MINI-SYMPOSIUM: THE PAEDIATRIC FOOT

(iii) The foot in cerebral palsy

C. M. Duffy and A. P. Cosgrove

Musgrave Park Hospital, Stockman’s Lane, Belfast BT9 7JB, Northern Ireland

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Summary
The foot in cerebral palsy is subject to the normal forces applied during walking, but its means of resisting these forces is altered as a result of the muscle weakness and spasticity inherent to this condition. Abnormal muscle activity permits the foot to deform in response to the forces applied to it, as well as causing deformities in their own right. The classical equino-valgus posture adopted by the diplegic foot is principally due to spasticity of the gastrocnemius and weakness of the peroneals. The equino-varus deformity more commonly seen in the hemiplegic foot is associated with spasticity of tibialis posterior.

While still flexible, the deformities of the cerebral palsy foot may be dealt with by a variety of conservative measures including casting, orthotics and botulinum injection. Once established, however, bony surgery is required to correct these deformities.

INTRODUCTION

Minear classified cerebral palsy according to the involvement of limbs.1 In this classification those with involvement of one side of the body are defined as hemiplegic, and those with involvement of both legs as diplegic. In hemiplegia the upper limb is usually more involved than the lower, while in diplegia the upper limbs are relatively spared.

Cerebral palsy is a central nervous system disorder, not primarily a disease of the musculo-skeletal system. The altered innervation of the muscles produces either spasticity or weakness, causing imbalances of muscle activity across joints. This leads first to correctable deformity but eventually to a fixed deformity. Changes in the foot occur mainly as a result of weakness or spasticity of the muscles attached to the long tendons which cross the ankle joint, and to a lesser extent of the intrinsic foot muscles. While normal muscle development in children depends on activity, bone is less affected and grows regardless of activity, although the affected leg in hemiplegia is often shorter than the contralateral normal side. If muscle is to keep up with bony growth it requires both passive stretch and active contraction, which should occur during normal activity, e.g. walking. Children with cerebral palsy may not be able to engage in normal childhood activity or even to walk.2 Those who do walk, tend to do so late. The loss of normal muscle stretch leads to muscle shortening, which in turn leads to the loss of the normal balance of agonists and antagonists. This is particularly important where there is spasticity of larger muscle groups, e.g. the gastrocnemius and soleus in the calf. Given that in the normal leg the plantarflexors are six times stronger than the dorsiflexors,3 spasticity of the plantarflexors, resisted only by weak dorsiflexors, results in serious miss-match, and equinus deformity ensues.

THE NORMAL FOOT

The foot is a complex structure, whose component parts must act smoothly together for normal gait to be achieved. The muscles controlling the foot and ankle complex can act either concentrically, i.e. by shortening to effect an action, or eccentrically, i.e. by ‘paying out’ or lengthening in a graduated fashion to control an action. The long tendons crossing the ankle to reach the foot may be divided according to function as plantar-flexors, dorsiflexors, invertors and evertors. Thus, any tendon passing behind the axis of the ankle, regardless of its primary function, will contribute to plantarflexion and any in front of it to dorsiflexion. Similarly, those tendons passing to the medial side of the subtalar joint will tend to invert it, and those passing laterally will evert it. Additionally, the Achilles tendon crosses the subtalar as well as the ankle joint and so acts on both, Soleus tending to invert the subtalar joint. Any disturbance in the properties of these muscles must disrupt the smooth function of the foot.
THE FOOT IN THE NORMAL GAIT CYCLE

The priorities of gait have been defined by Gage as:
- stability in stance;
- clearance of the foot in swing;
- appropriate pre-positioning of the foot;
- adequate step length;
- conservation of energy.

The first three requirements all relate directly to the foot so it can be readily appreciated that disturbance of any of the above mechanisms will cause disruption of normal walking ability.

During the stance phase of the gait cycle body weight moves sequentially from the heel to the toe of the planted foot. On an unyielding surface the force exerted by the foot will be opposed by an equal and opposite ground reaction force, which can be readily measured. This force has a vector quality. As body weight passes along the foot the ground reaction force will exert forces on the joints of the lower limb, dictated by its position in relation to them. The joints of the foot and ankle must be stable in order to resist these forces, and not deform under their influence. It is the role of the muscles of the normal foot and ankle to control the effect of the ground reaction force to ensure its smooth progression from heel to toe.

This is best understood if the foot is considered as a series of ‘rockers’ (Fig. 1). The first rocker is that period around heel strike when the heel contacts the ground first followed by the forefoot being lowered to the ground. At this time the fulcrum of movement of the foot is the heel. The ground reaction force acts through the heel and pushes it to the floor, which tends to plantar-flex the ankle. The pre-tibial muscles, acting eccentrically, control the resultant plantar-flexion movement. The second rocker occurs at mid-stance. The foot is flat on the floor and the fulcrum of movement moves to the centre of the ankle. The ground reaction force advances forwards along the foot so that the load axis passes in front of the knee. This is a position of great passive stability, in which quadriceps may be inactive. Both first and second rocker are associated with deceleration of the body as it descends onto the planted foot.

