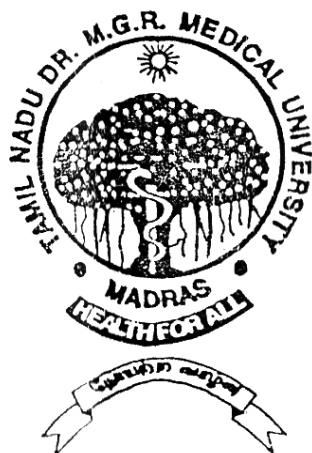


**TREATMENT OF IDIOPATHIC CONGENITAL  
TALIPES EQUINO VARUS BY PONSETI METHOD  
- A SHORT TERM FOLLOW UP STUDY**

*Dissertation submitted for*

**M.S. DEGREE EXAMINATION  
BRANCH – II ORTHOPAEDIC SURGERY  
Department of Orthopaedics and traumatology  
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Thanjavur.**



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**MARCH 2007**

## CERTIFICATE

This is to certify that this dissertation entitled  
**“TREATMENT OF IDIOPATHIC CONGENITAL TALIPES  
EQUINO VARUS BY PONSETI METHOD – A SHORT  
TERM FOLLOW UP STUDY”** is a bonafide record work  
done by **. K. MANIVANNAN** submitted as partial fulfillment  
for the requirements of **M.S. Degree Examinations  
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**Congenital talipes equinovarus** probably the most common congenital pathological condition is a descriptive term. The term was first described by Hippocrates. It was Nicolas Andry in his “Orthopaedicia” described the term “Pedis Equinal” which meant the foot resembling the foot of the horse. The term “talipes equinovarus” is derived from latin : Talipus , a combination of words- Talus ( ankle) and pes (foot) ; equinus meaning “horse like”(the heel in plantar flexion) and varus meaning inverted and adducted.

The incidence of CTEV is approximately 1 – 1.4 cases per 1000 live births<sup>3</sup>. Boys are affected twice as often as girls<sup>3</sup>.The etiology of club foot is still obscure although too many theories have been proposed. A higher incidence of CTEV was also noted in patients with a positive family history<sup>2,3</sup>.

The theories proposed in the etiology of CTEV are mechanical factors in utero, neuromuscular defect, primary germ plasma defect, arrested fetal development, hereditary, etc;

Irrespective of the etiology, the pathoanatomic changes associated with CTEV include ankle equinus, a calcaneum that is in equinus and inverted position beneath the talus and the talar head prominence at the dorsolateral midfoot, navicular medial and plantar to the talar head, cuboid medial and in

front of calcaneum, medial tilting of anterior part of talus, shortened talar neck, narrow posterior ankle mortise, talar tilt out of ankle mortise <sup>4</sup>.

The goal of treatment is to reduce or eliminate these deformities so that the patient has a functional, painfree, plantigrade foot with good mobility and without calluses and does not need to wear modified shoes.

The recommended treatment of CTEV ranges from non-operative casting & stretching to complete peritalar surgical release and bony procedures for neglected CTEV cases.

The methods of J.H.Kite <sup>4</sup>, Ignacio V. Ponseti <sup>6</sup> and French methods as described by Masse & Bensahel <sup>7</sup> are examples of non-operative methods of correction of CTEV.

The technique of gradual and simultaneous correction of all deformities of CTEV using manipulation and casting at weekly interval described by Dr. Ignacio V. Ponseti has gained wide acceptance throughout the world.

Ponseti opined different from others in that he described about the interdependent movements of tarsal bones and considered the view that tarsal

joints move on a fixed axis of motion to be incorrect. He described the Kite's method of correction in which the abduction of calcaneus under the talus was prevented by applying counter pressure over the calcaneocuboid joint as "Kite's error". This is very essential in correction of heel varus as the calcaneus cannot be everted unless it is fully abducted under the talus.

In this study, we have attempted to analyse the functional outcome of Idiopathic clubfoot using Ponseti's technique in children presenting to us within the first two years of age without any prior treatment.



## **AIM**

The aim of treatment in Idiopathic CTEV is to obtain:

Painless

Pliable

Plantigrade and

Cosmetically acceptable foot.

With various treatment modalities so far available for the treatment of Idiopathic CTEV, we are not able to obtain a plantigrade foot with either after single stage or multiple staged procedures. Most of the cases end up with stiff, small and painful foot.

The present study is aimed at evaluating the functional outcome of CTEV correction by Ponseti method at the end of initial correction and at six months follows up.

## **HISTORICAL REVIEW**

The earliest documentation of clubfoot comes from the ancient Egyptian people. Paintings on the walls of their ancient tombs depict the clubfoot deformity, and statue of diastrophic dwarf with a clubfoot can be found in the Tutankhamen collection<sup>4</sup>.

Hippocrates was the first to start treatment in cases of CTEV as soon as possible after birth, before bony deformities are established<sup>5</sup>. According to him, intrauterine malposition was the cause and adaptive changes in surrounding muscles and bones lead to articular malalignment<sup>5</sup>.

Areaus, Pare and Fabrig recommended stretching of the foot by specialized apparatus as early as 17<sup>th</sup> century<sup>2</sup>.

In 1836 Mcguerin was the first to use plaster of paris in the treatment of CTEV<sup>2</sup>.

Surgical methods of management of CTEV was initiated by Little by doing subcutaneous tenotomy of tendo achilles<sup>2</sup>.

In 1857 Solly was the first to introduce a bony procedure – partial cuboidectomy for correction of the deformity<sup>2</sup>.

Ditrich forwarded the theory that primary failure was in the peripheral nerve to the peroneal muscles and he found a sluggish electrical reaction in the peroneii in premature infants with clubfoot.

In the 18<sup>th</sup> and 19<sup>th</sup> centuries, the general trend was to treat CTEV only after the child has passed early infancy. Most preferred method was a single stage correction of all the components of the deformity.

The current trend has reverted towards the non operative treatment of Idiopathic CTEV as soon as possible after birth<sup>3,4</sup>.

## **AETIOLOGY**

The exact aetiology of this condition is not known, but deforming forces are well understood<sup>3</sup>. Various theories had been put forward with regard to the aetiology of CTEV but none have succeeded in explaining the same conclusively.

### **1. Mechanical pressure in utero :**

This is an oldest theory described by Hippocrates. He believed that the foot was held in a position of equinovarus by external uterine pressure leading to development of CTEV. Oligohydromnios prevents fetal movements and makes feet vulnerable to external pressure<sup>4</sup>.

But when foot is forming i.e. first few months of pregnancy, fetus is floating in liquor amnii and result in uniform distribution of pressure. There was absence of increased incidence of CTEV in twin pregnancies in which the uterus was supposed to be overcrowded leading to unacceptability of this theory<sup>4</sup>.

## **2. Neuromuscular defect**

Issac<sup>9</sup> proposed a prominent neurogenic factor in the causation of CTEV. He believed that CTEV is a resistant form of Arthrogryposis multiplex congenita. He concluded that the anomalies observed at the time of dissection were independent of immobilisation, stretching, relaxation of muscles and were not influenced by previous treatment. He also proposed that fibrosis observed in clubfoot muscle specimen should not be considered as a primary aetiological factor<sup>4</sup>.

## **3. Arrested development:**

Heuter and Volkman regarded CTEV as an arrest of fetal development. Bessel, Hagen opposed the theory of arrested fetal development as they said that there is no such physiological stage in the development of fetus that resembles CTEV<sup>4</sup>.

Mau wrote that the embryonic foot does not show the distortion of bones about tarsal joints, which is found in CTEV<sup>4</sup>.

A. Victoria Diaz said “embryonic foot position changes with movements of talus and calcaneus due to growth spurt in distal tibia and fibula. In fibular phase, foot goes into usual fetal position of equinovarus and in tibial phase it is pronated into usual fetal position. Any arrest in the tibial phase without growth spurt results in persistent equinovarus deformity”<sup>10</sup>.

#### **4. Blastemal defect in the development of tarsal cartilage analogue:**

Waisbord<sup>11</sup> described a defect in the cartilage analogue of tarsal bones as cause of the deformity. Ponseti did not find any defect in the cartilage analogue in specimen dissected by him<sup>3</sup>.

Irani and Sherman<sup>12</sup> described a primary germ cell defect in head and neck of talus, but were unable to explain germ cell defect in unilateral clubfoot and correction of deformity by realigning the navicular and calcaneus on talus without any correction in the talus<sup>4</sup>.

