Acta Ortopédica Mexicana

Volumen *Volume* **18** **Suplemento** *Supplement* **1** **Julio-Diciembre** *July-December* **2004**

Artículo:

Conservative treatment of idiopathic congenital clubfoot. Efficiency assessment

Acta Ortopédica Mexicana 2004; 18(Suppl. 1): Jul.-Dec: S63-S69

Case report

Conservative treatment of idiopathic congenital clubfoot. Efficiency assessment

Enrique Espinosa-Urrutia,* Alfredo Penagos-Paniagua**

"Victorio de la Fuente Narváez" Orthopedic Hospital, IMSS

SUMMARY. A clinical assessment is presented on the response of idiopathic congenital clubfoot, classified as rigid, to serial manipulations and immobilization in casts with the Ponseti technique. The study was conducted on 22 newborns (37 feet). Variables in observation were four factors making the deformity (adduction, supine, equinus, and varus). The response is charted against time through statistical testing including the Kaplan-Meier efficiency test and Survival Function. Adducted and supine clubfeet were determined and corrected with an average of 6.5 manipulations having an 89.2% success rate while varus and equinus required an average of 12 manipulations with a 35% to 43% possibility of success. In some cases it became necessary to perform subcutaneous elongation of the Achilles tendon with good response in only 41% of cases. This procedure, therefore, is not recommended. Although conservative treatment does have a possible success rate of over < 50%, it still is the treatment of choice to correct this problem in newborns or as preparation for surgery.

Key words: clubfoot, treatment, assessment.

RESUMEN. Se presenta una evaluación clínica de la respuesta del pie equino varo congénito idiopático, clasificado como rígido a las manipulaciones seriadas e inmovilizaciones en moldes de yeso con la técnica propuesta por Ponseti. El estudio se realizó en 22 recién nacidos (37 pies). Se tomaron como variables en observación los cuatro factores que integran la deformidad (aducto, supino, equino y varo). La respuesta se graficó contra tiempo a través de las pruebas estadísticas: de eficiencia de Kaplan-Meier y Survival function. Se determinó que el aducto y el supino se corrigen con un promedio de 6.5 manipulaciones con un porcentaje de éxito de 89.2%, mientras que el varo y el equino requieren de un promedio de 12 manipulaciones con una posibilidad de éxito de 35 y 43% respectivamente. En algunos casos fue necesario realizar alargamiento subcutáneo del tendón de Aquiles con buena respuesta en sólo 41% de los casos, por lo que no resulta un procedimiento recomendable. No obstante que el tratamiento conservador tiene una posibilidad de éxito < 50%, sigue siendo el tratamiento de elección para corregir este problema en el recién nacido o bien como preparatorio a la cirugía.

Palabras clave: pie, equino varo, tratamiento, evaluación.

* Head of the Medical Education and Research Division of the "Victorio de la Fuente Narváez" Orthopedic Hospital, IMSS (Spanish acronym for Mexican Social Security Institute) and Consulting Physician of the Rio de la Loza Gynecology-Obstetrics and Perinatology Institution.

** Orthopedics, with a Master of Science degree in Medical Sciences assigned to the "Victorio de la Fuente Narváez" Orthopedic Hospital, IMSS.

Mailing address:
Dr. Enrique Espinosa-Urrutia. Cto. Misioneros No. 4 Cd. Satélite Naucalpan Edo. Mex. CP 53100. Telephone Numbers: 57 47 35 00 Ext. 1420-1422. E-mail: espinosa@doctor.com

Most authors agree that treating idiopathic congenital clubfoot should start at the time of birth with serial manipulations and casts.[3,8,13,15,17] Other authors such as Dimeglio, Bensahel and Yamamoto[1,5,21] only agree on the need to manipulate the foot serially and posteriorly instead of putting it in a cast. They have performed several physiatric measures until the deformity is corrected. However, whatever the procedure selected success rates with conservative treatment only are reported within a very broad range: Kite, 90%; Dimeglio, 68%; Ponseti, 56%; Bensahel, 48%; Dangelmager, 40%, Fripp and Sahw, 19%.[2,13,15,18]

Figure 1. Adducted, flexible clubfoot. Slightly marked deformities. See the prominent peroneal malleolus separated from the calcaneum.

