CLUB FOOT

An Anatomical and Experimental Study of Muscle Growth

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The investigator of a human congenital abnormality finds himself faced with two great difficulties: firstly, limited anatomical material for study; and secondly, an experimental field fraught with considerable difficulties. In the case of club foot etiological theories abound—the majority pure conjecture without anatomical support. Nutt (1925) mentioned five etiological hypotheses attributing the deformity to anomalies of the gene, the nerves, the bones, to pressure and to abnormal foetal movements. These can be resolved into theories of either intrinsic or extrinsic etiology. German authors, particularly Mau (1927), subscribed to the "germ" or "gene" defect but admitted that an extrinsic factor might exist. Little (1839) and Adams (1873) both believed that the deformity resulted from a nerve lesion, likening it to that sometimes acquired in the neurological lesions of post-natal life.

Hippocrates and Galen preferred the now popular theory of extrinsic pressure of which Browne (1937) is a protagonist. This theory receives considerable support from the veterinary surgeons, who find club foot in lambs born to mothers suffering from oligo-hydramnios or hydramnios (Roberts 1929, Whittem 1957).

This paper is concerned with the description of anatomical material obtained from infants with club foot encountered during an infant mortality survey in the county of Oxfordshire during the spring of 1958. It is partly interpreted in the light of experiments on the growth of muscles in rabbits, which were performed by other workers (Alder, Crawford and Edwards 1959).

The anatomical material and experimental material resemble each other and provide further evidence that in club foot the primary cause of the deformity is an extrinsic factor maintaining the foot in a deformed position and causing growth deficiencies in the long muscle. We have reviewed the previous anatomical literature in order to arrive at the most critical analysis of our available material, and, having studied the measures necessary to release the deformity in the newborn, we have a plea for early surgical intervention in the management of the fresh case.

REVIEW OF THE LITERATURE

There have been many descriptions of club foot, but comparatively few relate to infants. Fewer still make any reference to either the macroscopic or microscopic findings in the muscles, and of this small number the findings are frequently suspect because no reference has been made to methods of histological preparation, nor is it certain that post-mortem autolysis had not supervened. We consider this a grave objection to the collection and study of pathological material from museums.

After the first year of life adaptive changes produce anatomical changes of their own, and the picture becomes confused. Therefore our search concerned only descriptions of the anatomy of equinovarus in infants.

Little in 1839 examined many specimens himself. He described, in a classic monograph, one infant in whom the tibialis anterior muscle had a "broad insertion" into the medial cuneiform bone. In another the muscles and tendons on the affected side were "small." Little pointed out that the muscular change is variable and occasionally widespread, but found that it varied directly with the extent of the deformity. Little mentioned that Hippocrates...
thought that the muscles maintained the club foot. In Little's monograph Mackeever (1820) is quoted as having dissected three specimens of club foot: he found the tendons and muscles "of usual bulk." Tourtual (1832) had a seven-months foetus with equinovarus in whom the tendon of the tibialis anterior was "twisted and inserted into the first cuneiform."

Adams (1855) described three interesting dissections— one in a newborn infant, one at ten weeks only and one at six months old. He found in one that the gastrocnemius was asymmetrically developed, the lateral head being "small and fatty." The tendo calcaneus was "large and tight," the tibialis anterior and posterior "very tense and possibly hypertrophied." All three muscle bellies were shortened. The extensor digitorum longus was very "small," and Adams described it as being "microscopically degenerate." The peroneal tendons showed adaptive displacement. In a second of Adams's cases the muscles were said to be normal in structure but to show several tendinous abnormalities.

More descriptions of isolated dissections of club feet followed in the German literature (Hueter 1863b, Kocher 1878): emphasis then, and until comparatively recently, was on the bony changes in club foot.

In 1884 Parker and Shattock reported a series of six dissections in infants, three of whom had talipes equinovarus and the others talipes calcaneus and varus. In only one specimen is there a reference to the muscles, which were described as histologically normal.