#### **5. Primary retracting fibrosis:**

Zimmy et.al<sup>3</sup> showed fibroblast, cells resembling myofibroblast, mast cells in fascia from medial and lateral side of clubfoot and speculated that contractures of ligaments were due to myofibroblast like cells and enhanced histamine released by mast cells.

Fukuhara observed myofibroblast like cells in the spring ligament and speculated fibromatosis in the medial tarsal ligaments as the cause of CTEV <sup>11</sup>. Ippolito and Ponseti I.V. similarly described primary retracting fibrosis as primary cause of CTEV deformity <sup>14</sup>.

## **6. Hereditary and environmental factors:**

The literature regarding inheritance pattern of CTEV is confusing. There is confusing evidence for multifactorial aetiological factors including environmental and genetic factors <sup>15</sup>.

Pedigree studies have established that the disease is certainly not inherited in a single autosomal dominant or autosomal recessive mendelian fashion, although a mendelian component of inheritance cannot be fully excluded <sup>15</sup>. A genetic predisposition operating on a polygenic or in some cases autosomal dominant basis was thought to manifest as Idiopathic CTEV when a threshold for expression is exceeded. When the genetic predisposition alone does not exceed the threshold, environmental factors may act alone or synergistically to reduce the threshold for expression to the point at which Idiopathic CTEV is manifest <sup>15</sup>.

In CTEV, Palmer initially favoured the theory of autosomal dominant gene with reduced penetrance as the cause of deformity but later he supported the multifactorial system of inheritance <sup>16</sup>.

Wynne Davis <sup>16</sup> concluded that a decreasing incidence of CTEV disorder as the relationship of the parents become remote could be indicative of dominant gene with reduced penetrance or multifactorial inheritance system. However the manner in which the occurrence rate decreases is suggestive of a multifactorial model.

A preponderance of the condition among those patient with first degree relative affected increasing the frequency to 2.9 per 1000 live births is highly suggestive of a heritable component <sup>15</sup>.

Chung provided strong corroboration of the polygenic model of inheritance in his study of incidence of CTEV according to race, conducted in the population of Hawaii <sup>16</sup>.

Syndromic CTEV has either Autosomal dominant i.e. Craniocarpotarsal Dysplasia or Whistling face Syndrome, or Autosomal recessive pattern i.e. Diastrophic Dwarfism <sup>16</sup>. It is also associated with Larson syndrome and Smith- Lemli- Opitz syndrome <sup>16</sup>.

Gorlin R.J. <sup>16</sup> described clubfoot associated with X- linked recessive pattern. He wrote on Pirre-Robin syndrome with congenital heart malformation and CTEV.

## **7. Cytological abnormalities:**

Cytological abnormality produces syndromes that include CTEV with maternal unbalanced 6 : 11 translocation as reported by Clark <sup>16</sup>. Insley reported a case of association of CTEV with a deficiency of a part of long arm of chromosome 18 <sup>16</sup>.

## **PATHOANATOMY**

Antonio Scarpa was the first to describe the vivid anatomy of CTEV. He described the twisting of the calcaneus and navicular around the talus as “congenital dislocation of the talocalcaneonavicular joint” <sup>3</sup>.

William Adams <sup>17</sup> called attention to abnormal shape of the head and neck of talus, which he felt was secondarily due to the acquired deformities i.e. adaptation to the altered position of the os calcis and navicular, this deformed shape being result than cause of the deformity.

Evance D. said that the essential abnormality lies in the midtarsal joints and other elements of the deformity were due to secondary adaptive changes <sup>4</sup>.

Attenborough said “the fundamental deformity is plantar flexion of the talus” <sup>18</sup>.



Severity of the CTEV depends upon the degree of bony displacements whereas the resistance to treatment is determined by the rigidity of soft tissue contractures <sup>3</sup>.

The adapted alteration in the shape of tarsal bones are acquired in accordance of the Wolff's law <sup>4</sup> i.e. every change in the use of static function of bone causes a change in the internal form and architecture as well as alteration in its external formation and function according to mathematical law. The soft tissue contractures are acquired in accordance with law of Davis <sup>4</sup> which states, "when ligaments and soft tissues are in a lax state, they gradually will shorten".

### **DEFORMITIES IN CTEV**

The deformities in CTEV are:

1. Fore foot adduction
2. Hind foot varus
3. Hind foot equinus
4. Cavus

The foot in CTEV is always smaller in size than the normal foot in cases of unilateral CTEV due to small muscle mass and connective tissue fibrosis<sup>3</sup>.

## **OSSEOUS DEFORMITIES**

### **TALUS:**

This is least displaced but most deformed bone in CTEV. As it has no muscle attachments, it is forced into equinus by its articulations and attachment to calcaneum and navicular<sup>3</sup>. It appears to be subluxated anteriorly out of ankle mortise.

Body of talus is wide anteriorly as only posterior part of trochlea is in articulation with tibial plafond. Schiltz<sup>4</sup> observed that only the posterior half was normal and having normal rounded contour. The anterior half was wide, abnormal and important cause of limitation of dorsiflexion and persistent equinus. The posterior part of the talus which was not covered with cartilage is intra-articular.

The neck of the talus is directed plantarwards and medially<sup>4</sup>. The head-body angle as measured by Paturet is strikingly smaller in CTEV<sup>11</sup>. The neck is usually foreshortened and the usual constriction is absent.

The head of the talus is wedge shaped. It shows two articular facets. The anterolateral surface is left uncovered by displaced navicular, which extends over the talar neck. Talonavicular joint is oriented in a more sagittal plane compared to normal coronal orientation of the facets<sup>3</sup>. Three facets on the inferior surface of the head appear as a single continuous flat surface. Posterior concave facet of the body is less developed and shallow. Medial surface is underdeveloped but congruent with oblique surface of calcaneus.

#### **CALCANEUS:**

Calcaneus is involved in all the three deformities of CTEV i.e. equinus, varus and adduction. Clinical deformity is due to abnormal position rather than abnormal shape of the calcaneus. Posterior tuberosity is displaced upwards and medially. Anterior end of the calcaneus is displaced downwards, medially and inverted under the head of the talus<sup>3</sup>. Sustentaculum tali is displaced medially and underdeveloped. Medial surface is underdeveloped but congruent with corresponding articular surface of the talus. Posterior facet is underdeveloped while anterior and medial facets are flat and continuous. The longitudinal axis of talus and calcaneus are parallel to each other.

## **NAVICULAR**

This is most severely displaced bone in CTEV <sup>4</sup>. It is grossly medially displaced and adducted, inverted over the head of the talus. It is in close contact with sustentaculum tali and medial malleolus. Medial tuberosity of the navicular is large and provides large area of insertion for enlarged, thickened tibialis posterior tendon. It is wedge shaped with wide dorsal and narrow plantar lateral surface.

## **CUBOID**

It is medially displaced and inverted in front of the calcaneus. It is not as much medially displaced as the navicular. Only the medial part of anterior part of the calcaneus articulates with the cuboid.

## **CUNEIFORMS AND METATARSALS**

Cuneiform and Metatarsals are always adducted but are normal in shape. 1<sup>st</sup> metatarsal is always in plantar flexion as compared to other metatarsals and accounts for the cavus deformity in CTEV <sup>3</sup>.

## **TIBIA**

Lower end of tibia articulates only with posterior part of talus which is devoid of articular cartilage. Tibia has half the amount of external rotation as compared to normal foot <sup>19</sup>. It has been the usual convention to suppose

that the tibia was medially rotated. This however has been challenged by Swann et al.(1969), who demonstrated lateral rotation of the tibia which indicated the need for a rotational osteotomy in some cases <sup>52</sup>.

Lateral malleolus is displaced posteriorly <sup>4</sup>. This brings the tendo achilles in close relation to lateral malleolus mainly due to thickening of the fascia enclosing peroneal tendons and the calcaneofibular ligament <sup>20</sup>.

### **SOFT TISSUE ANATOMY**

CTEV foot is always shorter than the normal foot <sup>3</sup>. Reduction in the girth and length of leg muscles is a common finding <sup>3, 4, 10, 11, 14</sup>. Increase in fibrous connective tissue in the muscles and tendon sheath is common finding during dissection <sup>3</sup>. Few authors have observed abnormalities in the insertion of tendon during anatomical dissection and at surgery <sup>5</sup>. Most authors have found that the ligaments on the posterior and medial aspect of the tarsal joints are thick and short <sup>3, 10, and 14</sup>.

