An approach to the assessment of cavus deformity

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Introduction
Pes cavus is a descriptive term referring to an abnormally high arch in the foot which does not flatten on weight-bearing. It covers a spectrum of deformity. It may be simply an exaggerated longitudinal arch, or, more commonly be associated with other deformities. These may include clawing of the toes, metatarsal callosities, pronation and adductus of the forefoot, a pronounced metatarsal boss, fixed varus of the hindfoot and equinus of the ankle. In the majority of cases, there is an accompanying neurological abnormality. The aim in assessment of the cavus deformity is to define potential aetiology, delineate existing deformity and to formulate a treatment plan. Treatment goals would be a balanced plantigrade foot, free from point loading, maintaining midfoot and hindfoot mobility where possible.

Anatomical pathology
Pes cavus is a descriptive term, referring to a high longitudinal arch of the foot. The literature on the description and assessment of pes cavus can be confusing, and non-uniform.1-4 Although all affected feet have a high arch, most in addition, have other deformities. These range in severity from simple clawing of the toes to severe rigid deformities. In order to simplify description of these deformities, it may be convenient to describe the individual components. Mann has described the bony deformity as having two components, namely anterior and posterior.1 The posterior component comprises the hindfoot, while the anterior is subdivided into forefoot and metatarsophalangeal joint.

The hindfoot may exhibit a varying magnitude of dorsiflexion as assessed by the dorsiflexion pitch of the calcaneus. In the normal foot this is less than 30°.1 The hindfoot may be calcaneus or equinus.2 In addition it commonly has a varus component, which may be fixed or mobile. It may however be neutral or valgus. This is generally assessed clinically.

The forefoot may have plantar flexion of all metatarsals, or only the first may be plantar-flexed. In addition, there may be adduction of the metatarsals. Once again these deformities may be fixed or mobile.

The metatarsophalangeal joint (MTPJ) may demonstrate varying degrees of clawing, from flexible extension of the MTPJ and flexion of the interphalangeal joints (IPJ) to a rigid claw with a plantar-flexed metatarsal.

Once the individual components have been appreciated, three patterns of deformity emerge.4,7 It must be noted that no single theory to explain the various deformities exists, and in reality a combination of mechanisms probably plays a role.

Cavovarus
This is the commonest type, and is frequently what is implied when the term cavus is used. The usual cause is Charcot-Marie-Tooth disease,¹ and spinal dysraphism (L5 myelomeningocele), but may also result from a variety of other conditions (Table I). The foot deformity represents the end-point of muscle imbalance between the intrinsic muscles of the foot and between the groups of extrinsic muscles themselves.5,10 Initially the first metatarsal becomes plantar-flexed. This is initially flexible but later becomes fixed with contraction of the plantar fascia. The normal tripod structure of the foot becomes unbalanced. With weight bearing, the hindfoot forces the subtalar joint into varus.11
The cavovarus foot thus has two apposing rotational deformities: pronation of the forefoot on the hindfoot and supination (or varus) of the hindfoot (Figure 1).

Calcaneocavus
Less common, this results from an imbalance between the triceps surae and the ankle dorsiflexors. Chuinard and Baskin proposed a theoretical right-angle triangle of muscle pull. If any part is weakened, deformity occurs. If the gastrocnemius-soleus is weakened, the relatively unopposed planar flexors, along with the oblique pull of the extensor group create cavus. This is classically seen in poliomyelitis. Conversely if the tibialis anterior is weakened, the long toe extensors are recruited to oppose gastrocnemius-soleus, but instead of dorsiflexing the foot, tend to dislocate the MTPJ dorsally and depress the metatarsal heads. Clinically there is balanced flexion of the forefoot with calcaneus of the hindfoot (Figure 2).

**Cavus (Plantaris)**
This is relatively seldom referred to as a separate entity. It is thought to result in global weakness in calf musculature with weakness in the foot dorsiflexors, and, in addition insufficient strength in the plantar flexors to produce true equinus. Relative sparing of the short flexors may also play a role. The deformity presents with isolated flexion of the forefoot on hindfoot.

**Aetiology**
The appearance of a cavus foot is often the initial symptom of a neuromuscular disorder. At least two-thirds will have an underlying disorder, and of these, half will have Charcot-Marie Tooth disease. Even in cases of known disorders, sudden progression warrants investigation. This is especially true in spinal dysraphism, where it may be an indication of a tethered cord. Table I, modified from Lovell and Winter’s *Pediatric Orthopaedics* and *The Art and Practice of Children’s Orthopaedics*, lists the commoner causes.

