ETIOLOGY OF PERONEAL SPASTIC FLAT FOOT

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A type of rigid flat foot which is accompanied by contraction of the peroneal muscles has long been recognised. The etiology is obscure. Correction of the deformity seems to be prevented by tension of the peroneal muscles, and there has been wide acceptance of the thesis that the deformity is caused by peroneal muscle spasm induced by painful stimuli arising from the tarsal joints. It has been assumed that these stimuli result from abnormal stresses thrown upon the tarsal joints by severe degrees of weak flat foot. The concept, therefore, has been that of a weak flat foot which originally was flexible but which was transformed later into a rigid flat foot by the development of peroneal spasm.

This theory does not explain all the features of peroneal spastic flat foot. In many cases it can be demonstrated by electromyographic studies that there is no spasm of the peroneal muscles; the peronei react in every way as do normal muscles. The apparent spasm, in reality, is an organic shortening of the muscles adaptive to long-continued deformity. If all possibility of peroneal spasm is eliminated by novocaine block of the peroneal nerve, by section of the peroneal nerve, by section of the peroneal tendons, or by anaesthesia supplemented with curare, the deformity still persists in many cases.

It is difficult to believe that peroneal spastic flat foot develops from flexible flat foot by reason of the stresses which fall upon the tarsal joints. Flexible flat foot is comparatively common in childhood, adolescence, and young adult life, and it is not at any time accompanied by peroneal spasm. On the other hand peroneal spastic flat foot is rare, and when it does occur it often dates back to early childhood; the foot has always been stiff. This suggests that it is a separate type of flat foot, probably congenital in origin, and rigid from the onset.

In 1921 an important contribution was made by Sloman. This was supplemented in 1927 by Badgley. Both authors observed that certain severe cases of the deformity were associated with a structural anomaly, namely fusion of the anterior process of the calcaneus to the navicular—coalescentia calcaneonavicularis, or calcaneonavicular bar (Fig. 1). Little attention has been paid to these important contributions. Lapidus (1946), in his extensive report, made no reference to them, nor did the papers he reviewed include any suggestion that rigid flat foot might be due to structural anomalies of the tarsus.

In this paper we shall show that most cases of so-called peroneal spastic flat foot are due to tarsal anomalies. The calcaneonavicular bar, described by Sloman and Badgley, is

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**Fig. 1**
Calcaneonavicular bar (in this case synostosis calcaneonavicularis) which is sometimes the cause of rigid flat foot.

**Fig. 2**
Lipping of the talus in rigid flat foot almost always indicates a tarsal anomaly limiting subtalar movement and thus distorting tali-navicular movement.
an occasional cause of the deformity. Much more frequently, however, the deformity is due to an anomaly which hitherto has been unrecognised by surgeons, though it has been recorded by anatomists, namely, coalescentia talocalcanea or talocalcaneal bridge. Both anomalies are occasionally present in the same foot, and it is possible that other tarsal anomalies may also lead to rigid flat foot.

INCIDENCE OF PERONEAL SPASTIC FLAT FOOT

Examination of 3000 Canadian males who presented themselves for enlistment in the Canadian Army showed that seventy-four had peroneal spastic flat foot—an incidence of 2 per cent. This was one-third the incidence of flexible flat foot (217 cases—6 per cent.). There was one case of calcaneonavicular bar, revealed by radiographic examination (Fig. 1). It did not cause symptoms or interfere with training as a soldier. Peroneal spastic flat foot caused serious disability. It interfered with military training. In nearly all cases the lateral radiograph showed lipping of the superior margin of the head of the talus—an anomaly of particular significance which will be discussed later.

TARSAL ANOMALIES—THE CAUSE OF RIGID FLAT FEET

Two years ago, in developing a medial approach for the treatment of peroneal spastic flat foot by subtalar and talonavicular fusion, we noted a bridge of bone springing from the

Radiographic projection necessary to reveal talocalcaneal bridge. The central X-ray beam is projected downwards and forwards at an angle of 45 degrees, through the heels which have been freed of the leg shadow by flexing the knees.

