DOI: https://doi.org/10.30795/jfootankle.2026.v20.2006

Special Article

Clubfoot: an updated review

Alexandre Francisco de Lourenço[1]

1. Escola Paulista de Medicina, Universidade Federal de São Paulo, São Paulo, SP, Brazil.

Abstract

Congenital talipes equinovarus, commonly referred to as clubfoot, is one of the most frequent congenital deformities of the lower limb. Over the last few decades, a major paradigm shift has occurred in its management, with the Ponseti method becoming the gold standard for treatment worldwide. However, other treatment modalities, including selected surgical procedures, may still be required. This review aims to provide an updated overview of the epidemiology, etiology, pathoanatomy, clinical assessment, treatment principles, recurrence management, and long-term outcomes of clubfoot, emphasizing current evidence-based practices relevant to foot and ankle specialists.

Level of evidence V; Expert opinion; Therapeutic studies - investigating the results of treatment.

Keywords: Clubfoot; Congenital talipes equinovarus; Ponseti method; Pediatric foot deformities.

Introduction

Congenital clubfoot is a complex condition in which there is an evident deformity of the foot present at birth, and that also involves the leg, where calf atrophy can be observed. This atrophy is present from the neonatal period, and it becomes more evident with growth. Although this atrophy is less noticeable in bilateral cases, it is important to inform parents from the outset that it is an intrinsic feature of clubfoot and not a consequence of the treatment. Placing a silicone implant to augment the calf may be considered after skeletal maturity; however, this is purely for aesthetic purposes, as the atrophy does not impair limb function (Figure 1)[1,2].

The main characteristics of clubfoot are equinus and varus of the hindfoot, cavus of the midfoot, and adduction of the forefoot. Clubfoot is a condition that has long been surrounded by controversy in almost all aspects, from its etiology and pathological anatomy to methods of evaluation and types of treatment. In fact, regarding treatment methods, clubfoot has undergone an exciting shift in recent years. Many orthopedic centers have reported a marked decline in the number of patients requiring extensive surgical releases for clubfoot. We have observed a historical shift toward abandoning an almost exclusively surgical approach by orthopedists treating this severe deformity, in favor of a more functional and less aggressive approach, driven by the enormous worldwide impact of the conservative Ponseti method, as will be discussed throughout this review[3-7].

Nevertheless, as Ponseti himself noted, recurrence occurs; some feet do not respond as well to his method, and surgical releases may be required[3].

Incidence

Clubfoot is one of the most common congenital deformities of the foot. Its incidence varies widely throughout the world according to race and sex. It is particularly high in Southeast Asia, where it may reach up to 7 per 1,000 live births.

In Brazil, particularly in São Paulo, an incidence of 2.17 per 1,000 births was observed[8], while in the United States, the incidence is around 1 per 1,000 births. Males are affected more often than females in a 2:1 ratio. Bilateral involvement occurs in approximately 50% of cases, and when only one side is affected, the right side has a slightly higher incidence than the left[7-9].

Study performed at Escola Paulista de Medicina, Universidade Federal de São Paulo, São Paulo, SP, Brazil.

Correspondence: Alexandre Francisco de Lourenço. Rua José Maria Lisboa, 1370, Apto 41, 01423-002, São Paulo, SP, Brazil. **Email:** alex.dot@uol.com.br.
Conflicts of interest: None. **Source of funding:** None. **Date received:** March 31, 2026. **Date accepted:** March 31, 2026.

How to cite this article: Lourenço AF. Clubfoot: an updated review. J Foot Ankle. 2026;20(1):e2006.

Etiology

The etiology of clubfoot remains unknown, with limited substantive advances reported in the literature to date. Several theories have been proposed involving neurological, muscular, mechanical-postural, and hereditary factors.

There is even a theory of arrest of embryonic development proposed by Bohm (1929)[10], who noted that the characteristics of the feet of a fetus between six and eight weeks of gestation are very similar to those of clubfoot, including equinus, supination, forefoot adduction, and medial deviation of the talar neck. On the other hand, talonavicular dislocation, which is one of the most recognized alterations in clubfoot, is not present at any stage of normal foot development[7,10].

Irani and Sherman dissected 11 clubfoot specimens and concluded that the primary defect was deviation of the anterior portion of the talus. They suggested that the abnormality of the talus resulted from a germplasm alteration[11].

MacNicol and Nadeem studied 95 feet initially diagnosed as idiopathic clubfoot and observed that 46% showed abnormalities in somatosensory evoked potentials, suggesting an underlying neurological pathology[12]. Undoubtedly, there is an association between several neuromuscular disorders and equinocavovarus deformity of the feet, such as arthrogryposis and myelomeningocele. On the other hand, the occurrence of isolated deformity in children without other abnormalities, as observed in idiopathic cases, argues against an exclusively neurological origin for clubfoot.

Rigidity of the soft tissues on the medial side was demonstrated by Ippolito and Ponseti, who identified an increase in collagen fibers in the ligaments and tendons of clubfoot. They studied five clubfeet and three normal feet from aborted fetuses between 16 and 20 weeks of gestation and found alterations in the shape, size, and articulations of the tarsal bones. They also noted a decrease in muscle fibers in the posteromedial portion of the distal third of the leg and an increase in connective tissue in the adjacent muscles, tendons, and fascia. Thus, they concluded that fibrotic retraction might be the primary etiological factor of clubfoot[13]. However, this deformity can also be observed in patients with marked ligamentous laxity, such as those with Down syndrome.

Regarding genetic contribution, it is known that heredity follows a polygenic pattern, since the risk decreases with lower degrees of kinship, increases when both parents have clubfoot, and also increases when more than one family member is affected. Recent evidence suggests that genes involved in limb development, apoptosis, and muscle function may contribute to the condition. A systematic review on the etiology of clubfoot has highlighted several of these gene families and pathways; however, a major candidate gene has not yet been identified[14].

Environmental factors, including maternal smoking, have also been associated with the development of clubfoot[15].

Pathology

Despite the lack of new evidence on the pathological anatomy of clubfoot, several characteristics should be recognized, including thickening and contracture of soft tissues, such as ligaments, tendons, and joint capsules. For many authors, the main alteration is talonavicular dislocation or subluxation, although medial deviation of the cuboid and calcaneus also occurs, producing hindfoot varus. The entire structure remains in equinus, resulting in the characteristic equinovarus deformity of clubfoot.

A circulatory alteration has also been described, consisting of hypoplasia or absence of the anterior tibial artery, which may be present in many cases and has important implications in surgical anatomy, since the medial surgical approach may compromise the posterior tibial artery, which may represent the main vascular supply to the clubfoot[16].

Many years ago, even before the use of sophisticated imaging techniques, it was already described that medial and plantar deviation of the navicular, cuboid, and calcaneus occurs around the talus. There is also a parallelism between the axes of the talus and calcaneus, both in the anterior and lateral views.

The Kite angle, a radiographic parameter formed by the axes of the talus and calcaneus on the anteroposterior view, is decreased, demonstrating hindfoot varus.

