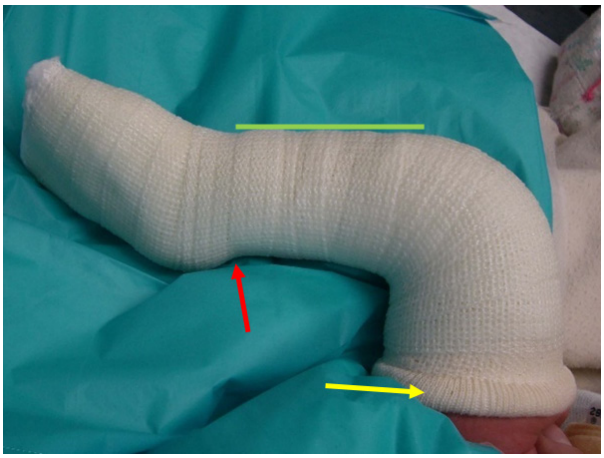

Figure 57. Long leg cast with no step off between the short leg cast and the extension over the knee to the hip (green line). Well-padded proximal trim line with stockinette folded over the edge (yellow arrow). Also note careful molding behind tendo-Achilles immediately proximal to the heel pad (red arrow).

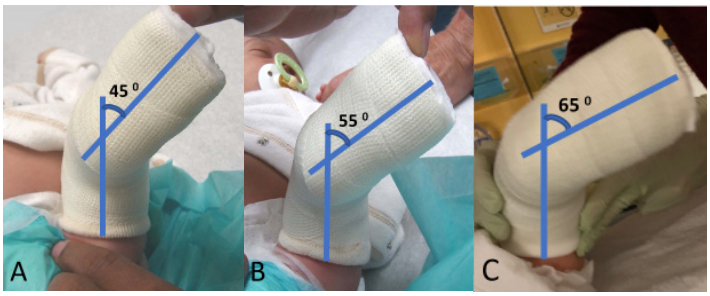

Figure 58. Progressive external rotation measured by the thigh-foot angle. A. 4th cast. B. 5th cast. C. 6th cast applied following tendo-Achilles tenotomy.

thigh-foot angle of 50-60 degrees external. This is not, in fact, an overcorrection. It is actually full correction of the foot. Most babies have physiologic flexible flatfoot deformities for which the natural history in most is development of a longitudinal arch by age 10-12 years. Twenty to 25% of adolescents and adults maintain the flatfoot shape as the contour of a strong and stable foot that is of little consequence as a cause of disability. Achieving this shape in a clubfoot helps prevent recurrence of deformity and does not create a pathologic overcorrected foot [Figure 61].

Complications of Casting

Using careful technique, as described, complications are uncommon but, unfortunately, NOT "never" events, even in very experienced hands.

Crowded toes are due to tight casting around the toes. This will be uncomfortable for the baby and could create pressure sores on the great and/or 5th toes. Ensure that the positions of the fingers holding the forefoot during application of the padding and the cast material are wide enough to prevent medial-to-lateral compression, but not so wide or tall as to prevent appropriate cast molding [see Figures 53, 60].

Flat heel pad will occur if the casting material is wrapped too tightly around the heel or if the molding is behind the heel pad rather than above it over the tendo-Achilles [Figure 28 C, D]. A flat heel pad will contribute to cast slippage and the associated risk of pressure sores (see below). If this occurs to a significant extent, take a 1-2 week cast holiday to allow the fat to reform/expand. Then reinitiate casting with more careful padding, cast application, and molding techniques [Figures 49, 52].

Superficial sores (slight pink coloration) are managed by applying additional padding over the site and applying minimal pressure molding with the next cast.

Fissures in the skin creases over the sinus tarsi or antero-lateral ankle [Figure 20]. These are inevitable in most feet, especially in those that are the most severely deformed and those that correct quickly. The skin over the deformed dorso-lateral midfoot/hindfoot (sinus tarsi) fits perfectly on the original foot deformity. As the deformities correct, the skin over the sinus tarsi and antero-lateral ankle becomes relaxed and redundant. It will eventually contract or shrink but, in the short term, creases or folds develop in Langer's lines. There is no possibility to stretch the redundant skin while applying the casts [Figure 47], so irritation and inflammation may develop at the base of the fissures. If they become swollen, tender, or result in a break in the skin, a 1-2 week cast holiday is required before more serious soft tissue injury develops.

Pressure sores (bright red, or purple, or dark and necrotic) are due to excessive pressure that was applied over bony prominences during cast application or cast slippage that changed the location of the bony prominences in relationship to the molds. Common sites include the head of the talus, the antero-lateral corner of the ankle joint, the back of the heel, and under the first metatarsal head [Figure 62].

A cast holiday must be observed until the tissues return to normal, which is usually 1-2 weeks. Families need to know that there will be some mild recurrence of deformities resulting in the need for 1 or more additional casts than might have otherwise

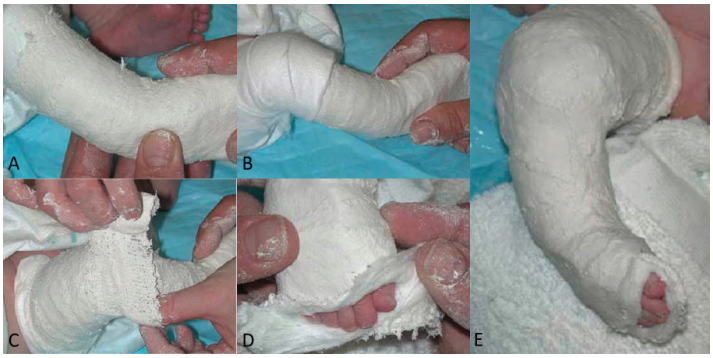

Figure 59. Plaster of Paris cast application.

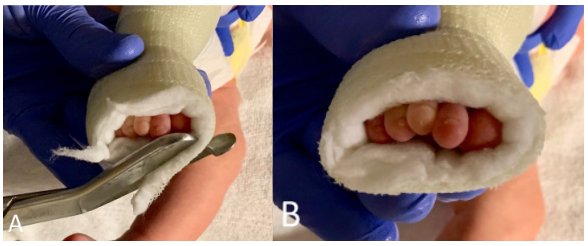

Figure 60. A. It is better to trim the cast back after application than risk making it too short or too thin at the end. B. Ideal length and shape of the distal end of the cast.

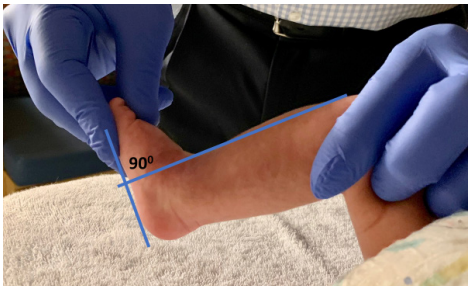

Figure 61. Clubfoot ready for tendo-Achilles tenotomy following 6 serial casts with low longitudinal arch, straight lateral border, physiologic valgus of the hindfoot, and dorsiflexion to approximately neutral, i.e. 90 degrees.

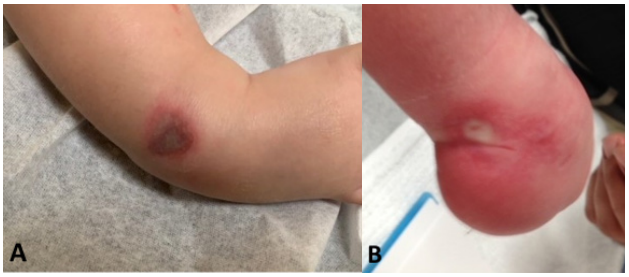

Figure 62. A. Pressure sore on the antero-lateral corner of the ankle joint. B. Pressure sore on back of heel cord.

Figure 63. Donut-shaped, adhesive-backed cushion used to offload a bony prominence that is applied directly to the skin under a cast.

been required. But the risks of more serious injury to the foot mandate temporary discontinuation of casting. Deformity correction with the Ponseti method is not a race. It is better to potentially apply more casts than it might have taken Dr. Ponseti to achieve full deformity correction than to cause pressure sores.

In older children with severe and rigid deformities, donut-shaped cushions can be applied to the skin surrounding a bony prominence to avoid a pressure sore or to enable ongoing casting if a very superficial sore has already been created [Figure 63].

Diffusely swollen and warm foot with shiny, hypersensitive and hyperemic skin may develop in association with a pressure sore or may develop independently [Figure 64].

The foot appears to have complex regional pain syndrome. These occur because of aggressive manipulations and castings. Clubfoot deformity correction is not a race. The deformities correct at the rate determined by the individual collagen characteristics of the foot being treated. The average number of casts is 6, but the range is 3-10, or more. Overly aggressive manipulation and casting tears, rather than stretches, the collagen in the tendons and ligaments. That results in inflammation and possibly bleeding. Inflammation and bleeding create scar tissue that, like surgery, causes stiffness which is exactly what the Ponseti method is intended to avoid. A cast holiday must be observed until the tissues return to normal, which is usually 1-2 weeks.

Irritation in the popliteal fossa transverse skin crease can be caused by perspiration, hyper-flexion of the cast, or bunching up of the cast padding [Figure 65].

A thin layer of baby powder can be applied to the site if mild irritation is noted at a cast change. If the irritation is more significant, an antibiotic cream or salve can be used. In severe cases, a cast holiday is indicated. Avoid bunching the padding in the popliteal fossa by always applying the cast and the cast padding in 2 parts, first below the knee and then extended above the knee. Also avoid flexing the knee beyond the position established when the padding was applied.

Irritation at the proximal thigh trim line can be caused by insufficient padding over the end of the cast or urine soaking. A cotton stockinette folded back upon itself over abundant cast padding creates a soft trim line [Figure 57]. Educate the parents to avoid overlapping the diaper on the cast, because urine will transfer from the diaper to the cast padding and progress distally. The non-drying acidic moisture will be irritating to the skin [Figure 66].

Rocker-bottom deformity is due to forcefully dorsiflexing the foot against a very tight Achilles tendon resulting in hyper-dorsiflexion of the midfoot. This usually occurs after the cavus has been corrected and the subtalar joint has everted beyond neutral. The collagen in the tendo-Achilles is much more resistant to stretch than that in all the other tendons and ligaments in the foot. Dr. Ponseti said that one should not intentionally and forcefully attempt to stretch the tendo-Achilles. In a small percentage of cases, the tendo-Achilles stretches completely by the time the subtalar joint is everted. If not, he recommended not trying to stretch it, but instead to perform a tendo-Achilles tenotomy. If rocker-bottom deformity is suspected [Figure 20B], confirm it with a maximum dorsiflexion lateral x-ray of the foot and ankle [Figure 67].

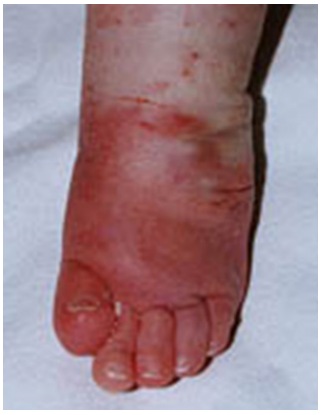

Figure 64. Diffusely swollen and warm foot with shiny, hypersensitive and hyperemic skin. These iatrogenic soft tissue injuries are due to overly aggressive manipulations and casts that may be exacerbated by cast slippage.

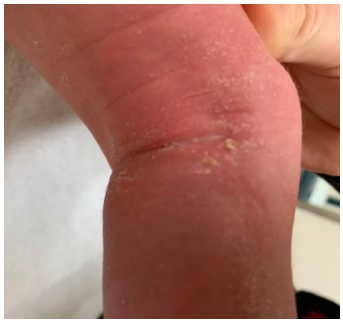

Figure 65. Irritation in the popliteal fossa transverse skin crease.

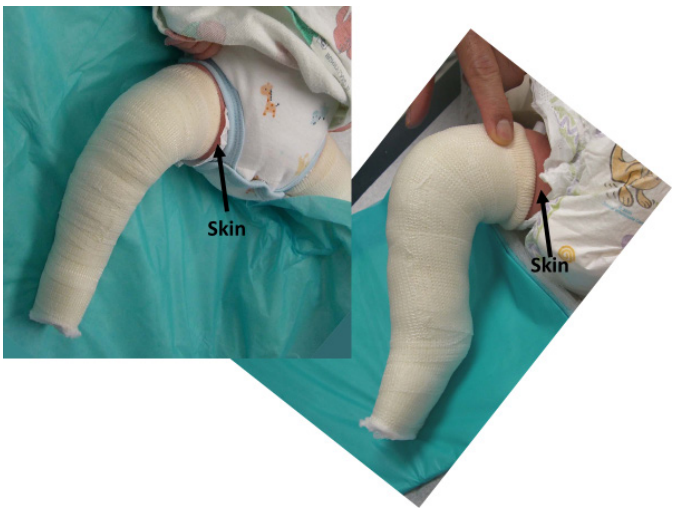

Figure 66. Skin between the top of the cast and the diaper must be seen at all times to prevent urine from transferring from the diaper to the cast padding and wicking down the cast.