The appearance of a cavus foot is often the initial symptom of a neuromuscular disorder

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**Table I: Causes of cavus feet (with common causes listed in bold)**

<table>
<thead>
<tr>
<th>Causes</th>
<th>Bilateral</th>
<th>Unilateral</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CAVOVARUS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Charcot-Marie Tooth (HSMN)</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>- Myelomeningocele (S1)</td>
<td>✓ ✓</td>
<td></td>
</tr>
<tr>
<td>- Friedreich’s ataxia</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>- Muscular dystrophy</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>- Polynuiritis</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>- Roussy-Levy syndrome</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>- Spinal dysraphism</td>
<td>✓ ✓</td>
<td></td>
</tr>
<tr>
<td>- Syringomedia</td>
<td>✓ ✓</td>
<td></td>
</tr>
<tr>
<td>- Compartment syndrome</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>- Trauma</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>- Residual clubfoot</td>
<td>✓ ✓</td>
<td></td>
</tr>
<tr>
<td><strong>CALCANEOCAVUS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Myelomeningocele (L5 with weak S1)</td>
<td>✓ ✓</td>
<td></td>
</tr>
<tr>
<td>- Poliomyelitis</td>
<td>✓ ✓</td>
<td></td>
</tr>
<tr>
<td>- CP</td>
<td>✓ ✓</td>
<td></td>
</tr>
<tr>
<td>- Syringomedia</td>
<td>✓ ✓</td>
<td></td>
</tr>
<tr>
<td><strong>CAVUS (PLANTARIS)</strong></td>
<td>✓ ✓</td>
<td></td>
</tr>
</tbody>
</table>

The cavovarus foot thus has two apposing rotational deformities: pronation of the forefoot on the hindfoot and supination (or varus) of the hindfoot (Figure 1).

**Figure 1: Lateral X-ray demonstrating cavovarus**

**Figure 2: Lateral x-ray demonstrating calcaneocavus**
Presentation

Just as the patho-anatomy can vary, so can the clinical presentation. A minority is asymptomatic or only mildly affected. Mild complaints include difficulty in shoe-fitting, and accelerated wearing away of the lateral border of the shoe. Clawing of the toes may lead to painful dorsal callosities. Pressure under the metatarsal heads and base of the fifth metatarsal may also lead to painful callosities.

The midfoot becomes stiff with inversion. This is due to the fact that, whereas in eversion the axis of rotation of the midtarsal joints (talonavicular and calcaneocuboid) are parallel, allowing subtalar motion, in inversion the axes are divergent, locking movement in the midtarsal joint. As a result, patients may complain of repeated sprains and hindfoot pain. A combination of a stiff, varus hindfoot and weak evertors may also lead to complaints of multiple sprains and instability. This may eventually lead to degenerative change in the ankle joint.

Because of the importance of not missing occult neuromuscular disorders, there must be a specific enquiry into the family history.

Clinical evaluation

Presenting complaints should be noted, including pain, callosities, instability and shoe wear. Patients should be questioned about hand weakness/clumsiness, indicating intrinsic muscle involvement. A detailed family history must be obtained.

A brief global examination should include examination of the spine, and neuromuscular examination with emphasis on the intrinsics of the hand, as well as signs of proximal muscle weakness (Gower test). Sensory alterations should be sought.

A detailed family history must be obtained

The feet should be examined – with the patient walking and standing – both from the front and behind. Subtle evidence of foot drop may be evident, as may calf wasting (stork leg deformity) and recruitment of secondary ankle dorsiflexors (cock-up toes in swing phase). With the patient seated shoe wear pattern, dorsal and plantar callosities, ulceration and sites of pain are noted. Active and passive range of ankle, subtalar and metatarsophalangeal (MTP), and interphalangeal joints should be evaluated along with an examination of the mobility of the midfoot. The power of the extrinsic muscles of the foot must be tested and graded. This is particularly important if tendon transfers are to be contemplated.

The various components of the foot (hindfoot, midfoot and MTP joints) in their relationships to each other as previously described are assessed, and an attempt made to gain an impression of the overall pattern of deformity.