Figure 2. *Rigid* clubfoot. Severe deformity, navicular bone stuck to the medial malleolus. No difference is seen between the peroneal and the calcaneal malleolus.

Figure 3. Rigid clubfoot associated to constricting bands.

Figure 4 Dorsoplantar X-ray of a newborn patient with adducted clubfoot. See the difficulty in identifying ossification nuclei and their orientation.

This may be probably so because of a great diversity of environmental, structural, and tissue type factors that may come together in determining this foot deformity. They are: genetic proneness (4:1 people with clubfoot have a family history of the deformity);[16,19,20] changes in germinal plasma of their bone structures,[10,11] type I fiber prevalence over Type IIB in the muscles and retractile disorders in their soft tissues conditioned by an increased production of collagen, contractile proteins and degradation of fibroblasts and myofibroblasts.[6,7,23] Handelsman and Glasser attribute this disproportionate group of muscle fibers to a possible denervation[6] albeit studies reported on electromyography conducted on patients with clubfoot detect no such changes (Bill PL).

The possibility for some of these factors, all of them or a few others, such as vascular disorders as proposed by Hootnick,[3] to determine the presence of a clubfoot lead to infer that there may be a great variety of types and many possibilities of response in a single nosologic entity with an apparently equal clinical manifestation. Thus, when starting conventional treatment, even on a newborn, with serial

Figure 5. Reduction maneuver of the navicular bone as proposed by Ponseti, maintaining the forefoot in traction and supine, aligned with the varus of the hindfoot to prevent the deformity from clawing.

Figure 6. Rocking chair arch foot caused by an attempt to correct the clubfoot before correcting the adduction and varus.

manipulations every eight days and immobilization of the correction achieved with circular Paris casts, making a prognosis on the amount of casts necessary to achieve a correction or to warn about the imminent need for surgical treatment becomes very difficult.

In an attempt to provide guidance about this, Harrold[8] proposed in 1983 a classification identifying three types of clubfoot according to the degree of deformity and correction possibilities. Based on these same parameters, Dimeglio[4] proposed another 20 point classification in 1985 allowing for the pinpointing of four kinds of foot: from grade I or postural, to grade IV or teratologic, varieties over which a correction and treatment alternative prognosis is established. However, for the authors of this paper, these varieties determine, only in a somewhat subjective manner, the severity of the problem but hardly allow us to establish a defined treatment plan or a correction prognosis against time. We, therefore, consider only two types that may be clinically differentiated with greater accuracy: the *postural* and *rigid* types.

In postural clubfoot *(Figure 1)*, factors may be reduced passively from the beginning to a neutral or further position. A space between the medial malleolus and navicular bones can be seen. The peroneal malleolus is not stuck against the calcaneum, No muscular atrophy of the calf is seen and will be corrected with four to six manipulations with casts changed every eight days so that within a month to a month and a half, the problem is fully resolved. On the other hand, the factors in rigid feet *(Figure 2)* are very difficult to change. The bone, joint and muscle structures show a completely different layout from those described above. Thus it is very difficult to estimate how long, what type of treatment and to what extent, will be needed to achieve correction of these factors.[3]

Of all the means of conservative treatment published, those seeming to have greater acceptance are the one proposed by Kite and Lovell and the one proposed by Ponseti.[13,17] The former begins treatment by stretching the foot by longitudinal traction, not described by Ponseti. Both, however, suggest reducing first the talonavicular dislocation. Kite does this by a lateralization maneuver of the forefoot, started by putting pressure with his thumb outside, on the tarsal sinus and, at the same time, starting lateral translation of the navicular bone over the head of the talus, with

Figure 7. Schematic showing the technique to elongate the Achilles tendon by sliding it, subcutaneously.

the index finger of the same hand. Ponseti, on the other hand, suggests longitudinal traction of the forefoot with the other hand by putting traction on the navicular bone to translate it over the head of the talus, trying always to maintain the anterior portion of the foot in supine position during this maneuver *(Figure 3)* and thus trying to align the forefoot to the hindfoot in varus. He feels this helicoidal movement may result in a claw foot.