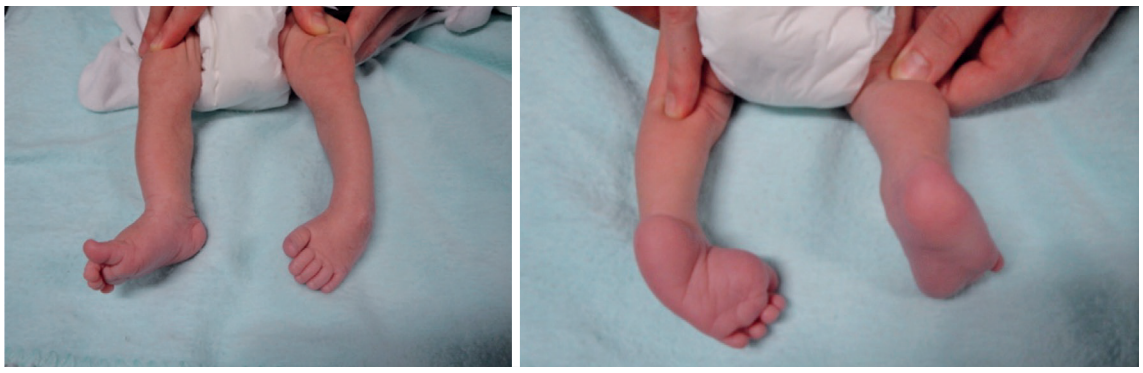

Figure 1. Calf atrophy is readily apparent at initial presentation, before treatment begins.

Although all soft tissue alterations involving the tendons and ligaments of the posterior and medial portions of the foot are important, the main component of the deformity in clubfoot is the medial displacement of the navicular-calcaneus-cuboid complex relative to the talus.

The talus itself presents with its distal extremity deviated plantar and medially, and its declination angle, formed by the axes of the neck and body, is significantly decreased. In the normal talus, this angle measures approximately 160°, whereas in clubfoot it is close to 90°.

Despite the supinated appearance of the foot, the forefoot is pronated relative to the hindfoot (plantar flexion of the first metatarsal), which is responsible for the cavus deformity. Soft tissue contractures maintain the disarrangement among bones and joints.

Intrinsic bone deformities are also present, such as the altered shape of the talus, which has a shortened neck due to its altered declination angle. This leads to alterations in the relationships between the bones of the clubfoot.

Because of the intrinsic talar deformity and the malposition of the navicular–cuboid–calcaneus complex, the talar head becomes dislocated relative to the so-called "acetabulum pedis."

As a result of medial deviation of the navicular, a false articulation may occur with the medial malleolus, and the lateral part of the talar head remains uncovered.

At the inferior portion of the talus, alterations of the subtalar joint facets may also occur, and the calcaneus may present with a poorly developed sustentaculum tali.

Although there is still controversy regarding the presence of internal tibial torsion associated with clubfoot, it is more likely that this represents a clinical appearance resulting from malposition of the talus within the ankle mortise[3,17,18].

Herzenberg et al.[19], using three-dimensional reconstructions of fetal specimens, demonstrated marked talar dysmorphism, including increased internal rotation of both the talus and the calcaneus relative to the ankle mortise. Although the talar body may appear externally rotated, the overall alignment is one of internal rotation, driven by deformity of the talar neck and medial displacement of the articular surface.

Diagnosis

Currently, prenatal diagnosis can be made using ultrasonography. The advantages of prenatal diagnosis include the possibility of determining whether the deformity is isolated or associated with other abnormalities, as well as allowing counseling about the condition and explaining the treatment to the parents. The disadvantages include the stress that may affect the family, as well as a relatively high rate of false-positive diagnoses, meaning that after birth, the diagnosis is not confirmed. Even when prenatal ultrasonography is combined with genetic testing in the evaluation of clubfoot, distinguishing isolated cases from those associated with additional structural or genetic anomalies remains challenging (Figure 2)[20-22].

However, it is at birth that the diagnosis can effectively be made through physical examination. In evaluating a newborn with clubfoot, the initial priority is to rule out associated anomalies. The deformity is quite characteristic and involves both the leg, which shows calf atrophy, and the foot, which presents the equinocavovarus deformity. A complete physical examination is important for ruling out other abnormalities and classifying the type of clubfoot. It is important to differentiate idiopathic clubfoot from cases associated with neuromuscular or syndromic disorders. Among the aspects evaluated, we must always consider the deformity's rigidity and the presence of medial and posterior skin creases.

While in true clubfoot the deformity is obvious, sometimes normal feet may present a positional equinovarus posture, which can confuse the neonatologist or pediatrician. However, orthopedic examination easily demonstrates that these postural deformities are not a cause for concern and should not be labeled as congenital clubfoot.

Radiography or any other complementary examination is not necessary because a large part of the bones of the newborn are still cartilaginous, not visible on radiographs.

The association between clubfoot and developmental dysplasia of the hip (DDH) remains controversial, as does the need for routine ultrasound screening[23,24].

Despite the lack of consensus, it is prudent to screen for DDH, as evidence suggests that physical examination alone may fail to detect even severe cases[25]. When DDH is associated with clubfoot, management of the hip should take priority. It is well established that clubfoot does not require urgent treatment. In fact, it may even be preferable to begin casting at around one month of age (Figure 3)[26,27].

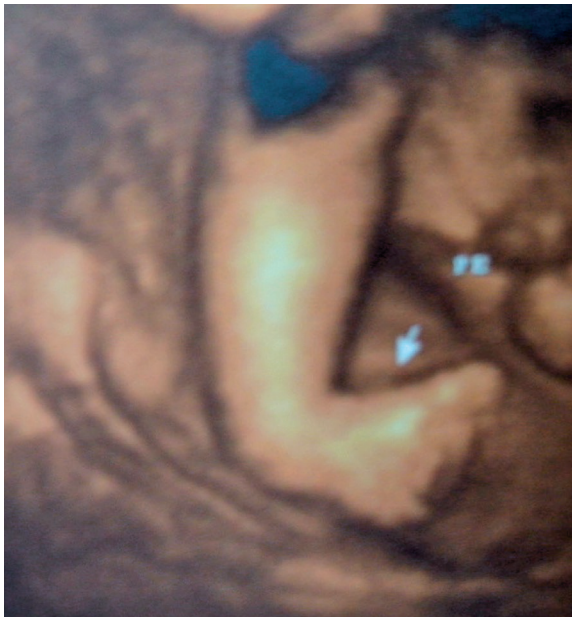

Figure 2. Clubfoot may be detected on a prenatal ultrasound.

Classification

Clubfoot may be an isolated deformity (idiopathic) or may be associated with other conditions. Thus, the following classification can be considered[6]:

Idiopathic

Neuromuscular

- Arthrogryposis
- Myelomeningocele

Syndromic

- Larsen syndrome
- Moebius syndrome
- Freeman-Sheldon syndrome
- Streeter syndrome

Postural

- Not a true clubfoot; usually does not require treatment and should not be referred to as congenital clubfoot.

In addition to the general classification described above, there have long been attempts to classify clubfoot according to the severity of the deformity, and numerous classifications exist in the literature.

Among the several classifications described, the most commonly used today are those proposed by DiMeglio and Pirani. Both authors suggest that their classifications can be used not only for the initial assessment but also for treatment follow-up[28,29].

DiMeglio classification

The DiMeglio classification is based on clinical evaluation through inspection and palpation. A scoring scale is used for each item evaluated. Four main parameters are assessed, each graded from 0 to 4, and four additional parameters are scored 1 point each if present. The four main parameters evaluated and graded according to reducibility are: 1) degree of equinus; 2) hindfoot varus; 3) forefoot adduction; 4) medial rotation of the calcaneopedal block (relationship between the axis of the foot and the axis of the leg when viewed from the front).