### **POSTERIOR CONTRACTURES**

The contracture of tendo achilles, ankle capsule, subtalar capsule, posterior talofibular ligament and calcaneofibular ligament prevents correction of equinus deformity <sup>5</sup>.

Posterior capsules of the ankle and the subtalar joints are thickened and contracted. Posterior subtalar capsule contractures are more severe than the posterior ankle contractures.

The Achilles tendon is always contracted and shortened. This prevents downward extrusion of posterior tuberosity of the calcaneus, which is necessary for dorsiflexion. Its calcaneal attachment is broader and wider. Its insertion is more on the medial side of the calcaneal tuberosity as compared to normal foot, resulting in varus position of the calcaneus. This medial attachment must be divided while performing posterior release or posteromedial soft tissue release, to aid in the correction of heel varus.

Posterior talofibular ligament and calcaneofibular ligaments become thickened and shortened as per Law of Davis <sup>4</sup>. This results in prevention of movements of the fibula, which were very essential for normal dorsiflexion at the ankle joint. This must be excised in posteromedial soft tissue release to obtain good correction <sup>20</sup>.

The dorsiflexion of the talus is prevented by the contracted and shortened posterior capsule of the ankle and tight achilles tendon and posterior talofibular ligament. These structures prevent the downward exit of

the back portion of the trochlea out of the ankle mortise, a prerequisite for the dorsiflexion.

### **MEDIAL PLANTAR CONTRACTURE**

**(Tibialis Posterior tendon, Deltoid ligament, Spring ligament, Talonavicular capsule)**

The fibrosis of the above mentioned structures form a mass of indistinguishable scar, which obscures the midtarsal, subtalar joints. It maintains sustentaculum tali, medial malleolus and medial tuberosity of the navicular in close proximity. This mass of scar tissue prevents the forward and lateral migration of the navicular as well as lateral movement of anterior end of the calcaneus. This fibrous tissue forms deep layer of Deltoid ligament, which is located between contiguous surfaces of the medial malleolus and medial articular surface of the talus <sup>14</sup>.

The tibialis posterior tendon is short and its tendon sheath is thick and hypertrophied. It has abnormal attachments to spring ligament, sustentaculum tali and navicular. It has very wide insertion on navicular tuberosity. It also blends with common mass of scar tissue, which maintains sustentaculum tali, medial malleolus and medial tuberosity of the navicular in close proximity.

Both flexor hallucis longus and flexor digitorum longus are short and contracted which causes flexion contractures of the digits. The *Master knot of Henry*<sup>4</sup> where these two structures cross is an important plantar contracture that resists mobility of the navicular by virtue of its attachment to the undersurface of the navicular.

**Spring Ligament:** This is an important structure, which supports talar head on its plantar aspect. It is always contracted, short and inelastic. This is because of the equinovarus deformity present in CTEV, which brings the navicular in close relation to sustentaculum tali resulting in relaxation of the spring ligament and subsequent contracture as per the law of Davis.

According to the law of Davis<sup>4</sup>, the talonavicular joint capsule also contracts, which is in a lax state due to the equinovarus position.

## **TALOCALCANEAL INTEROSSEOUS AND BIFURCATED**

### **LIGAMENTS:**

These are underdeveloped, stretched and thin<sup>7</sup>. They are contracted in cases of neglected CTEV in the older children<sup>4</sup>. Release of these ligaments during surgery may lead to over correction as a complication at a later date<sup>4</sup>.



## **PLANTAR CONTRACTURES:**

The contractures of abductor hallucis, intrinsic toe flexors and plantar aponeurosis are more prominent in older children and are less prominent in children less than four years of age <sup>4</sup>.

Abductor hallucis is considered to be an important structure which maintains persistent forefoot adduction. It has an accessory abnormal attachment to tendon sheath of tibialis posterior, navicular and Master knot of Henry.

Peroneal tendons are weak. Intrinsic toe flexors are shortened. Calf muscles and extrinsic toe flexors are also shortened.

The ligaments on medial and posterior aspect of the ankle joint are pulled into the joint by severe plantar flexion and varus displacement of talus. There is marked thickening and shortening of the tibionavicular and plantar calcaneonavicular ligament.

## **BIO-KINEMATICS:**

The correction of the severe displacements of the tarsal bones in CTEV requires a clear understanding of the functional anatomy of talus.

There are controversies regarding axis of motion of subtalar joints. According to Farabuef, Virchow H, Huson and Siegler, there is no fixed axis of motion of subtalar joint. This is in contrast to the concept by Hicks, Elfnan and Inman which emphasis that subtalar joint moves around a fixed axis <sup>3</sup>.

A better understanding of the tarsal mechanics in the normal foot was given by Huson in his thesis “A functional and anatomical study of tarsus”. He demonstrated that tarsal joints do not move as a single hinge but rotate about a moving axis as in the case of the knee. Each joint has its own specific motion pattern. These are described by means of discrete arcs, representing the successive portion of a particular moving axis. This successive position is followed by a fixed pattern which is characteristic for the joint concerned <sup>6</sup>.

He described “Constrained Mechanism” in which motion of the tarsal joints occur simultaneously. If one of the joint movements is blocked the other joint movements also get blocked. The ligaments play an important role as “Kinematic Constraints” of joints apart from their share in forced transmission to support the elastic vault structure of the foot <sup>3</sup>.

The concept of passage of axis of rotation from anteromedial to posterolateral was given by Inman <sup>21</sup>.

Seigler<sup>3</sup> described “Kinematic Coupling” as there is no separation between the motion of the ankle joint and subtalar joint in living objects. Motion of the foot shank complex in one direction occurs by the combined motion of both joints. Contribution from ankle joint in dorsiflexion and plantar flexion is more than that of subtalar joint while subtalar joint has more contribution in inversion and eversion than that of ankle joint. Both joints contribute equally in internal and external rotation.

Ponseti<sup>53</sup> gave a new concept to the kinematics around the talus. He described that, in the clubfoot, the anterior portion of the 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 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 will correct the heel varus deformity of the clubfoot.

He emphasized that 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, closed 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. No single axis of motion (like a metered 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 clubfoot necessitates a simultaneous gradual lateral shift of the navicular, cuboid and calcaneus before they can be everted into a neutral position<sup>53</sup>.

## **TREATMENT**

The spectrum of treatment options for CTEV is large .It ranges from non-operative methods including manipulation, strapping, repeated stretching and POP casting on one side to operative methods like soft tissue surgery and bony procedure.

### **FORCIBLE MANIPULATION**

The concept of forcible manipulation was first described by Bruckner<sup>5</sup>. Thomas did immediate forcible correction with a wrench and application of a splint to hold the foot in corrected position<sup>5</sup>. Forcible

corrections at one or two sittings was carried out by Lorenz using a modified Thomas wrench and later used a padded pyramid correcting a deformity over its apex.

Tubey was the first person to give details of the manipulation technique. He advised abduction and eversion at talocalcaneonavicular, calcaneocuboid joint with dorsiflexion of whole foot at ankle <sup>5</sup>.

Harreustein feared damage to distal tibial and fibular epiphysis during forcible manipulation <sup>5</sup>.

## **SPLINT**

Pare advocated splint alone as a device to correct all or part of the deformity <sup>5</sup>. Scarpa used shoes to correct the deformity and emphasized that varus should be converted into equinus. Trelat, Shaffer have described various devices for manipulative correction.

In 1897, Gibnery practiced wrenching to convert the equinovarus into equinovalgus. He then reduced the equinus by tenotomy and manual force, immobilizing the foot in plaster of paris cast long enough for the bones on the outer side to atrophy and for those on the inner side to hypertrophy <sup>5</sup>.

Dennis Brown in 1934 gave a breakthrough by introducing metal splint for the correction of the deformity<sup>5</sup>.

Forcible manipulation has fallen to disrepute owing to the stiffness of the joints, deformities of bones and spurious correction providing a rocker bottom foot which developed following this form of treatment.

### **REPEATED STRETCHING:**

The emphasis on treating newborn with CTEV was first given by Hippocrates who advocated repeated manual correction and application of strong bandages during manipulation. Over correction was considered to be an essential part of the procedure<sup>5</sup>.

Sofield departed from forcible manipulation and started using elastic traction for the correction of the deformity<sup>5</sup>. Brown supported this principle and claimed that useful feet and leg can be obtained without use of the force. He based his thoughtful account on three well known hypotheses: continuous traction will gradually tire a muscle, a contracted muscle put on stretch will gradually lengthen, if relaxed, will shorten and return to the contracted state as per the Law of Davis<sup>4</sup>. Hence over correction is a must.

