INTRODUCTION

Talipes equinovarus (TEV) more commonly referred to clubfoot is a development disorder of the foot and ankle that affects one (unilateral) or both (bilateral) feet The foot is in an incorrect anatomical position, and is inclined inward, axially rotated outward, and points downward[1]. True TEV does not resolve itself, and the affected infant will likely need to undergo treatments including casting, manipulation, and, possibly, surgery in order to place the foot in the correct anatomical position [2]. These corrective actions usually allow the infant to walk and run normally.

This deformity has been described since the birth of medicine. It was documented as early as ancient Egypt. Smith and Waren in 1924 found that Pharaoh Siptah of the XIX Dynasty was affected with clubfoot [3]. Talipes Equinovarus was first introduced into the medical literature by Hippocrates in 400 B.C.V.[3,4]. He recognized that some clubfeet were congenital, while some were acquired in early infancy. The term talipes equinovarus is derived from Latin: talus (ankle) and pes (foot); equinus: "horse like" (the heel in plantar flexion) and varus: inverted and adducted. [4].

Kite recognized TEV was composed of forefoot adductus, heel varus and equinus. He noted foot and ankle equinus, and believed forefoot cavus and posterior ankle contracture made up total equinus[5,6]. He did not identify subtalar supination as a third component of equines.

The incidence of clubfoot varies with race and gender. The incidence varies from 0.39 per 1000 among Chinese to 1.2 per 1000

among Caucasians to 6.8 per 1000 among the Polynesians [7]. The male to female ratio[8] is 2.5: 1 Siblings of affected individuals have up to 30-fold increase in the risk of clubfoot deformity. There is also increased incidence in both the twins in monozygotic twins compared to dizygotic twins[9].

The incidence of recurrent clubfoot following Ponseti treatment has been reported between 11% and 41%[10].

Neglected clubfoot is still a relatively common deformity in developing countries, reflecting the socioeconomic problems of the population[11].

The etiologic hypotheses of clubfoot are as varied as are the treatment methods used. The theories of causation vary from intra-uterine molding, germplasm defects, intra-uterine developmental arrest to neurogenic, myogenic and vascular explanations. Abnormalities have been found in almost all the tissues of affected feet including bones, muscles,ligaments, blood vessels etc [9].

The equinovarus deformity is classified into congenital and acquired. The congenital is further classified into idiopathic and non-idiopathic types. The idiopathic type is typically an isolated skeletal anomaly, usually bilateral, has a higher response rate to conservative treatment and a lower tendency towards a late recurrence. The causes of the non- idiopathic type include deformity occurring in genetic syndromes, teratologic anomalies, neurological disorders of known (e.g., spina bifida) and unknown etiology and myopathies. The non-idiopathic type is characterized by opposite deformities in the feet (calcaneovalgus in one foot and equinovarus in the other), presence of other anomalies and

a poor response to conservative or operative treatment. Acquired equinovarus has neurogenic causes (e.g, poliomyelitis, meningitis, sciatic nerve damage) and vascular causes (Volkmann Ischemic Paralysis)[12].

Hippocrates[4] suggested that the treatment of clubfoot should start as soon as possible after birth with repeated manipulations and fixations by strong bandages which should be maintained for a long time to achieve over correction. His teaching principles of treatment are as valid today as they were 2300 years ago.

Kite corrected each component separately. He corrected forefoot adductus first and proceeded to correct heel varus, which he understood as a single plane rotation around a longitudinal axis through the calcaneus. Then he addressed the equinus. Kite's own articles described correction of forefoot adductus and heel varus as a unit. His cast consisted of a slipper cast with a leg component for infants over age 1 and an above-knee portion for infants less than1year old. Ponseti made similar observations about the components of the deformity, but also noted plantarflexion of all or part of the medial column and forefoot supination as well as varus [13,14] His technique differs substantially from Kite's technique.

Ponseti began the casting sequence by supinating the forefoot even further as the adductus component is manipulated. Doing so places the entire foot distal to the talus on the same supinated plane. Then Ponseti would manipulate the equinus. He used above knee casts with the knee flexed for all ages. If Ponseti achieved successful reduction, he would place the child in an externally rotated transverse bar and shoe apparatus in order to maintain reduction. He managed unyielding equinus via an Achilles tenotomy[15].

Lengthening of the Achilles tendon by Stronmeyer was the first description of a surgical correction for the disorder[16]. Kite [17] described the idea of manipulation and serial casting; this remains the standard of care for first-line therapy.

The opinion regarding the treatment of idiopathic clubfoot is that the initial treatment should be non-operative. This includes stretching and adhesive strapping, physiotherapy, Denis Browne splint, manipulation and casting. Some feet cannot be adequately corrected with conservative treatment[18].

Surgical treatment became an option when the principles of general anaesthesia were established. Numerous surgical procedures were used during the past sixty years for treatment of clubfoot and several are still in use [19].

About fifty to sixty percent of the children with clubfoot present with a fixed and rigid deformity after conservative treatment[20]. Operation is indicated when conservative treatment fails to correct the deformity or when initially good results gradually fail, due to the persisting imbalance between the inverting and everting muscular forces. The first series of surgically treated patients and operative procedures appeared in the literature during the 1950s and 1960s[21].

The indications for surgical treatment are based on the severity and rigidity of the deformity[22].

Corrective surgery for idiopathic clubfoot in children up to 1 year of age usually presents satisfactory results.[23] In approximately 20% of these patients, however, additional corrective surgery may be necessary because of recurrence of the deformity.

The treatment of recurrent and neglected clubfoot is challenging even for experienced surgeons. This deformity has a tendency to recur even after several corrective surgeries. Additional surgeries focused exclusively on the soft tissue may lead to greater joint stiffness[24].

The tridimensional aspects of clubfoot deformity and the severe joint stiffness with retraction of soft tissues significantly limit the use of conventional corrective methods, such as osteotomies, triple arthrodesis, or talectomy. A limitation of the corrective capacity of surgery is the typical shortened medial column of the foot associated with a tense neurovascular bundle[24] When aggressive corrections of all the deformities are attempted in a single surgical procedure there is a great risk for damage to the neurovascular bundle, which usually is surrounded by and adhered to scar tissue Therefore, one-step surgical corrections are considered high risk[25].

Potential risks to the soft tissues include extensive skin necrosis, secondary infection, neurovascular compromise, and ischemia, [26] all of which may lead to amputation.

In the past decade the use of circular external fixators has become a treatment option for recurrent and neglected clubfeet. This treatment is based on the distraction-osteogenesis principles described by Ilizarov and colleagues.[27] The progressive distraction offered by the external fixator allows simultaneous correction of all components of the clubfoot deformity.[27] During the course of correction, a gradual lengthening of the blood vessels, nerves, muscles, connective tissues, and skin occurs, reducing the risks for neurovascular damage, skin necrosis, and secondary infection [16].

The Ilizarov method is safe and minimally invasive, requiring minimal or no bone resection[28]. The use of this method also offers more predictable and satisfactory results[29]. Another advantage of the Ilizarov external fixator over conventional methods is preservation of foot size or lengthening of a baseline shortened foot. Osteotomies of the midfoot or hindfoot should be performed during the distraction process. When such osteotomies are performed, the treatment period is prolonged and the device must be kept in place until bone consolidation is complete[24].