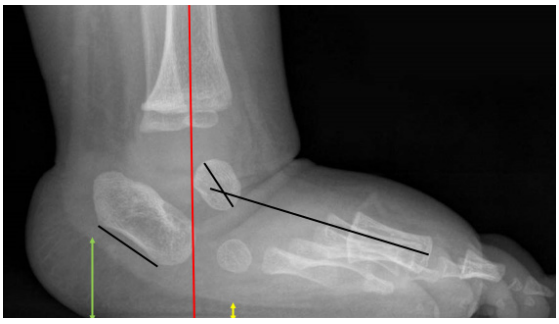

Figure 67. Lateral x-ray of a rocker bottom clubfoot deformity. There appears to be neutral ankle dorsiflexion with the axis of the tibia (red line) perpendicular with the bottom of the foot. But the talus and calcaneus are planar flexed and the 1st metatarsal is dorsiflex in relation to the talus. This is the definition of a rocker bottom deformity. The deception is due to the excessive thickness of the heel pad (double headed green arrow) compared with the thickness of the fat under the midfoot (double headed yellow arrow). Note that the difference between a rocker bottom deformity and a flatfoot is the position of the calcaneus. In both deformities, there is dorsiflexion of the 1st metatarsal in relation to the talus. But, in a flatfoot, the calcaneus is no more plantar flexed than 90 degrees to the tibia.

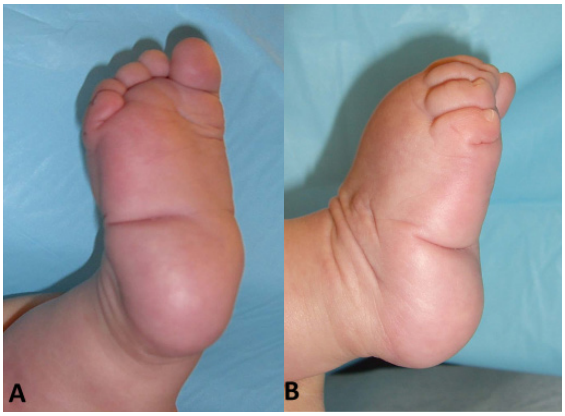

Figure 68. Hyper-abduction iatrogenic deformity. A. Plantar view. B. Lateral view

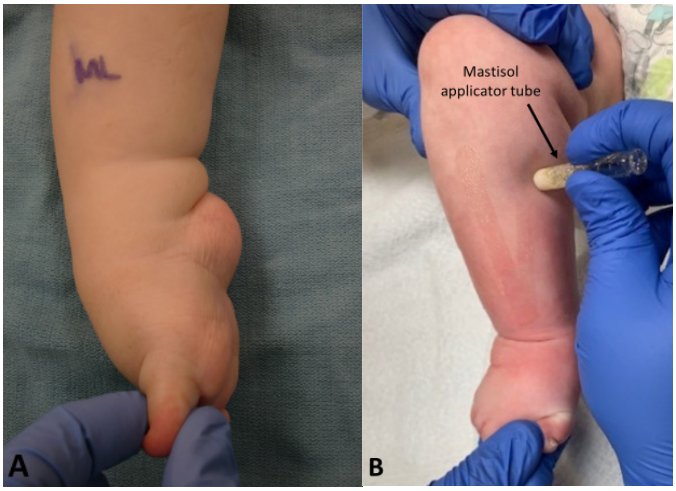

Figure 70. A. A "fat" clubfoot in a child who is not fat. Casts tend to slip down and off these feet. B. Using Mastisol liquid adhesive, or a similar product, on the skin will usually prevent the cast from slipping down and obviate the need for hyper-flexing the knee.

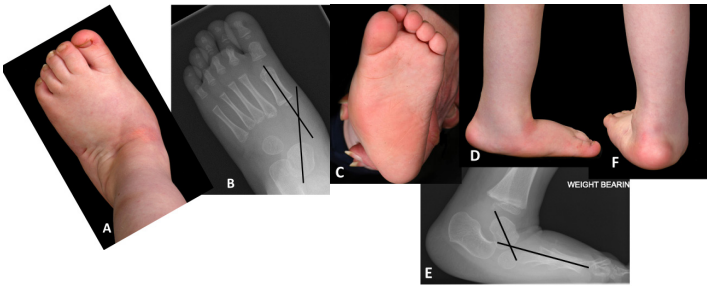

Figure 69. A. Hyper-abducted clubfoot in a 3-year-old who had been serial casted. B. AP radiograph of the foot showing the foot-CORA in the unossified medial cuneiform, i.e. metatarsus ABductus. The talo-navicular joint is likely reduced in this case, based on the location of the foot-CORA. C. Plantar view. D. Standing lateral view. E. Weightbearing lateral radiograph. The hyper-dorsiflexed foot-CORA at the talonavicular joint indicates an associated rocker bottom deformity. F. Posterior view of the foot. (Images courtesy of Donald Campbell, MD from Dundee, Scotland who was consulted regarding management of this iatrogenic deformity that was created elsewhere)

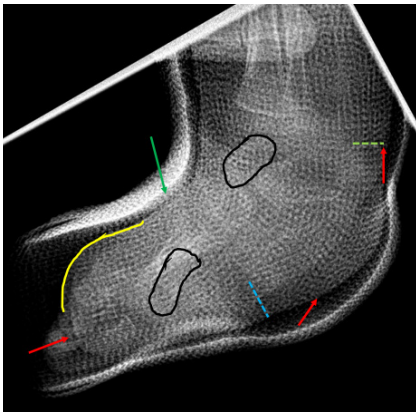

Figure 71. Fat, complex clubfoot slipping back in cast (red arrows). Talus and 1st metatarsal are outlined in black. Trans-tarsal cavus exists with a plantar midfoot crease (blue dashed line). Yellow curved line outlines abundant fat on the dorsum of the foot. Anterior ankle mold is now over mid-forefoot (green arrow). Posterior ankle crease (green dashed line) is now proximal to the posterior ankle mold in the cast.

If rocker-bottom deformity is identified, take a cast holiday and wait for the iatrogenic deformity to resolve. If it does not, perform reverse casting. Then try more gentle casting or perform an early tendo-Achilles tenotomy.

Hyper-abduction of the midfoot occurs when excessive and forceful abduction forces are applied to a midfoot in which the hindfoot inversion is unyielding. The clinical manifestation is a concave lateral border, often with a deep dorsal-plantar skin crease [Figure 68].

The navicular usually remains adjacent to the medial malleolus while the cartilaginous anlage of the mid-tarsal bones deform into an abduction deformity according to the Hueter-Volkmann law. If confirmed with x-rays, take a cast holiday and reinitiate more gentle and focused casting when the deformity resolves. If the metatarsus ABductus deformity does not resolve, perform reverse metatarsus adductus casting. If the diagnosis is delayed, the iatrogenic deformity may not respond to these measures and surgery will be needed [Figure 69].

Cast slippage occurs most commonly in the first 1-2 casts when the severe inward J-shaped or L-shaped curvature is converted to pure equinus before eversion/dorsiflexion recreates an acute angle at the ankle. Pressure sores often develop over the anterior ankle and/or the back of the calcaneus [Figure 62] because the molded indentations are no longer in the proper locations and the foot is firmly wedged in a position in which the "hills and valleys" meet Slippage can be avoided in most cases by using a thin layer of padding, a thin layer of cast material, molding carefully behind the tendo-Achilles above the heel pad, flexing the knee to 90 degrees, and extending the cast proximally on the thigh almost to the groin crease. Dr. Ponseti recommended hyper-flexion of the knee to prevent cast slippage, but that technique puts exaggerated pressure in the skin folds in the popliteal fossa [Figure 65]. Mastisol liquid adhesive, or a similar product, can be used on the skin as a gentle adhesive to avoid the need to hyper-flex the knee. It is particularly helpful for the "fat" feet and legs in which the pressure points are less effective in reaching their targets through the abundant soft tissues [Figure 70].

Creation of a complex, or atypical, clubfoot often occurs with cast correction of a "fat" foot [Figure 20]. These casts tend to slip down because the pressure points are less effective in reaching their targets through the abundant soft tissues. When the cast slips down, the anterior ankle mold indentation presses on the dorsal mid/forefoot thereby flexing the forefoot on the hindfoot creating trans-tarsal cavus with a deep plantar

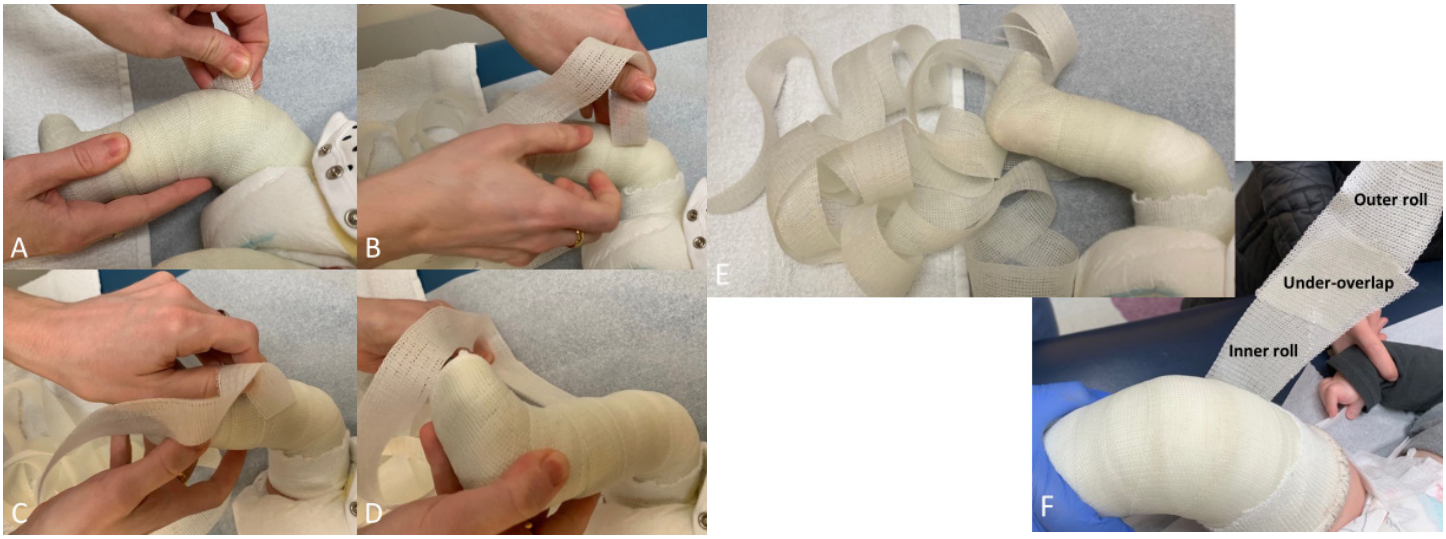

Figure 72. A – E. Removing a semi-rigid fiberglass cast. F. Under-overlapping the ends of the rolls during application makes removal even easier. Removal of one roll initiates removal of the next roll.

transverse midfoot crease, the so-called complex or atypical clubfoot [Figure 71].

In the attempt to apply effective pressure points, pressure sores may develop, thereby causing swelling on top of already large, round feet. A cast holiday may be indicated to allow the swelling to resolve leaving only the fat foot. Then, one should use the double thumbs technique of manipulation and casting [Figure 29]. Dr. Ponseti recommended hyper-flexing the knee to prevent repeat cast slippage, but irritation in the popliteal fossa of these large legs is almost guaranteed [Figure 65]. I have found that applying Mastisol to the skin without hyper-flexing the knee is helpful to prevent the casts from slipping down and off these feet [Figure 70].

Purple toes in the first hour after cast application are due to the cooling effect on the skin of evaporation of the water in the deepest layers of the cast. Inform the parents that the purple toes will return to pink in an hour.

Skin sensitivity from application of the cast padding is often seen after the first 2-3 casts. The child generally does not fuss during the manipulation stage or even when the forefoot and leg are held in preparation for application of the cast padding. As soon as the soft cotton padding touches the skin, the child cries. The crying often stops during or after molding of the short leg cast, so the child is calm and quiet during application of the upper portion of the long leg cast.

Cast removal

Semi-rigid fiberglass casting tape removal

Semi-rigid fiberglass casting tape is applied by underlapping the beginning of each successive roll with the end of the previous roll [Figure 50]. To remove the cast, scratch up the end of the final roll with a fingernail and simply unravel the entire cast. Because of the under-overlapping of the rolls, the entire cast will unravel continuously. The end of 1 roll starts the removal of the previously applied roll [Figure 72].

The cast padding can then be easily removed [Figure 73].
It will only take a few minutes to remove the cast. Ask the family

Figure 73. Removing semi-rigid cast and cast padding takes 2-3 minutes.

to remove the cast about an hour before they plan to leave home for their next clinic appointment. They can give the baby a full bath before leaving home. Ask them not to remove the cast the night before because there may be some diffuse reactive swelling of the foot overnight that will make subsequent manipulations and casting more challenging. These clinic visits are very quick and efficient: walk in, assess progress in deformity correction, manipulate the foot, and apply the next cast.

Another advantage of semi-rigid fiberglass casting tape is that the family can remove the cast immediately if they notice that the cast has slipped down or the baby is particularly uncomfortable, typically a sign of an impending pressure sore.

Plaster of Paris cast removal – [according to Dr. Jose Morcuende from Iowa]

Remove each cast in clinic just before a new cast is applied.