J. Hiram Kite <sup>2, 3, 5</sup> was a strong advocator of non operative treatment of clubfoot. His original technique consists of manipulation and casting followed by wedging of the cast to correct individual deformities. Later he advised repeated change of the whole cast with manipulative stretching at each stage. He said “Whatever is gained without force is achieved without harm”.

Jones and Lovett <sup>6</sup> said that: “In very young children it is probable that every case can be cured without operation with the exception of a possible tenotomy of the tendo achilles in the final stage after constantly repeated manipulations by the parents carefully taught by the surgeon”.

#### **PLASTER OF PARIS CASTS:**

Guerin was the first to describe the use of plater of paris casts in the treatment of CTEV <sup>5</sup>. This was followed by Thomas, Jones, Litle, Bradford and Lovett (1899) and Whitman (1910). Soule <sup>5</sup> practiced manipulative reduction followed by retention in adhesive strapping incorporating the strapped limb in plaster of paris cast (1930). Elmslie used plater of paris casts without splinting. Trethowan and Dunn said that it is practically impossible to maintain the correction by POP cast <sup>5</sup>. Lord introduced the above knee cast to avoid slipping and to aid in the correction of inversion.

### **ADHESIVE STRAPPING:**

It is not known who first described adhesive strapping to retain the correction, but Whitman <sup>5</sup> was one of the most effective advocates of adhesive strapping for correction of the deformity. Masse and Bensahel has popularized this concept in recent times <sup>1</sup>.

### **KITE'S METHOD:**

The initial technique of Kite as described above was modified by himself in which he advocated repeated stretching and applying a new cast instead of wedge correction for individual deformities. After full correction, Phelps splint is used for maintenance of CTEV correction <sup>1, 2</sup>. This method was derived from the concept three-point pressure, where manipulations are done done by applying counter pressure over calcaneocuboid joint and abduction of whole foot under the talus. Ponseti described this as 'Kite's error' as by applying counter pressure over calcaneocuboid joint he blocked abduction of the calcaneus under the talus. This is very essential in the correction of the heel varus as the calcaneus cannot be everted unless it is fully abducted under the talus <sup>3</sup>. Although this method is effective in most cases, due to long duration of treatment, the practice changed and surgical management is recommended for those patients with residual deformity after three months of manipulation and casting <sup>1</sup>.



## **FRENCH METHOD:**

This nonoperative method of correcting CTEV was developed by Masse and Bensahel in France in 1970<sup>1</sup>. It is also known as “Functional Method” of CTEV deformity correction. Followers of this method believe that retraction of posterior tibial muscle and weak peroneal muscle are the primary factors responsible for clubfoot. It consists of daily manipulation of the newborn clubfoot, stimulation of weak peroneii, and temporary immobilization with non-elastic adhesive strapping. Daily treatment is continued for approximately two months and then sessions are progressively reduced to three sessions per week for an additional six months, after which strapping is continued until becomes ambulatory. Night time splinting is used for an additional two to three years<sup>1</sup>. In 1990 a continuous passive motion machine was developed in France only for clubfoot treatment<sup>23</sup>. Manipulations are done on daily basis by the trained physiotherapist. Daily two sittings of continuous passive motion for foot and ankle are advocated. This treatment is very lengthy, expensive and a lot depends on the skill of the physiotherapist. For those who still require surgery, the procedures are usually restricted to posterior structures only.

This method fails to correct the deformity in a quarter of the cases<sup>1,24</sup>. Parents’ compliance is very essential as daily visits to the clinic are

required for the treatment and if patient is living far from the hospital, successful outcome becomes less likely.

#### **PONSETI TECHNIQUE:**

Ponseti published his first article on CTEV correction in *The Journal of Bone and Joint Surgery* in March 1963 which was not widely accepted. However his article in 1995 on the long term follow up of CTEV cases by his technique created a new path in the treatment of CTEV by nonoperative method <sup>53</sup>.

It consists of serial manipulation and casting with gradual and simultaneous correction of all deformities of CTEV. Manipulations and casting are done at weekly intervals with POP immobilization. Equinus is the only residual deformity, which is to be corrected by percutaneous tenotomy of tendo Achilles <sup>7, 28, 29</sup>. This is followed by POP casting for three weeks. Then the baby is subjected to bracing protocol which consists of open toe high-top straight last shoes attached to a bar for full time for the first three months and twelve hours at night and two to four hours in the middle of the day for a total of fourteen to sixteen hours during each twenty four hour period <sup>53</sup>.

## **SEQUENCE OF DEFORMITY CORRECTION IN PONSETI**

### **TECHNIQUE :**

#### **CAVUS :**

The first element of management is correction of the cavus deformity by positioning the forefoot in proper alignment with the hindfoot. The cavus which is the high medial arch is due to the pronation of the forefoot in relation to the hindfoot. The cavus is always supple in newborns and requires only supinating the forefoot to achieve a normal longitudinal arch of the foot. 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 for effective abduction of the foot to correct adductus and varus.

#### **MANIPULATION :**

##### **Location of the head of the talus:**

The head of the talus is palpated in front of the lateral malleolus as its lateral part is barely covered by the skin. The anterior part of the calcaneus is felt beneath the talar head.

##### **Stabilize the talus :**

Stabilizing the talus provides a pivot point around which the foot is abducted.

**Manipulation of foot :**

Next with the foot in supination and talus stabilized , the foot is abducted as far as can be done without causing discomfort to the infant. The correction is held with gentle pressure for about 60 seconds and then released.

**Subsequent casts :**

During this phase of treatment, the adductus and varus are fully corrected. The equinus deformity gradually improves with correction of adductus and varus. This is part of the correction because the calcaneus dorsiflexes as it abducts under the talus. No direct attempt at equinus correction is made until the heel varus is corrected.

**Decision to perform tenotomy :**

A major decision point in management is determining when sufficient correction has been obtained to perform a percutaneous tenotomy to gain dorsiflexion and to complete the treatment. This point is reached when the anterior calcaneus can be abducted from underneath the talus. It has to be confirmed that the foot is sufficiently abducted to safely bring the foot into 0 to 5 degrees of dorsiflexion before performing tenotomy. This abduction allows the foot to be safely dorsiflexed without crushing the talus between the calcaneus and the tibia .If the adequacy of the abduction is uncertain, another cast or two is applied to be certain.

## **MAINTANENCE OF DEFORMITY CORRECTION:**

The brace is applied immediately after the last cast is removed, three weeks after tenotomy. The brace consists of open high-top straight last shoes attached to a bar. For unilateral cases, the brace is set at sixty to seventy degrees of external rotation on the clubfoot side and thirty to forty degrees of external rotation on the normal side. In bilateral cases, it is set at seventy degrees of external rotation on each side. The bar should be of sufficient length so that the heels of the shoes are at shoulder width. The bar should be bent five to ten degrees with convexity away from the child, to hold the feet in dorsiflexion.

The brace should be worn full time (day and night) for the first three months after the last cast was removed. After that the child should wear the brace for twelve hours at night and two to four hours in the middle of the day for a total of fourteen to sixteen hours during each twenty four hour period. This protocol continues until the child is three to four years of age<sup>53</sup>.

The rational behind this bracing is that the medial soft tissues remain stretched out only if the brace is used after the casting. In the brace, the knee are left free, so that the child can kick them straight to stretch the gastrocnemius tendon. The abduction of the feet in the brace, combined with the slight bend causes the feet to dorsiflex. This helps maintain the stretch on the gastrocnemius muscle and Achilles tendon.

## **RELAPSE :**

Relapse is detected when slight equinus and varus deformity of the heel is observed, usually without increased cavus and adduction deformity of the fore foot <sup>4</sup>. Relapses are rare after five years and extremely rare after seven years of age regardless of whether the deformity is fully corrected or not<sup>3</sup>. Following are the guidelines described by Ponseti for treatment of relapsed CTEV <sup>3</sup>.

A. For correction of second or third relapses in children older than two-and-half years of age, when tibialis anterior has a strong supinatory action, transfer of tibialis anterior to third cuneiform is advocated. Transfer of the tibialis anterior tendon averts further relapse, maintains the correction of heel varus and thus greatly reduces need for medial release operation. The tibialis anterior tendon should never be split so as not to lose its eversion power, nor should it be transferred to fifth metatarsal or to the cuboid since this may excessively evert the foot causing severe forefoot pronation and heel valgus. To prevent bow stringing of tendon under the skin in front of the ankle, the tendon must be left under the superior retinaculum.