Bissell (1888) described the equinovarus feet of a male infant who had died of pneumonia. This child had had elongation of the tendo calcaneus at four months. The calf muscle was said to be "small" and its tendon inserted "higher up" than normal. The tendons of the tibialis anterior and posterior were shortened and displaced inwards. The muscle bellies of the leg were examined histologically and found normal. Like many other authors Bissell went on to describe in detail the directional changes in the angulation and facets of the bones of the feet.

Burrell (1893) examined specimens in which there was apparently no difference between the muscle bellies of the normal and abnormal side, and Nichols (1897) in another dissection noted only directional changes and shortening in the tendons of tibialis anterior, posterior and gastrocnemius.

In 1920 Pfrang published a description of a club foot which he found in the dissection room at Würzburg. The rarity of these dissections is well illustrated by the details given by the author of his encounter with this specimen, which was already half dissected. The bones received great attention. Of the muscles the description covers directional changes in the tendons. The tendon of the flexor hallucis longus was pinched between the tibia and calcaneum and partly divided. The peroneus longus tendon was embedded in the lateral surface of the calcaneum—it was said to be "enormous." The tendo calcaneus was "weak" and the belly of the extensor hallucis longus was "atrophied."

Hackenbroch (1923) visited a number of museums and collected eight specimens of club foot associated with other congenital abnormalities including spina bifida. He referred to the muscles in only one: the tibialis anterior was "very obvious" and the other muscles "thin."

Böhm (1929), arguing that club foot resembles in position an embryonic foot in the second month, referred to two dissections of infants by von Meyer. Here the muscles on the affected side were "small" but normal histologically.

Dittrich (1930) described the dissection of an infant with spina bifida and bilateral club foot. He concluded from this one dissection, which was performed months after death, that the position and form of the feet were determined by the soft tissues. The muscle histology was given in detail: the fibres of the tibialis anterior were wavy in outline, there was an increase of sarclemma nuclei in the tibialis posterior, and the fibres of the gastrocnemius were narrow, faintly striated with some loss of sarcoplasm.

One of the most important studies on the anatomy of congenital deformities was that of Middleton (1932, 1934), who collected pathological material illustrating congenital tibial
kyphosis and myodystrophia foetalis (arthrogryposis multiplex congenita). One of his specimens had an equinus and metatarsus varus deformity. Middleton stated that the "sural" calf muscles of this specimen were pale and the muscle fibres irregular but otherwise fully differentiated. Middleton evidently suspected the presence of immature muscle fibres in his sections but none was definitely seen.

Mau (1938) gave much thought to a study of three foetuses with club foot and other congenital abnormalities. Macroscopically the muscles appeared to be normal. Histologically, however, apparently all the muscles showed varying degrees of scattered abnormality. There was a loss of striation. The diameter of the muscle fibres varied, and some were coiled. There were abnormalities of staining. Mau labelled these changes "myelodysplasia." He did not actually mention which change was found in which muscle, but his contribution shows a section of the extensor digitorum longus muscle showing gross degenerative change: methods of preparation were not described. In this study, the most detailed and relevant yet appearing in the literature, Mau concluded that as each muscle in his specimen showed changes it was impossible to "attribute the deformity to any particular muscle." He therefore assumed that the club foot was caused by an endogenous disorder.

In 1950 Bechtol and Mossman recorded the anatomy of a four-months foetus and of a three-months foetus, each with equinovarus deformity. They suspected that the older foetus had arthrogryposis. The muscles of the affected foot were "deficient in volume" and varied histologically in their degree of maturity, though all the fibres of any single muscle were at the same stage of development. No histological abnormality of nerves or end plates was seen. The authors concluded that the more mature muscles exerted greater tension (unspecified) and pulled the foot into a deformed position.

In their second specimen, microscopic examination of the muscles showed the calf muscles to be abnormal, with dying muscle cells and faint striation. "Abnormal" straight fibres were seen and "normal" coiled fibres. The muscles involved were the gastrocnemius, soleus, tibialis posterior, flexor hallucis longus and flexor digitorum longus.