Cast knife removal Soak the cast in water for about 20 minutes, and then wrap the cast in wet cloths before removal. This can be done by the parents at home just before their visit. Use the plaster knife [Figure 74].

Soaking and unwrapping This is an effective method but requires more time. Soak cast thoroughly in water and when completely soft unwrap the plaster. To make this process easier, leave the end of the plaster free for identification [Figure 75].

When parents should be concerned during the casting phase – and call the provider!

- Inconsolable pain, especially if it develops after an initial period of comfort.
- Any foul-smelling odor or drainage coming from inside the cast.
- Red, sore, or irritated skin at the edges of the cast.
- Urine and/or stool that are known, or presumed, to have significantly soaked the padding
- Poor circulation in the toes.
- Cast slipping off.

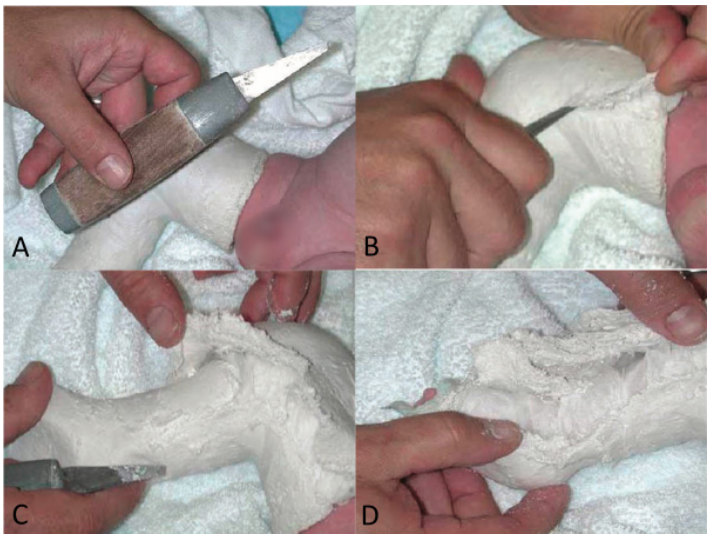

Figure 74. A. Use the plaster knife. B. Cut obliquely to avoid cutting the skin. C. Remove the above-knee portion of the cast first. D. Finally, remove the below-knee portion of the cast.

Figure 75. A. Soaking cast in water in a resource challenged country. B. Unwrapping the cast.

CHAPTER 6

Tendo-Achilles tenotomy (TAT)

The ankle is usually in neutral (0 degrees) dorsiflexion after 6 casts that focused on and achieved full correction of cavus, adductus, and varus. Tendo-Achilles tenotomy (TAT) is indicated when cavus, adductus, and varus are fully corrected but ankle dorsiflexion remains less than 10 degrees above neutral with the knee extended. The need for the tenotomy is not due to a failure of the method. Dr. Ponseti's histologic studies found that, unlike the tarsal ligaments that are stretchable, the tendo-Achilles is made of non-stretchable, thick, tight collagen bundles with few cells. He determined that a tendo-Achilles tenotomy is needed in 90-95% of clubfeet. Attempts to avoid the tenotomy by aggressive and forceful dorsiflexion casting can inadvertently create a breech, or rocker bottom, deformity.

It is a tendo-Achilles tenotomy (TAT), **not** a tendo-Achilles lengthening (TAL)! The tendon is completely divided, thereby separating and leaving a gap between the 2 ends of the tendon. A lengthening indicates that the tendon is cut in such a way that the 2 ends can be reattached to maintain continuity of the musculo-tendinous unit after elongating the distance between the origin and insertion of the musculo-tendinous unit.

Indications for tendo-Achilles tenotomy

Cavus – converted to a flat-to-low normal longitudinal arch without rocker bottom deformity.

Adduction - converted to straight lateral border.

Varus – converted to physiologic valgus of the hindfoot. It is challenging to accurately determine valgus alignment of the subtalar joint due to the excessively fat heel pad. But the hindfoot must appear to be in valgus. Note that the ankle joint is in valgus alignment in all newborns. The tibio-talar (ankle) joint does not assume a neutral or perpendicular relationship with the tibia until age 4 years. Therefore, if the hindfoot appears to be in neutral axial alignment when viewed from behind, the subtalar joint has residual varus deformity, the two opposite deformities cancelling each other out.

Family preparation

Prepare the family for the almost inevitable need for a tendo-Achilles tenotomy starting at their initial encounter with you, which could be their prenatal consultation visit. And reinforce the necessity and safety of the procedure frequently during the weekly castings. Explain that the tendo-Achilles tenotomy is a minor outpatient procedure.

Physical location for performing the tenotomy

Dr. Ponseti recommended performing the tendo-Achilles tenotomy under local anesthesia in the outpatient clinic. It can, of course, be performed in the operating room, but there is increasing evidence that general anesthetics in babies may have negative consequences on the developing brain. And for a procedure that is tolerated even better than a circumcision, the risks are hard to justify. I was recently influenced to perform the procedure under nitrous oxide sedation. I found that both children I treated in this way were extremely agitated throughout the tenotomy and cast application, much more agitated than any of the hundreds of children in whom I have performed the tenotomies in clinic with the consoling mother at her baby's head. Whether the mother/father is present for the clinic procedure is based on the comfort level of the surgeon. I believe it is completely appropriate for a surgeon, particularly a less experienced surgeon, to ask the parents to leave the exam room for a few minutes during the tenotomy and return soon thereafter for comforting the child and during cast application. In the clinic, the child typically cries when my assistant holds the leg and foot firmly to prevent me from inadvertently injuring the neurovascular bundle that is millimeters away from the tendo-Achilles. With skin numbed by the 4% lidocaine cream, I rarely hear any increase in the volume of crying when the tendon is cut, literally a 3 second procedure. Two cc of 1% lidocaine are immediately injected, but the child typically stops crying within seconds of the mother picking him/her up. And the final justifications for avoiding anesthetic risks and believing in the comfort of the procedure is the post-procedure analgesic requirement. Although not formally studied, it is my impression that approximately 90% of parents give their child 1 dose of acetaminophen later that day, 5% give 2-3 doses within 24 hours, and 5% give none when given the option to give as many doses as they feel necessary.

Equipment

Prepare all the materials in advance. Select a tenotomy blade. I prefer a #6700 Beaver blade because the curved end can be used as both a probe and a cutting device. A pointy blade or needle can engage the tendon prematurely without your tactile knowledge of exactly where it is. The #6700 Beaver is essentially a mini-

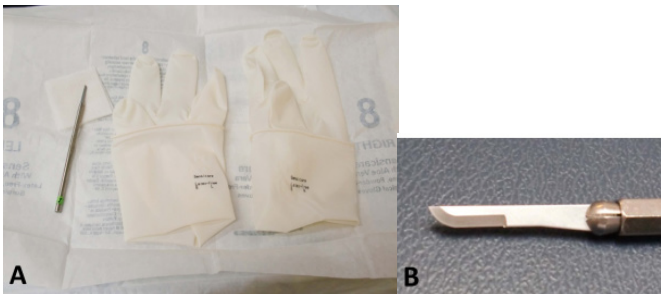

Figure 76. A. On a Mayo stand, open sterile gloves, the scalpel of your choice, and a 2x2" gauze pad. B. My choice is a #6700 Beaver blade.

#15 BP scalpel, a blade I'm very accustomed to working with. Use a scalpel with which you are most comfortable. [Figure 76].

Anesthesia

Apply lidocaine cream to the back and sides of the tendo-Achilles. Within 20-30 minutes, the local anesthesia penetrates to a depth of 1 cm. which is more than enough to numb the skin and the subcutaneously positioned tendon. Dr. Ponseti recommended injecting a small amount of local anesthetic near the tendon, but there is a risk that too much liquid local anesthetic will make palpation of the tendon difficult and the procedure more complicated [Figure 77].

Skin preparation

With the patient at the end of the exam table and your assistant on the ipsilateral side of the table, have your assistant hold the forefoot with one hand and the thigh with the other hand while abducting the hip and positioning the foot and leg parallel with the table [Figure 78A].

Wipe off the lidocaine cream after approximately 20". Prep the hindfoot and ankle with Betadine [Figure 78B].

Risk considerations related to the location of, and approach to, the tenotomy

The posterior tibial neurovascular bundle is only a few millimeters antero-medial to the tendo-Achilles. The anatomy of the posterior ankle must be well known to the surgeon to help avoid injury to this and other perhaps less important, but still significant, structures [Figure 79].

Do not cut the tendon at the posterior skin crease. That is the location of the insertion of the tendo-Achilles on the calcaneus. If the calcaneal apophysis rather than the tendo-Achilles is cut inadvertently, it will not heal well. The calcaneal apophyseal fragment will migrate proximally with the tendon eventually ossifying and creating a bony prominence that will be irritated by the counter of a high-top shoe or boot.

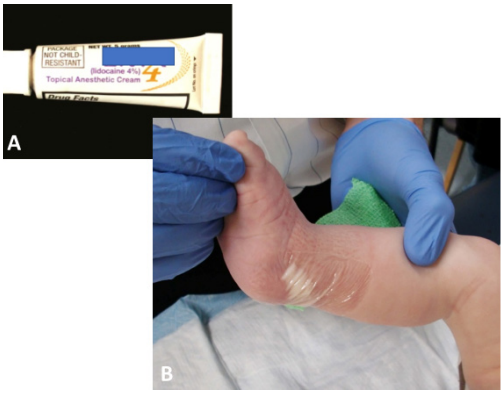

Figure 77. A. Lidocaine cream. B. Lidocaine cream surrounding the tendo-Achilles covered by clear plastic occlusive dressing.

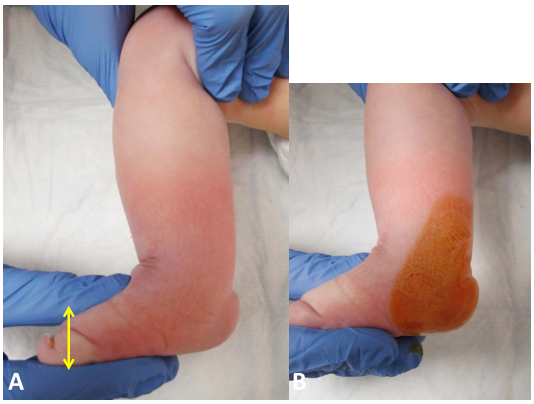

Figure 78. A. The assistant abducts the hip with one hand on the thigh while the other hand holds the forefoot firmly. The knee is flexed 90 degrees. The sagittal planes of the lower leg and foot are parallel with the table. The assistant will dorsiflex and plantar flex the ankle (double headed yellow arrow) stopping when the tendo-Achilles is most easily palpated. Some surgeons perform the TAT by hyper-dorsiflexing the ankle with one hand and performing the tenotomy with the scalpel in the other hand. Hyper-dorsiflexion may pull the tendo-Achilles anteriorly and closer to the posterior tibial neurovascular bundle, thereby putting it at risk for injury. At a minimum, hyper-dorsiflexion makes the tendon harder to palpate. B. Prep the midfoot, hindfoot, and ankle with Betadine.

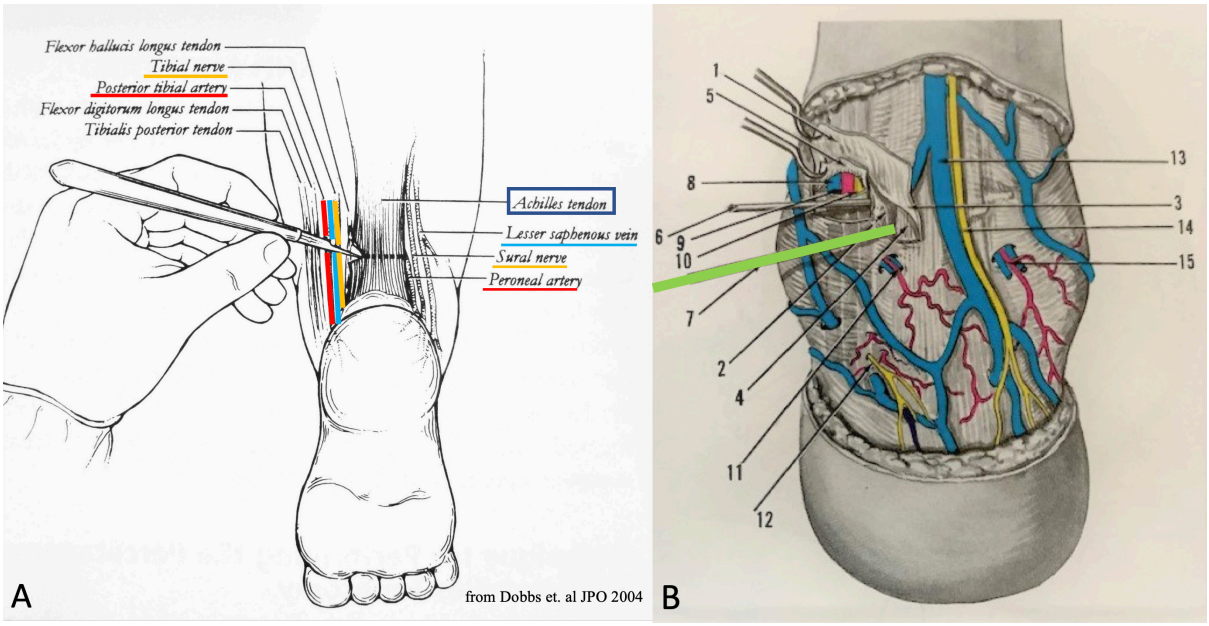

Figure 79. A. Enhanced sketch from Dobbs et al. JPO 2004. The posterior tibial neurovascular bundle is a few millimeters antero-medial to the tendo-Achilles and must be carefully avoided to protect it from inadvertent injury during this percutaneous procedure. B. Posterior ankle sketch from Sarrafian 3rd ed 2011, page 472. Green line represents tenotomy scalpel.

