TALIPES CAVUS

BY

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This deformity is one in which the plantar arch is increased in depth by the approximation of the heads of the metatarsal bones to the posterior tuberosity of the calcaneus. Under the head of talipes cavus there have been described several distinct pathological conditions having in common a deepening of the plantar arch. Thus in talipes equinus, equinovarus, and calcaneus the plantar arch may be much increased in depth. An increase in the depth of the arch may occur independently of these affections, both as a congenital and as an acquired deformity.

There are two classes to be considered: (1) congenital talipes cavus, and (2) acquired talipes cavus.

Congenital Talipes Cavus

An increase in the depth of the arch with corresponding high instep is met with as a congenital affection which is in most cases hereditary. It was attributed by Redard to a primitive malformation of the ligaments and muscles of the plantar surface. In slight cases no inconvenience is experienced and walking is quite easy. At times, however, pain is felt in the instep or about the heads of the metatarsal bones, especially after much exercise or long standing; but it is possible that this pain and discomfort is not so much dependent upon the increase in the arch of the foot as it is upon the tendency of the talus and calcaneus to be tilted medially as the result of long standing or improper weight-bearing when walking—that is, the pains complained of are those of the beginning of flat-foot.

In the congenital class I am including the cases of a short tendo calcaneus, as so many of these develop a cavus later on in life. It is not recognized that a diminution of dorsal flexion is very common. Generalizations are dangerous, but I have an impression that apart from wearers of high heels about 20% of the population have less than 75 degrees of dorsal flexion. Another large section have good dorsal flexion but have thickened superficial plantar fascia somewhat akin to the thickened palmar fascia so often seen in later life without any evidence of Dupuytren's contraction.

Unless a special examination be made with the leg fully extended it is quite easy to miss slight cases of diminution of dorsal flexion, as the foot looks normal. As a rule nothing is noted until about 7 years of age, when either an awkward gait or valgus ankle often appears. The child also does not run as well as its mates, and at times gets easily tired. Later on a talipes cavus may develop, with clawing of toes. Callosities appear on the sole of the foot, especially about the head of the first or the fifth metatarsal.

Owing to want of freedom in walking with the foot straight the toes are turned out and the talus and calcaneus are tilted medially. I consider a short tendo calcaneus is the commonest cause of a weak or flat foot, and practically the only cause of valgus ankles and genu valgum. It is also the determining cause of many sprains of ankles and knees. From the point of view of industrial compensation this is important, as rupture of ligaments of ankles and knees is very common in those employed on railways and tramways; and these anatomical deficiencies should be looked for and eliminated either by rejection or by operation. Any gross pathological condition may take years to develop, and may be delayed till the end of adolescence or later. Besides the onset of flat-foot the commonest disabilities are swelling around ankles and fatigue when walking uphill or upstairs.

In young children and in the milder types of the deformity it is sufficient to try to obtain lengthening of the tendo calcaneus and to stretch the shortened plantar muscles and fascia. I have in this way overcome the diminution of dorsal flexion as late as 21 years of age.

In patients who are old enough instruct them to bend their knees over the foot, which is placed on a block of wood one to one and a half inches high. The heel must be kept on the ground. When the diminution of dorsal flexion approaches the right angle the exercises should be done without the block of wood. The foot is placed two feet in advance of the other. With younger patients the foot must be dorsiflexed by someone pressing it with knee flexed from below the heads of the metatarsal bones. This helps to stretch the shortened plantar muscles and ligaments as well as to lengthen the tendo calcaneus.

Except in adults it is rarely necessary to lengthen the tendo calcaneus by operation. I would here note that the safest and best method of lengthening the tendon is to cut it from its insertion to the calcaneus by passing a tenotome beneath the skin and cutting downwards upon the fan-like expansion of the tendon. This method seems to avoid entirely the production of a weak tendon or wasting of the gastrocnemius.

The proportion of cases of diminution of dorsal flexion that develop a talipes cavus is not easy to determine. We may see a very definite equinus without any cavus or a slight diminution of dorsal flexion with a definite cavus, or even full dorsal flexion with a gross cavus. Again, while many of these may develop into grosser deformities as life proceeds, others will remain *in statu quo*. Both diminution of dorsal flexion and a tendency to talipes cavus are certainly hereditary in 75% of cases, if not more. As slight cases of talipes cavus are seldom seen by the surgeon it is difficult to estimate how far treatment by stretching would be successful.

As regards its pathology, we have not advanced upon Redard, and we must for the present assume with him that certain muscles are short and prevent a proper development of the foot. Shortness of the hamstrings and eye muscles is frequently noted, so we are not putting forward an extravagant theory regarding the formation of talipes cavus.