In 1951 Stewart presented his findings in the muscles of twenty patients operated upon for club foot and in one necropsy on an affected infant. Stewart frequently found an abnormal insertion of the tendo calcaneus into the medial side of calcaneum. He described abnormal insertions of the peroneus brevis into the dorsum of the lateral metatarsals—a "negative deforming force" producing adduction of the metatarsus—and of the peroneus longus to the lateral metatarsals, preventing normal postural depression of the first metatarsal. He found that the tendon of the tibialis anterior in his foetal specimen and certain operative cases was inserted abnormally far forward. He even noted that the plantar fascia was once attached dorsally. Stewart attributed club foot to insertional peculiarities of postural muscles.

A further American contribution came from Flinchum (1953), who dissected a six-and-a-half-months premature infant with right club foot. He found that the tendo calcaneus on the affected side was longer than on the normal and was inserted into the medial side of the calcaneum. The anterior tibial tendon on the affected side was longer than on the unaffected side and had a normal insertion. The posterior tibial muscles appeared similar on both sides. The "peroneal muscle mass" on the affected side was only one half the size of the unaffected side. The overall muscle lengths were not determined. A good deal of attention was directed towards the shape of the foot bones in this specimen.

Adams (1958) recorded that in 1952 Vander Eeken, Pearson and he had an opportunity to examine two separate specimens of club foot in the infant. One of these had a deficiency of anterior horn cells in the lumbo-sacral cord and a corresponding atrophy of certain muscle groups. No conclusions could be reached from the other, whose deforming muscles were apparently free from signs of atrophy and all the muscles were histologically normal.

Comment—In forty-two descriptions of club foot in the foetus and infant collected from the literature we can detect no constant macroscopic or microscopic abnormal finding in the
muscles of the deformed foot, nor, apart from directional changes, is there a regular pattern of abnormality in the case of the tendons and their insertions. No investigator, though possibly aware of the significance of the reduction in length and girth of the individual long muscle bellies, has made an adequate and acceptable histological study with the material available. The precise etiology of the condition has been further confused.

### Table I

**Dimensions of Principal Long Muscles and Tendons**

<table>
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<tr>
<th>Specimen</th>
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<th>Abnormal side</th>
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<td>Length (cm.)</td>
<td>Diameter (cm.)</td>
<td>Length (cm.)</td>
<td>Diameter (cm.)</td>
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<td>3.5</td>
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<td>2.3</td>
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<td>2.5</td>
<td>2.5</td>
<td>2.2</td>
</tr>
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<td>6.8</td>
<td>5.6</td>
<td>4.7</td>
</tr>
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<td>1.0</td>
</tr>
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<td>5.1</td>
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<td>5.5</td>
</tr>
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<td>8.5</td>
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<td>Total</td>
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</table>

**ANATOMICAL MATERIAL**

The material collected consists of eight specimens of which six had talipes equinovarus, one had talipes calcaneo-valgus and one had metatarsus varus. One specimen, with calcaneo-valgus deformity, was from a premature infant. All the others were from infants at term.

For purposes of comparison we have also examined histologically the muscles of six further specimens as follows: one case of equinovarus in an older child, one case of a child who had never walked and had bilateral pes equinovarus, a normal sixteen-weeks foetus, a normal eighteen-weeks foetus, and an infant with normal feet who had died at birth from respiratory insufficiency.

Each specimen was obtained within one to three days of death. Two specimens were not fully dissected because mutilation of the cadaver was forbidden. These specimens were examined unfixed in the necropsy room. The remainder were fixed in 10 per cent formol saline after disarticulation through the knee and were dissected some days later. After dissection specimens of the muscles were removed and immersed in 10 per cent formol saline before sectioning and staining.
FINDINGS

Specimen 1—A stillborn male infant with a cranial defect and right talipes equinovarus. Muscles on the affected side were dissected out and photographed together with those of the normal side (Fig. 1). In the case of each principal long muscle the length and the diameter at its widest point were measured. The results are shown in Table I.