B . Ligament and joint release surgeries are necessary only in few cases. It should not be done before the age of six months. Ponseti advocates sectioning of only tight ligaments to achieve proper alignment of bones, since

a perfect reduction is unattainable owing to the incongruity of the joint surfaces and changes in the shape of the bones. Lengthening of tendon of tibialis posterior was done by technique described by Coleman<sup>40</sup>.

C . Cavo-varus is the commonest residual deformity of treated CTEV, in which tarsus remained in some degree of varus while forefoot is pronated. The plantar fascia becomes shortened and thickened, thus aggravating the deformity<sup>3</sup>. The rigidity of heel varus is assessed by Coleman's lateral block test<sup>40</sup>. For correction of cavovarus deformity, if heel varus corrects within five degrees of the neutral position with the Coleman's block test, following series of procedures advocated by Reginald R. Cooper<sup>3</sup> is used for best correction of the deformity-

1. Severance of plantar fascia percutaneously.
2. A small dorsolateral wedge of bone is resected from the base of the first metatarsal.
3. Jones procedure.
4. The tendon of peroneus longus is severed in the plantar aspect of the foot and sutured to the tendon of peroneus brevis.
5. Transfer of tendon of tibialis anterior to the third cuneiform.
6. Lengthening of the tendo achilles.

#### **D . TRIPLE ARTHRODESIS :**

This is a salvage procedure. This is to be done in patients at or nearing the skeletal maturity. It is indicated when ankle joint motion is fairly good but the tarsal joints are very rigid in supination<sup>3,4</sup>.

#### **E . TALECTOMY :**

It is indicated in severe cases of very stiff club foot with little or no ankle motion that have relapsed after extensive tarsal release operation. It gives satisfactory results when performed between ages of one to six years<sup>3</sup>. TALECTOMY can be done as a primary procedure in patients with severe club foot and poor or absent leg muscles, who are suffering from arthrogryposis or myelomeningocele.

Due to structural abnormalities of the talar bones and joints, a clubfoot cannot be corrected fully and hence completely normal foot is neither desirable nor expected<sup>5, 7, 39, and 41</sup>.



Patients were selected from the Out Patient section of the Department of Orthopaedics and Traumatology, Thanjavur Medical College , Thanjavur for correction of Idiopathic CTEV using the Ponseti technique from October 2004 to November 2005 .Cases of Idiopathic CTEV of age upto 2 years were selected. These patients were followed up in a prospective manner for a period of six months.

**INCLUSION CRITERIA :**

1. Adduction,Supination and varus deformity of the foot with or without wasting of calf muscles.
2. Age less than two years.
3. Virgin club foot.

**EXCLUSION CRITERIA :**

1. Postural club foot.
2. Syndromic club foot.
3. Neglected club foot.
4. Relapsed club foot.

Thirty four patients entered the study after explaining the study protocol and the possible necessity for Achilles tenotomy and foot abduction orthosis till the age of four years. Appropriate informed written consent was obtained from the parents (Appendix II). Twenty eight patients (twenty three

unilateral and five bilateral CTEV) completed serial castings with or without Achilles tenotomy and were given foot abduction orthosis and were followed up for six months. Four patients who did not achieved the required degree of correction at the end of ten castings were considered as failure cases and were referred for posteromedial soft tissue release. Two patients were non – compliant and dropped out in the middle of the treatment.

These patients are followed-up in a prospective manner for a period of six months.

All infants were in the age group of four days to two years with a mean age of presentation of 192 days. All were assessed for associated syndromic pathology and only those infants with idiopathic CTEV were included in the study. Before cast application every week the degree of deformity was graded according to Pirani severity scoring system.

The Pirani severity scoring system consists of midfoot score and hindfoot score with grading of the deformity between 0 and 3 in each category. (Appendix I).

There were twenty one male and eleven female infants included in our study. Three male infants and two female infants had bilateral deformities. Of the eighteen male patients with unilateral deformity, fourteen

infants had involvement of right foot and four had involvement of left foot. Out of nine female infants with unilateral involvement, one had involvement of left foot.

### **PONSETI TECHNIQUE**

Initially a layer of cast padding was applied from groin to toe and the surgeon held the foot in corrected position. An assistant applied the cast using fast setting plaster in two sections. The first one comprised of below knee plaster to hold the foot in corrected position. The next section consisted of extending the cast above knee to convert it into a groin to toe plaster cast. During this, the knee was held in 90 degree flexion. After application of the cast the child was observed for about 30 minutes for any signs of limb ischemia. The parents were educated about possible complications like cyanosis, swelling, excess cry and the contact number in case of emergency were provided. They were then advised to report for the next cast after 7 days.

The first cast was aimed at correcting the cavus deformity by supinating the forefoot thereby bringing the forefoot in alignment with the hindfoot.

In the second and subsequent casts, the foot in supination was abducted while the surgeon applied counter- pressure on the head of the talus.

The calcaneus abducts by rotating and sliding under the talus. Simultaneously it extends and everts thereby correcting the heel varus. To stretch the medial tarsal ligaments fully, the foot was severely abducted to an angle of about 60 degrees. A maximum of 10 casts were fixed as endpoint for correction of cavus, hindfoot varus and adduction deformity.

After correction of the above deformities, passive dorsiflexion of the foot to 15 degree above neutral with the examiner applying a single finger pressure was attempted; If achieved, a final cast was applied in the final corrected dorsiflexed position for three weeks. If dorsiflexion more than 15 degrees was not possible, a percutaneous tenotomy of the tendo achilles was done under general anaesthesia. After this tenotomy, the foot was placed in the final corrected dorsiflexed position for three weeks.

After the last cast was removed, correction was maintained by using Dennis-Browne splint. The brace was worn full time (day and night) for the first three months after the last cast was removed. After that, the child should wear the brace for 12 hours at night and 2-4 hours in the middle of the day for a total of 14-16 hours during each 24-hour period. This protocol continues until the child is 3-4 years of age.

The patients were reviewed at 14 days after application of Dennis- Brown splint to assess the compliance of the parents. In subsequent visits patients were reviewed once in three months. The parents were given contact numbers and were advised to contact us regarding the maintenance of Dennis Browne splint.

### **Statistical analysis**

The results were analysed using SPSS 10 software.

T-Test paired samples analysis was done to find out the difference between the means of values (before casting, after casting and follow up castings).

In this study full correction of the deformity was obtained in thirty three feet (23 unilateral and 5 bilateral CTEV). In this study, the end point for castings was taken as ten casts. Percutaneous tenotomy was done, if needed, once adequate abduction is achieved.

Out of 37 feet, 6 feet achieved full correction at the end of initial casting without percutaneous tenotomy and 27 feet were fully corrected with percutaneous tenotomy. Four feet were not corrected with Ponseti method and were considered as failure cases. They were referred for posteromedial soft tissue release. Two patients were non-compliant and dropped out in the middle of the study.

The mean age at initiation of treatment for 32 patients (37 feet) was 192 days (range 4 days to 2 years).

The mean initial Pirani severity score for 37 feet was 4.30 (out of maximum possible score of six). After full correction by ponseti technique (with or without percutaneous tenotomy) the final mean score was found to be 0.17 and the mean change in score was found to be 4.13. This was analysed by the paired t test and the p value was <0.0005 which is significant. The mean value of Pirani score at 6 months follow up was 0.11 which shows

a change of 4.19 from the initial score. This change also has a p value of <0.0005 which is significant.

**Table: 1. Distribution of age**

	Frequency	Percent
0 –6 months	12	37.5
6 – 12 months	7	21.9
12 – 8 months	9	28.1
18 – 24 months	4	12.5
Total	32	100

The most common age group was 0 – 6 months with 12 (37.5%) patients and most of the patients (59.4 %) were less than 1 year of age.

**Table: 2. Details of age of subjects in days**

	Age in days
Mean	192.84
Std. Error of mean	33.35
Median	135
Std. Deviation	188.68
Minimum	4
Maximum	730

The minimum age – 4 days

The maximum age – 730 days (2 years).

The mean age at initiation of treatment for the 32 patients was 192 days  
(range 4 days – 730 days).

**Table : 3. Distribution of Sex**

Sex	Frequency	Percentage
Female	11	34.4
Male	21	65.6
Total	32	100

There were 11 females (34.4%) and 21 males (65.6%).

The male to female ratio was 1.9: 1

**Table: 4 Side of involvement**

	Frequency	Percent
Bilateral	5	15.6
Unilateral	27	84.4
Total	32	100

5 cases were bilateral (15. 6 %) and 27 (84.4 %) cases were unilateral.