Acquired Talipes Cavus

Though clawing of toes is not always present in congenital talipes cavus it is invariably so in acquired cases unless either the dorsal flexors or the plantar flexors are severely paralysed. Most cases of acquired talipes cavus occur after an attack of anterior poliomyelitis in which the dorsal flexors are more involved than the plantar flexors. In consequence the fore part of the foot is plantar-flexed at the medial tarsal joint. Upon recovery of the dorsal flexors the toes become clawed. This condition of clawing may be explained as follows. Owing to the plantar flexion of the foot from paresis of the dorsal flexors the points of attachment of the anterior muscles are abnormally separated. The anterior muscles themselves, having nearly recovered their original strength, try to overcome this abnormal separation; but, their proximal attachment to the fibula being more or less a fixed point while their distal attachment to the toes is to a great extent a movable one, in endeavouring to approximate their attachments they draw the toes nearer to the fibula. Thus contraction of the anterior muscles causes dorsiflexion of the first phalanx; but as the plantar flexor muscles at the same time act strongly and are attached to the two distal phalanges they hold the latter in a position of plantar flexion.

This explanation is in accordance with what occurs in a dissected foot, in which the toes will assume a clawed condition if the long flexor and extensor tendons are strongly pulled in the direction of their proximal attachments. It is further borne out by the fact that severe cases of clawed toes are generally met with in the slighter cases of equinus—that is to say, in cases of rectangular talipes in which the anterior muscles have almost entirely recovered their normal strength and tone, and therefore are acting strongly. In moderately severe cases clawing is not often seen except in the great toe, and we may explain the frequency of clawing of this digit in severe forms of equinus by the fact of its very powerful dorsal muscle. We see this when a child's foot is recovering from paresis of the anterior muscles: the great toe is then the first digit that assumes a position of clawing and is always more clawed than any of the others.

This clawing of the toes is attributed by some authors to paralysis of the interossei; by others, to spasmodic contraction of the anterior muscles. With regard to the clawing being due to the paralysis of the interossei, these muscles are difficult to put into action by galvanism or faradism even under favourable conditions. In talipes equinus they are placed at a great disadvantage on account of the contraction and adaptive shortening of their opponents, and are therefore much more difficult to stimulate by electricity. Hence, as might be expected, they are often reported to be paralysed when it is probable that they are not thus affected. Moreover, contraction of the dorsal interossei is seen to be definitely present in a large number of clawed toes on stimulating the motor points, and this contraction will be found to increase in strength as the toes are made to assume their ordinary position after tenotomy and manipulation of the toes.

When their contraction is feeble no reaction of degeneration can be demonstrated, and no evidence of the wasting so commonly seen and so well marked in the hand is to be found in the foot in these cases. Except, then, that their contraction is feeble and in some instances is not obtained, no data are available to uphold the theory that the condition of clawing is due to paralysis of the interossei.

Against the paralysis theory the following facts may be of use: (1) That in some cases the dorsal interossei act well. (2) That in many cases we at first obtain slight contraction of some of the dorsal interossei and then increased contraction upon correction of the deformity by tenotomy, which thus removes a large amount of resistance opposed to their action, showing clearly that the weakened contraction or absence of contraction is due to such opposing force. (3) That the interossei are supplied by the same nerve as the posterior muscles and therefore would be more likely to suffer with them than with the anterior muscles: it would be extremely improbable that the interossei alone of all the muscles supplied by the posterior tibial would be so often picked out in conjunction with the anterior muscles. (4) In the dissected specimen of clawed toes No. 3514A, St. Bart's Museum, the interossei are healthy and hypertrophied. I would further point out the feebleness of contraction of the interossei in comparison with the long muscles of the foot, which makes it difficult to believe that, conversely, it is the normal action of the interossei that prevents the formation of clawed toes in the undeformed foot.

Operative Measures

In severe cases in which the deformity is causing disability operation must be undertaken. As the methods in vogue forty-five years ago were quite unsatisfactory I devised, in 1893, an open incision for dealing with contracted plantar muscles and ligaments. Mr. Walsham (Walsham and Kent Hughes, 1895) refers to it, but did not

give an account of the operation. I mention this in order to establish the fact that I have not copied from Steindler (1920). In 1937 I showed a case at the Paediatric Society, Melbourne, of a man who at the age of 7 had been operated upon by me for cavus in 1902.

