50 Years Ago in CORR

Dural and Intradural Compression as a Cause of Clubfoot

NJ Giannestras MD CORR 1953;1:28–32

Calcaneovalgus Foot in the Newborn and Its Relationship to Developmental Flatfoot

CF Ferciot MD CORR 1953;1:22–27

This month's issue of CORR highlights the astonishing advancements in the causes and treatment of clubfoot. It is now well recognized that while most cases of clubfoot are considered "idiopathic," others arise from or are associated with spinal anomalies. This was not always the case although the association with other specific conditions such as cerebral palsy, arthrogryposis, various syndromes—e.g., prune belly, Freeman-Sheldon syndrome, Larsen syndrome—was better recognized given the other obvious abnormalities. Giannestras, in 1953, reported two patients with spinal anomalies associated with clubfoot and stated, "The possibility of intrathecal nerve root compression due to a tumor or a transverse constricting band has not been given much mention in the American literature" [9]. (I was able to find only one earlier paper [13] in the literature in my searches of PubMed and Google Scholar and that paper was in Turkish.) In the two cases Giannestras reported, neither had spontaneous correction of the clubfoot when the spinal cause was

removed surgically. Doctors examining patients with clubfoot now well know to search for other causes, although they remain relatively uncommon.

The studies of Ruth Wynne-Davies documented the substantially higher risk of occurrence of clubfoot when first and even second degree relatives had clubfoot [14–16]; she proposed a dominant inheritance pattern with either reduced penetrance or multifactorial genetic predisposition. The recent search for causative genes has revealed some interesting associations, but no definitive causes [3, 4, 6, 7, 12, 17]. This is perhaps not unexpected since clubfoot appears to occur in utero: fetal ultrasound studies suggest the deformity likely does not occur before 12 weeks of gestational age [1, 2, 11]. Therefore, there is likely some environmental influence in addition to the genetic.

While most patients with clubfoot have equinovarus deformities, a small percentage have other sorts, including calcaneovalgus (so-called, "reverse clubfoot"). Ferciot, in 1953, called attention to this other variety [8],

although he thought it not particularly uncommon, finding it in about 14% of 356 newborns. The deformity is typically "positional" and either corrects spontaneously or with early splinting. Kasser stated, "Surgical correction of calcaneovalgus foot is not required" [10]. Ferciot also recognized these patients' deformities could be corrected by proper splinting, and recommended treatment in the more severe forms. This deformity, however, can be clinically confused with congenital vertical talus, an important distinction given the natural history and treatment of the two entities are different. Many patients with vertical talus eventually undergo surgery, although recently the use of a modified Ponseti method has been used with promising corrections in most patients [5].

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