The tendo-Achilles tenotomy (TAT)

Don sterile gloves. Ask your assistant to hold the limb firmly as described above [Figure 78]. Standing at the end of the exam table, completely divide the tendo-Achilles 1 cm. proximal to its insertion on the calcaneus (and the deep posterior heel crease) from a medial/antero-medial approach. It should take longer than 3-4 seconds [Figure 80].

To avoid injury to the posterior tibial neurovascular structures, insert the scalpel through the skin directly medial to the Achilles tendon and perpendicular to the foot in the coronal plane with the face of the blade parallel with the fibers of the tendon. Since the foot and leg are parallel with the table, the scalpel will be inserted perpendicular with the table which is the coronal plane of the ankle. Once through the skin, use the tip of the scalpel as a probe, slowly translating it anteriorly (towards the toes) until it falls into the fat immediately anterior to the tendon. Rotate the scalpel blade 90^0 and translate it posteriorly to divide the tensioned tendo-Achilles while the assistant simultaneously dorsiflexes the ankle [Figure 81].

Further clarification of the technique is shown in Figure 82.

There must be a sudden and visible release that is best described as a "pop" accompanied by dorsiflexion of at least 15-20 degrees. The release will be experienced by the surgeon and the assistant (and often by observers). If a complete release is not experienced and at least 15-20 degrees of dorsiflexion not observed, reinsert the scalpel to release the remaining fibers. Immediately inject a small volume of 1% lidocaine that can disperse further in the area. Apply a pressure dressing for several minutes before applying a long leg clubfoot cast with the ankle dorsiflexed at least 10^0 and with a 70^0 external thigh-foot angle [Figure 83].

If there is noticeable bleeding other than a few drops of blood, maintain direct manual pressure for longer. With this approach, there should be minimal-to-no risk of cutting the posterior tibial artery. Note that there are several large to small veins and the small peroneal artery in the area [Figure 79] that, if cut, can be well controlled with prolonged manual compression.

The other possible percutaneous approaches to the tendo-Achilles are 1) lateral to medial in the same coronal plane and 2) posterior to anterior in the sagittal plane. Both approaches risk greater injury to the posterior tibial NV bundle. It is obvious that a lateral to medial approach runs that risk. But a posterior to anterior approach likewise risks NV injury particularly with a windshield wiper rotation of the scalpel.

Post-tenotomy cast

This cast holds the foot for 3 weeks. During that time, the gap between the tendon ends fills with scar tissue that is structurally sound, but not yet completely histologically normal. Nevertheless, it is sufficiently healed and reformed in an elongated state at that time that rigid immobilization is no longer necessary. The upper age limit at which 3 weeks is enough to maintain immobilization has not been studied.

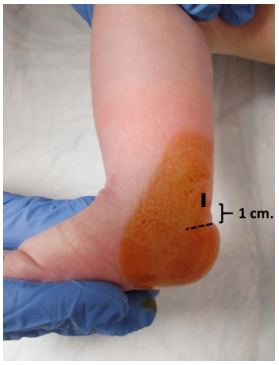

Figure 80. The tendo-Achilles tenotomy is performed percutaneously from a medial/antero-medial approach 1 cm. proximal to the deep posterior heel crease (dashed transverse line). The scalpel blade is inserted longitudinally (short, straight black line).

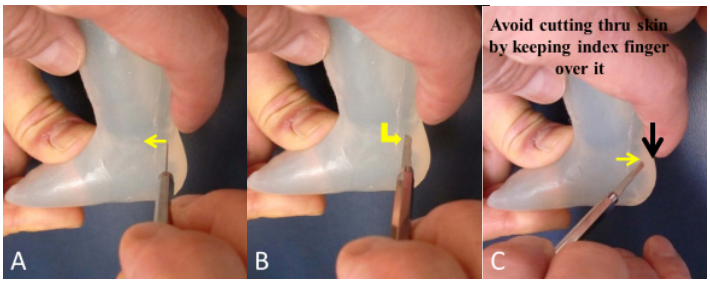

Figure 81. A. Insert the scalpel blade 1 cm proximal to the calcaneus (posterior skin crease), parallel with the tendon, perpendicular to the ankle/table, and directly towards but not through the tendo-Achilles. By doing so, you can be assured that there is no way the neurovascular bundle can be between the skin and the tendon. Using the scalpel as a probe, slowly translate it anteriorly (yellow arrow) until the blade falls into fat immediately anterior to the tendon. B. Insert the scalpel approximately 1 cm. Then rotate the blade 90 degrees until it points directly posteriorly. C. Cut the tensioned tendon completely - anterior to posterior – until it suddenly releases and the ankle dorsiflexes. Place a finger posterior to the tendon either in line with the scalpel or immediately proximal to it (black arrow and fingertip). This will help ensure that you do not cut so forcefully that you cut through the posterior skin – and your finger.

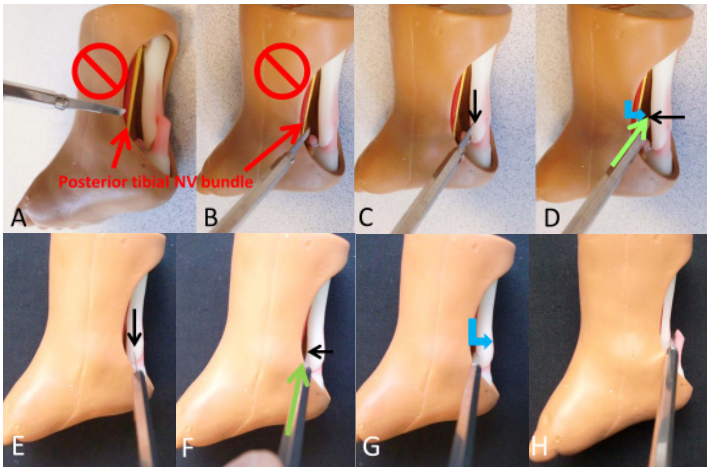

Figure 82. A and B. Random insertion of the scalpel risks injury to the posterior tibial neurovascular (NV) bundle. C. Insertion of the scalpel in line with the tendon and directed at the tendon in the coronal plane (black arrow) perpendicular with the long axis of the foot ensures that the NV bundle will not be injured. Skin to tendon. D. The scalpel is slowly translated anteriorly (black arrow) until it easily passes anterior to the tendon into fat (green arrow). The blade is rotated 90 degrees posteriorly and translated posteriorly to cut the tendon (blue angled arrow). E. Different point of view with the scalpel inserted perpendicular to the foot (coronal plane) through skin directly at, but not through, the tendo-Achilles. F. The scalpel is slowly translated anteriorly (black arrow) until it passes into the fat anterior to the tendon (green arrow). G. The scalpel is rotated 90 degrees posteriorly and then translated posteriorly to cut the tendon - H.

The cast should last the entire time without the need to change it to accommodate growth of the foot and leg. The baby and mother may go home immediately. As noted earlier, although not formally studied, it is my impression that *approximately* 90% of parents give their child 1 dose of acetaminophen later that day, 5% give 2-3 doses within 24 hours, and 5% give none when given the option to give as many doses as they feel necessary. This is usually the last cast required in the treatment program.

Cast removal

After 3 weeks, the cast is removed. At least twenty degrees of dorsiflexion is now possible. The tendon is clinically healed. The operative scar is minimal. The foot is ready for bracing. The foot appears to be over-corrected to a flatfoot shape. This is often a concern to the parent. Explain that this is not an overcorrection, only full correction.

Errors related to tenotomy

Premature equinus correction. Attempts to correct the equinus before the cavus, adductus, and varus are corrected will result in a rocker-bottom deformity [Figures 20B, 67]. Equinus through the subtalar joint can be corrected only if the calcaneus everts beyond neutral. Tendo-Achilles tenotomy is indicated only after cavus, adductus, and varus are fully corrected in idiopathic clubfeet. That proscription may be modified in severe, resistant arthrogrypotic clubfeet in which early TAT plus or minus other superficial tenotomies and plantar fasciotomy are followed by further serial casting [Figures 33 and 34].

Failure to perform a complete tenotomy. The sudden release with a "pop" or "snap" signals a complete tenotomy. Failure to achieve this may indicate an incomplete tenotomy. Repeat the tenotomy maneuver to ensure a complete tenotomy if there is no "pop" or "snap" [Figure 84].

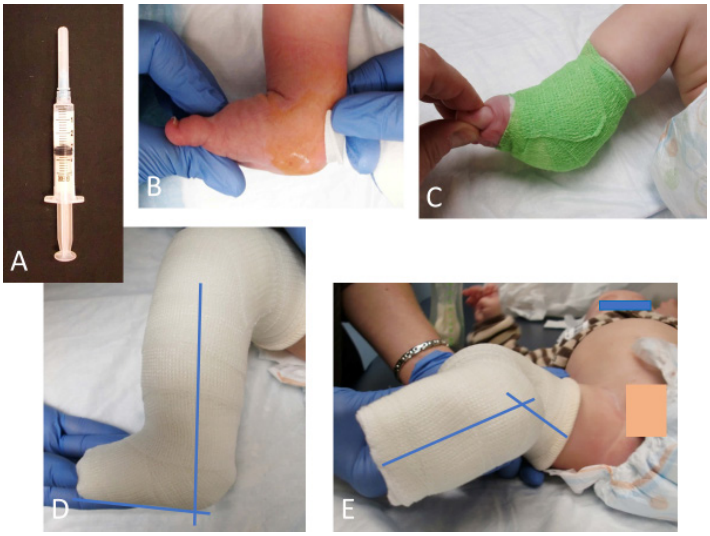

Figure 83. A. Immediately inject 1-2 cc of xylocaine. B. Apply pressure. Note ankle dorsiflexion above neutral. C. Cover with 2x2" gauze, circumferential cast padding, and Coban. Maintain for 5-10". D and E. Apply a long leg cast with 10-15 degrees of ankle dorsiflexion and a thigh-foot angle of approximately +70 degrees.

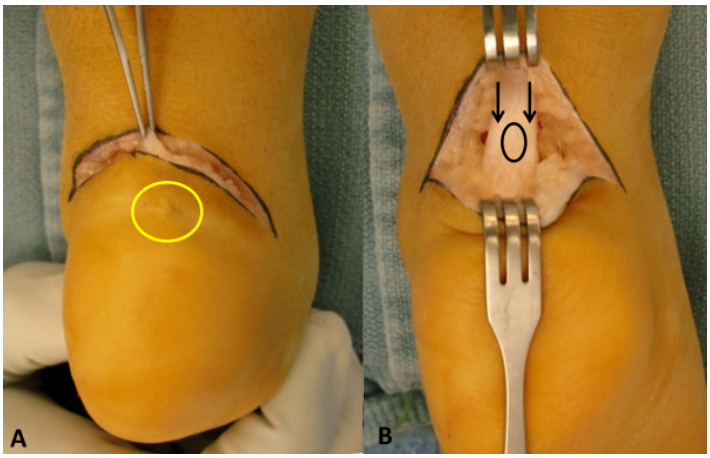

Figure 84. The rocker bottom deformity foot x-ray shown in Figure 55 was created by a surgeon who attempted a percutaneous TAT from a direct posterior to anterior approach in the sagittal plane using a #11 BP pointy scalpel. Believing that it was a complete tenotomy, the surgeon then applied the FAB 3 weeks later. Four months later, the foot appeared to be a rigid rocker bottom deformity resembling a vertical talus. Several reverse Ponseti casts were required to correct the iatrogenic deformity, at which point the joints in the foot were anatomically corrected but there was extreme ankle equinus. A. Open exposure through a short Cincinnati incision was chosen to enable visualization of the pathoanatomy with the option of performing a posterior release if necessary. The yellow oval surrounds the small scar from the attempted posterior percutaneous tenotomy 6 months previously. B. Although difficult to see in this photo, but obvious under direct visualization, there was an oval-shaped scar (black oval) in the center of the tendo-Achilles flanked on both sides by pristine, virgin collagen that had never been cut (distal to the 2 black arrows). Z-lengthening of the tendo-Achilles was performed. The ankle did not quite dorsiflex to neutral. To avoid recreating a rocker bottom deformity with serial postoperative casting, a posterior ankle release was performed concurrently.