Right: Left ratio was found to be 3.6:1



**Table 5 : Correlation between side and sex**

Section	Bilateral	Unilateral	
		Right	Left
Male	3	14	4
Female	2	4	1

**Table: 6. Details of Percutaneous tenotomy done**

Tenotomy	Frequency	Percent
Done	24	75
Not done	8	25

75 % of patients needed percutaneous tenotomy of tendo achilles at the end of casting.

**Table: 7. Correlation between Percutaneous tenotomy and sex**

Tenotomy	Sex		Total
	Female	Male	
Done	8	16	24
Not done	3	5	8
Total	11	21	32

76 % of male patients and 72.7 % of female patients needed percutaneous tenotomy.

**Table 8: Details of PMSTR done**

	Frequency	Percent
Not done	28	87.5
Done	4	12.5

PMSTR: Postero Medial Soft Tissue Release.

**Table 9: Details of Pirani score – Paired samples**

		Mean Pirani Score	N	Standard Deviation	Standard Error of mean
Pair I	Before Treatment	4.30	32	0.61	0.11
	After Treatment	0.17	32	0.30	0.053
Pair II	Before Treatment	4.30	32	0.61	0.11
	At Follow up	0.11	32	0.21	0.037
Pair III	After Treatment	0.17	32	0.30	0.053
	At follow up	0.11	32	0.21	0.037

1. Mean Pirani score before treatment - 4.30 (range – 3.5 - 5)

2. Mean Pirani score after treatment - 0.17 (range – 0 – 0.5)

3. Mean Pirani score at 6 months follow up - 0.11 (range – 0 – 0.5)

4. Mean change in Pirani score - 4.13  
(before treatment and after treatment)

P value < 0.0005 (highly significant)

5. Mean difference in Pirani scores - 4.19

(before treatment and at follow up)

P value < 0.0005 (highly significant)

6. Total number of casts required for the study was 284 with a mean of 8.88.

**Table 10: Paired samples test for three pairs**

	Paired differences					T	Df	Sig (2-tailed)
	Mean Pirani score	S.D	S.E.M.	99% confidence interval of the difference				
Pair I	4.13	0.49	0.087	3.89	4.36	47.440	31	<0.0005*
Pair II	4.19	0.56	0.099	3.82	4.46	41.970	31	<0.0005 *
Pair III	0.067	0.25	0.043	-5.68	0.11	1.438	31	0.161

S.D : Standard Diviation

S.E.M : Standard Error of Mean

Df : Degree of freedom

Pair I - Before Treatment

After Treatment

Pair II - Before Treatment

At follow up

Pair III - After Treatment

At follow up

\* - statistically significant

**Table 11: Cast Complications**

<b>Complication</b>	<b>No. of cases</b>
Pressure sores	3
Skin blisters	1
Slippage of casts	2
Eczema	1

The treatment options for Congenital talipes equinovarus has gone a full circle and reached the earlier concept of non-operative treatment, as it is associated with improved results.

As it is evident from our study, the results of deformity correction are better if treatment is started within first month of life and results are statistically significant. Harold <sup>30</sup> and Porter <sup>43</sup> gave similar reports. The visco-elastic properties of infant's soft tissues respond to properly directed mechanical stimuli with gradual remodeling of joint surfaces, resulting in gradual and simultaneous correction of the deformities <sup>3</sup>.

The sequence of deformity correction was most important to avoid complications like Rocker-Bottom foot, persistent cavus and locking of the calcaneus under the talus leading to persistent heel varus. Frick <sup>38</sup> has emphasized on the importance of maximal forefoot supination in the initial casting, failure of which results in persistent rigidity and incomplete correction of the deformity. During manipulations the foot is never pronated in order to prevent bean shaped deformity and incomplete correction of heel varus.

The fact that the navicular moves towards its normal position following manipulation was confirmed by Kuhns in his study using

ultrasonography<sup>44</sup>. Pirani confirmed similar results in clubfoot treated by Ponseti method<sup>46</sup>.

32 children with congenital clubfoot participated in the study. Total number of clubfeet was 37. All the patients were of age 0 to 24 months (range: 4 days to 2 years) at initial casting. Mean age of the group was 192 days. Morcuende et al. had retrospectively analysed the records of 157 patients (256 clubfeet). In this study also all the patients were of the age group 0 to 24 months. There were 21 male children and 11 female children in the present study and the male: female ratio is 1.9:1. Morcuende et al. reported a male female ratio of 2.13: 1. The male preponderance found in this study is in agreement with other studies.

The feet are evaluated using Pirani severity scoring system which was easy to use and simple and fairly reproducible. In our study the scoring was done by a faculty who was not involved in the study and casting was done by the author throughout the period of study. The points in the Pirani scores are allotted on the basis of inspection findings of the sole of the foot, lateral border, posterior and medial creases, palpability of the talus and emptiness of the heel as well as correctability of equinus.

In about twenty four patients (75%), percutaneous tenotomy of tendoachilles was done in order to achieve complete correction. Ponseti himself has observed that percutaneous tenotomy was needed in most of the patients.

Though Ponseti advises tenotomy under local anaesthesia, we found the child to be frightened and uncooperative to procedure under local anaesthesia. Hence general anaesthesia was preferred and tenotomy was done 1.5 cm above the calcaneus with the foot held in maximum dorsiflexion by the assistant. The tenotomy was performed with a size eleven surgical blade.

In our study, we observed that the earlier the child is started on casting by ponseti technique, the results are better without any need for surgery. In few of our infants, pressure sores developed due to the delicate skin .However the pressure sores healed by skipping one week of casting and then reapplying the cast a week later. One of our patient was very obese and presented with frequent slipping of POP casts. Another patient developed repeated eczema and was referred for posteromedial soft tissue release. We also observed that as the child gets older, the prominence over the calcaneocuboid junction in the lateral column prevents complete correction.

The most common residual deformity which occurred at the end of treatment was forefoot adduction in about five patients who completed Ponseti method of treatment with or without percutaneous tenotomy.

The rate of posteromedial soft tissue release can be drastically reduced by using Ponseti technique and hence the complications of surgery were avoided. Colburn reported similar finding following treatment of CTEV by Ponseti method.

We found the following factors contributed to the success of CTEV correction by Ponseti technique:

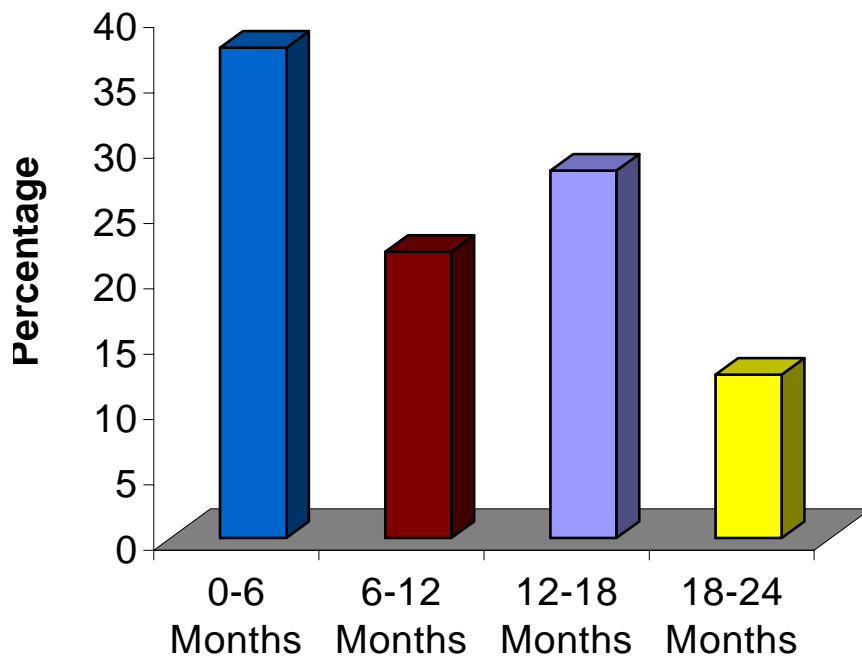
- ❖ Earlier the child was started on treatment better were the results
- ❖ The milder the severity of deformity
- ❖ Strict adherence to the sequence of correction as advised by Ponseti.
- ❖ Removal of the cast just before applying the subsequent cast.
- ❖ Regular follow-up by the patients.
- ❖ The compliance of the parents in maintaining the cast as well as the Dennis Browne splint.
- ❖ Absence of complications.