In addition to these parameters, four additional findings are evaluated, each receiving 1 point: 1) medial crease; 2) posterior crease; 3) cavus deformity; 4) generalized hypertonia of the child[28].

The total score ranges from 0 to 20, and according to this score, the deformity is classified as:

- 0-5: benign foot
- 5-10: moderate foot
- 10-15: severe foot
- 15-20: very severe foot

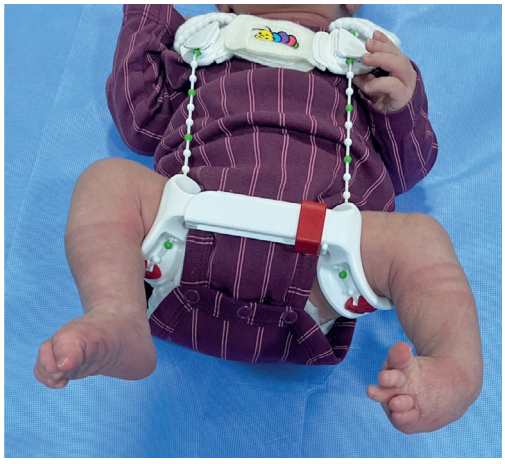

Figure 3. In case of the association of clubfoot and developmental dysplasia of the hip, treatment of the hip takes priority, and the Tubingen brace may be preferable to the Pavlik harness in this context.

Pirani classification

The Pirani classification is based on the clinical evaluation of hindfoot and midfoot deformities. For each parameter assessed, a score is assigned: 0 (normal), 0.5 (mild/moderate deformity), or 1 (severe deformity). The total score ranges from 0 (normal foot) to 6 (severe deformity). Both the hindfoot and the midfoot contribute a maximum of 3 points each[29].

This classification has been widely used by practitioners who follow the Ponseti method, and its main advantage is that it is simple to remember and appears to have limited subjective influence and low interobserver variability. Actually, both the DiMeglio and Pirani classification systems have consistently demonstrated good interobserver reliability (Figure 4)[30].

Treatment

Historically, when surgical treatment was not considered sufficiently safe, nonoperative methods predominated. With the development of new surgical techniques and the increased safety afforded by advances in anesthesia, there was a marked expansion in the surgical management of clubfoot, which remained the predominant approach from the 1960s through the late 1990s (Figure 5).

The publication of long-term follow-up studies of children treated with extensive surgical releases revealed high rates of complications, stiffness, and pain, combined with the general trend toward less invasive approaches in medicine, which led to renewed interest in conservative treatment methods[31].

In fact, for a long time, most orthopedists agreed that early conservative management should be the initial approach for congenital clubfoot. However, few considered conservative treatment to be the definitive treatment for most children with this deformity. In many cases, the casting used during conservative treatment was inadequate to achieve correction, and the child simply waited until reaching an age appropriate

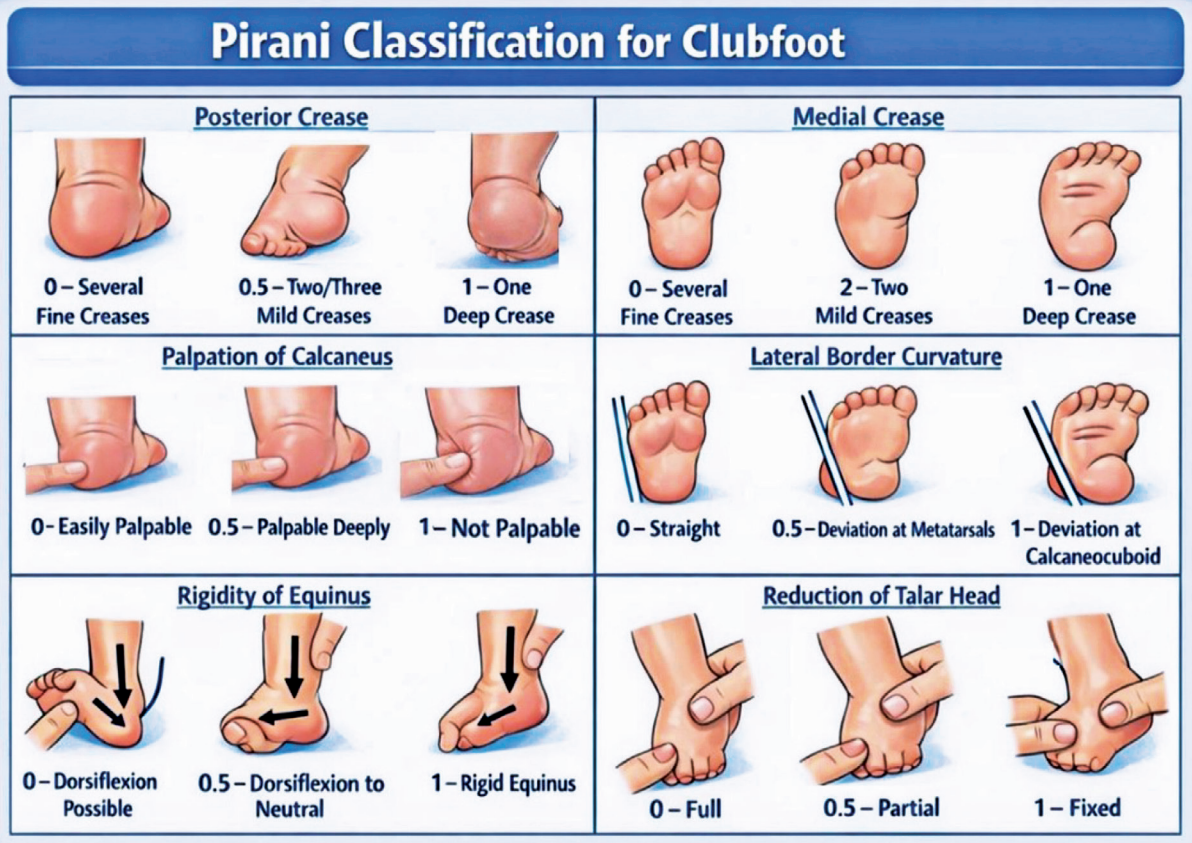

Figure 4. Pirani classification.

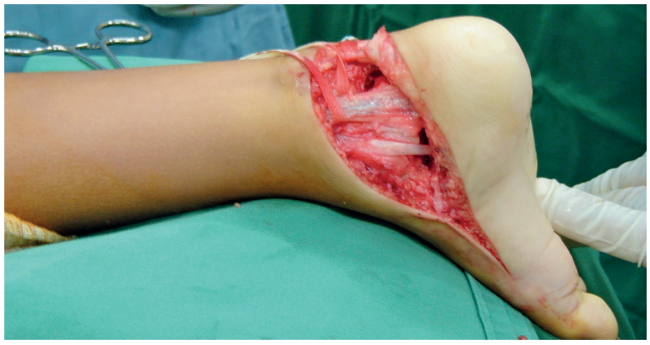

Figure 5. Extensive posteromedial and lateral releases for clubfoot is rarely indicated in current practice.

for surgery while undergoing temporary treatment with casts. This was particularly the case of the method described by Kite, who reported correction in up to 80% of cases. However, such correction rates were never consistently replicated by other authors, even though this method remained the most widely adopted conservative treatment worldwide for many years[32-34].