CHAPTER 7

Foot abduction bracing (FAB)

Bracing is essential

At the end of casting, the foot is everted and externally rotated to an exaggerated degree, which should measure a thigh-foot angle of 60 to 70 degrees external. After the tenotomy, the final cast is left in place for 3 weeks. Ponseti's protocol then calls for a brace to maintain the foot in maximum eversion and dorsiflexion. Huson's findings relating to the kinematic opposite direction linkage between rotation in the tibia and the subtalar joint apply to bracing as they do to casting. That is the biomechanical justification for the design of the brace recommended by Ponseti, in which the feet are externally rotated on a bar that connects them. The bar is a proxy for the tibia that maintains the linkage while enabling knee flexion and extension. The gold standard brace, that is comprised of straight-last open-toe shoes that are attached to a bar, is called a foot-abduction brace (FAB) or foot-abduction orthosis (FAO), although it should more accurately be called a foot-eversion brace (FEB) [Figure 85].

Sixty to 70 degrees of external foot rotation on the bar are required to maintain the eversion of the subtalar joint, abduction of the midfoot, and supination of the forefoot to prevent relapse. The medial soft tissues remain stretched out only if the brace is used after the casting phase of treatment. In the brace, the knees are left free, so the child can kick them "straight" to stretch the tendo-Achilles. The eversion of the feet in the brace, combined with the slight bend in the bar (convexity away from the child), causes the ankles to dorsiflex. This helps maintain the stretch on the triceps surae muscles and the tendo-Achilles.

The FAB shown in Figure 85 is the one most used in the US. The cost of that brace is prohibitive in many resource-challenged countries. There are many versions of the foot-abduction brace that have been created in those countries due to financial necessity. The most important features of FAB's, regardless of cost, are: 1) high top shoes or boots that can control the position of tiny feet, 2) a bar of adequate length that connects the shoes/boots and maintains external rotation and dorsiflexion of the shoes/boots [Figure 86].

For unilateral cases, the brace is set at 60 to 70 degrees of external rotation on the clubfoot side and 40-50 degrees of external rotation on the normal side. In bilateral cases, it is set at 70 degrees of external rotation on each side. The bar should be of enough length so that the heels of the shoes are at shoulder width and the bar should be bent 5 to 10 degrees with the convexity away from the child, to hold the feet in dorsiflexion [Figure 87].

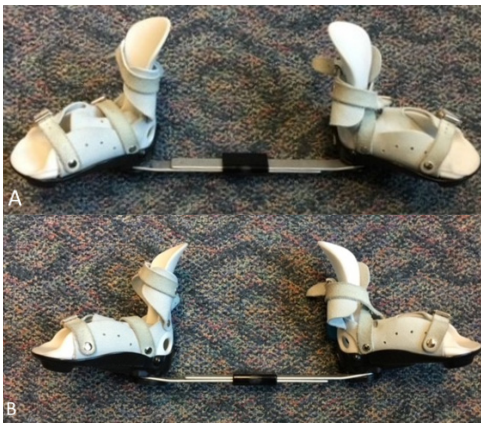

Figure 85. Mitchell-Ponseti foot-abduction brace (FAB) - www.mdorthopaedics.com. A. The boots are externally rotated 60-70 degrees to maintain subtalar joint eversion. The bar is extended until the heels are shoulder width apart for comfort and biomechanical advantage. B. The upward angulation at the ends of the bar, when combined with external rotation of the boots, creates ankle dorsiflexion.

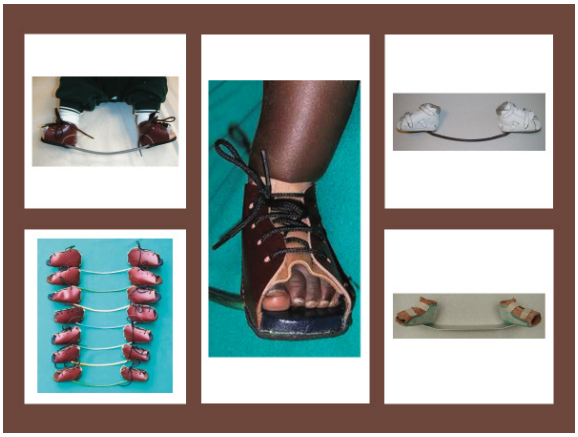

Figure 86. A variety of FAB's made in resource-challenged countries.

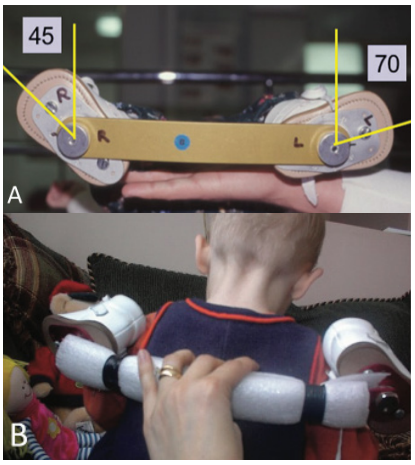

Figure 87. A. Externally rotate a clubfoot 60-70 degrees and a non-clubfoot 40-50 degrees. B. The heels of the shoes/boots are shoulder width apart. The bend in the bar, apex distal, can be appreciated. It is better appreciated in Figures 85 and 86.

A common error is to prescribe a bar that is too short, which is uncomfortable for the child. A narrow brace is a common reason for a lack of compliance [Figure 88].

Ankle-foot orthoses (AFO's) are not a substitute for the FAB because, although they can attempt to maintain dorsiflexion, they have no control of subtalar joint eversion, which is critical to the success of bracing. No other independent single leg brace, not even a fairly well-known hinged version, has been shown to be comparable to the FAB in maintaining clubfoot deformity correction. This particular brace was designed with a full and accurate understanding of the motions of the ankle and subtalar joints but, in my experience, the springs are inadequate to maintain deformity correction against the strength of the muscles even when the child is sleeping.

In my experience, a FAB with hinged connections of the boots to the bar is better tolerated in some children than those FAB's with solid connections. To my knowledge, there have been no prospective randomized controlled studies comparing the efficacy, safety, and tolerance of the hinge-connected bars to those with solid connections. Therefore, except in cases in which the child will absolutely not tolerate the standard FAB, the higher cost of the hinge-connected FAB is not justified.

Bracing protocol

Three weeks after the tenotomy, the cast is removed and the FAB is applied immediately. The brace should be worn full time (23 hours per day) for the first 3 months after the last cast is removed. The 1 hour in which the child is not wearing the brace is not a continuous hour. The FAB should be removed for a few minutes several times per day. These are opportunities to fully reseat the heels into the boots. Tiny feet tend to plantar flex out of the boots despite appropriate tension on the laces and buckles. Over-tightening the laces and buckles can cause soft tissue injury. Reseating the feet every few hours is safer. During a few of the re-seatings, the parents can passively stretch the tendo-Achilles for 30-60 seconds [Figure 23]. Although not a part of the Ponseti method, this does no harm and can only be beneficial. It costs nothing and makes biologic sense.

After the first 3 months of bracing, the child should wear the FAB for 12 hours at night and, if possible, 2 to 4 hours in the middle of the day during napping. It is not practical for the ambulatory child to wear the FAB when awake. This protocol continues until the child is 3 to 5 years of age, preferably until the child is at least 4 years old.

Occasionally, a child will develop excessive heel valgus and external tibial torsion while using the brace. In such instances, the physician should reduce the external rotation of the shoes on the bar from approximately 70 degrees to as little as 40 degrees.

Importance of bracing

The Ponseti manipulations and casts combined with the percutaneous tenotomy (TAT) almost routinely achieve excellent deformity correction. However, without a rigorous post-correction bracing program, relapse of deformities occurs in 31-56% of cases. It is even higher in children who do not wear braces at all. This contrasts with a relapse rate of only 6% in children who strictly follow Dr. Ponseti's bracing protocol (see above). Maintaining deformity correction with bracing may be considered equally important to achieving deformity correction with the casting and TAT.

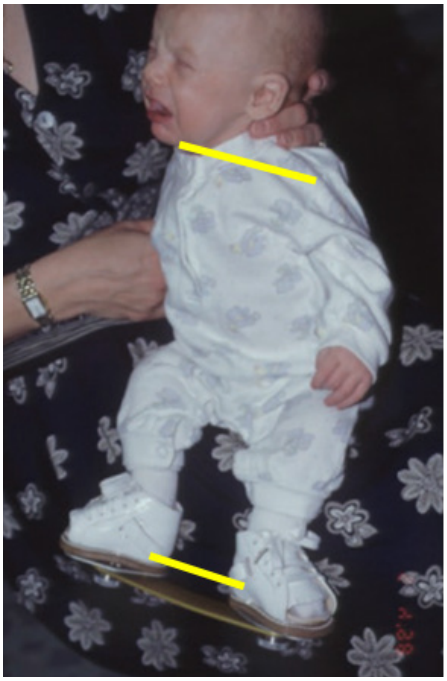

Figure 88. The bar on the FAB is too narrow, causing discomfort. Although the child's shoulders are rotated, they are clearly wider than the distance between his heels on the bar.

Figure 89. Awkward sleeping positions due to the FAB are not surprising. And the FAB most certainly restricts rolling over, especially under covers. But there is no evidence that harm can come to the knees, hips, or spine.

It is important to acknowledge the parents' concerns about the awkward positions children assume in bed, reassuring them that there have never been any studies reporting associated knee, hip, or spine problems in the short or long term [Figure 89].

When to stop bracing

How long should the nighttime bracing protocol continue? Bracing is the most challenging aspect of the Ponseti method to study. For that reason, the bracing protocol has been generalized to all clubfeet regardless of their initial severity, response to casting (number of casts), need for a TAT, or any other feature. This results in some feet being braced longer than they need and others shorter. Dr. Ponseti initially recommended 2 years of nighttime bracing and gradually increased to 5 years. The general recommendation is for 4 years of nighttime bracing.

Most children get used to the brace and it becomes part of their lifestyle. If, after 3 years of age, compliance becomes a problem, it may become necessary to discontinue the brace. The child is closely followed for evidence of relapse whenever it is discontinued, whether by the physician or the child/family. Should early relapse of deformity be observed, bracing should be promptly restarted. If significant relapse of the deformities has already occurred, repeat long leg serial casting is required before reinitiating the nighttime brace. Practically speaking, once the FAB has been discontinued, reinitiation is extremely difficult for the child to accept.

Increasing Brace Compliance

The most compliant families are those who understand Ponseti management and the role of bracing. Take every opportunity to educate the family about the importance of every aspect of Ponseti management, including bracing. It's never too early to start. The prenatal consultation visit should include discussion of the protocol and importance of bracing, and the message should be reiterated during almost every casting visit, on the day of the TAT, and during every follow-up visit during the bracing years. The message should clarify that it is your responsibility, as the physician, to achieve deformity correction with the casts and tenotomy, and it is their equally important responsibility, as parents, to use the brace as instructed to maintain deformity correction.

Written material is very helpful when available. Often written material is more convincing than information given verbally. You can direct the family to online resources and/or create your own.

Instructions for bracing

Demonstrate to the parents how to apply the brace after the post-tenotomy cast is removed. Remove the brace and ask the parent to apply the brace while being supervised. Make certain the infant is comfortable in the brace. If the infant appears to be uncomfortable and not merely upset, remove the brace and examine the skin for evidence of irritation with reddening of the skin.

Most children appear to be uncomfortable in the brace at first. That is to be expected because the legs are, for the first time, tethered together. There will be a period of gradual acceptance of the brace by the child. Parents should avoid the natural tendency to remove the brace if the child is crying. Instead, the parents should cajole, distract, and emotionally comfort the child to help increase tolerance. If the infant learns that by crying the brace will be removed, the pattern will be difficult to correct. Encourage the family to make the bracing a part of the normal life of the infant.

Follow-up

Schedule a return visit in 10–14 days to assess the appropriate use of the brace and the parents' comfort with its use. A telehealth virtual visit can substitute for an in-person visit. If the bracing is going well, schedule the next in-person visit in about 3 months. At that time, the bracing may be discontinued during the day. The brace can, thereafter, be applied during long naps during the day, but must be used for 12 hours at night when sleeping.

Should the family have trouble with bracing, encourage them to call or to return to clinic.

CHAPTER 8

Anterior tibial tendon transfer (ATTT)

Indications

Anterior tibial tendon transfer (ATTT) is indicated in a clubfoot with full correction of all deformities and with good flexibility, but with muscle imbalance that is characterized by overpull of the tibialis anterior in relation to the peroneal muscles, in particular, the peroneus longus and peroneus tertius [Figure 90].

This inherent peroneal muscle weakness is present in 38 – 54% of clubfeet and is, no doubt, due to different genetics than is present in those clubfeet with normal muscle balance. As such, it cannot be corrected by external interventions like casting or bracing. There is no data confirming the efficacy of peroneal strengthening exercises, although it is reasonable to prescribe physical therapy towards that end. Prolonged nighttime bracing has been shown to prevent or delay structural deformity relapses even in those feet with muscle imbalance. Definitive treatment is by means of anterior tibial tendon transfer.

The muscle imbalance is manifest as dynamic supination of the foot during the swing phase of the gait cycle that might persist during early stance but is not a structural deformity [Figure 91].

Although often referred to as dynamic forefoot supination, the dynamic deformity is actually supination/inversion of the subtalar joint, so it is foot supination [represented statically in Figure 13B]. Supination of the forefoot, when it exists, is a structural deformity within the CPU and that almost never exists in a clubfoot except, perhaps, as an iatrogenic deformity. There are no dynamic deformities within the CPU because there is very little motion between the bones in the CPU. Structural forefoot supination may coexist with structural and/or dynamic supination/inversion of the subtalar joint, but structural forefoot supination is not corrected by ATTT. Dynamic and/or structural foot supination may be accompanied by callus formation along the plantar-lateral border of the foot.