With the Kite and Lovell technique, once the talonavicular dislocation is reduced, a cast is placed in the form of a slipper. Once it hardens, the entire foot with the heel is turned outwards and the foot is flattened to prevent it from clawing. While Ponseti used the maneuver described above to correct adducted clubfeet, Kite, once the slipper is dried, begins translating the forefoot outwards. This has been called by Ponseti as "Kite's error". He claims that any force applied on the distal portion of the calcaneum to reduce adduction of the forefoot prevents the distal end of the calcaneum from moving laterally against the talus. In both techniques, care is taken not to make an effort to correct the deformity until the hindfoot adduction and the talipes varus have been corrected. Otherwise, a "rocking chair foot" *(Figure 4)* could result. According to Ponseti, if you have corrected the forefoot and hindfoot and you are unable to correct the clubfoot, you need to perform a subcutaneous tenotomy of the Achilles tendon and continue with serial manipulations and casts. With this additional procedure, Ponseti reported having increased his success rate to 89%.

In this study, we tried to establish the behavior of each one of the four factors (adducted, supine, equinus, and varus) using a statistical evaluation, by classifying the clubfoot as rigid and estimating how many maneuvers make it possible to achieve correction, with Ponseti's technique as a basis.

Material and methods

In this study, we observed 2,162 newborns at the "Rio de la Loza Gynecology-Obstetrics and Perinatology Institution, January 1998 and December 1999. Among these, 36 newborns were detected with idiopathic clubfoot: 13 with a flexible clubfoot diagnosis and 23, with a rigid clubfoot diagnosis.

All 13 patients with flexible clubfoot and one with rigid clubfoot associated to constricting bands *(Figure 5)* were eliminated. The sample was, therefore, made of 22 patients with rigid clubfoot: 15 bilateral and 7 unilateral feet, for a total of 37 feet; 19 left feet and 18 right feet. These patients were included based on clinical criteria only. X-rays in newborns are not considered as assessable[3] because of the difficulty of placing a foot with severe rigidity on a chassis so visible ossification nuclei do not maintain their normal shape and in newborns only the calcaneum and talus are ossified *(Figure 6)*.

All patients in this series were treated from birth by the author using manipulations with Ponseti's technique.[17] They were immobilized with casts from thigh to foot. It was decided to put these casts over the knee in order to correct the tibial torsion, prevent the casts from spontaneously sliding outwards and mask the muscle hypotrophy of the calf. In those cases when no modification of this factor was seen even after four correction attempts by passive maneuver and casts, subcutaneous tenotomy and elongation of the Achilles tendon by sliding it *(Figure 7)* was performed.

The number of casts placed on each patient to achieve correction, was recorded. Patients requiring subcutaneous tenotomy were also recorded. Each patient was followed up for two years after achieving correction of all factors or until the first relapse.

Patients requiring open elongation of the Achilles tendon or syndesmotomy because of the lack of correction or relapse of any factor were considered as a failure. The success rate, according to the average of maneuvers detected to correct each factor was estimated with the Kaplan-Meier's efficiency statistical test and the progress of each patient was charted based on the lack of response against maneuvers made by using the *Survival Function* method.