Interestingly, two other conservative and far more effective treatment approaches—the French functional method and the Ponseti method—were overshadowed for many years. The French functional method was adopted in very few centers outside France. Even among its original developers, the reported rate of initial correction was approximately 75%, which is lower than that achieved by most centers using the Ponseti method worldwide[35,36].

French Functional Technique

Bensahel et al. (1980) developed a functional treatment technique for clubfoot based on daily manipulations performed by physiotherapists, followed by immobilization using adhesive taping[35,36].

This method was later refined by DiMeglio et al. (1996), who introduced a passive motion machine that could be applied to the infants' feet for up to 16 hours per day. Using this treatment approach, these authors reported success in approximately 75% of patients (Figure 6)[37].

Although similar correction rates have been reported by other authors (even without using the passive motion machine), caution should be exercised when applying this

technique. The deformity in clubfoot is extremely complex, and treatment may require surgical interventions that physiotherapists are not trained to evaluate or perform.

Ponseti method

Although described many years ago, the Ponseti technique remains one of the most modern contributions to the treatment of clubfoot[3,4].

The Ponseti technique involves manipulations and serial casting with long-leg casts changed weekly. This is perhaps the only similarity between this method and the Kite technique. Herzenberg et al.[34] conducted a comparative study of the two techniques and demonstrated the superiority of the Ponseti method.

The first major difference compared with the Kite method concerns the fulcrum used during foot abduction. Ponseti recommended that the head of the talus, palpated on the lateral side of the foot, should serve as the fulcrum (Figure 7).

The talus is stabilized within the ankle joint, and the entire foot is progressively abducted. This simultaneously corrects the talonavicular dislocation, the hindfoot varus, and the forefoot adduction. This simultaneous correction is another major difference compared with the Kite method. Casting should be performed by two people: the surgeon manipulates and maintains the foot in position while the assistant applies the cast. After manipulation, the foot should be immobilized in abduction with a long-leg cast, with the knee flexed at 90°. The cast must be well molded, and the toes should remain free to allow dorsiflexion. A relevant point to remember is that cavus should be corrected in the first cast, by stretching the plantar structures while maintaining the forefoot in supination relative to the hindfoot.

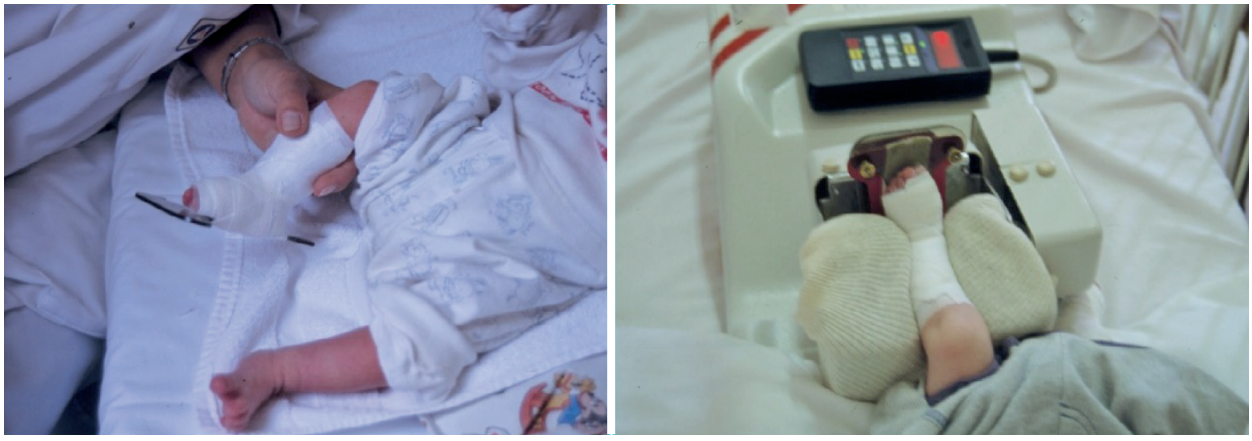

Figure 6. Continuous passive motion device used in Montpellier, France (courtesy of Alain DiMeglio).

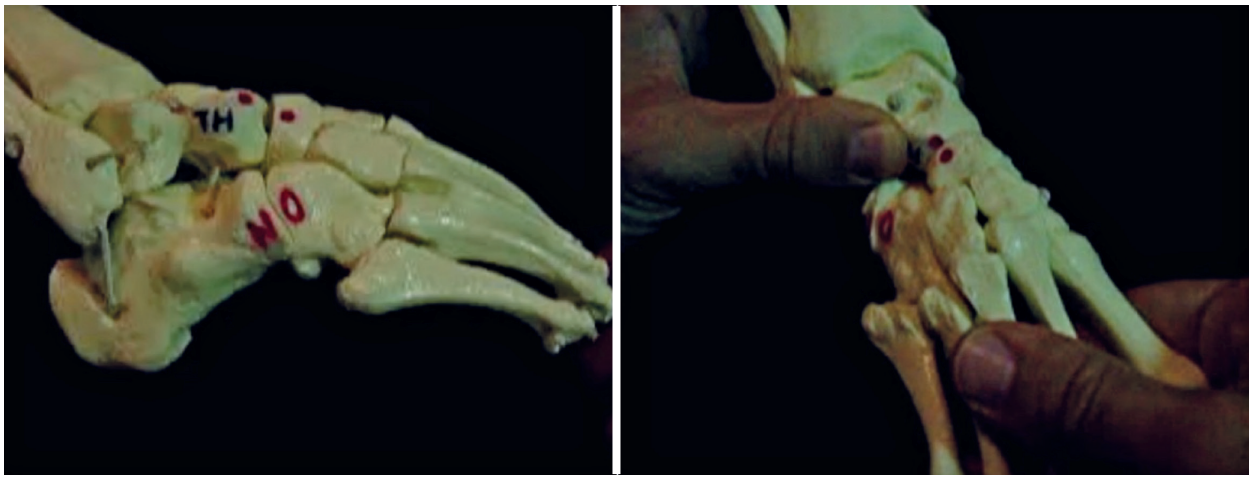

Figure 7. The correct fulcrum to correct clubfoot is the talar head, not the calcaneocuboid joint, as demonstrated in the model developed by Dr. Ponseti.

The casts should be changed weekly, and parents are usually instructed to remove the cast at home shortly before the new appointment. Some practitioners prefer removing the cast at the clinic, depending on logistical considerations. Usually, about five casts are required even for severe deformities (Figure 8).

When correction of equinus is not achieved with manipulation and serial casting, percutaneous Achilles tendon tenotomy is indicated and is required in over 90% of cases. In the original technique, tenotomy is performed under local anesthesia, but many surgeons have preferred to perform it under general anesthesia. The advantage of the original technique is that it enables treatment in settings with limited hospital access. However, under general anesthesia, palpation of the tendon and casting are easier[38].

Another point of discussion concerns the type of blade used to cut the Achilles tendon, which can be performed using the tip of a No. 11 blade, an ophthalmic blade, or even a needle (Figure 9)[39,40].

After tenotomy, the foot is immobilized with a long-leg cast, with the ankle in 20° dorsiflexion and approximately 70° abduction, for three weeks.

Important points for successful treatment include:

- The child should remain calm; breastfeeding during casting is ideal but not always feasible
- Casting should be performed by two practitioners
- The foot should never be pronated
- The calcaneus should never be forced, only properly molded
- With progressive abduction, the initial supination gradually returns to neutral
- Achilles tenotomy should only be performed when equinus is the only remaining deformity.