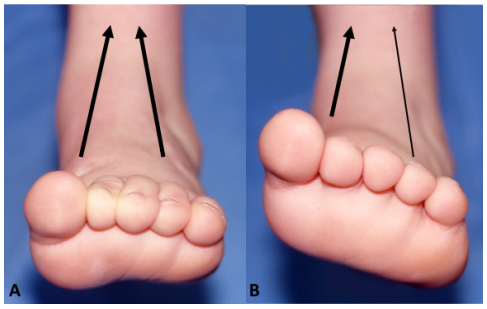

Figure 90. A. Normal foot alignment during active/dynamic dorsiflexion due to balanced muscle forces. The tibialis anterior and peroneus tertius muscles have equal power/strength represented by equal sized black arrows. This muscle balance is identified in 46 – 62% of clubfeet following serial casting, TAT, and bracing. B. This foot has intrinsic muscle imbalance and is dynamically supinating/inverting through the subtalar joint during active dorsiflexion due to normal anterior tibial muscle power but weaker than normal peroneus tertius muscle power (represented by a thinner arrow over the peroneus tertius), as seen in 38 – 54% of clubfeet. Although obviously not apparent on this still-action photo, this foot does not have structural deformity and, in fact, looks exactly like the foot in A at rest. Note that a clubfoot may have both structural and dynamic supination deformities.

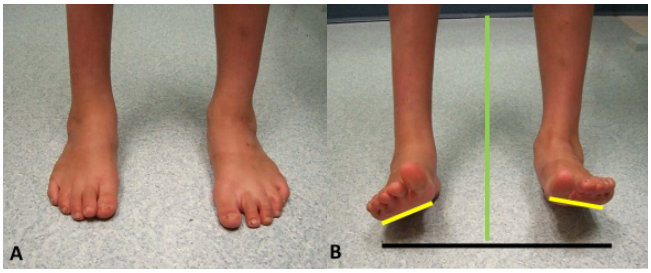

Figure 91. A. Bilateral corrected clubfoot during double leg stance with no apparent structural deformities. B. Dynamic supination during heel standing that is also noted during the swing phase of gait when the child walks. Yellow lines are the transverse planes of the metatarsal heads shown as supinated in relationship to the sagittal plane (green line) and plantigrade position (black line).

Correct deformity first

1. If residual or recurrent deformities (cavus, adductus, varus, equinus) co-exist with apparent muscle imbalance, preoperative serial casting should be performed to correct them before performing an isolated ATTT.
2. If any of the segmental deformities cannot be corrected by a series of preoperative long leg casts, they should be surgically corrected concurrent with the tendon transfer. Most often, this merely involves a repeat TAT and/or a plantar fasciotomy.
3. With severe, resistant deformities that do not respond significantly to preop serial casting and in which a la carte capsular release surgery is required, it is advisable to correct the deformities alone without concurrent ATTT. In this situation, there is risk of creating over-correction with the performance of a simultaneous ATTT.

    a. The apparent muscle imbalance in this scenario could, in fact, be normal balanced muscle power that is being misinterpreted due to the structural deformities. Following rehabilitation from the surgical correction of the deformities, muscle balance can be reassessed and treated secondarily if identified.

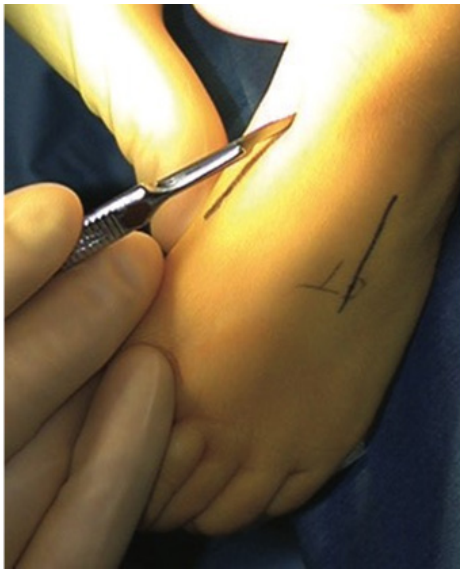

Figure 92. Incision over the dorso-medial midfoot in line with the tibialis anterior tendon. The planned dorso-lateral incision is marked.

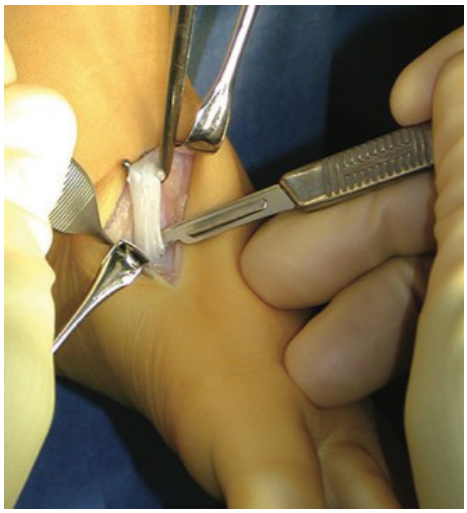

Figure 93. The tibialis anterior is released from the medial cuneiform without injuring the 1st metatarsal physis.

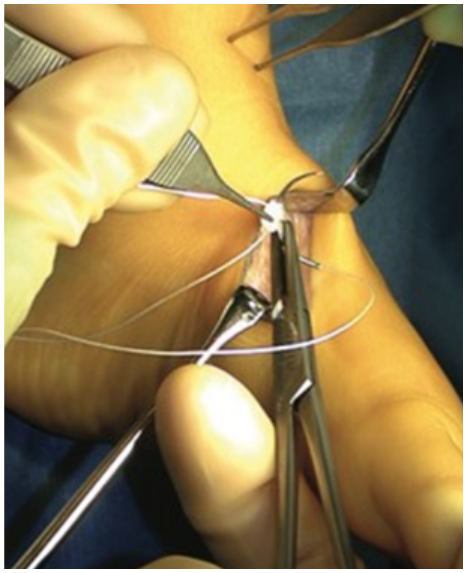

Figure 94. A Bunnell-type 0 Vicryl suture is inserted leaving long tails.

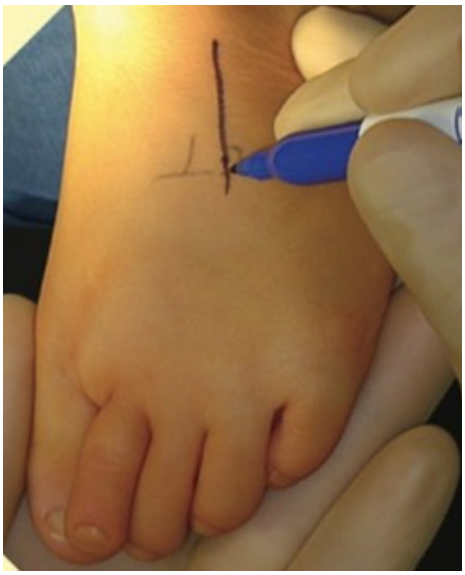

Figure 95. Position of dorsal-central midfoot incision over the lateral (3rd) cuneiform.

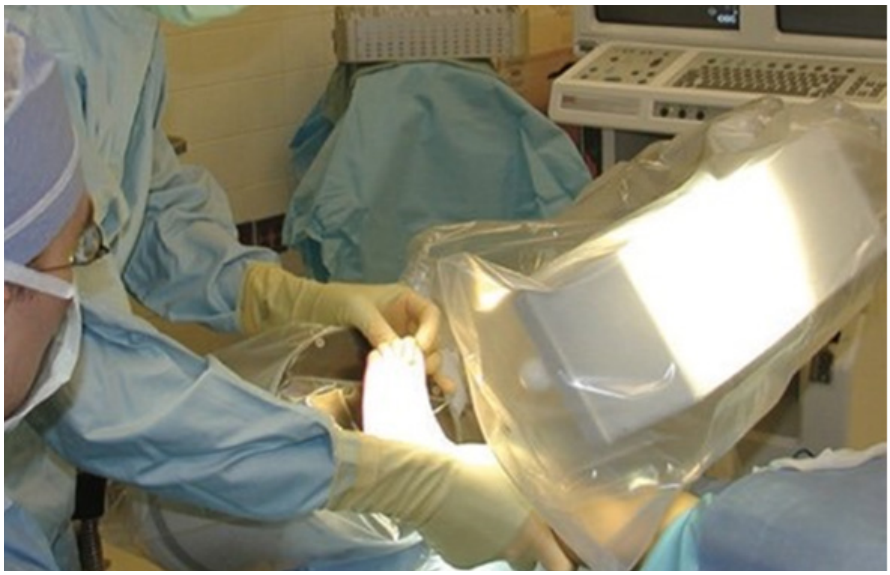

Figure 96. Use mini-fluoroscopy and a 25-gauge needle to identify the lateral cuneiform.

Tendon transfer technique

1. Under a general anesthetic and with peripheral nerve blocks placed, position the child supine with a folded towel under the ipsilateral buttock to internally rotate the limb to neutral.
2. Use a high thigh tourniquet.
3. Make a 4 cm longitudinal incision over the dorso-medial midfoot in line with the tibialis anterior tendon [Figure 92].
4. Expose and isolate the tibialis anterior tendon from the distal edge of the extensor retinaculum to its insertion on the medial cuneiform. Avoid dissecting so far distally that the 1st MT physis is exposed and possibly injured.
5. Taper the flared end of the tendon to the thickness of the more proximal visible portion and release it from the medial cuneiform [Figure 93].
6. Insert a Bunnell-type #0 absorbable suture in its end, leaving long tails on both limbs of the suture [Figure 94].
7. Make a 4 cm longitudinal incision over the central midfoot in line with the 3rd MT/lateral (3rd) cuneiform. Avoid/retract the superficial peroneal nerve [Figure 95].
8. Bluntly expose the lateral cuneiform between the EDC and the peroneus tertius.
9. Using a 25-gauge needle and mini-fluoroscopy, identify the lateral cuneiform [Figure 96].
10. Make a cruciate incision in the periosteum of the lateral cuneiform and elevate the 4 triangular corners with a Freer elevator [Figure 97].

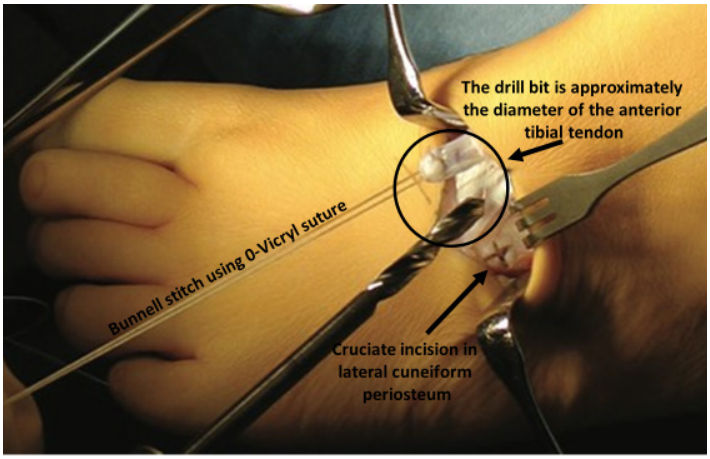

Figure 97. Cruciate incision in dorsal periosteum of lateral cuneiform marking the intended site for the drill hole. The drill bit and the tibialis anterior tendon are of equal diameter.

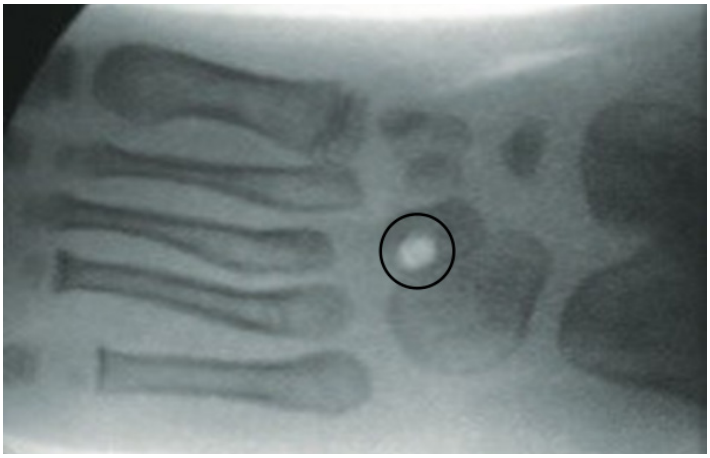

Figure 98. Mini-fluoroscopic image of drill hole (white circle within black circle) in lateral cuneiform.