Radiograph obtained by the technique depicted in Fig. 3, in a patient who had a talocalcaneal bridge on the left side (A). On the right side (B), which is normal, the joint between the sustentaculum tali and the neck of the talus can be seen.

medial surface of the talus, spanning the subtalar joint, and meeting a mass of bone from the medial surface of the calcaneus at the posterior end of the sustentaculum tali. Since then we have found this abnormality in twelve of the seventeen cases of peroneal spastic flat foot which have come under our care. The remaining five cases included three with calcaneonavicular bars and two of tarsal rheumatoid arthritis. In this small series, therefore, tarsal anomalies were the cause of deformity in 88 per cent. of cases: in more than two-thirds of the cases there was a talocalcaneal bridge; and in nearly one-fifth there was a calcaneonavicular bar.

Radiographic demonstration of talocalcaneal bridge—Failure to recognise this anomaly, and to associate it with peroneal spastic flat foot, must be attributed to the difficulties of radiographic visualisation. It is not seen in ordinary projections of the foot; a special projection is necessary if clear pictures are to be secured (Figs. 3 and 4).
In a normal foot this projection passes between the sustentaculum and neck of the talus so that the joint space is shown clearly; when there is talocalcaneal fusion the joint space is replaced by a bone bridge. A secondary change is seen in lateral radiographs, namely marginal lipping of the talonavicular joint on its dorsal surface (Fig. 2). This is present to some degree even in children (Fig. 9). With advancing years the lipping becomes still more marked (Fig. 13). Such lipping on the dorsal margin of the talonavicular joint in a case of peroneal spastic flat foot is invariably associated with anomalies of the tarsal structure which limit movement of the subtalar joint—either a calcaneonavicular bar or, more commonly, a talocalcaneal bridge. The anchoring effect of these bone fusions deranges
the normal inversion-eversion movement of the subtalar and midtarsal joints and throws abnormal stresses upon the talonavicular joint, thus causing impingement of the articular margins and the development of osteophytes.

**Descriptions of the talocalcaneal bridge by anatomists**—Anatomists have been aware of this talocalcaneal bridge. The best account we have found is in Pfitzner's *Beiträge zur Kenntniss des Menschlichen Extremitäten skelets vii. Die Variationen in Aufbau des Fusskelets*, published fifty years ago. He reported two cases and added seven others from the literature. He noted variations including an accessory joint between the sustentaculum and talus (articulatio talocalcana accessoria) a fibrous bond between the two bony masses (coalescencia talocalcanae) and bone fusion (synostosis talocalcanae). In several cases there was also synostosis calcaneonavicularis (calcaneonavicular bar). This combination existed in one of our cases. Pfitzner relates the anomaly to the accessory tarsal bone—os sustentaculi,

![Fig. 8](image)

Specimen of right talocalcaneal bridge (from the anatomical museum of the University of Toronto by courtesy of Professor J. C. B. Grant). It is exactly similar to the specimen illustrated by Pfitzner. Below—medial view showing syndesmosis between talus and posterior end of sustentaculum (Z), and lipping of the superior margin of the articular surface of the head of the talus characteristic of rigid flat foot due to tarsal anomalies (X). To the right—posterior view showing marked valgus tilt of the calcaneus.

occasionally found at the posterior end of the sustentaculum tali. Certainly this is the position of the mass of bone which spans the subtalar joint and fuses with the body of the talus. Pfitzner's illustration of one case of talocalcaneal bridge is reproduced in Fig. 5. Through the courtesy of Professor J. C. B. Grant we have been able to identify a similar case in the anatomical museum of the University of Toronto which is illustrated in Fig. 8.