After removal of the final cast, maintenance of correction is achieved using an abduction brace (often called the Denis-Browne brace).

The brace is applied with:

- 70° external rotation of the affected foot
- 10° dorsiflexion

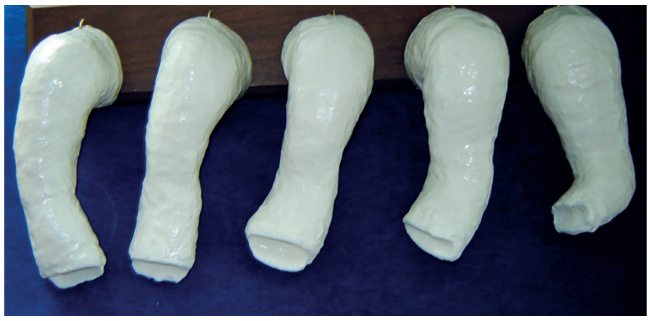

Figure 8. Most feet require approximately five serial cast changes to achieve correction before tenotomy.

It is worn full-time for three months, followed by nighttime use for three to four years.

In unilateral cases, the normal foot should be positioned in approximately 40° of external rotation.

The main difficulty with the method is brace compliance. Failure to use the brace is one of the most common causes of recurrence. Parents must therefore be strongly advised of the importance of proper brace use (Figure 10)[41].

Today, the Ponseti method is considered the standard treatment worldwide. However, even Ponseti acknowledged that about 5% of feet have very rigid ligaments that do not respond to manipulation and therefore require more extensive surgical correction[4].

Accelerated Ponseti

In the classic Ponseti method, the cast changes are done at weekly intervals, but to reduce the treatment time and less burden for families traveling long distances, some centers started using an accelerated protocol in which the manipulation and casting are done twice per week (every 3-4 days) with similar results[42,43].

Complex clubfoot

Complex (or atypical) clubfoot, as described by Ponseti et al.[44], represents a distinct subset of deformities, often observed in patients who have undergone prior manipulation or casting. It is characterized by severe equinus, a short

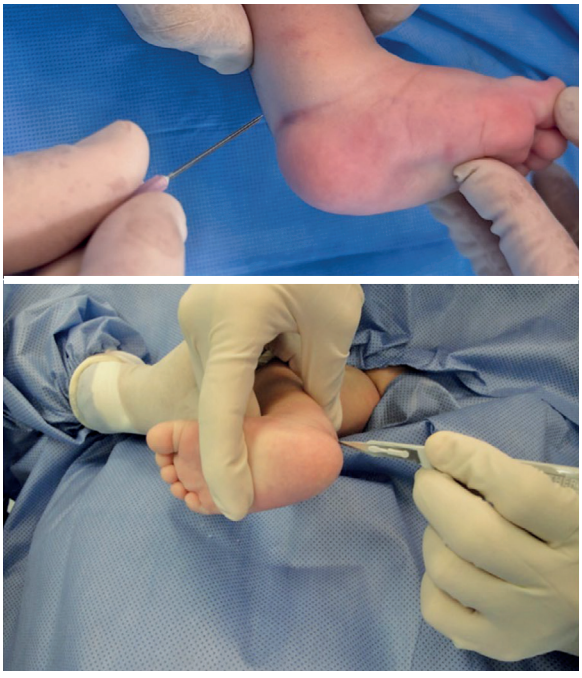

Figure 9. A needle or a scalpel may be used to perform the tenotomy.

and stubby forefoot with a shortened first metatarsal, hyperextension of the great toe, marked plantarflexion of all metatarsals, and deep transverse plantar and posterior ankle creases.

Although many cases appear idiopathic, this pattern is frequently associated with improper casting technique, particularly slippage of the foot within the cast. Inadequate molding of the long-leg cast may allow slippage, resulting in abnormal forces that displace the calcaneus proximally, promoting further contracture and ultimately exacerbating the equinus deformity (Figure 11).

Early recognition of this condition is critical, as successful correction requires modification of the standard Ponseti method, typically employing the four-finger technique with careful attention to forefoot supination and controlled abduction. The knee should be maintained in 100° to 110° of flexion within the cast. Most cases can be corrected with a series of cast changes followed by Achilles tenotomy; however, the recurrence rate is higher in this type of clubfoot[45,46]. Even after correction, a complex clubfoot may present a lateral "break" at midfoot, and the abduction brace should initially be set at approximately 40° of abduction and gradually increased over subsequent weeks to 60°–70º (Figure 12).

Recurrence

Ponseti emphasized that clubfoot deformity tends to recur until approximately seven years of age. With careful supervision and adequate parental adherence, recurrences can be prevented in nearly 50% of patients. In the remaining cases, recurrence may occur between 10 months and 7 years of age.

When recurrence occurs, treatment should follow the same principles as the initial correction, with serial casting reintroduced. However, casts are typically changed at longer intervals (every 10-14 days) compared with the standard weekly protocol. Repeat Achilles tenotomy is frequently required, and in many cases, transfer of the tibialis anterior tendon to the lateral cuneiform is performed to address dynamic supination and reduce the risk of further recurrence. Therefore, when Ponseti reported correction rates approaching 90%, it is important to consider that these outcomes included management of recurrences, which often required additional procedures (Figure 13)[3,4].

Noncompliance with the use of the abduction brace is often cited as the primary cause of recurrence; however, this is not invariably the case, as highlighted by Mahan et al.[47]. In their study, recurrences in children younger than two years were strongly associated with inadequate brace use, whereas in children older than two years, recurrences occurred even in the presence of appropriate compliance, indicating that additional factors contribute to recur in this age group.

Figure 11. Poor casting technique is a major cause of complex clubfoot. A cast applied with less than 90° of knee flexion and inadequate molding can slip easily, exerting abnormal pressure on the calcaneus.

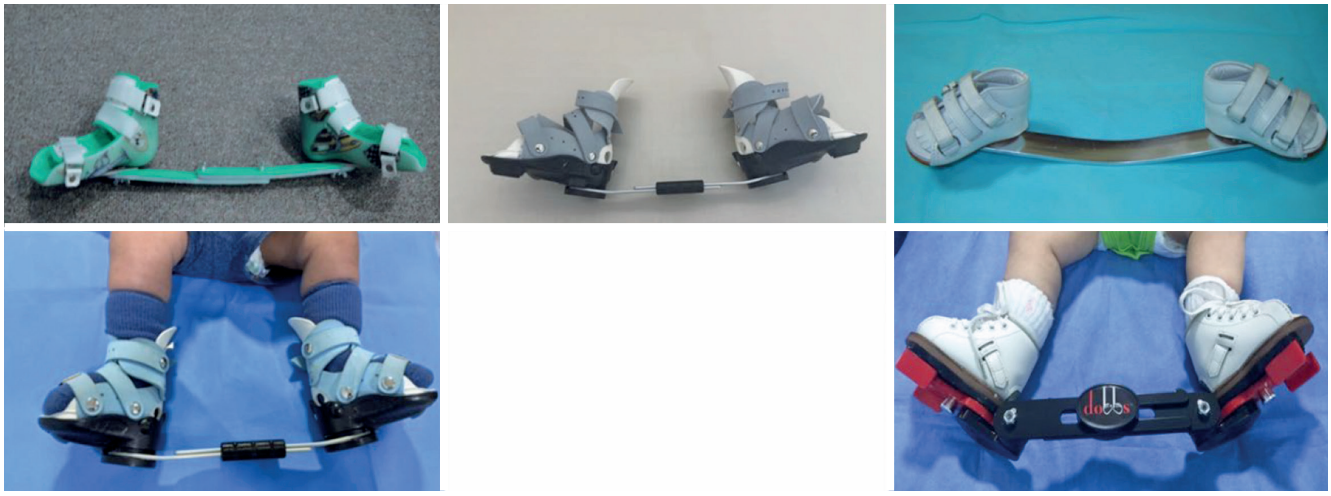

Figure 10. Brace compliance is a major challenge in the Ponseti method, regardless of the type used.