![Fig. 1](image)

Muscles from a right club foot compared with those of the normal left side.

With the exception of the gastrocnemius-soleus, where the difference was considerable, the total length of each muscle and tendon was very little shorter on the abnormal than on the normal side. Nevertheless the muscle belly was shorter and narrower; thus the abnormal tendon was relatively longer than its normal counterpart. The significance of this finding will be referred to later.
The anterior ligament of the ankle was very thickened, particularly between the malleoli and the neck of the talus. The deltoid ligament was, however, a mere thin sheet.

Of the bones, the tibia showed torsion in its lower half; the talus was in equinus and its neck was angled medially. The navicular bone lay on the medial side of the head of the talus, where it had a separate facet. Its tuberosity was very close to the medial malleolus. The tibial articular surface of the talus was normal, though the fibular facet was enlarged. The articular facet on the lower end of the fibula was placed forward, presumably from contact with a persistently equinus talus.

The calcaneum was small and folded with a concavity opening medially; it was inverted at the subtalar joint and its main talar facet was moulded into a saddle shape. The calcaneocuboid and metatarso-cuboidal joints were normal. The cuneiforms and the cuboid bone itself were normal, but the entire metatarsus was adducted.

**Specimen 2**—A stillborn infant with rachischisis and talipes equinovarus on both sides.

*Right foot*—Each long muscle was dissected out with its tendon. The muscle bellies appeared normal but, of course, could not be compared for size with a normal side. The tendon of the gastrocnemius and of the tibialis anterior were narrow. The tendons of the gastrocnemius, soleus, tibialis anterior and posterior and of the peronei were normally inserted.

*Left foot*—The muscles appeared normal but on manipulation high tension in those of the calf was obvious. The plantar fascia seemed short but the direction of its slips was normal. The tendon of the tibialis anterior curved gently beneath the extensor retinaculum to a normal insertion. The flexor digitorum longus tendon seemed to be constricted by the flexor retinaculum, above and below which it bulged. This tendon was of a peculiar hard consistency like cartilage. No other tendon in this specimen showed this consistency. The tendons of tibialis anterior and posterior swung gently to a normal insertion.
The findings at this dissection suggested that the tibialis anterior and posterior muscles and tendons had taken up a position in relation to a pre-existing deformity rather than, by angling around their respective retinacula, brought about a supination of the foot. We were satisfied of this by an experiment, simply repeated, whereby inversion can readily be produced in the foot of an infant cadaver by pulling either on the tibialis posterior or, when the heel is in equinus, on the tibialis anterior. The appearance of the tibial muscles then in no way resembles that seen in congenital equinovarus.

*The bones (left and right foot)*—The bony changes in both the left and right foot resembled those found in Specimen 1. There was, in addition, torsion of both tibiae and there was a well marked groove for the tendon of tibialis anterior across the lower end of the left tibia. Also in these two specimens the calcaneum was so deformed and the midtarsal joint so supinated that the calcaneo-cuboid joint was directed medially instead of antero-posteriorly: this directional change assumed considerable importance in attempts to correct the whole deformity (see below). In each foot the adducted forefoot also lay in moderate cavus. This infant had an overriding fifth left toe (Figs. 2 and 3).

*Specimen 3*—A stillborn infant showing multiple congenital abnormalities, talipes equinovarus on the right and a varus left forefoot.

*Right foot*—Compared with the left side the bellies of the long muscles on the right were shorter and thinner, particularly the gastrocnemius, soleus, tibialis anterior, tibialis posterior and the peronei. These muscles and their tendons were measured and tabulated with those of the earlier specimen (Table I). It will be seen in this specimen that the total length of the tibialis muscles and tendons, as well as that of the gastrocnemius and soleus, was shorter on the abnormal than on the normal side. Even the abnormal peroneus longus showed shortening.

The tendon of the tibialis posterior ran vertically downwards, grooving the posterior aspect of the medial malleolus deeply, and ended in the deep plantar tissue slightly posterior to the navicular bone. The tibialis anterior tendon swung in a gentle curve across the lower tibial shaft and beneath the extensor retinaculum to its insertion. Again there was no suggestion that this was a deforming muscle.