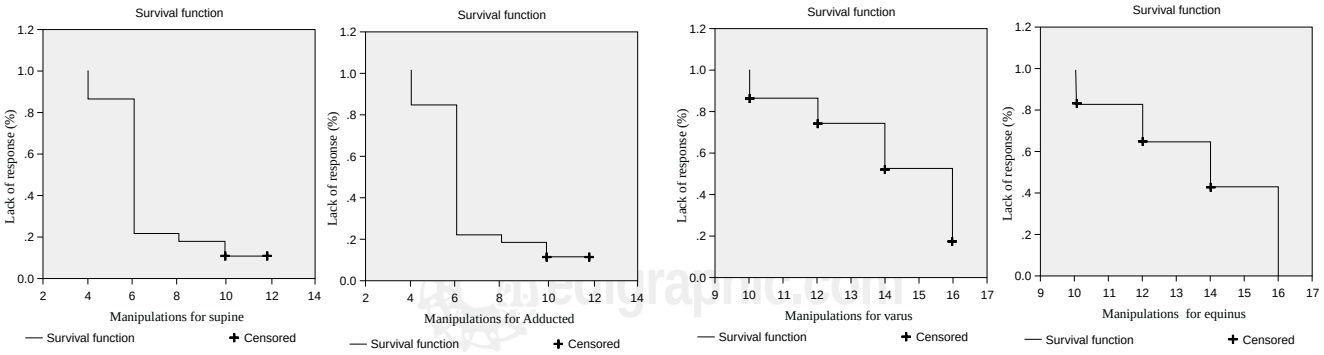

Chart 1. Chart illustrates the response to treatment of supine clubfoot, charted against weeks.
Chart 2. Chart illustrates the response to treatment of adducted clubfoot, charted against weeks.
Chart 3. Chart illustrates the response to treatment of varus clubfoot, charted against weeks.
Chart 4. Chart illustrates the response to treatment of equinus clubfoot, charted against weeks.

Figure 8. Adducted and supine feet corrected. The lateral projection shows the difficulty to correct the equinus foot although proximal ends of the calcaneum and talus tend to converge.

Of all 36 patients, 23 were males (63.8%) and 13 females (36.2%) corresponding to a 1.7:1 ratio. Of these, 21 cases were bilateral (56%) and 15 were unilateral (41%).

The clubfoot incidence in the population seen was 16.6/1000 in two years, with a prevalent male population in a 1.7:1 ratio, a bilaterality ratio of 2.1:1 and a prevalence of left feet of 1.05:1.

The summary of responses of clubfeet to the treatment chosen, determined by the Kaplan-Meier test, may be seen in *Table 1*.

Considering a study population of 37 rigid club feet (N = 37), the average weekly maneuvers necessary to correct the supine foot was 6.6 with a standard deviation of 2.1 allowing for about one month and a half to correct this problem in 33 feet (89.2%). *Chart 1* shows how the Survival Function test indicates a greater correction rate of this factor on the sixth week only.

Adducted clubfoot correction was also highly satisfactory around the 6 manipulations with an average of 6.5 weeks, a standard deviation of 2.1 and, as with the supine foot, an 89.2 efficiency rate. *Chart 2* shows a tracing very similar to the one in Chart 1 allowing us to infer that correction of these two factors is almost simultaneous and the failure rate is only 10.8%.

In 16 cases we were able to correct the varus. There were, however, 3 relapses between 12 and 18 months after their apparent correction, resulting in only 13 cases corrected of the 37 in our study.

Unlike the relative ease for correcting the forefoot within an average of six weeks, talipes varus offers great resistance and a tendency to relapsing. Final correction of this factor in this series was 13/37 (35.1%) only after 12 manipulations and casts, i.e. about three months. *Chart 3* shows a ladder shaped tracing highlighting the poor and late response to correct this problem with the technique we used.

Correcting the equinus, albeit also low, is more encouraging 16/37 (43.2%). Of these 16 feet, eleven were corrected with manipulations and cast around the eighth week with subcutaneous elongation of the Achilles tendon in 12 cases between weeks 10 and 18 because of the lack of response to manipulations *(Chart 4)*. Of these twelve patients, only five (41%) were corrected, six had to be operated again through open elongation of the Achilles tendon and posterior syndesmotomy, and one of them required left unilateral medial release at the age of eleven months.