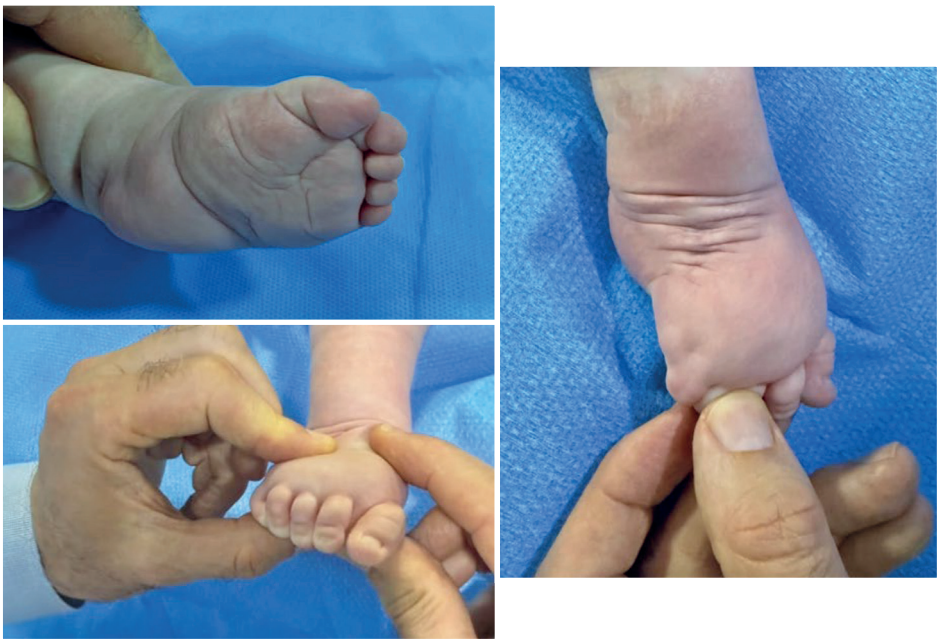

Figure 12. Early recognition of complex clubfoot is essential, and modification of the Ponseti manipulation protocol is required, including the use of the four-fingers technique. The abduction brace should be set at a lower degree of abduction to prevent the increase of the lateral midfoot "break".

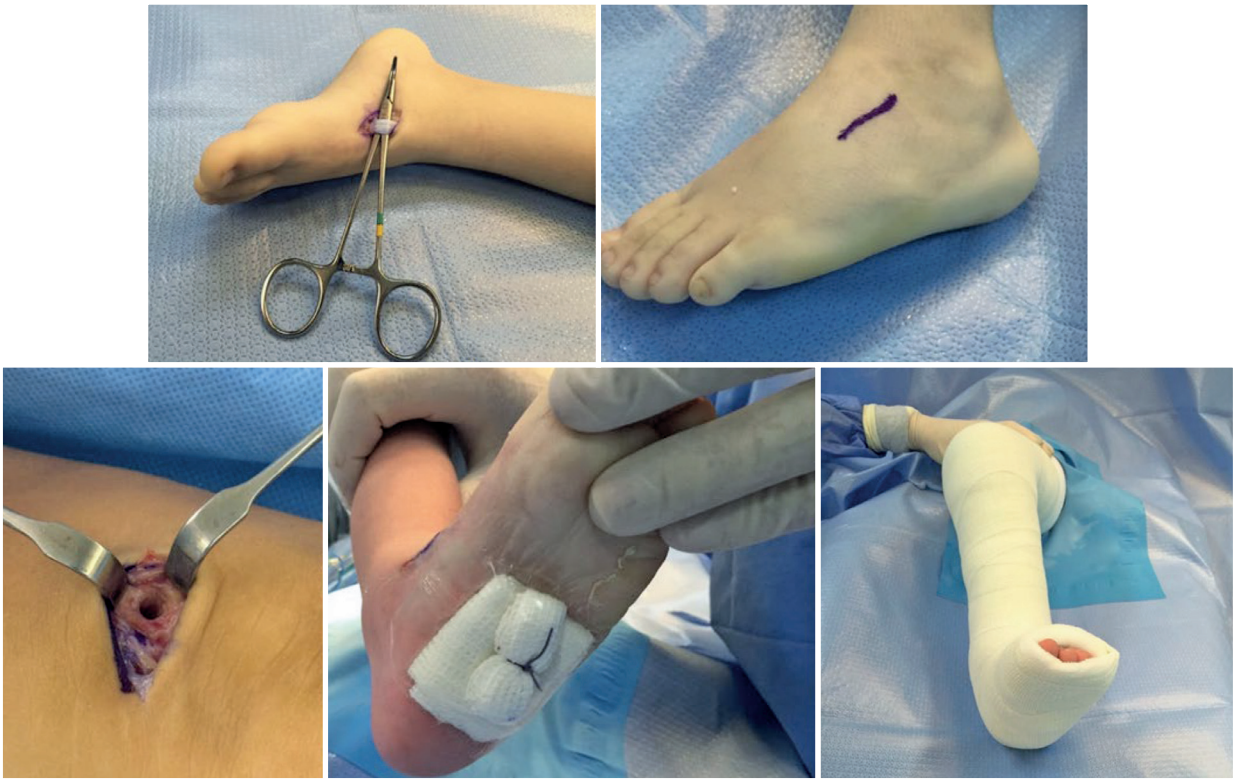

Figure 13. Anterior tibialis anterior transfer to the lateral cuneiform is often performed in cases of recurrence, preferably using the pull-out technique, followed by six weeks of long-leg casting.

One potential contributing factor to recurrence is insufficient activity of the evertor muscles, as suggested by Little et al.[48]. Notably, most components of the Ponseti method rely on passive interventions, including manipulation, casting, and bracing. In this context, strategies to stimulate muscle function may play an important role. In infants, this may be achieved through reflex-based stimulation, whereas in older children, active exercises can be introduced to promote foot eversion. Such approaches may be more effective than relying solely on prolonged brace use. Although this concept requires further investigation, it aligns with the hypothesis proposed by several authors and reflects my clinical experience, suggesting that targeted muscle activation may help reduce recurrence rates (Figure 14)[49,50].

The role of surgery in contemporary clubfoot management

Although the Ponseti method is widely described as a conservative treatment for clubfoot, it may be more accurately characterized as a minimally invasive surgical approach. While the initial correction relies heavily on serial manipulation and casting, approximately 90% of patients require a percutaneous Achilles (calcaneal) tenotomy to achieve adequate correction of equinus. This procedure, although minor, is undeniably surgical.

Furthermore, a subset of patients will subsequently require additional surgical intervention, most commonly a tibialis anterior tendon transfer, to address dynamic supination and reduce the risk of recurrence.

Therefore, rather than being purely conservative, the Ponseti method should be understood as a comprehensive treatment strategy that combines non-operative techniques with essential minimally invasive surgical procedures to optimize outcomes.