In this operation I want you to look upon the plantar fascia as a solid mass occupying the under surface of the foot, through which the tendons, nerves, and blood vessels penetrate. Dividing the superficial layer of this mass is of little benefit, and dividing all structures attached to the calcaneus does not deal adequately with the contracted solid central mass, which has numerous strong attachments to the capsule and ligaments of the joints on the under surface of the bony framework of the foot. The superficial plantar fascia is a broad band which divides into a large medial and smaller lateral portion just anterior to the calcaneus.

First make a curved incision from the first metatarsophalangeal joint along the medial border of the foot to the head of the talus, then descend backwards to the anterior edge of the calcaneus midway between the tip of the malleolus and the sole of the foot. Dissect up this flap to expose the medial edge of the superficial part of the plantar fascia, taking the subcutaneous tissue with the skin. Cut through the fascia, exposing the flexor brevis hallucis as it arises from the deep surface of the fascia, and remove it freely with an elevator, pushing the muscle anteriorly and thus exposing the intermuscular septa as they pass down to the deeper tissues. Incise these and free the outer strip of superficial plantar fascia from its deep attachments. Divide both portions of the superficial plantar fascia near the anterior edge of the calcaneus. Now proceed to lift the abductor hallucis from its attachment to the first metatarsal bone and free it from its origin from the fascia posteriorly. With a couple of broad elevators, somewhat curved, work your way across the foot, keeping close to the bony framework and dividing the thickened bands of the fascia attached to the structures on the plantar surface of the bony framework, taking care not to injure the peroneus longus in its fibrous sheath as it passes round the lateral border to its medial attachment.

The intermuscular septa of the plantar fascia will be found much hypertrophied and contracted. Passing from the fore part of the foot and proceeding backwards, divide the long and short plantar ligaments. The only haemorrhage likely to give trouble is from an artery near the head of the fifth metatarsal. You have now freed the plantar fascia superficially and deeply from its attachments and have a medial portion containing the vessels, nerves, and tendons. Except for stripping the flexor brevis from its fascial origin and the abductor hallucis from its bony and fascial attachments, you have not injured any muscles.

If you are not able to unfold the foot and abolish the cavus you have three other situations to attack:

1. Prolong the original skin incision backwards along the level of the plantar surface of the calcaneus. With a blunt chisel or elevator lift the periosteum off the medial surface of the calcaneus, carrying the skin and soft structures towards the malleolus. This will loosen the tight skin in this neighbourhood which is often an obstacle to restoration. Next pass the chisel along the anterior border of the calcaneus, dividing everything except skin, and bare the bone.

2. Another position to be attacked if necessary is the contracted capsule of the talo-navicular, navicular-cuneiform, and cuneiform-metatarsal joints, which must be opened up freely. You can now determine how much lengthening, if any, of the tendo calcaneus is required to obtain full dorsiflexion.

3. I prefer to divide the tendo calcaneus at its insertion to the tuberosity of the calcaneus, as this avoids any possibility of

a weak tendon. Insert the knife subcutaneously between the skin and the fan-like expansion of the tendinous insertion, and cut deeply to the bone.

If the toes are severely clawed the dorsal tendons, long and short, are divided subcutaneously, and if necessary the long plantar tendon near its insertion. It is seldom necessary to divide the capsule of the joints on the dorsal surface as they usually give on being stretched. I get the patient out of bed the first day after operation, and, according to his condition, sit him up on a chair for a few minutes to a few hours. On the third day I dress the foot again, and allow the patient to take more or less freedom, depending on the appearance of the wound. If possible I like my patients to bear most of their weight on the foot by the end of the first week: it all rests upon the condition of the skin incision.

I think the use of a tourniquet, which I seldom employ in any operation, is distinctly contraindicated here. Slight sloughing of the skin will often occur in operations on feet even on the medial surface, and the use of a tourniquet will increase this tendency.

REFERENCE

Walsham, W. J., and Hughes, W. Kent (1895). *Deformities of the Human Foot*, p. 494, London.

Clinical Memoranda

Intense Toxic Reaction from Sulphapyridine

The following case, in which severe toxic reactions resulted from the use of sulphapyridine seems worthy of record.