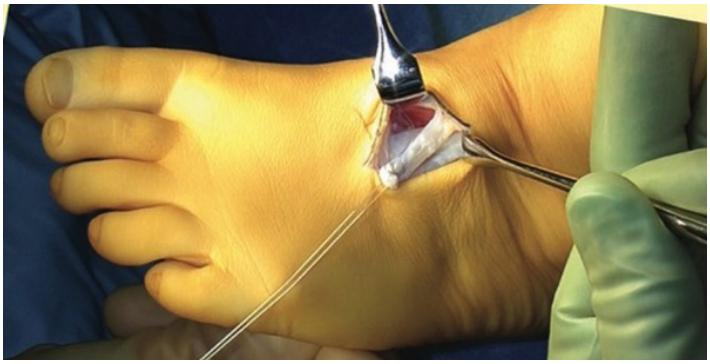

Figure 99. The tibialis anterior tendon has been passed from dorso-medial to dorsal-central deep to the extensor tendons and the extensor retinaculum.

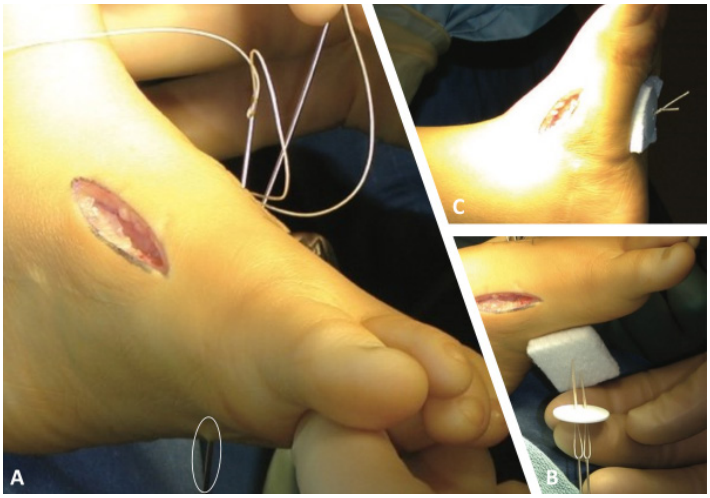

Figure 100. A. Keep 1 Keith needle in the hole and sticking out the bottom of the foot (white oval) while passing the 2nd needle to avoid spearing the 1st suture with the 2nd needle. Then pull both needles through the hole. B. Pass the Keith needles through a thick felt pad and plastic button. C. With the ankle dorsiflexed 5-10 degrees and firm tension pulling the tendon into the hole, tie the sutures together. Upon briefly releasing the dorsiflexion force on the foot, it should drop down to no more than 5-10 degrees.

11. Make a drill hole through the lateral cuneiform, including the plantar cortex, aimed somewhat lateral to the mid-arch. The diameter of the hole should be slightly greater than the diameter of the tendon [Figures 97 and 98].
12. Transfer the tendon laterally from the dorso-medial incision to the central incision remaining deep to the extensor tendons and deep to the extensor retinaculum. Release fatty or fibrous bands that prevent the tendon from assuming a reasonably straight vector from proximal to distal in its new location [Figure 99].
13. Thread 1 of the long suture tails into each of 2 large Keith needles. Pass one of the needles through the hole until the tip pierces the plantar skin, but don't pull it through yet. Leave the needle shaft in the hole. Pass the other needle, exiting 5-7 mm away from the 1st on the plantar surface of the midfoot. If the 1st suture were left bare in the hole, the 2nd Keith needle would almost certainly pierce and weaken it. Pull both needles and sutures through the hole and out the plantar surface of the foot [Figure 100A].
14. Pass the needles through a thick felt pad and through different holes in a large button [Figure 100B].
15. With the foot held in at least 5-10 degrees of dorsiflexion, pull the tendon firmly into the drill hole and tie the sutures over the felt pad and button on the plantar surface of the midfoot [Figure 100C].
16. Supplement the button fixation by suturing the tendon to the dorsal periosteum of the lateral cuneiform with a 2-0 absorbable suture [Figure 101].

17. Approximate the skin edges with interrupted subcutaneous 3-0 absorbable sutures and a running subcuticular 4-0 absorbable suture [Figure 102].
18. IF significant cavus and/or equinus did not correct with serial preoperative casting, perform a mini-open plantar fasciotomy and/or percutaneous tendo-Achilles tenotomy BEFORE setting the tension on the tendon transfer.
19. Apply a long leg, bent knee clubfoot cast with maximum dorsiflexion and abduction/eversion molding [Figure 103].
20. Remove the cast 6 weeks later
21. A CAM boot can be used for an additional 2 weeks in children over 4 years of age.
22. In children under age 4, resume nighttime FAB until age 4 years.
23. In older children, and in particular those who required a concurrent TAT, consider 6 months of nighttime bracing with an adjustable dorsiflexion AFO.
24. In some cases, physical therapy is required to regain strength and normalize gait.

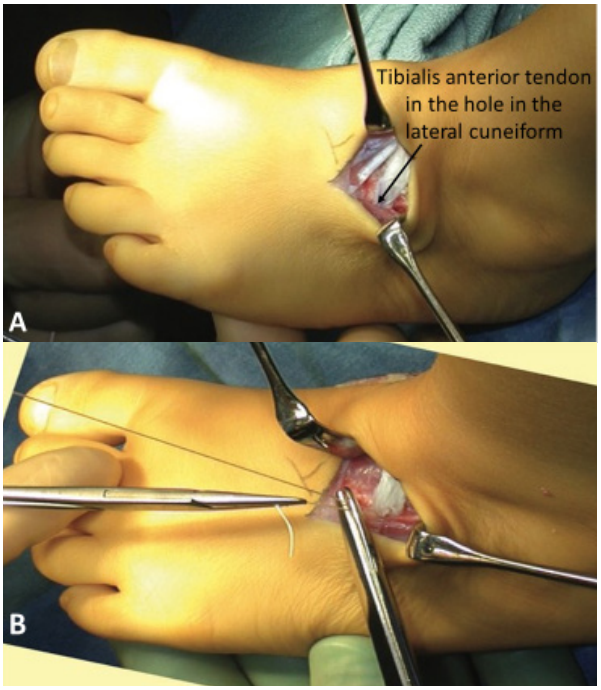

Figure 101. A. Tibialis anterior has been pulled into the hole in the lateral cuneiform with its lead sutures which were then tied over a thick felt pad and button. B. Further secure the transfer with a figure-8 absorbable suture between the tendon and the dorsal periosteum.

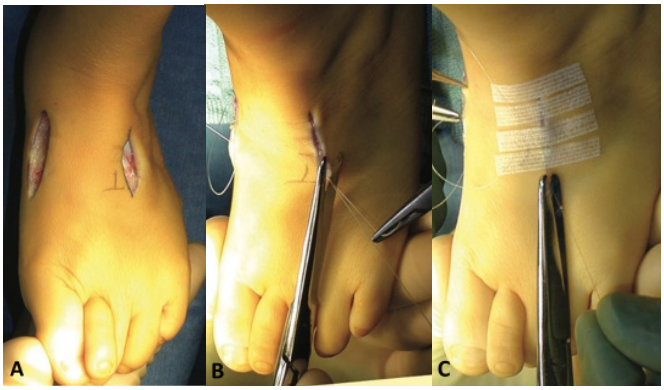

Figure 102. A. Good static muscle balance. B. Close incisions with absorbable subcutaneous and subcuticular sutures. C. reinforce with Steri-Strips.

Figure 103. Long leg bent knee cast.

CHAPTER 9

Relapses

Recognizing relapses

Once the final cast is removed and the bracing is started, the child should be seen in clinic regularly to check on compliance with bracing and to identify evidence of relapse of the deformities. The term relapse means recurrence of previously corrected, or even incompletely corrected, structural deformities, i.e. recurrent cavus, adductus, varus, and/or equinus. It has been reported to occur in 31-56% of clubfeet. Dynamic deformity, specifically dynamic supination of the foot, is due to muscle power imbalance and is not a relapse. It is an inherent, genetically determined feature of some clubfeet that may lead to structural relapses. Structural relapsed deformities are amenable to the same external interventions that are used to correct the original deformities, i.e. serial casting and TAT; however, they may not be as responsive, due to the advanced age of the child. Dynamic deformities are not amenable to correction by external interventions, because those interventions cannot balance muscle forces. Bracing appears to delay the development of structural deformities in feet with dynamic deformities. The ultimate treatment for dynamic deformity is anterior tibial tendon transfer (see chapter 8).

A recommended follow-up schedule after the last cast is removed is:

At 2 weeks (optional) to check on the parents' ability to apply the FAB appropriately, compliance with full-time bracing, and skin tolerance of the brace.

At 3 months to graduate to the nights-and-naps schedule.

Every 3-4 months until age 2 years to monitor compliance and check for relapses.

Every 6 months until age 4-5 years to monitor compliance and check for relapses.

At 6 months and 1 year after discontinuing the brace to check for relapses.

Thereafter, consider every 1-2 years until skeletal maturity. Truthfully, most families choose not to return unless they are concerned about a possible relapse.

Reasons for relapses

The most common cause of relapse is noncompliance with the bracing program. If relapse occurs in infants who are braced according to the protocol, the cause is an underlying muscle imbalance of the foot. Because compliance cannot be reliably monitored, the cause is difficult to ascertain.

Pattern of relapse

Relapses, i.e. recurrent deformities, usually occur in the opposite order of deformity correction, which is cavus, adductus, varus, and equinus. Equinus is usually the first deformity to recur followed by varus, then adductus and cavus. The order may, however, vary.

Management of relapses

Do not ignore relapses! At the first sign of relapse, whether in Ponseti-treated clubfeet or those treated surgically, apply one to three (or as many as it takes) long leg casts to stretch the recurrent contractures and regain deformity correction. The longer one waits, the more severe and resistant the deformities become, thereby increasing the duration of casting and perhaps leading to the need for more extensive surgery to achieve deformity correction. This cast management is the same as the original Ponseti casting program. Once the deformities are corrected with casting, the bracing program is restarted. Even in the child with a severe recurrence, casting is often very effective.

Weekly casting is continued until the foot can be brought to at least 10˚ of dorsiflexion with full correction/slight over-correction of cavus, adductus, and varus. If this is not achieved in 4–5 casts in children under 4 years of age, percutaneous TAT is repeated. Once the equinus is corrected, resume the nighttime bracing program.

Management of residual deformity

If cast correction is incomplete and the residual deformity is unacceptable, operative correction is required. Start with Ponseti casting. Even if cast correction is incomplete, the severity of the deformity is reduced, and less extensive surgery will be required to complete the correction. Less surgery means less stiffness, weakness, and pain in adult life.

Description of the surgical techniques for correction of recurrent or residual clubfoot deformities is beyond the scope of this monograph but will be summarized here.

Select the procedure(s) based on the age of the child and the severity and type(s) of deformity. Be aware that clubfeet requiring operative correction are prone to recurrence throughout childhood (25–50%).

A la carte postero-medial-lateral soft tissue releases are indicated in infancy and early childhood for correction of resistant residual deformities.

Bony procedures are indicated in later childhood if soft tissue releases result in residual structural deformities. The options include osteotomies and, rarely, joint fusions.

Gradual deformity correction using external fixation devices may be indicated in older children in select circumstances and, perhaps, environments.

Structural deformity relapse and dynamic muscle imbalance

1. If residual or recurrent deformities (cavus, adductus, varus, equinus) co-exist with apparent muscle imbalance, preoperative serial casting should be performed to correct them before performing an isolated ATTT.
2. If any of the segmental deformities cannot be corrected by a series of preoperative long leg casts, they should be surgically corrected concurrent with the tendon transfer. Most often, this merely involves a repeat TAT and/or a plantar fasciotomy.
3. With severe, resistant deformities that do not respond significantly to preop serial casting and in which a la carte capsular release surgery is required, it is advisable to correct the deformities alone without concurrent ATTT. In this situation, there is risk of creating over-correction with the performance of a simultaneous ATTT.
    a. The apparent muscle imbalance in this scenario could, in fact, be normal balanced muscle power that is being misinterpreted due to the structural deformities. Following rehabilitation from the surgical correction of the deformities, muscle balance can be reassessed and treated secondarily if identified.

Addendum: The Ponseti Method – Staying true to it!

(from: Mosca VS. The Ponseti Method: Staying true to it! JPOSNA 2021;3).

I was trained to believe that the definition of a clubfoot was a congenital equino-cavovarus foot deformity "that required surgery". And that didn't mean a simple percutaneous tendo-Achilles tenotomy (TAT) that could be safely and effectively carried out in an outpatient clinic room with local anesthesia. It meant a la carte surgery that would require a tendo-Achilles lengthening (TAL) and posterior ankle capsulotomy at least, and a circumferential postero-medial-lateral release at most.

Following my orthopedic residency and pediatric orthopedic fellowship, both at highly regarded institutions, I started my clinical and academic practice in 1985 at Children's Orthopedic Hospital (as it was known at the time) and the University of Washington School of Medicine in Seattle. And for the next 10 years, I treated 60-70 clubfoot deformities per year, at least 90% of which underwent a la carte surgery. I never performed a simple, isolated TAT. It wasn't what I was taught. And the serial manipulation and casting techniques that I had learned and then carried out for the first 3-6 months of each child's life never seemed to result in residual deformity for which a simple TAT would be enough surgical treatment.