With the proper application of the Ponseti technique, the need for extensive surgical procedures has markedly decreased. The method has also been increasingly used for non-idiopathic and neglected clubfoot, with many patients benefiting from it. However, maintenance of correction in these groups is less predictable, and the need for additional surgical interventions remains higher[51-53].

Figure 14. Exercises may help maintain correction, initially with passive motion and stimulation of reflex evertor activity and later active motion and strengthening.

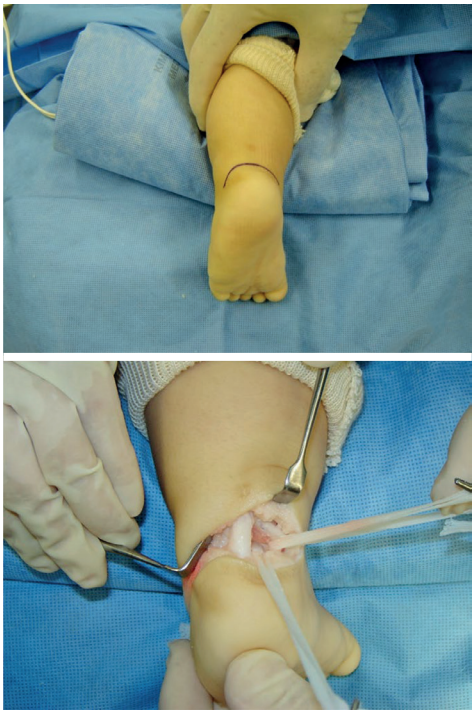

Figure 15. Surgery is limited to less extensive, targeted procedures, in accordance with the principle of "à la carte" release.

Therefore, despite these advances, surgery remains an important adjunct in selected cases. Indications for operative intervention include resistant deformities that fail to respond to appropriate Ponseti casting, recurrent clubfoot—often associated with poor brace compliance or persistent muscle imbalance—and residual deformities such as dynamic supination or persistent equinus. In these situations, modern surgical strategies favor limited, targeted procedures rather than extensive releases.

In addition, surgery may be required more frequently in non-idiopathic clubfoot, including neuromuscular and syndromic cases, which are characterized by greater rigidity and higher recurrence rates despite initial correction with the Ponseti method. Similarly, neglected or late-presenting cases often necessitate a combination of casting and surgical intervention, including osteotomies or gradual correction techniques.

Nevertheless, before considering surgical intervention, it is advisable to perform a period of manipulation and casting as a preoperative measure to reduce the extent of the procedure.

An individualized approach, as proposed by Bensahel, may represent the optimal strategy. In this concept of "à la carte" surgery, operative treatment is directed only at residual deformities that persist after conservative management, thereby avoiding extensive surgical exposure to preserve joint mobility and minimize long-term morbidity (Figure 15)[54].

Author contributions: The author contributed individually and significantly to the development of this article: AFL *(https://orcid.org/0000-0002-0557-9176) conceived and planned the activities that led to the study, data collection, interpreted the results of the study, wrote the article and participated in the review process. The author read and approved the final manuscript. *ORCID (Open Researcher and Contributor ID).

References

1. Ippolito E, De Maio F, Mancini F, Bellini D, Orefice A. Leg muscle atrophy in idiopathic congenital clubfoot: is it primitive or acquired? J Child Orthop. 2009;3(3):171-8.
2. Andjelkov K, Llull R, Colic M, Atanasijevic TC, Popovic VM, Colic M. Aesthetic Improvement of Undeveloped Calves After Treatment of Congenital Clubfoot Deformity. Aesthet Surg J. 2018;38(11): 12009.
3. Ponseti IV, Smoley EN. The classic: congenital club foot: the results of treatment. 1963. Clin Orthop Relat Res. 2009;467(5):1133-45.
4. Ponseti IV. Treatment of congenital club foot. J Bone Joint Surg Am. 1992;74(3):448-54.
5. O'Shea RM, Sabatini CS. What is new in idiopathic clubfoot? Curr Rev Musculoskelet Med. 2016;9(4):470-7.
6. Mange TR, Bram JT, Scher DM, Doyle SM. Idiopathic Talipes Equinovarus: Current Concepts. J Am Acad Orthop Surg. 2025; 33(24):1372-81.
7. Riccio AI. Disorders of the foot. In: Herring JA, editor. Tachdjian's Pediatric Orthopaedics. 6th ed. Philadelphia: Elsevier; 2022. p. 706-41.
8. Laredo Filho J. Contribuição ao estudo clínico-estatístico e genealógico- estatístico do pé torto congênito equinovaro [thesis]. São Paulo: Escola Paulista de Medicina, Universidade Federal de São Paulo; 1968. 104 p.
9. Smythe T, Rotenberg S, Lavy C. The global birth prevalence of clubfoot: a systematic review and meta-analysis. EClinicalMedicine, 2023; 63:102178.
10. Bohm M. The embryologic origins of clubfoot. J Bone Joint Surg. 1929;11:229-59.
11. Irani RN, Sherman MS. The pathological anatomy of idiopathic clubfoot. Clin Orthop Relat Res. 1972;84:14-20.
12. Macnicol MF, Nadeem RD. Evaluation of the deformity in club foot by somatosensory evoked potentials. J Bone Joint Surg Br. 2000;82(5):731-5.
13. Ippolito E, Ponseti IV. Congenital clubfoot in the human fetus: a histological study. Journal of Bone and Joint Surgery. 1980;62:8-22.
14. Pavone V, Chisari E, Vescio A, Lucenti L, Sessa G, Testa G. The etiology of idiopathic congenital talipes equinovarus: a systematic review. J Orthop Surg Res. 2018;13(1):206.
15. Werler MM, Yazdy MM, Kasser JR, Mahan ST, Meyer RE, Anderka M, et al. Maternal cigarette, alcohol, and coffee consumption in relation to risk of clubfoot. Paediatr Perinat Epidemiol. 2015;29(1):3-10.