*Ligaments and bones*—There was equinus at the ankle joint and inversion at the subtalar and midtarsal joints. The anterior capsule of the ankle joint and its medial and lateral ligaments had undergone major directional changes. The deltoid ligament was short, tough, contracted and folded in under the medial malleolus.

The neck of the talus was angled medially and the navicular bone articulated with a separate facet on the medial surface of its head.

The forefoot was adducted at the transverse tarsal joint. There was clawing of the second, third and fourth toes and underriding of the fifth toe.

There was medial rotation of the lower tibial shaft which showed a well marked impression for the tibialis anterior tendon.

*Specimen 4*—A stillborn foetus showing a pronounced calcaneo-valgus deformity. No other skeletal abnormalities were seen.

In this case a study of the vascular anatomy of the affected limb was carried out by injecting 100 cubic centimetres of a fine barium suspension (Micropaque) and 10 per cent Berlin blue solution (4 : 1) into the common iliac artery. The limb was then radiographed using a beryllium x-ray tube. This study showed that the position of the talus and the main vascular pathways in the limb were normal.

The condition of the muscles on the affected side was the reverse of that seen in the specimens with equinovarus deformity—that is, the gastrocnemius, soleus, long flexors and tibialis posterior had large muscle bellies and well formed tendons, whereas the extensor digitorum longus was reduced in bulk and had a narrow tendon. The insertions of the long muscles were normal. The talus was dorsiflexed, the tarsus was pronated at the midtarsal joint and the longitudinal arch was flattened.

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Specimen 5—A male infant aged three days. Cause of death unknown. The child had ear pits, irregular teeth, hypoplastic kidneys and right talipes equinovarus.

Right foot—The dissection was performed in the necropsy room. The gastrocnemius and soleus muscle bellies on the affected side were short and thin, and their tendon was narrow. The tibialis anterior and posterior were similarly affected. These muscle bellies, however, as in all previous specimens, looked a normal colour. The tendo calcaneus was normally inserted.

Bones—The ankle was in equinus, there was inversion at the subtalar joint and marked supination at the midtarsal joint. The whole forefoot was adducted at the transverse tarsal joint.

Specimen 6—A stillborn male infant with partial rachischisis and left talipes equinovarus.

Left foot—We noted abnormalities like those in Specimen 5. Having attempted the corrective manoeuvres described later we removed sections of muscle for histology.

Comment—The severity of the deformity obviously varied from case to case. In the severe cases the abnormal musculature was noted throughout the long muscles (compare Specimen 1 and Specimen 3). In talipes equinovarus the principal muscles affected were the gastrocnemius and soleus, the tibialis posterior and the tibialis anterior, in that order.

None of the insertional abnormalities previously described (Stewart 1951) were present in these infant specimens.
HISTOLOGICAL EXAMINATIONS

Longitudinal sections were taken from the gastrocnemius-soleus muscles, the peronei and the tibiales anterior and posterior of all specimens, from the foot of the "normal" stillborn infant, from the sixteen-weeks foetus and from the tibialis posterior of the older child with club foot. All were stained with phosphotungstic acid and haematoxylin and eosin. Sections were also prepared from the remaining long muscles of certain of the specimens only. From the left leg of specimen 2 the posterior tibial, anterior tibial and musculo-cutaneous nerves were extracted, stained, examined and photographed. In another (Specimen 5) nerve endings and end plates were sought in the muscles, using luxol fast blue stain.

![Fig. 6](image)

Muscle fibres of tibialis posterior taken from a sixteen-weeks normal human foetus.

In comparable specimens—Specimens 1, 3 and 5, where a normal and abnormal side could be compared, and also in the case of the muscles of the foetus, "normal" foot and calcaneo-valgus foot—the slides of the muscles so prepared were photographed to enable accurate comparison and measurement (Figs. 4 to 6).