Dwight (1907), from a study of the Harvard anatomical material, mentioned the occurrence of coalescencia talocalcana. He quoted Pfitzner's observations and commented on page 17 of his monograph: "The first of these exceptional bones, the os sustentaculi, forming the hind end of the sustentaculum, is very uncommon. I have never seen it separate. It is of no practical importance except as concerned in fusion of the astragalus and os calcis and would be hard to show in an X-ray." On page 22 we may read: "Fusion of the astragalus and os calcis occurs at the posterior end of the sustentaculum and may be attributed to the os sustentaculi fusing with both bones. The bones may be firmly
Osteoarthritic lipping in a thirteen-year-old child, demonstrating that structural changes due to rigid flat foot from tarsal anomalies may appear early in life. Fig. 9 shows a lateral projection of the involved left foot, the lipping of the head of the talus being marked X. Fig. 10 shows the normal right foot. Fig. 11 is the postero-superior oblique projection showing the talocalcaneal bridge on the left side (A) in contrast with the normal talosustentacular joint on the right side (B).
co-ossified or they may be united by gristle, the opposed surfaces showing the characteristic irregular finish. Either of these conditions is decidedly uncommon."

Nomenclature—A few words regarding nomenclature are necessary. Pfitzner's strict adherence to B.N.A. usage is precise and valuable. If the bone bridge is complete it is designated—synostosis talocalcanea. If the bridge is interrupted it is designated—coalescentia talocalcanea; this term may be further modified by the terms syndesmosis or synchondrosis to designate the type of tissue which fills the gap. There is need, however, for a simple all-inclusive title and we propose the term "talocalcaneal bridge." Similar words are used to describe fusion of the calcaneus and navicular, namely, synostosis calcaneonavicularis and coalescentia calcaneonavicularis. The all-inclusive term is "calcaneonavicular bar."

TYPES OF PERONEAL SPASTIC AND RIGID FLAT FOOT

The term peroneal spastic flat foot is often applied indiscriminately and inaccurately to certain rigid flat feet which arise from quite different causes. There are at least three such entities and there may be others. Two are related to each other, since in each there is anomaly of tarsal structure with fusion of the calcaneus to the navicular, or to the talus. In these cases there is a deformed rigid foot without peroneal spasm; the peroneal muscles are shortened adaptively in consequence of valgus deformity. The third entity is inflammatory arthritis of the tarsal joints with reflex peroneal muscle spasm which twists the foot into valgus. In the early stages this may fairly be called peroneal spastic flat foot, though it would be more informative to emphasize the nature of the pathological process which gives rise to the spasm rather than the mechanism whereby deformity is produced. The term peroneal spastic flat foot should be abandoned in favour of more precisely descriptive designations of the different varieties of the deformity. For that purpose, a detailed description of the clinical picture of the three different types is necessary.

1) Rigid flat foot due to talocalcaneal bridge (Synostosis talocalcanea or coalescentia talocalcanea)—In this tarsal anomaly there is fusion of the accessory os sustentaculi to the talus and calcaneus. The complete form is represented by a bone bridge arising from the calcaneus immediately behind the sustentaculum, spanning the subtalar joint and fusing with the body of the talus (synostosis talocalcanea). The bone bridge is not always complete, in which case there may be a fibrous bond between the calcaneus and talus (syndesmosis talocalcanea) or a cartilaginous bond (synchondrosis talocalcanea). The bridge may be a synostosis in one area, and a syndesmosis or synchondrosis in another; or the two masses may establish contact by means of an accessory joint (articulatio talocalcanea accessoria). In every variation of the anomaly, except the last, there is some fixation of the talus to the calcaneus; it is complete if synostosis is present, and nearly complete if the lesion is a syndesmosis or synchondrosis. This complete or incomplete fixation of the talus to the calcaneus interferes with normal freedom of inversion-eversion movement, and movements of the talonavicular joint are distorted. In consequence there is impingement of the articular margins of the talus and navicular, and very characteristic osteoarthritic lipping develops on the supero-lateral margin of the head of the talus (Fig. 8).

For reasons which are obscure, the talocalcaneal bridge forces the calcaneus into valgus and often produces marked deformity. Commonly the deformity is limited to a valgus tilt of the heel without depression of the longitudinal arch, but severe deformities combine valgus at the midtarsal joint with valgus of the heel. Our impression is that the most severe deformities are associated with synostosis rather than coalescentia talocalcanea. There may be no parallel between deformity and symptoms. Patients who have synostosis may have surprisingly few symptoms even when deformity is great.
Since the basis of this rigid flat foot is a congenital tarsal anomaly it is first observed in early life. It may well be present from birth, but it is seldom detected until the child begins to walk. In such cases the diagnosis of congenital club foot of the calcaneovalgus type is often made. The true lesion is not revealed in routine radiographic projections but it can be visualised by special projections (Figs. 3 and 4).