16. Sodre H, Bruschini S, Mestriner LA, Miranda F Jr, Levinsohn EM, Packard DS Jr, et al. Arterial abnormalities in talipes equinovarus as assessed by angiography and the Doppler technique. J Pediatr Orthop. 1990;10(1):101-4.
17. Westberry DE, Davis RB, Binkley-Vance R, Westberry A, Westberry A, Wack LI. In-toeing gait in children with clubfoot and the effect of tibial rotation osteotomy. J Pediatr Orthop B. 2020;29(4):348-54.
18. Lichtblau S. External rotation tibial osteotomy in clubfoot: adverse late effects. Clin Orthop Relat Res. 1978;(136):225-9.
19. Herzenberg JE, Carroll NC, Christofersen MR, Lee EH, White S, Munroe R. Clubfoot analysis with three-dimensional computer modeling. J Pediatr Orthop. 1988;8(3):257-62.
20. Brasseur-Daudruy M, Abu Amara S, Ickowicz-Onnient V, Touleimat S, Verspyck E. Clubfoot Versus Positional Foot Deformities on Prenatal Ultrasound Imaging. J Ultrasound Med. 2020;39(3):615-23.
21. Radler C, Myers AK, Burghardt RD, Arrabal PP, Herzenberg JE, Grill F. Maternal attitudes towards prenatal diagnosis of idiopathic clubfoot. Ultrasound Obstet Gynecol. 2011;37(6):658-62.
22. de Vries JM, Arduc A, van Leeuwen E, Tan-Sindhunata MB, Struijs P, Witbreuk M, et al. Can Prenatal Ultrasound and Genetic Testing Reliably Exclude Non-Isolated Clubfoot? Prenat Diagn. 2026;46(1):30-8.
23. Ibrahim T, Riaz M, Hegazy A. The prevalence of developmental dysplasia of the hip in idiopathic clubfoot: a systematic review and meta-analysis. Int Orthop. 2015;39(7):1371-8.
24. Håberg Ø, Foss OA, Lian ØB, Holen KJ. Is foot deformity associated with developmental dysplasia of the hip? Bone Joint J. 2020;102-B(11):1582-6.
25. Harper P, Joseph BM, Clarke NMP, Herrera-Soto J, Sankar WN, Schaeffer EK, et al. Even Experts Can Be Fooled: Reliability of Clinical Examination for Diagnosing Hip Dislocations in Newborns. J Pediatr Orthop. 2020;40(8):408-12.
26. Alves C, Escalda C, Fernandes P, Tavares D, Neves MC. Ponseti method: does age at the beginning of treatment make a difference? Clin Orthop Relat Res. 2009;467(5):1271-7.
27. Liu YB, Li SJ, Zhao L, Yu B, Zhao DH. Timing for Ponseti clubfoot management: does the age matter? 90 children (131 feet) with a mean follow-up of 5 years. Acta Orthop. 2018;89(6):662-7.
28. Diméglio A, Bensahel H, Souchet P, Mazeau P, Bonnet F. Classification of clubfoot. J Pediatr Orthop B. 1995;4(2):129-36.
29. Pirani S, Hodges D, Sekeramayi F. A reliable & valid method of assessing the amount of deformity in the congenital clubfoot deformity. Orthop Procs. 2008;90-B(Supp_I):53-53.
30. Flynn JM, Donohoe M, Mackenzie WG. An independent assessment of two clubfoot-classification systems. J Pediatr Orthop. 1998;18(3):323-7.
31. Smith PA, Kuo KN, Graf AN, Krzak J, Flanagan A, Hassani S, et al. Long-term results of comprehensive clubfoot release versus the Ponseti method: which is better? Clin Orthop Relat Res. 2014;472(4):1281-90.
32. Kite JH. Principles involved in the treatment of congenital club-foot. 1939. J Bone Joint Surg Am. 2003;85(9):1847.
33. Kite JH. The treatment of congenital clubfeet. JAMA. 1932: 99(14:)1156-62.
34. Herzenberg JE, Radler C, Bor N. Ponseti versus traditional methods of casting for idiopathic clubfoot. J Pediatr Orthop. 2002;22(4):517-21.
35. Bensahel H, DeGrippes Y, Billot C. À propos de six cents pieds bots. Chirurgie Pédiatrique. 1980;21()5:335-42.
36. Bensahel H, Guillaume A, Czukonyi Z, Desgrippes Y. Results of physical therapy for idiopathic clubfoot: a long-term follow-up study. J Pediatr Orthop. 1990;10(2):189-92.
37. DiMeglio A, Bonnet F, Mazeau P, De Rosa V. Orthopaedic treatment and passive motion machine in clubfoot. J Pediatr Orthop B. 1996;5()3:173-80.
38. Rangasamy K, Baburaj V, Gopinathan NR, Sudesh P. Techniques, anaesthesia preferences, and outcomes of Achilles tenotomy during Ponseti method of idiopathic clubfoot correction: A systematic review. Foot (Edinb). 2022;52:101922.
39. Dhingra M, Cazorla Bak Y, Edokpayi F, Chong HH, Shyamsundar S. A systematic review and single center experience with percutaneous needle tenotomy in tongenital talipes quinovarus (CTEV). Cureus. 2022;14(12):e32812.
40. Alkan H, Tuncel CY, Üner Ç, Koçyiğit İA, Erdoğan Y, Yaşar NE, et al. Ultrasonographic and elastographic comparison of needle versus scalpel Achilles tenotomy in clubfoot: A clinical and morphological study. Foot Ankle Surg. 2025 S1268-7731(25)00239-5.
41. Ramírez N, Flynn JM, Fernández S, Seda W, Macchiavelli RE. Orthosis noncompliance after the Ponseti method for the treatment of idiopathic clubfeet: a relevant problem that needs reevaluation. J Pediatr Orthop. 2011;31(6):710-5.
42. Morcuende JA, Abbasi D, Dolan LA, Ponseti IV. Results of an accelerated Ponseti protocol for clubfoot. J Pediatr Orthop. 2005;25(5):623-6.
43. Llombart-Blanco R, Mariscal G, Altabbaa H, Polevoi E, Barrios C, Llombart-Ais R. Accelerated versus standard Ponseti method for idiopathic clubfoot: A systematic review and meta-analysis of efficacy and safety. Foot Ankle Surg. 2026:S1268-7731(26)00001-9.
44. Ponseti IV, Zhivkov M, Davis N, Sinclair M, Dobbs MB, Morcuende JA. Treatment of the complex idiopathic clubfoot. Clin Orthop Relat Res. 2006;451:171-6.
45. Loose O, Fernandez Fernandez F, Langendoerfer M, Wirth T, Eberhardt O. Complex, atypical clubfoot: follow-up after up to 16 years reveals a high risk of relapse but good functional and radiological outcomes. Arch Orthop Trauma Surg. 2023; 143(10):6097-104.
46. Masquijo J, Arana E. Complex clubfoot: my 5 tips for appropriate evaluation and treatment with the Ponseti method. Acta Ortop Mex. 2023;37(4):233-6.
47. Mahan ST, Spencer SA, May CJ, Prete VI, Kasser JR. Clubfoot relapse: does presentation differ based on age at initial relapse? J Child Orthop. 2017;11(5):367-72.
48. Little Z, Yeo A, Gelfer Y. Poor Evertor Muscle Activity Is a Predictor of Recurrence in Idiopathic Clubfoot Treated by the Ponseti Method: A Prospective Longitudinal Study With a 5-Year Follow-up. J Pediatr Orthop. 2019;39(6):e467-71.
49. Greve C, Killen BA, Bos GJFJ, Houdijk H, Moerman S, Murgia A. Muscle force imbalances predict clubfoot recurrence risk: A musculoskeletal modelling approach. Comput Methods Programs Biomed. 2025;270:108972.
50. El-Banna G, Baskar D, Segovia N, Frick S. Clubfoot Activity and Recurrence Exercise Study (CARES). J Pediatr Orthop. 2022; 42(1):e916.
51. Ishizuka T, Hung YY, Weintraub MR, Kaiser SP, Williams ML. Ponseti Idiopathic and Nonidiopathic Clubfoot Correction With Secondary Surgeries. J Foot Ankle Surg. 2021;60(4):742-6.
52. Lourenço AF, Morcuende JA. Correction of neglected idiopathic club foot by the Ponseti method. J Bone Joint Surg Br. 2007;89(3):378-81.
53. Nogueira F, Poggiali P. Review Article: Current Concepts in the Treatment of Congenital Clubfoot. Rev Bras Ortop (Sao Paulo). 2024;59(6):e821-9.
54. Bensahel H, Csukonyi Z, Desgrippes Y, Chaumien JP. Surgery in residual clubfoot: one-stage medioposterior release “à la carte”. J Pediatr Orthop. 1987;7(2):145-8.