FINDINGS

In each microscopic section we searched for four changes: 1) irregularities in fibre diameter; 2) nuclear changes; 3) irregularities in staining; and 4) absence of cross striations. Muscle diameter measurement was calculated against a scale on each photomicrograph. The results are set out in Table II. When two or less of these changes were present we considered the section to show mild abnormality; when more than two, moderate abnormality. It will be seen that the distribution of the abnormal muscles is irregular throughout the entire material and even amongst each specimen.
TABLE II
STATE OF THE MUSCLES EXAMINED

<table>
<thead>
<tr>
<th>MUSCLES</th>
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<tr>
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In retrospect we interpret most of our abnormal sections as showing merely post-mortem autolysis—there was a loss of striae and nuclear pyknosis, and fibres having a large diameter were affected by post-mortem swelling. A study of the histology of Specimen 2—a bilateral club foot (not included in the Table) further supported this view, for on one side, fixed later than the other, there were moderate changes through each of the long muscles of the calf.

EXPERIMENTAL STUDIES ON MUSCLE GROWTH

Immobilisation of the foot of the young rabbit (two and a half to four and a half weeks old) in dorsiflexion for four to twenty-five weeks by the method illustrated in Figure 7 decreases the longitudinal growth of the muscle belly of the tibialis anterior, which thus remains shorter than normal in the adult at the end of the experiment (Alder, Crawford and Edwards 1959) (Fig. 8). In consequence, the "resting tension," which is exerted by the experimental muscle when not contracting, develops at a considerably shorter length of the belly than does tension of similar magnitude in the normal muscle. In fact resting tension becomes appreciable in the experimental belly as it is stretched beyond the length that it occupied when the foot was immobilised in dorsiflexion; whereas in the normal limb similar tension is not developed until the belly attains the length it occupies with the foot fully plantarflexed. At the end of the experiment the freed foot can be only slightly plantarflexed because of this premature development of resting tension in the muscles. The girth of the muscle is little affected by its prolonged immobilisation and it exerts almost as great an isometric tetanic tension as the normal. Histological examination fails to reveal any significant differences between the structure of the experimental and control muscles.
Crawford (personal communication) has immobilised one foot of each of a series of adult rabbits in dorsiflexion. This again causes resting tension to develop at a shorter length than normal, so that there is increased resistance to full plantarflexion of the foot. However, the effect is less than that produced in the young rabbit, nor is there any alteration in the length of the belly of tibialis anterior when the experiments are performed after the cessation of growth.

In both the adult and young rabbits the premature development of resting tension in the experimental tibialis anterior must be a consequence of its immobilisation with the belly held at the short length which obtains when the foot is dorsiflexed.

![Diagram to show method of fixation of foot of infant rabbit.](image)

**CORRECTABILITY OF DEFORMITY**

Before dissection of each human specimen a simple trial of correction was attempted, the tibial shaft being held firmly with one hand and the affected foot manipulated with the other. With the exception of the left foot of Specimen 2, correction was obtainable but the force required was considerable and when sustained seemed liable to tear the soft parts of the foot.

**Effect of tenotomy**—Simple tenotomy of the tendon calcaneus greatly facilitated correction of two of the deformities (Specimens 3 and 5) (Figs. 9 and 10). Additional tenotomy of the tibialis anterior was required to correct another (Specimen 2, right foot) and of both tibialis anterior and tibialis posterior in a fourth (Specimen 1). The calcaneo-valgus deformity was more easily corrected after division of the extensor digitorum longus tendons, but the deformity in Specimen 2 (left foot) could not be fully corrected even after division of the tendon calcaneus and of both the tibialis anterior and tibialis posterior tendons. This specimen was later dissected down to the bones, whereupon it was found that there existed a bony block to correction, consisting of the inwardsly inclined cuboid facet of the calcaneum (Fig. 11). Resection of this facet permitted full correction.

We noted that, after correction, the small bones of the feet, particularly the navicular bone, lay in a relatively normal anatomical arrangement with one another, even in the left foot of Specimen 2, although here, as previously noted, the alignment of the calcaneo-cuboid joint was already permanently altered.