The mobility of the subtalar joint/hindfoot is assessed by the block test described by Coleman and Chestnut. The patient’s heel and lateral border are placed on a block approximately 2.5 cm thick. The first to third/fourth metatarsals are allowed to fall into pronation, eliminating their effect on the tripod.
During weight bearing, the hindfoot that returns to valgus is flexible and those that do not are considered rigid (Figures 3a and 3b). Price and Price describe an alternative where the patient is placed prone and the knee flexed to 90°. With the foot no longer weight bearing, the forefoot is allowed to pronate, and, if flexible, the heel can come out of varus.18

Radiographic features
Anteroposterior (AP) and lateral radiographs are routine, and it is essential that they are taken standing. The cavus may be quantified by Meary’s angle, the angle between the long axis of the first metatarsal and the long axis of the talus.19 This should normally be 0° (Figure 4).

The calcaneal pitch is assessed by the angle formed between a line drawn along the inferior border of the calcaneus and the weight bearing surface.16 An angle of greater than 30° is abnormal, and consistent with cavovarus/calcaneocavus (Figure 5).

Other investigations
When there is a suspicion of spinal abnormalities, radiographs of the spine and/or Magnetic Resonance Imaging (MRI) should be performed.

If Charcot-Marie-Tooth is suspected EMGs and nerve conduction studies should be performed as an initial investigation. These are diagnostic for type I disease, where there is a slow nerve conduction velocity and prolonged distal latencies.20 Patients with type II disease have relatively normal nerve conduction studies. To make the diagnosis they require, in addition, nerve biopsy, which reveals Wallerian degeneration. Consultation with a neurologist is appropriate.

Principles of management
The overall goal of management is to provide a plantigrade stable foot.
Conservative management is appropriate in the minimally symptomatic mild case or in cases where surgical treatment is contraindicated. Well-fitting, pliable shoes, pliable metatarsal bar orthoses, and ankle-foot orthoses may all be appropriate. Most cavus feet progress with time or become symptomatic, and surgery will become necessary. Just as each case must be individually assessed, an individualised surgical plan is necessary. Indications for surgery include progressive deformity, painful callus, or ulceration, symptomatic clavering and ankle instability. As a guide, those feet with supple deformities require only soft tissue release with or without tendon transfer. In the younger patient in whom deformities may change, several procedures may be necessary to accommodate growth or compensate for altering neurology.

Where a specific fixed bony deformity exists in an otherwise supple foot, which prevents plantigrade loading, then an osteotomy to correct this deformity along with the appropriate soft tissue corrections is indicated. The two most common examples would be the plantar-flexed first ray, where a dorsal closing wedge osteotomy of the first metatarsal or cuneiform is performed, and the fixed varus hindfoot, which would then require a lateral closing wedge, or lateral displacement osteotomy to correct this deformity along with the appropriate soft tissue corrections.

With progression of stiffness and deformity, midfoot osteotomies may become necessary to correct the cavus. They all remove a wedge, or truncated wedge and result in a degree of shortening. In addition, depending on the level, the tarsometatarsal joints may be sacrificed. The pathology requiring these osteotomies is more severe than simple soft tissue procedures and the results predictably poorer.

In the mature foot, with deformities not correctable by the above means, a triple arthrodesis is indicated. The advantage is that multiplanar deformities may be corrected in a single procedure — typically hindfoot varus and equinus and midfoot plantar flexion and pronation. The disadvantages are shortening of the foot, stiffness, and accelerated degenerative changes in the ankle joint. The procedures are also technically demanding.

In addition to procedures to correct the cavus itself, attention must be paid to callus, ulcers, claw toes and equinus where present. A simplified protocol summarising the above is presented in Figure 6.

**Summary**

- Assess individual components of the foot:
  - hindfoot
  - forefoot
  - MTPJ
- Assess overall deformity type:
  - cavus/planteris
  - cavovarus
  - calcaneocavus
- Seek a cause:
  - family history, intrinsics, drop foot – Charcot-Marie-Tooth
  - hairy naevi, back abnormalities, progressive deformity – spinal defects
  - Formulate an individualised plan.

This article was not submitted to an ethical committee for approval. There was no patient involvement, and it was purely a review/instructional article. The content of this article is the sole work of the author. No benefits of any form have been derived from any commercial party related directly or indirectly to the subject of this article.

**References**

15. Dwyer PC. The present status of the problem of pes cavus. CORR 1975;106:254-75.