2) Rigid flat foot due to calcaneonavicular bar (synostosis or coalescentia calcaneonavicularis)—This type of flat foot is due to fusion of the anterior process of the calcaneus to the navicular (Fig. 16). The fusion may be a complete bone bridge (synostosis) or there may be a dense fibrous band (coalescentia). The anomaly is believed to result when the rare accessory tarsal bone, calcaneus secondarius, fuses to the calcaneus and the navicular. When bone fusion is not complete it is often possible to recognise that the calcaneus secondarius has fused to the calcaneus or sometimes to the navicular.

A calcaneonavicular bar, by fixing the calcaneus to the navicular, restricts and distorts inversion-eversion movements. The foot is rigid, and abnormal stresses are thrown on the midtarsal joint. The clinical picture varies greatly. Unlike the talocalcaneal bridge, which probably always causes deformity and disablement, the calcaneonavicular bar may cause no deformity and no disability. This is particularly true when there is fusion by bone. The one instance of calcaneonavicular bar found in the Canadian Army Foot Survey was such an example: there was no deformity and no disablement: nor did it interfere with military training.

When there are symptoms, they may be grouped into well-defined syndromes. 1) There may be long standing deformity and disablement dating back to early childhood. The deformity is severe and it is associated with persistent pain and weakness, thus resembling cases of talocalcaneal bridge. This is the type of case which was described by Sloman and Badgley. 2) There may be no deformity and no disability until the patient sustains a severe wrenching injury which gives rise to persistent pain and weakness. Such cases are due to avulsion of the calcaneus from the navicular at the fibrous bond. During the acute phase there may be peroneal spasm with rigidity of the foot. The peroneal spasm and the fixation subside rapidly with rest but they may recur if early exercise is permitted. The symptoms tend to persist. 3) There is some evidence to suggest that the calcaneonavicular bar may be fractured and even that such fractures may unite.

3) Arthritic flat foot with peroneal spasm—When rheumatoid arthritis affects the tarsal joints it induces peroneal spasm with resulting valgus deformity. In the later stages there is permanent damage of the joints with fixation in the deformed position. A clear distinction should be drawn between these cases and those with tarsal anomalies. This group of foot deformities must be recognised as part of the problem of arthritis. It would be desirable to recognise their true nature and use the accurate designation ‘arthritic flat foot with peroneal spasm.’
A twenty-six-year-old woman who had a left rigid flat foot from infancy with marked valgus of the heel (Fig. 12) but without great flattening of the longitudinal arch. Fig. 13 is a lateral projection of the involved left foot showing severe lipping of the talonavicular joint. Fig. 14 shows the normal right foot. In Fig. 15 the postero-superior oblique projection shows a talocalcaneal bridge on the left side (A) and a normal talosustentacular joint on the right side (B).
An example of calcaneonavicular bar, complete on the right side (Fig. 18—synostosis calcaneonavicularis) and incomplete on the left side (Fig. 17—syndesmosis calcaneonavicularis). The patient was a fifteen-year-old boy with moderately severe deformity and increasing disabling symptoms worse on the left side than on the right. In each foot the postero-superior oblique radiograph (on the right) shows a normal talosustentacular joint with no talocalcaneal bridge.

Severe congenital calcaneovalgus deformity of the left foot (Fig. 18), treated for twelve years by manipulations and plasters. Postero-superior oblique radiographs of the feet (Fig. 19) show a talocalcaneal bridge in the left foot (A) and a normal talosustentacular joint in the right foot (B). The bridge was demonstrated at operation; the deformity was corrected and the subtalar and talonavicular joints fused.
Congenital calcaneovalgus—The conception that most cases which are described as peroneal spastic flat foot are due to congenital anomalies of tarsal structure has led us to recognise that some, and perhaps all, cases of congenital calcaneovalgus are in reality severe examples of deformity due to a talocalcaneal bridge, sufficiently conspicuous to be recognised at birth, and totally unrelated to congenital talipes equinovarus. Review of one of our cases in the light of newly acquired knowledge revealed synostosis talocalcanea by radiography (Figs. 18 and 19) and this was confirmed at operation. Badgley records a similar case due to a calcaneonavicular bar. We are inclined to believe that tarsal anomalies may prove to be the cause of most cases of congenital calcaneovalgus.