CASE REPORT

The patient, a boy aged 6, was admitted to hospital on July 16, 1940, suffering from acute tympanico-mastoiditis on the right side and fairly severe general toxæmia. The following day a right conservative mastoid drainage, Stage 1, was performed, when pus was evacuated from the antrum, the lateral sinus being healthy. On July 19 his condition was still not satisfactory, there being a severe toxic state, with continued rise of temperature. Very soon, however, the fever began to abate by lysis, though the pulse rate did not fall in proportion; on July 25 the temperature began to rise again, and the patient complained of frontal and napal pain. He was now very drowsy and lay with his legs flexed on the abdomen. Kernig's sign was positive on both sides, Babinski's sign was positive on the right and negative on the left, while both knee-jerks were absent. There was no vomiting, and the tongue was clean and moist. The right mastoid region looked very unhealthy, there being sero-pus, and but little healthy granulation tissue. The left ear was sound, nor was there any acute pharyngitis or any rash on the body. The condition suggested early meningitis. Sulphapyridine (M & B 693) in 0.25-gramme doses was given by mouth at 9.42 p.m. on July 28, at 3.15, 7.15, and 11.15 a.m. and 5.40 and 9.45 p.m. on the 29th, and at 5 a.m. on the 30th. He therefore received 1.75 grammes in thirty-one hours eighteen minutes.

On July 29 some small red papules appeared on the back; these were non-irritating, and disappeared within twenty-four hours. On July 31 an erythematous rash, soon intensified by small red papules, each surrounded by a narrow and much paler area (almost white in places), was noticed on the back. On August 3 the rash was spreading and had involved the palms and soles. On the 4th the papules were becoming vesicular, being still surrounded by the whitish areas. The conjunctivæ of the lids and eyeballs were red and injected, the former being somewhat oedematous, while small red spots had appeared in the mucosa of the mouth and lips, the latter being much swollen. On the next day the conjunctivitis was beginning to diminish on both sides, but there were very definite erythematous patches in the mouth. Most of the vesicles on the back had now dried up and coalesced, forming

an extensive deep red covering. The general health was obviously improving, there being much less drowsiness, with no return of the mental irritability that had been a marked feature on his admission. On August 6 scaling on the back had begun, scabs forming in two places, whilst everywhere the involved skin was slowly recovering, its colour paling into a rose-beige. There was also less swelling of the eyelids and lips, and rapid improvement in the general health. By August 7 the rash had almost disappeared, leaving universal desquamation, with dry blisters here and there; the patient complained of pain on defæcation, and a small fissure-in-ano was discovered. On the 9th his general condition was very encouraging, there being no untoward signs; he sat up in bed and ate well. The dermal epithelium was still desquamating in large flakes, with fading pigmentation. On August 12 several large brownish dermal patches, with desquamation (especially on the palms) and a few blisters, could be seen. On the 14th a rapid subsidence of the dermal lesions was noted, the affected areas now showing only a slight brownish tinge, but desquamation was still proceeding on the palms. On the 16th his condition was so encouraging that I was able to perform Stage 2 of his mastoid operation and, concomitantly, remove the tonsils and nasopharyngeal excess, which tissues were very purulent. By August 28 the right ear had completely healed, and, his general health being very satisfactory, he was discharged home.

As regards some of the other treatment carried out, on August 5 10 grains of calcium lactate thrice daily was started, while for the dermal lesion the following lotion was used:

Calamin. præparat.	..	..	..	..	gr. xxx
Zinc oxid.	..	..	..	..	gr. xxx
Liq. calcis	..	..	..	..	5iv
Liq. hamamelidis	..	..	..	..	5j
Ol. olivæ	..	..	..	..	ad 5j

The pathological report on the aural pus recorded the presence of *Staph. aureus*. Lumbar puncture was not carried out, as I did not consider it to be necessary.

I am greatly indebted to my dresser, Mr. Backus, for his careful notes.

London, W.1.

WILLIAM IBBOTSON, F.R.C.S.Ed.

Coeliac Disease in Twins

This disorder occurring in twins seems rare enough to be put on record.

CLINICAL HISTORY

The children, two boys, are apparently identical twins—that is, they are difficult to tell apart, and the mother looks carefully at them to see which she is speaking about. They both have all the symptoms of a rather mild degree of coeliac disease. They are under weight, being respectively 23 lb. and 21 lb. 4 oz. at 2 years of age.

When I first saw them they were pale and flabby, with very large abdomens, were listless and languid, and had poor appetites. The mother had not noticed diarrhoea, but questioning elicited that the stools were bulky and offensive. Analysis of the stools showed an excess of fat. The split fat was 44% in the case of one child and 37% in that of the other. After two months' treatment on a fat-free diet the children are both improving steadily.

The occurrence of this disorder in identical twins raises some speculation as to the aetiology, giving weight to the supposition that the inability to absorb fat is an inborn digestive fault.

My thanks are due to Dr. Adamson for his analysis of the faeces.

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The New South Wales Health Department proposes to appeal for at least 10,000 residents of both sexes between the ages of 18 and 60 to participate in a blood-transfusion scheme to provide a twenty-four-hour service of blood donors to hospitals and doctors and a supply of blood for transfusions in any emergency.