Children's Orthopedic Hospital, now called Seattle Children's Hospital, was then and remains the only children's academic full-service hospital that provides services to the five northwestern US states of Washington, Wyoming, Alaska, Montana, and Idaho (WWAMI). Those states comprise 27% of the land mass of the United States, though not 27% of the population of the US. Because so much of the land mass is rural and remote, general orthopedic surgeons in those states have historically treated clubfoot deformities as a service to their local populations. But with low volumes, their clubfoot casting and surgical skills were perhaps not as well developed as those who practiced in the higher volume centers in the WWAMI region and around the rest of the country. In those early years, there were no fellowship-trained pediatric orthopedic surgeons in the WWAMI region except in Seattle, Spokane, and Boise. When things didn't go well, the local orthopedic surgeons generally did not feel comfortable performing the next operation on their patients with clubfeet, so many referred their patients to me and other fellowship-trained pediatric orthopedic surgeons in the major population centers.

From the day I started in practice, it seemed that I was performing more second, third, and fourth clubfoot operations than primary procedures. There were under-corrections, recurrences, over-corrections at the talonavicular and subtalar joints, dorsal bunions, flat-top tali, and many iatrogenic deformities that could only be named by stringing together the terms for each of the segmental deformities (eg. abducto-calcaneo-valgus). It was the large number and variability of these iatrogenic surgically created deformities in former clubfeet that initiated my quest to truly understand the biomechanics of the child's foot and develop the assessment principles for managing them. I felt that only those understandings would enable me to develop an algorithmic approach to management.

But the worst problems were not correcting the deformities. The real problems were the pain, stiffness, and weakness created by "another" operation. Another operation (or two) could "correct" the iatrogenic deformities and at least temporarily relieve pain, but nothing could be done for the stiffness and weakness that increased with each subsequent operation. And stiffness equals pain and functional disability. There were many times I felt that an amputation would provide the child far better long-term comfort and function than another salvage operation.

It was with great interest and incredulity that I read the landmark 1995 article by Cooper and Dietz (1) in which they reported on the appearance, comfort, and function of a large number of Dr. Ponseti's patients who were, at that time, between 25 and 42 years of age (average 34 years). Their feet were essentially indistinguishable from the normal controls that were used in the study. Dr. Ponseti's was a non-surgical technique. I couldn't imagine a comparable series of clubfeet that had been surgically treated by me or any high volume and highly regarded clubfoot surgeon anywhere in the world. Although I and others had been reading Dr. Ponseti's articles going back to 1963 (2), we never "got it". Kite's method (3) didn't work as well in our hands as it apparently did in his, so why should Ponseti's? Then came the second of the one-two punch, the 1996 publication of Dr. Ponseti's now classic landmark "green book" entitled Congenital Clubfoot: Fundamentals of Treatment (4). Here, for the first time, we could read in detail and in one source exactly what he had been saying and doing, things that are generally not the purview of individual journal articles. And the details matter.

In 1996, I (and others who had never been to Iowa) almost immediately adopted the Ponseti method. Some of us committed that we would not modify the method

at all unless the part that we considered modifying was subjected to careful scientific study. There are many parts to the method beyond the basic concept of manipulate and cast. Changing any part could, inadvertently, eliminate or seriously modify the key element(s) to the proven success of the overall method. And it wouldn't be apparent until too late. I've developed, modified, and written about many pediatric foot procedures during my career but have taken great efforts not to modify this method. Twenty-five years later, I still have not modified the method as I learned it from Dr. Ponseti and his book, except for the type of casting material I use, which has been shown to have no effect whatsoever on the outcome of treatment. In his final years, Dr. Ponseti would tell my patients who contacted him, concerned about my use of semi-rigid casting tape, that it was "OK, because Dr. Mosca knows how to use it".

It is important to emphasize that the Ponseti method is not a panacea for this most common congenital musculoskeletal deformity. Dr. Ponseti never promised that his method would yield complete anatomic correction, just that the resulting preservation of range of motion and strength would enable long-term comfort and strength for most normal activities. Clubfeet do not and cannot become normal, because they are genetically programmed congenital deformities. However, the reliably achievable stated goals are laudable. Importantly, if true to the method, the need for open joint surgery with its associated pain, stiffness, and limited function in adulthood is largely eliminated. There has never been another clubfoot treatment method with comparable reported short- and long-term outcomes.

According to a survey of members of the Pediatric Orthopedic Society of North America (POSNA) published in 2012 by Zionts, et al., nearly all (96.7%) of those surveyed stated that they use the Ponseti treatment method (5). Although this was not a treatment outcomes study, the survey results indicated near universal adoption of Ponseti's techniques for initial clubfoot deformity correction and maintenance.

However, clubfoot treatment using the Ponseti method is not always easy, even for the highly skilled, highly experienced pediatric orthopedic surgeon. Six casts, percutaneous TAT, and tolerance of the foot-abduction-brace (FAB) for 4 years is typical, but not guaranteed. And relapses, defined as recurrent structural deformities, are common. Relapses may be due to non-compliance with bracing and/or muscle imbalance. Whereas muscle imbalance can contribute to structural deformity relapse, muscle imbalance itself is not a relapse. Instead, it is an inherent neuromuscular feature of some genetically different clubfeet that, so far, can only be identified clinically. Rates of relapse within the first 4 years of life reported by experienced and skilled pediatric orthopedic surgeons, including Dr. Ponseti, range from 31-56% of clubfeet (2, 6-10), not an insignificant percentage.

Recognizing the high rate of relapses, Dr. Ponseti included in his method not only the techniques to achieve and maintain initial deformity correction, but also the approach to managing the relapses that inevitably occur. A follow-up survey of POSNA members by Hosseinsedeh, et al, published in 2019, reported that Ponseti's recommended management of relapses was not closely adhered to (11). Specifically, Dr. Ponseti recommended repeat serial long leg casts followed by nighttime bracing for early relapses. Seventy-eight percent of survey respondents followed his recommendations, but over 11% went straight to surgery for a repeat TAT and/or an anterior tibial tendon transfer (ATTT). And he recommended preoperative serial cast treatment to correct recurrent structural deformities in feet with muscle imbalance prior to ATTT, with the possible addition of a concurrent repeat TAT in selected patients. Thirty-eight percent of respondents reported not performing preliminary casting.

It is important to reiterate that ATTT is a recognized part of Ponseti management and not a failure of treatment. Three studies from Iowa and one from Los Angeles with follow-up durations of 5-42 years reported rates for ATTT from 38-54% of clubfeet (1, 2, 6, 12, 13). However, a concerning finding in the second POSNA survey was that, in addition to ATTT, 43% of respondents reported performing concomitant posterior ankle capsular releases. Not only is posterior ankle capsulotomy not a part of Ponseti management, he discouraged all capsular releases. It is certainly possible that at least some of the 38% of respondents who did not cast preoperatively could have avoided the need for posterior capsulotomy by having done so.

Finally, the second POSNA study revealed great variability in the bracing protocol in comparison with Ponseti's latest recommendations. This finding also, no doubt, contributed to the relapses and frequent need for capsulotomies. There may, however, be some justification for this variability because Dr. Ponseti's recommended bracing protocol evolved from 2 years to 4 years over a period of decades and even within his book from cover to cover (4). But his final recommendation of 3 months full time and 4 years nighttime bracing is associated with the best treatment outcomes, so it must be learned and followed.

The reasons for apparent partial and perhaps decreasing dedication to all aspects of the Ponseti method are, no doubt, many. And it's a slippery slope from a little surgery to more extensive surgery.

First, it took several years after the 1995 article and 1996 book for most orthopedic surgeons to convert from

surgical management of clubfeet to the Ponseti method. It took at least as many years to eliminate most of the iatrogenic clubfoot deformities that were so pervasive in prior years. So, pediatric orthopedic surgeons who were trained in the last 15-20 years have not seen the many well-intended, but disastrous, outcomes of clubfoot surgery. They don't have the incentive that their teachers had/have to stay true to the Ponseti method and avoid clubfoot surgery. It perhaps seems easier and less morbid to "just operate" on a particularly resistant clubfoot than to persist with the Ponseti method. According to the writer and philosopher George Santayana (1905), "Those who cannot remember the past are condemned to repeat it". I believe the corollary is that "those who have not studied or lived the history are more likely to repeat it".

Second, and as noted earlier, clubfoot treatment using the Ponseti method is not always easy, even for those of us who have applied over 20,000 clubfoot casts. There are the "fat" feet that slip out of the casts or develop pressure sores and take many more than 6 casts before the TAT can be performed. And then these feet are challenging to maintain in the FAB because of the excessive fat. More slippage and pressure sores as well as recurrent deformities. It would seem to be easier to "just operate" than to cast for prolonged periods of time, brace, recast, reinitiate bracing, and so on.

Third, clubfoot casting is an art and not a science. It is a technical skill, but different than many surgical skills due to the wide variations in the severity and rigidity of clubfoot deformities and their responsiveness to treatment. Not all clubfeet are alike. And like any technical procedure, skill mastery is based on high volume repetition. So in smaller communities where the number of children with clubfeet is low, the well-intended and well-trained pediatric orthopedic surgeon cannot be expected to have the same success rate as the high volume, experienced practitioner in regard to number of casts required, need for more than a simple TAT, and the avoidance of cast slippage and pressure sores. Surgery is faster and may seem to be easier for the novice or low volume practitioner.

Fourth, there are wide variations in the tolerance of the child for the treatment and in the compliance of the family with all components of the method. Some of these variations are regional or cultural and not under the control of the pediatric orthopedic surgeon. Surgery may seem to be the only way to achieve and maintain deformity correction in these children through no fault of the surgeon.

Fifth, I know that not everyone treating clubfeet in the late 1990's truly embraced the Ponseti method. Their rate of joint surgery diminished, but not to the extent expected, i.e. less than 5% (this number does not include the TAT or ATTT). Some just liked operating on clubfeet. I understand that because the primary reason I became a pediatric orthopedic surgeon was that I liked operating on clubfeet. That's a fact! Yet, I was one of the first to abandon that passion when I realized it was not in the child's best interest. So, if some of the teachers in my generation did not and have not fully embraced the Ponseti method, how can we expect their students to fully embrace it?

Sixth, I am aware that some pediatric orthopedic surgeons who treat a large volume of clubfeet do not train the next generation of residents and fellows in the Ponseti method for fear of cast slippage, pressure sores, and internet scrutiny by the many parental clubfoot support groups. In fact, some of the internet parental support groups have taken on a life of their own, being excessively critical of both low and high-volume providers, thereby damaging their reputations and even leading some to abandon the treatment of clubfoot all together. For some surgeons, the feeling might be that a well-executed operation will not draw a family to the support groups in the same way that a cast pressure sore will. After all, Dr. Ponseti said that approximately 5% of clubfeet do not respond to his method. Neither the family nor anyone else could ever know if the index child undergoing surgery is the 1 in 20 or one of the 19 in 20 who are treated surgically by a particular practitioner.

Litigation has been brought against some pediatric orthopedic surgeons for creating generally benign and treatable complications of Ponseti casting. This has been promulgated by unethical online support for this inappropriate parental support group scrutiny by one or more pediatric orthopedic surgeons - who even profit handsomely for their support. That support adds to the decreased interest by some pediatric orthopedic surgeons in continuing to treat clubfeet. Then what? Who will treat clubfeet? Will families have to travel great distances every week to the few chosen Ponseti practitioners with the volume of patients to become and stay good at it?

We all eventually retire and/or move on to the great clubfoot clinic in the sky. The incidence of clubfoot is not changing. We need to support and offer ongoing education to those providers who may be struggling to master the Ponseti method but trying very hard to get it right. Chastisement and criticism of our colleagues are absolutely in the worst interest of the children with clubfeet and their families. We must train the next generation. A slipped cast can be reapplied. A swollen foot, shallow pressure sore, and mild rocker bottom deformity will resolve following a 1-2 week cast holiday in most cases. Those are undesirable but not "never

events", regardless of the practitioner's level of skill, training, experience, and proclamation. And they are not litigious occurrences. The families must be so-informed rather than incited. We must train the next generation in the most successful implementation of the Ponseti method. More repetitions of any technical skill are directly related to better outcomes, so patients should stay with their practitioner who can salvage the minor complication in the best interest of the child and learn from the experience. No technique is without potential risks and complications regardless of who is performing it.

We need to stay true to all components and aspects of the Ponseti method because, if this non-surgical method is unsuccessful, how successful can the infrequently performed surgery be? When surgeons were operating on clubfeet frequently, the results were often disastrous. If surgeons are now operating infrequently and only on the most severe and rigid clubfeet, the results should be even worse. My clinical fellows may see one or two clubfoot operations during their year in Seattle, not enough to become expert at this technically challenging procedure.

How then does one gain and maintain this intricate surgical skill that is rarely indicated? Should pediatric orthopedic surgeons participate in mission trips to underserved areas in the world to gain and maintain surgical experience? Should the rare child with a clubfoot that does not respond to Ponseti management be referred to the regional pediatric orthopedic surgeon with the most surgical experience with the technique? All hypothetical questions without answers.

Yes, there are consequences to mastering the Ponseti method, but they pale in comparison with the consequences of not mastering it.

Four hundred years BC, Hippocrates described a method for clubfoot management that, in all regards, was the early version of the Ponseti method. What followed were 2,400 years of increasingly complex clubfoot surgery because of disregard or ignorance of his teachings. Hopefully, the Ponseti method will not befall the same fate. Stay true to it. The kids deserve that commitment from all of us.

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CHAPTER 10

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400 BC Hippocrates

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