TREATMENT OF PERONEAL SPASTIC FLAT FOOT

Treatment of cases with tarsal arthritis—In general discussion of treatment, arthritic cases must be separated from tarsal anomalies. Treatment in the arthritic case must be directed to the systemic disease of which the foot problem is a local manifestation. Systemic treatment of the disease can be supplemented by local measures to correct or prevent deformity, and to protect the foot from further damage. Treatment of these cases is largely a medical problem but certain orthopaedic procedures can be valuable adjuncts. Of these the most important are: manipulation under anaesthesia to correct deformity; plaster casts to maintain correction; and foot supports of the Whitman type.

Treatment of rigid flat foot with tarsal anomalies—Rigid flat feet due to tarsal anomalies require entirely different treatment. Generally speaking, the deformity and disablement will not be corrected except by surgical measures. We are not impressed by the operation of removing the talocalcaneal bridge, or dividing the calcaneonavicular bar, in an effort to restore normal mobility. Better correction of deformity and better function is obtained by fusion of the subtalar and talonavicular joints, and this is the procedure we advocate when the disability is severe.

Treatment in childhood—Arthrodesis of the tarsal joints should not be undertaken in early childhood. If possible it should be postponed until late adolescence. Some plan of management must therefore be established which will tide the patient through childhood. Repeated manipulations under anaesthesia with immobilisation in the corrected position in plaster give the best results. When deformity is less severe, much can be done by consistent support of the foot with Whitman plates.

Operative treatment—The only surgical procedure we have found to be of value is arthrodesis of the subtalar and talonavicular joints. This is best accomplished through a medial approach which gives direct access to the talocalcaneal bridge and to the calcaneonavicular bar when one is present. The synostosis must be removed in order to free the calcaneus from the talus. Correction of the deformity is then accomplished by remodelling the opposed surfaces of the talus and calcaneus at the subtalar joint. If deformity is severe it may be necessary to make a separate lateral incision through which to lengthen the peroneal tendons and even the peroneus brevis and outer slips of the extensor digitorum brevis. The tubercle of the navicular is removed, and the navicular and head of the talus are denuded of cartilage. Cancellous bone is packed into the field of operation and when deformity has been corrected the wound is closed and plaster is applied.

SUMMARY

1. Peroneal spastic flat foot is a term loosely and often inaccurately used to describe rigid valgus feet developing from widely different causes.
2. The most common causes are two anomalies of the bones of the tarsus—the calcaneonavicular bar, and the talocalcaneal bridge. The first was described in 1921 by Sloman and in 1927 by Badgley; the other is described for the first time in this paper as an etiological
factor in rigid flat foot though it has been recognised by anatomists for fifty years as a skeletal variation. The term peroneal spastic flat foot, as applied to these cases, is inaccurate since there is no spasm of the peroneal muscles. The deformity is a fixed structural deformity due to anomalous bone structure, and the apparent spasm of peroneal muscles is in reality an adaptive shortening. A better term would be rigid flat foot due to talocalcaneal bridge or calcaneonavicular bar.

3. The smaller group of patients who suffer from inflammatory lesions of the tarsal joints, chiefly due to rheumatoid arthritis, do in fact develop valgus deformity from peroneal spasm. The resemblance between the two groups is superficial and it is limited to the apparent similarity of the deformity. Though it might be justifiable to designate this type as peroneal spastic flat foot, it would be better to use the more accurate title—arthritic flat foot with peroneal spasm.

4. Lipping of the upper margin of the talonavicular joint strongly suggests the existence of one or other of the congenital anomalies. Both anomalies are visualised only by special radiological projections.

REFERENCES


