

THE ETIOLOGY OF CONGENITAL CLUBFOOT

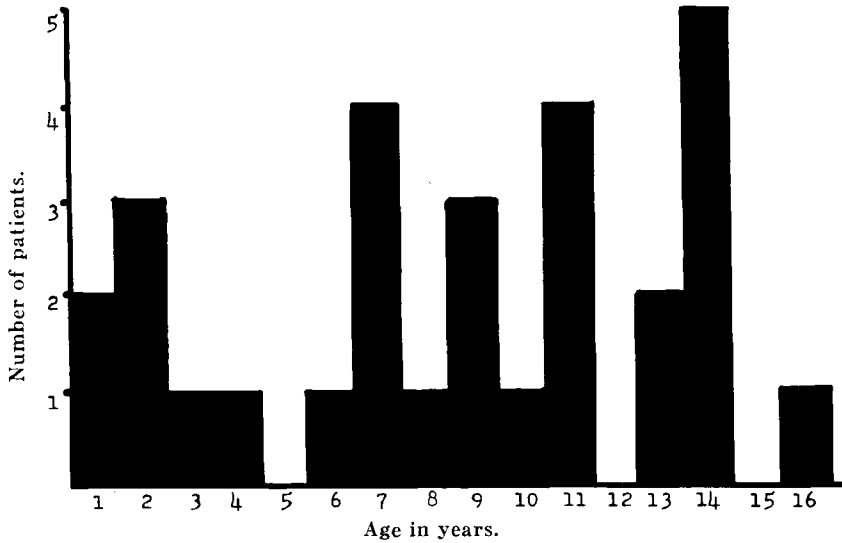
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The etiology of congenital clubfoot is the subject of continuing study at the Orthopaedic Hospital of Los Angeles. This report has to do with that portion of the study in which electrodiagnostic methods (i.e., electromyography and electrical stimulation) were used.

Twenty-nine children who had a congenital clubfoot (talipes equinovarus) without other neurological or muscular disorder were studied. These children were selected at random from those under observation or treatment in the Clubfoot Clinic of the Orthopaedic Hospital, Los Angeles. An attempt was made to have each age and method of treatment represented. With the exception of the children below one year of age, of which there were two, the cases were those of resistant or recurrent clubfoot. The remaining twenty-seven showed some residual deformity, either clinically or by X-ray. All but eight of this group had had one or more surgical procedures for the correction of the deformity. Muscle strength studies were not carried out, either because the child was too young to co-operate, or it was felt that the surgery and/or residual deformity would render the estimates valueless. Electromyographic studies were made of the anterior tibial, peroneus longus and medial head of the gastrocnemius muscles by means of an intramuscular needle electrode and the Meditron Electromyograph (Ref. 2). Although the electrode was inserted through the skin only once, approximately twenty different areas in each muscle were studied by varying the position of the electrode tip. In addition, the position of the electrode in the muscle was checked by electrically stimulating the area to be tested with the needle electrode. A normal electromyogram is dependent upon the presence of an intact neuromuscular unit (i.e., anterior horn cell, peripheral nerve and muscle fibers). Any disruption of this physiological unit will produce fibrillation potentials in muscle. These in-

CHART 1
Age Distribution.



voluntary discharges have been recorded in muscles forty-five years after the onset of a paralysis (Ref. 1). Following the completion of EMG studies, it was thought that some confirmation would be helpful. The twenty-nine patients were contacted, but only twenty-two responded. The same muscles were then tested by means of a constant current galvanic stimulator using skin electrodes. Rheobase and chronaxie studies were carried out on the same muscles previously tested with the electromyograph.

RESULTS

In all twenty-nine patients' normal motor units were noted in the muscles tested. No fibrillation potentials or polyphasic motor units were noted. In each there was an increase in resistance as the needle was inserted into the peroneus longus and medial head of the gastrocnemius, giving one the impression that muscle scarring and fibrosis had taken place. The muscle response to electrical stimulation was brisk and total. When the current was increased above the rheobase the action of the muscles was seen to approximate normal in that the anterior tibial dorsiflexed and inverted the foot, the peroneus longus depressed the first metatarsal head and the gastrocnemius pulled the foot into varus and equinus. Some modification of normal amplitude

of joint motion was the result of fixed deformity or triple arthrodesis. In no case was the rheobase greater than 7.75 milliamperes for the anterior tibial, 8 milliamperes for the peroneus longus and 9.5 for the gastrocnemius (Chart 2).

All muscles tested for the chronaxie responded to a current .1 milli-second in duration. Since this is well within normal limits no attempt was made to secure a response to a current of shorter duration.

CHART 2
Rheobase Values of Muscles Tested in Milliamperes.

Patient	Ant. Tibial	Peron. Longus	Gastroc.
RB	4.5	5	5
PH	7.25	5	6
RC	5	4.75	6.5
LLD	5.25	5	6
LD	5	5.5	7.75
JH	5.25	6.25	6.25
SA	4.75	5	6
MMcD	5.5	8	9.5
HA	4	4	4
ES	4	6.25	7.25
DO	7.75	7.5	7.5
SS	5	5.25	5.5
IC	3.75	4.75	5.5
TR	3.75	4.5	6.5
JS	4.5	6	5.5
SD	4.5	5	5
DC	5	5	6
SMcD	5.5	6.5	5.5
EH	4	5	5
EN	6	7	7.25
SC	5	5	6.5
DC	5.25	5.75	6.25

SUMMARY

No lower motor neurone lesion is demonstrated in these cases of congenital clubfoot.

The muscle scarring of specific groups (peroneal and calf), present by inference, is due to some factor other than a lower motor lesion and should be further investigated.

RESUME

Aucune lésion du neurone moteur inférieur n'a été démontrée dans ces cas de pied-bot congénital.

La cicatrice des muscles du groupe spécifique (péronier et triceps sural), présentée par inférence, est due à d'autres facteurs qu'à une lésion du moteur inférieur. Il convient donc de poursuivre les recherches.

ZUSAMMENFASSUNG

Eine Schädigung von unteren motorischen Neuronen kann in diesen Fällen von angeborenem Klumpfuß nicht nachgewiesen werden.

Die narbige Veränderung bestimmter Muskelgruppen (peroneus und Waden-muskeln), deren Vorhandensein man annehmen muss, ist durch einen anderen Faktor als durch eine untere motorische Schädigung bedingt und sollte weiterhin untersucht werden.

REFERENCES

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