Our results were successful in 87.5 % of the patients with no major adverse events and the results are certainly encouraging.

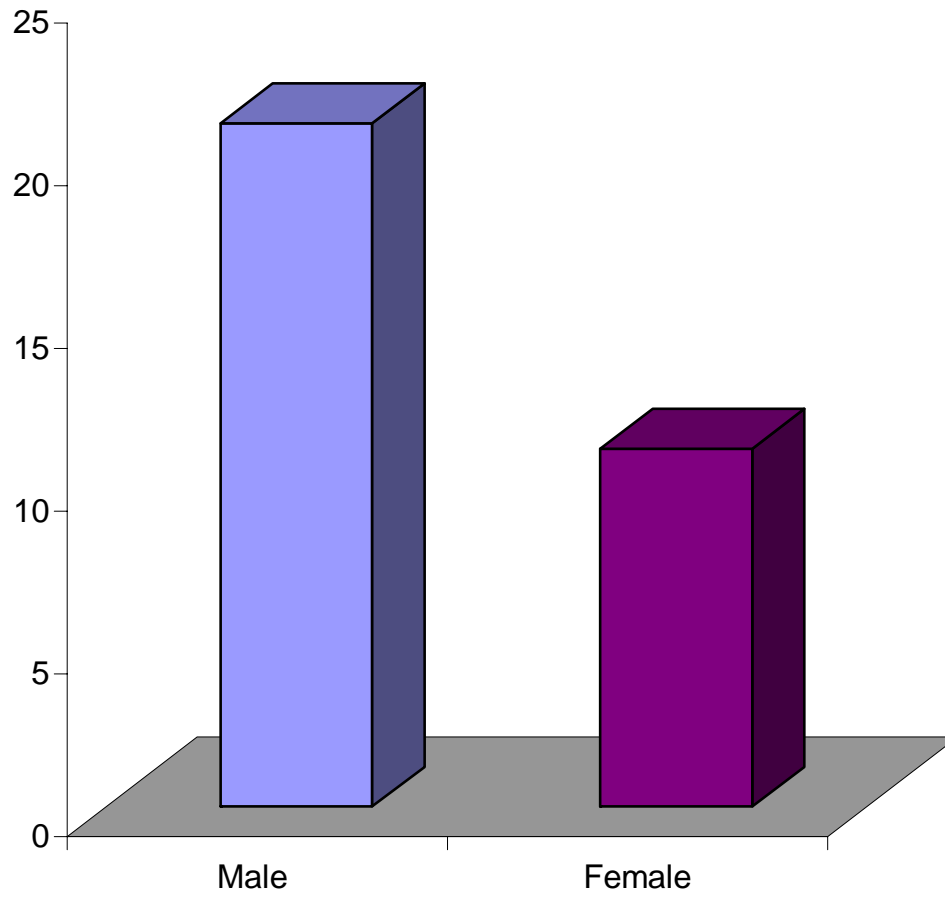


1. Early treatment of Idiopathic CTEV by Ponseti technique results in good correction of the deformity with minimal surgery.
2. Percutaneous tenotomy of Tendo Achilles was required in considerable number of cases to achieve good correction.
3. Forefoot adduction was the frequently observed residual deformity at the end of the treatment.
4. Extensive soft tissue release surgeries like posteromedial soft tissue release was rarely required for correction of the deformity avoiding long term complications.
5. The complication which we encountered frequently was pressure sore and was dealt successfully by skipping the casting for one or two weeks.
6. Parent compliance plays an important role in maintenance of the deformity correction.
7. It is an effective and affordable technique.
8. This non operative method of management of CTEV can be successfully implemented in centres where Infrastructure facilities are inadequate to perform operative procedures.
9. The results of this short term follow up study of management of congenital talipes equino varus are certainly encouraging.

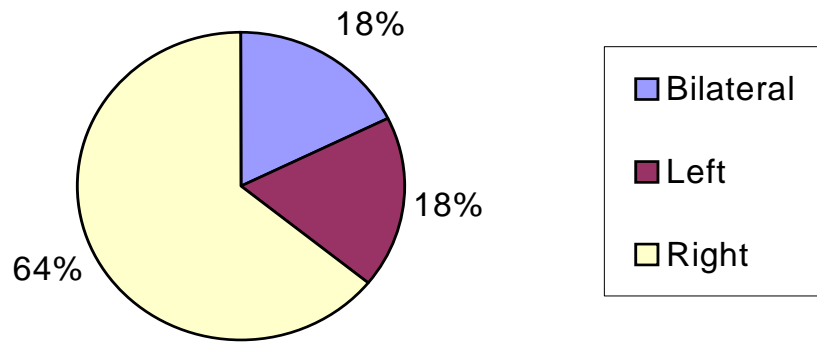
## Distribution of Age



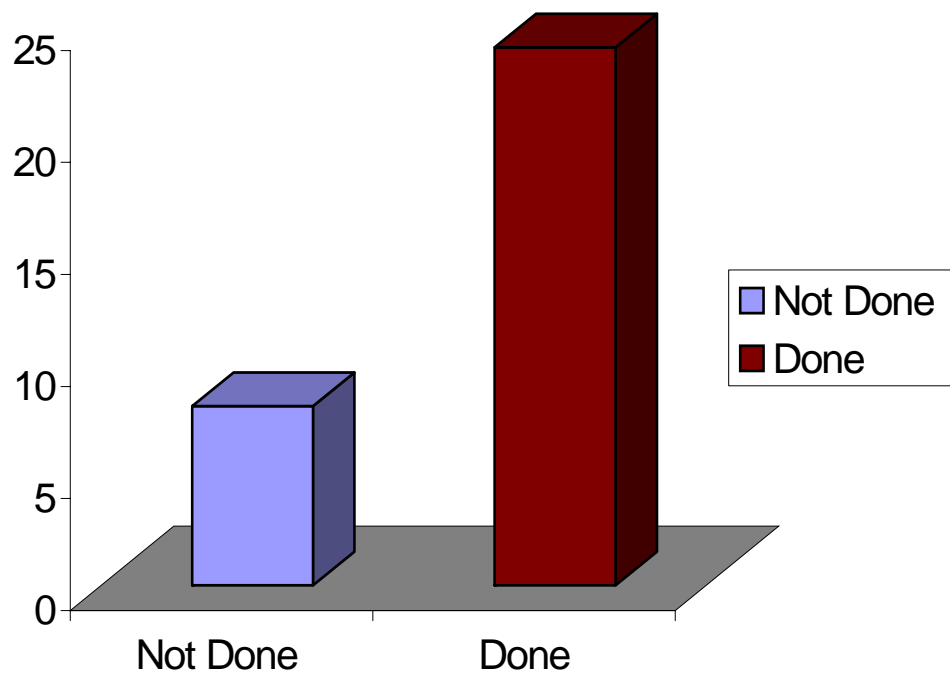
## Distribution of Sex



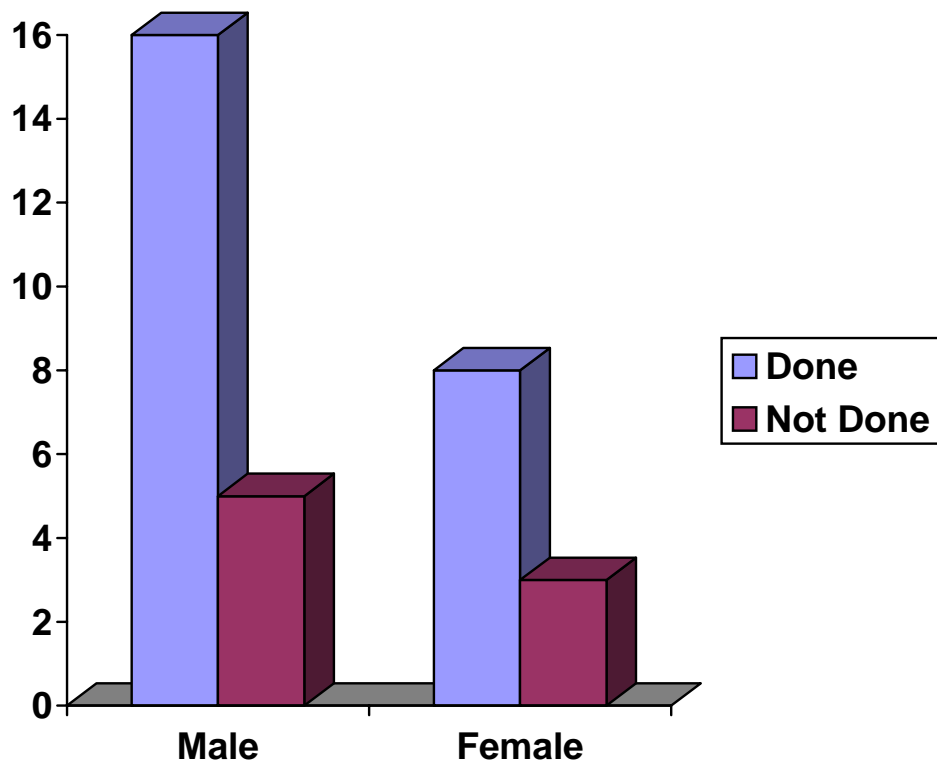
## Side of Involvement



## PERCUTANEOUS TENOTOMY



## Correlation Between Percutaneous Tenotomy and sex



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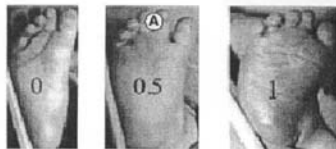
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## PIRANI SEVERITY SCORING