**Comment**—In this anatomical study we noted that the ease of correction of deformed feet varied. This is in keeping with clinical experience.

We were impressed with the ease of correction afforded by tenotomy of the tendon calcaneus and of the tibiales. In the most resistant case full correction could be obtained only by a bony procedure on the calcaneo-cuboid joint.

**DISCUSSION**

According to Böhm (1929) a club foot so resembles a foetal foot during the second month of pregnancy that the deformity may be merely the result of an arrest of development at an early stage. This is a contribution to the “intrinsic” theory. However, a glance at foetuses obtained by therapeutic abortion shows that an early foetal foot little resembles a...
FIG. 8
Tibialis anterior muscle of rabbit subjected to fixation during growth, compared with corresponding muscle from the normal leg. (Crawford, Personal communication)
club foot. Some further etiological factor is required. We were enabled by our own anatomical and histological studies to rule out the neural theory suggested by Adams (1855), Little (1839) and Adams (1958), for a neural change must be reflected in the musculature. There is experimental evidence that muscle fibres may evolve in the complete absence of any stimulus from the central nervous system (Adams, Denny-Brown and Pearson 1953). That club foot is the result even of a transitory, prenatal neuropathy seems unlikely when the number of types of club foot are considered. It remains therefore to search for a cause of club foot in the musculature.

We have considered carefully the views of Middleton (1932) and of Bechtol and Mossman (1950) that in club foot certain muscle groups may be immature.

Human foetal muscles at the tenth week show central nuclei and marginal striations. The nuclei later move to the normal peripheral position and the striations increase. At the fifteenth week, both types of fibre are seen. Even in the infant, mature and small, or “daughter,” fibres may be seen normally. The presence of foetal fibres (Bechtol and Mossman) must therefore be interpreted with reserve.

The changes in the muscle fibres described by Dittrich (1930) and suspected by Adams (1958) were sought but could not be identified with any certainty. Stewart’s emphasis on abnormalities of insertion receives no support from our anatomical studies. In previous reports of anatomical dissections there has been a lack of comparable studies of the normal.
and one cannot be satisfied, in the microscopic studies, that the material has been uniformly and freshly fixed and comparably stained. This difficulty was encountered in our own material, which was therefore reviewed very critically. The shape and length of muscle fibres as seen microscopically are particularly open to misinterpretation. We found that by teasing out our own sections we could easily produce coiled fibres. Assuming, therefore, that the muscle in club foot is normal, whether in the monster, in the severely deformed or in the mildly deformed foot, any theory that ascribes the deformity to neural or muscular disease is invalid.

The muscles in the limb of a young rabbit subjected to immobilisation fail to grow to the full extent. Histologically the muscle is normal. Resting tension, however, develops early when the muscle is stretched. This inhibits correction to normal when immobilisation ceases. These shortened and small muscles never regain full development although they can be forcibly stretched.

These short muscle bellies resemble those found in club foot in the human infant, and evidence has been submitted which suggests that the deformity is maintained by the same factors of increased resting tension in certain muscle groups. Ill developed calf muscles are found commonly in late cases of club foot, and the long-term anatomical and functional results of treatment are not uniformly satisfactory. These late findings will be the subject of a separate communication, but it must be stated here that the incidence of residual deformity coincided with delay in obtaining early full correction.

On anatomical grounds, and on the basis of our findings, tenotomy of the tendo calcaneus and, if necessary, of both the long tibial tendons, should be recommended in early infancy. This would enable the foot to be splinted in the over-corrected position at the earliest opportunity.

We are fully aware that this concept is a very old one, but we think in the light of this work that the aversion from it by present-day surgeons is unfounded. Most orthopaedic
surgeons would agree that division of the long tendons in the foot of a young infant can be performed with very little risk of complications. There is as yet no general agreement about the best time for such tenotomy. Our late results indicate that existing methods of treatment require revision, and anatomical evidence warrants the operation.

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