



# POSNA

## The Core Curriculum

### Clubfoot, infant

#### Objectives

1. Define clubfoot
2. Describe the pathological anatomy of clubfoot
3. Describe imaging of the infant clubfoot
4. Describe treatment of the infant with clubfoot
5. Discuss prognosis of the infant with clubfoot

#### Discussion points

1. What is a "true" clubfoot? How is it different from a postural deformation? Do you think some reported series may have different criteria for inclusion of an infant clubfoot? How would that alter results?
2. Should early surgery be performed for the infant with clubfoot? If so, what? Why or why not?
3. How do ultrasonographic findings suggestive of clubfoot correlate with postnatal clubfoot?

#### Discussion

There is perhaps no topic in children's orthopaedics more difficult to comprehend from a literature analysis than clubfoot. Trends in treatment have varied through the years. A half century ago, under the influence of Kite, Ponseti, and others; casting was the preferred treatment, even if it could take years to complete. About 30 years ago, authors such as Turco, Goldner, and Simons began writing about operative treatment, and the pendulum swung radically in the direction of surgery as the preferred treatment. Presently, the pendulum is swinging back, as the enthusiasm for the Ponseti method is building among pediatric orthopaedists. It is very difficult to correlate all the different opinions on pathoanatomy and treatment into a unified concept. I recall reading a textbook on foot disorders 30 years ago; there were 2 contributors to the chapter on clubfoot. First, I read on the Ponseti method emphasizing correction of the laterally rotated talus; then, later in the same chapter, I read Goldner's dictum that the fundamental goal of treatment was to correct the internal rotation of the talus. I doubt either author has modified his opinion to the present time, and there is still conflicted literature on this seemingly simple question of the direction of talar deformity. After all, the talus can be analyzed by gross dissection in addition to the sophisticated imaging techniques presently available. The debate continues as evidenced by a letter exchange following a report on longterm results from clubfoot treatment from Iowa. Except in the minds of true believers on either side of the issue, the question remains unresolved.

There are, however, more sophisticated immunochemistry techniques now available to examine structures in the clubfoot, and we will undoubtedly inch closer to a better understanding in the next generation. Consistent findings of analyses of clubfoot anatomy include smaller volume of the hindfoot, arterial dysgenesis (especially the anterior tibial artery), disordered structure of muscle, and a fibrous mass of tissue on the medial side of the foot. The latter has been consistently described in clinical dissections. Recent studies revealed haphazard orientation of collagen and cells in this scar tissue excised from fetuses and young children with clubfoot. Cytocontractile proteins and myofibroblasts are present during contraction of scar. Desmin, a marker of proliferative scar formation such as noted in proliferative Dupuytren's nodules (but not in the fibrous stage), was present in fetal specimens; but not in postnatal specimens. Vimentin, a marker of mesenchymal differentiation, was present in postnatal fibrous cells. Work such as this will help us to better understand the pathoanatomy of clubfoot. Arteriography consistently demonstrates deficiency of the anterior tibial artery. Somatosensory evoked potentials (SSEPs) have been studied, and a relationship between abnormal potentials and severity of involvement was noted.

Clubfoot is defined as a structural deformity of the foot, characterized by fixed cavus and adductus of the forefoot, and varus and equinus of the hindfoot. The ossification centers and cartilaginous anlagen of the hindfoot are smaller than normal. The calcaneus is rotated medially, with the result that the posterior aspect lies closer to the fibula. The rotation of the talus, and the placement of the articular surface for the talonavicular joint in relation to the long axis remains controversial. Carroll, Goldner, Ippolito, Ponseti, Settle, McKay, and Howard are a few contributors to our knowledge of the pathoanatomy of clubfoot. Hjelmstedt and Sahlstedt directed more attention to the ankle. Many others have used modern imaging techniques to indirectly examine the pathoanatomy of the clubfoot. These have not been routinely used because of the need for immobility of the infant during the study. There is no easy way to understand the difficulty in consolidating the information from the different authors perspectives without digging into the original papers to review the methods and results; only then should one formulate his/her own synthesis of the material. This cannot be accomplished by reading texts or review papers.

Examination of the newborn should include a complete musculoskeletal evaluation in addition to the foot exam. The foot should be evaluated for rotation (thigh foot angle), hindfoot posture (varus and equinus), and forefoot posture (adductus and cavus). The resistance of the soft tissue structures is subjectively noted. Despite numerous attempts, there still is not a reliable grading system for the infant clubfoot; an example being the impossibility of grading muscle strength of the peroneals and the tibialis anterior, both of which are stretched beyond physiologic limits.

Casting, or physiotherapy if the resources are available is standard treatment for the newborn. There are many methods of casting, Ponseti's method has been validated by longterm follow-up. The initial measures are designed to correct the forefoot cavus, with particular attention to avoiding pronation of the forefoot. Only then is hindfoot correction attempted. A percutaneous heelcord lengthening is done before 3 months of age for persistent hindfoot contracture. Posterior ankle capsulotomy is avoided, as is any measure that would add to the medial scar formation characteristic of clubfoot. The question of the effects of adding surgical trauma to the pre-existing medial scar in the infant's clubfoot is very unsettled.

A positive family history of clubfoot and a history of maternal smoking during the first three months of pregnancy increase the odds of congenital clubfoot twentyfold. In a recent study of

antenatal clubfoot diagnosis by ultrasound, 32% of 14 infants required no treatment, and were considered false positive findings.

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