Table 1. Correction rates and sessions necessary to achieve them, estimated by the Kaplan-Meier method.

	Results			
No. of Manipulations	Equinus	Varus	Adducted	Supine
N	37	37	37	37
Average (manipulations)	11.8	12	6.5	6.6
Standard deviation	1.5	1.6	2.1	2.1
Correction of 37	16	13	33	33
Correction %	43.2	35.1	89.2	89.2

Kaplan-Meier

Thus, a total of 16 corrected cases were considered, with an average of 11.8 manipulations necessary and a 1.5 standard deviation *(Table 1)*.

Of the 37 feet studied, eleven had a total correction of all four factors through a bloodless or non surgical procedure (29.7%), a rate increased to 43.2% by a subcutaneous elongation of the Achilles tendon.

Three more patients required posteriomedial release between the ages of 6 and 12 months, two bilateral and one left, which will also be a tributary of an anterior tibial transfer to the third wedge.

Discussion

The success rate to correct all four factors in the study on rigid clubfeet with Ponseti's technique was

very low in this hospital (43.2%) compared to the one reported by him, 89% of good to excellent results, albeit he does mention that 70% of his cases required subcutaneous elongation of the Achilles tendon. However, years later, he reported 50% of relapses requiring some additional treatment.[17]

With this technique, the rate of corrections of the adducted and supine foot is very reliable. It may achieve functional correction of the foot with an average of six manipulations, without damaging their bone structures. Ponseti's maneuver, of putting traction on the forefoot and reduce the navicular bone by maintaining the foot on a supine position to align it against the varus in the hindfoot, may be perhaps the cause for no foot having later additional bone and joint deformities *(Figure 8)*.

The difficulty found in this series to correct the varus and equinus may not be compared to others in the literature. These published evaluations only mention a global foot correction but we may infer this resistance is due to late mobilization, about six weeks after birth, of the posterior talotibial and talocalacaneal capsules and the talotibial and peroneocalcaneal ligaments, which early in treatment were always kept in a forced equinus position to prevent the transverse rupture and "rocking chair" deformity of the foot. This could also explain the little success achieved with only subcutaneous elongation of the Achilles tendon without releasing the upper joints.

In spite of the success rate being > 50% with conservative treatment in most series reported in the literature, it continues to be the first choice in treating rigid clubfoot in newborns. It is the best way, known today, to distend muscle contractures, retracted tissues, and to couple circulation to stretching suffered by the neurovascular bundle when the foot takes its normal position. The principle of this technique is to produce permanent plastic changes on shortened ligaments and tendons. The corrective principle of serial manipulations and immobilizations in casts is based on the viscoelasticity of the connective tissue to deform and adopt a final change. Very importantly, we need to consider that with a clubfoot, the supposedly hard structures (bones) are very fragile and the supposedly soft structures (ligaments and tendons) are very hard. Thus, correcting manipulations should be performed bearing in mind the anatomical structures being manipulated. Ligament and tendon stretching should be firm and sustained to get a change in orientation of the structures being manipulated. Casts should be placed by thinking of the new position of the structures and how to keep them steady.

It is important to consider that corrections are not achieved by adjusted casts pretending to correct the existing deformities since there is a risk of producing severe vascular and skin disorders.

With this study, we were able to define that the average time to reach correction of supine and adducted feet in a rigid clubfoot by serial manipulations and immobilization in a cast may be reached in about six weeks with the possibility that 10% of cases may require surgical release.

The possibility to correct varus and equinus by this means is very poor in rigid feet. It is estimated that only less than half of these cases reach full correction. Subcutaneous tenotomy does not seem to definitely solve this problem. While it may contribute to correcting the equinus it generally does not help to get the varus in remission and even when achieving it, it tends to relapse. A posterior ligament release involving the peroneocalcaneal tendon, the talotibial tendon and the talotibial portion of the deltoid ligament and the fibrosis presented by the talotibial and talocalcaneal capsule is preferred then, given the resistance of these factors, together with an elongation of the Achilles tendon by "Z-plasty".

