

CONGENITAL TALEPES EQUINOVARUS

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This is the commonest congenital foot abnormality and according to Lloyd Roberts, remains the most difficult to treat.

The incidence in the Sudan has not been established McKeown and Record (1960) put the incidence in Birmingham at 4.4. per 1,000 births, while Stewart (1951) reported an incidence of 1 per 1,000 births in Polyne-sians.

The function of the foot is to support the body weight in standing and accommodating to variations of surface on which it is placed. The bones of the foot and the various ligaments are important in maintaining its shape. So that any variation in the normal pattern may lead to deformity.

Aetiology:

This is still not clearly known and is the subject of many theories. A mixed genetic and environmental causes has been suggested by Wynne - Davies (1964). From the studies, if one child in the family has the deformity the chances of a second having it is 20 times that in the general population.

Intrauterine environmental causes have been suggested because of similarity of the deformity with that found in weakness of peroneal muscles of neurological origin or contracture of plantar flexor and inverter muscles. Browne (1936) suggested the cause to be due to increased intra uterine fluid tension. If this occur at all the deformity may only be a mild one.

Fripp and Shaw (1967) suggested the theory of arrested development. This is more acceptable since it relates to the embryonic situation where the foot goes through stages of equinus and inversion; and by 10 weeks develops equinus, inversion and metatarsal adduction.

Pathology of the deformity then consists of equinus, adduction and inversion of hind foot and adduction and inversion of the fore foot. In the bones CALCANEUS is inverted, its posterior end elevated approaching medial maleolus: TALUS is plantar flexed, neck medially deviated to 45° relative to the body (normal being 25°) NAVICULAR is rotated towards medial maleolus.

The muscle show at a later stage, wasting of calf muscles, tendo calca-neus become short and medial portion fans out and medially.

Clinically: it is important to perform general examination to exclude conditions that may give similar appearance. These include meningomyelocele, peroneal or dorsi-flexor weakness, arthrogyposis, anterior poliomyelitis. Other congenital deformities such as congenital dislocation of the hip are sometimes associated with it must be excluded.

Attenborough (1966) recognises two varieties which were designed by Hersh (1967) into *EXTRINSIC* which is posterior and is flexible and easily corrected, and *INTRINSIC* which is more rigid and only partially or slightly corrected by manual pressure.

Treatment:

This has passed through many stages, from repeated passive stretching advocated by Hippocrates, to semiforceful correction by Browne (1934). Nowadays the more popular methods of treatment include:—

(1) *Repeated passive manipulation*, first attempting to correct forefoot adduction, then later the inversion and equinus. The position is maintained by either Robert Jones Strapping or Plaster of Paris or Polythene and elastic splints. After 3 months if successful then use Denis Browne's splint. The failure rate can be high.

(2) *Surgical Treatment:* This may be directed to,

(a) *Soft Tissues:* To release tight ligaments, joint capsules and tendons elongation advocated Beckman (1930), Attenborough (1966) Clark (1968).

(b) *Bone:* This may be required in cases of relapse of previous operations, or late cases coming between 5-12 years of age. Dillyn Evans operation (1961) in which the tendo Achillis is lengthened, release of talonavicular joint medially and fusion of calcaneo-cuboid joint, or it may be combined with Dwyer operation (1963) which is a calcaneal ostiotomy.

(c) *Triple Arthrodesis* of the subtalar, talonavicular and calcaneocuboid joints. It is reserved as a last effort since it leaves a rigid foot.

(3) *Transplantation:* of the tibialis anterior or posterior may be performed but this requires careful assessment and is not always successful.

In our local situation we have found it necessary to modify our approach to treatment. Because our patients come from distant places, and are seen rather late, methods requiring repeated manipulations may not work well. Therefore we tend to rely more on Surgical methods of treatment.

REFERENCES

1. Mckeown T and Record R.G. (1960) Malformation in a population observed for five years after birth. In Ciba foundation symposium on congenital malformations, edited by G.E.W. Wolstenholme and C.M.O. Connor P.2 London Churchill.
2. Stewart S.F. (1951) Club foot: its incidence, cause and treatment. *Journal of Bone and Joint Surgery* 33A, 577.
3. Wynne Davies R. (1964a) Family Studies and Causes of Club foot. *Journal of B.J. Surgery* 46B, 445.
4. Browne D (1936) Congenital deformities of mechanical origin. *Proceedings of the Royal society of Medicine.* 29, 1, 409.
5. Browne D. (1934) Talepes Equino-Varvs *Lancet* II, 969.
6. Fripp A.T. and Shaw N.E. (1969) Club foot. Edinburgh and London. E & S livingstone, Ltd.
7. Attenborough G.G. (1966) Severe Congenital Talepes Equinovarus. *Journal of Bone and Joint Surgery* 44B, 31.
8. Hersh A. (1967) The role of Surgery in the treatment of club feet. *J.B.J.S.* 49 A,x,68
9. Evans D (1961) Relapsed Club foot *J.B.J.S.* 43 B, 722.
10. Brockman E.P. (1930) Congenital club foot. Bristol Wright.
11. Clark J.M.P. (1968) Treatment of Club foot. Early detection and management of the unreduced club foot. *Proceedings of the Royal Society of medicine* 61, 779.
12. Dwyer F.C. (1963) The treatment of relapsed club foot by insertion of wedge into calcaneo. *J.B.J.S.* 45 B. 67.
13. Lloyd Roberts G.C. (1971) Orthopaedics in infancy and Childhood, Butterworths