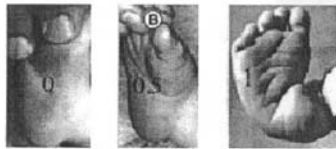
Scores six clinical signs

- 0 normal
- 0.5 moderately abnormal
- 1 severely abnormal



### Midfoot score

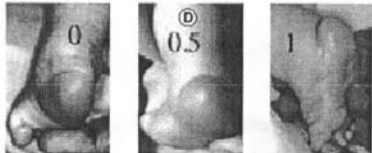
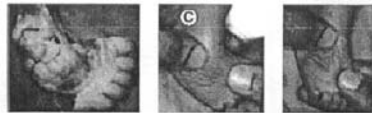
Three signs comprise the Midfoot Score (MS), grading the amount of midfoot deformity between 0 and 3.



*Curved Lateral border (A)*

*Medial crease (B)*

*Talar head coverage (C)*



### Hindfoot score

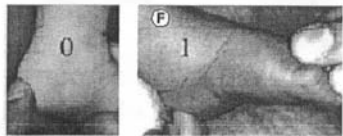
Three signs comprise the Hindfoot Score (HS), grading the amount of hindfoot deformity between 0 and 3.



*Posterior crease (D)*

*Rigid equinus (E)*

*Empty heel (F)*



# Consent Proforma

**Title :** Treatment of Idiopathic Congenital talipes equinovarus by Ponseti method –  
Short term follow up study.

**Aim :** To evaluate the functional outcome of Idiopathic CTEV correction by Ponseti  
method at the end of initial correction and at six months follow up.

**Consent :** I have been explained about the nature of the study and also about the nature  
of child's illness in my own vernacular language.

I here by give my consent for the inclusion of my child in the study

Signature

## PROFORMA

Name :

Age :

Sex :

Address :

Consanguinity of Marriage :

Antenatal H/O :

Natal and Post Natal H/O :

Birth Order :

Immunisation :

Unilateral/Bilateral :

Family H/O :

Any previous treatment :

Assessment of the deformity using Pirani severity scoring system:

❖ Before Treatment :

❖ After Treatment :

❖ At 6 months follow up :

Percutaneous Tenotomy done : YES / NO



**Surgery (PMSTR)** : Yes / No

Side	Casting	Percutaneous tenotomy	Posteromedial soft tissue release
Right			
Left			

Residual deformity at the  
end of treatment :

**Follow – up**

Residual deformity :

Supple / Stiff foot :

Rocker Bottom Foot :

Pain :

Wasting of calf muscle :

Nonspecific complaints :

## KEY TO MASTER CHART

<b>M</b>	-	Male
<b>F</b>	-	Female
<b>Y</b>	-	Yes
<b>N</b>	-	No
<b>R</b>	-	Right
<b>L</b>	-	Left
<b>D.O</b>	-	Dropped out cases
<b>PT</b>	-	Percutaneous Tenotomy
<b>PMSTR</b>	-	Posteromedial Soft tissue release.
<b>PSS-FP</b>	-	Pirani Severity Scoring at six months follow up.
<b>Rx</b>	-	Treatment
<b>MON</b>	-	Months

S.No.	Name	Age	Sex	Side UL/BL	Pirani Severity score		No. of Casting	PT	PMSTR	PSS - FP
					Before Rx	After Rx				
1	NATHIYA	12 MON	F	R	4.5	0.5	9	Y	N	0
2	NEELAKANDAN	8 MON	M	R	3.5	0	10	Y	N	0
3	JAYASREE	8 MON	F	L	4.5	0.5	9	Y	N	0
4	VIJAYARASAN	4 MON	M	R	4.5	0.5	7	Y	N	0.5
				L	3.5	0	7	Y	N	0
5	RAJAKUMARAN	6 MON	M	R	3.5	0	7	Y	N	0
6	IYYAPAN	3 MON	M	R	4.5	0	7	Y	N	0
7	SATHISH KUMAR	4 MON	M	R	4.5	0	6	Y	N	0.5
8	BARADWAJ	1.5 MON	M	R	3.5	0	6	N	N	0
9	VASANTH	22 DAYS	M	R	4	0	6	N	N	0
				L	5	0.5	9	N	N	0
10	DINESH	7 MON	M	R	4.5	0	7	Y	N	0
11	MAHESHWARAN	7 MON	M	L	3.5	D.O	D.O	D.O	D.O	D.O
12	SACHIN	7 MON	M	R	5	0	8	Y	N	0
13	HARIHARAN	7 MON	M	R	4.5	0	7	Y	N	0
14	RAJESHWARI	7 MON	F	R	3.5	0	7	Y	N	0.5
15	ARUN	1.5 MON	M	R	4.5	0	6	Y	N	0
16	MOUNICA	4 DAYS	F	R	4.5	0	7	Y	N	0
				L	3.5	0	6	Y	N	0
17	ASIN	12 DAYS	F	R	5	0	9	Y	N	0
				L	4.5	0	7	Y	N	0
18	PRABUDEVA	9 MON	M	L	4.5	0	8	Y	N	0
19	NANDHINI	45 DAYS	F	R	3.5	0	6	Y	N	0
20	NISHA	23 DAYS	F	R	3.5	0	6	N	N	0

S.No.	Name	Age	Sex	Side UL/BL	Pirani Severity score		No. of Casting	PT	PMSTR	PSS - FP
					Before Rx	After Rx				
21	ARULMANI	4 MON	M	L	4.5	0	7	Y	N	0
22	ANANTH	4 MON	M	R	4.5	0	7	Y	N	0
23	SUDHA	3 MON	F	R	3.5	0	8	Y	N	0
24	RANI	4 DAYS	F	R	3.5	0	6	N	N	0
25	MAHESHWARI	16 DAYS	F	R	4.5	0	7	Y	N	0
26	JAGANIVASAN	21 MON	M	L	5.5	1	10	N	Y	0.5
27	SUDHAN	5 MON	M	R	4.5	0	9	Y	N	0
				L	3.5	0	9	Y	N	0
28	KALIYAPERUMAL	6 MON	M	R	4.5	0.5	9	Y	N	0
29	DHARMARAJ	3 MON	M	R	3.5	0	7	Y	N	0
30	KALAIVANAN	9 MON	M	L	4.5	0	8	Y	N	0
31	GOWRI	18 MON	F	R	5.5	0.5	10	N	Y	0.5
32	JAYABAL	19 MON	M	R	5	0.5	10	N	Y	0.5
33	GILBERTRAJ	24 MON	M	R	5	1	10	N	Y	0.5
34	KRITHIKA DEVI	7 MON	F	R	5	D.O	D.O	D.O	D.O	D.O

## CASE - I



Before Treatment



Before Treatment



1st Cast



At the end of 6th Cast



7th Cast



After 9th Cast



After Percutaneous tenotomy



Denis Browne Splint



At 6 Months

**CASE - II**



**Before Treatment**



**Before Treatment**



**1st Cast**



**At the end of 7th Cast**



**8th Cast**



**After 10th Cast**



**Percutaneous tenotomy**



**Denis Browne Splint**



**Denis Browne Splint**

## PERCUTANEOUS TENOTOMY



**Before Tenotomy**



**Tenotomy**



**After Tenotomy**



**Cast application after tenotomy**



## COMPLICATIONS



Fatty Child



Fatty Child



Fatty Child - Slippage of Cast



Blister



Pressure Sore



Pressure Sore