This way, if conservative treatment is carried out efficiently, one may expect global correction of the clubfoot at about the fourth or fifth month of treatment in less than half the cases. In those patients where changes have been seen to continue, i. e. there is a response to treatment it may continue until the fifth or sixth month. Otherwise, it would be best to consider elective syndesmotomy before causing further muscular hypotrophy and deformation to the bone and joint structures.

Bibliography

1. Bensahel H, Guillaume A, Czukonyl Z, ThemarNoel C. The intimacy of club foot: The ways of functional treatment. J Pediatr Orthop B 1994; 3: 155-60.
2. Carwford AH, Gupta AK. Club foot controversies: complications and causes of failure. Instr Course Lect 1996; 45: 339-346.
3. Cummings RJ, Davidson RS, Armstrong PF, Lehman WB. Congenital club foot, instr course lect. J Bone Joint Surg 2002; 84: 290-308.
4. Dimeglio A, Bensahel H, Souchet P, Mazeau P, Bennet F. Classification of club foot. J Pediatr Orthop B 1995; 4: 129-36.
5. Dimeglio A, Bonnet F, Mazeau P, De Roza V. Orthopaedic treatment and passive motion machine: consequences of the surgical treatment of club foot. J Pediatr Orthop B 1996; 5: 173-180.
6. Handelsman JE, Badalamente MA. Neuromuscular studies in club foot. J Pediatr Orthop 1981; 1: 23-42.
7. Handelsman JE, Glasser R. Muscle pathology in club foot: The present and a view of the future. New York, NY: Springer Verlag, 1994, pp 21-31.
8. Harrold AJ, Walker CJ. Treatment and prognosis in congenital club foot. J Bone Joint Surg Br 1983; 65: 8-11.
9. Ippolito E, Ponseti IV. Congenital club foot in human fetus. A histological study. J Bone Joint Surg Am 1980; 62: 8-22.
10. Ippolito E. Update on pathologic anatomy of club foot. J Pediatr Orthop B 1995; 4: 17-24.
11. Irani RN, Sherman MS. The pathological anatomy of idiopathic club foot. J Bone Joint Surg Am 1963; 45: 45-52.
12. Isaacs H, Handelsman JE, Badenhorst M, Pickering A. The muscles in club foot-a histochemical and electron microscopic study. J Bone Joint Surg Br 1977; 59: 465-72.
13. Kite JH. The nonoperative treatment of congenital club foot. Clin Orthop 1972; 84: 29-38.
14. Kojima A, Nakahara H, Shimizu N et al. Histochemical studies in congenital club foot, en Simons GW: The club foot: The present and a view of the future. New York, NY: Springer Verlag, 1994, pp 16-21.

15. Laaveg SJ, Ponseti IV. Long-term results of treatment of congenital club foot. J Bone Joint Surg Am 1980; 62: 23-31.
16. Lochmiller C, Johnston D, Scott A, Risman M, Hecht JT. Genetic epidemiology study of idiopathic talipes equinovarus. Am J Med Genet 1998; 79: 90-6.
17. Ponseti IV. Treatment of congenital club foot. J Bone Joint Surg Am 1992; 74: 448-54.
18. Roye DP, Roye BD. Idiopathic congenital talipes equino varus. J Am Acad Orthop Surg 2002; 10: 239-248.
19. Wynne-Davies R. Family studies and the cause of congenital club foot. Talipes equinovarus, talipes calcaneo-valgus, and metatarsus varus. J Bone Joint Surg Br 1964; 46: 445-63.
20. Wynne-Davies R. Genetic and environmental factors in the etiology of talipes equinovarus. Clin Orthop 1972; 84: 29-38.
21. Yamamoto H, Muneta T, Morita S. Nonsurgical treatment of congenital club foot with manipulation, cast and modified Dennis Browne splint. J Pediatr Orthop 1998; 18: 538-542.