The third rocker is associated with acceleration of the body away from the planted foot and it occurs at the end of stance, just as the heel starts to rise off the ground in preparation for its advance into swing (toe off). At this point the fulcrum of movement has reached the level of the metatarsal heads. The ground reaction force is exerting a strong external dorsi-flexing influence which must be resisted and overcome by the plantar-flexors to propel the foot forwards into swing. After the foot has left the ground concentric contraction of the tibialis anterior lifts the foot into dorsi-flexion to facilitate clearance in swing phase, before dropping it back into the neutral position in terminal swing in preparation for initial contact.

There are three major joint complexes in the foot whose normal function is essential for normal walking. These are the subtalar joint (STJ), the midtarsal joints (MTJ) (calcaneo-cuboid (CC) and talo-navicular (TN)) and the first metatarso-phalangeal joint (MTPJ).

Inversion and eversion take place at the STJ, and thus accommodates the foot to uneven surfaces. It is also responsible for accommodating the ankle joint to the line of progression of the body as the axis of the ankle is at 20° of external rotation. This is overcome by heel eversion of the foot at initial contact, allowing the talus to internally rotate. Eversion of the subtalar joint has the effect of rendering the CC and TN joints parallel and free to move. In this position the foot is supple and able to act as a shock absorber. In midstance the STJ inverts and restores ankle alignment to external rotation. This action of inversion of the subtalar joint during stance also diverges and so locks the midtarsal joints and the tarsometatarsal joints, making the foot a rigid lever and allowing it to act as a firm support as the centre of gravity passes over it. It also has the effect of flattening the medial arch, which reconstitutes at the end of the stance phase. At this time the MTPJs dorsiflex to allow body weight to roll over the metatarsal heads rather than the tips of the toes.

The mechanical effects described could not be achieved without adequate muscle control. Tibialis anterior and posterior are eccentrically active during ST eversion to control its rate and extent. During stance tibialis posterior, soleus, the long toe flexors and the plantar fascia are all active concentrically in assisting single stance support. Towards the end of the stance phase the long toe flexors are again eccentrically active in pressing the toes to the ground to increase the area of

![Figure 1](Perry's rockers)
support available and facilitate body weight rolling over the metatarsal heads in third rocker.

**THE FOOT IN CEREBRAL PALSY**

**Equinus**

Equinus deformity of the foot often results in failure of the heel to make contact with the ground during walking (Fig. 2). However, more proximal abnormalities such as knee or hip flexion contracture can also raise the heel off the ground. Conversely, some individuals with an equinus deformity may get the heel down by allowing the tibia to incline posteriorly and hyperextending the knee (Fig. 3).

Equinus in cerebral palsy may result from altered control, spasticity or contracture of the calf muscles together with weakness of tibialis anterior. In diplegia, it is often only the gastrocnemius that is tight; the soleus is often not. This situation is demonstrated by the Langenskiold test in which, if it is positive, the foot is seen to dorsiflex more with the knee flexed than extended. This indicates that the gastrocnemius, crossing both knee and ankle, is tight, but not the Soleus, which crosses only the ankle complex. The tibialis anterior is likely to be weak and under poor voluntary control. Thus, while tibialis anterior may be functional, it cannot be called upon to activate ‘on demand’.

The confusion test is used to assess whether there is tibialis anterior function, even if not under voluntary control. The child is asked to dorsiflex the foot as a single action. Children who cannot do this may still be able to dorsiflex as part of a mass action when the child also flexes his hip and knee. Thus tibialis anterior can be shown to be active but not necessarily under voluntary control. However, the confusion test is a poor predictor of whether tibialis anterior will function during gait.5

Equinus means that initial ground contact is on the forefoot, or less commonly with flatfoot placement. Thus, the first rocker is abolished, as is the second rocker, and smooth transfer of weight from the heel to the forefoot.

This stretches the calf muscle and, if the knee is flexed, the gastrocnemius will tend to flex it further. In turn, premature heel rise may occur, or heel contact may not occur at all (Fig. 2). A beat of clonus may be detectable. If the knee is extended, it is likely that the ground reaction force acting on the forefoot will pass in front of the knee causing it to hyperextend, allowing the heel to come in
contact with the ground despite the equinus position of the ankle (Fig. 3).

The loss of smooth transfer of weight along the foot leads to increased muscle activity at the more proximal joints to maintain an upright posture, which is not energy-efficient, particularly with respect to the quadriceps. It is known\(^6\) that normal children who are idiopathic toe walkers have decreased velocity of walking and stride length, like children with spastic diplegia. However, such children do not demonstrate the increased knee flexion often seen in those with cerebral palsy. Such ‘normal’ toe-walking children do have reduced power generation of their gastro-soleus complex at terminal stance, indicating that this is a function of the biomechanical disadvantage at which this muscle group must work in equinus, and not just of cerebral palsy.

**Other effects**

During the stance phase of gait, if the ankle is in equinus, the abnormal pressures on the forefoot may result in callosities, particularly below the metatarsal heads. As weight is only borne on a small base in equinus gait this can significantly impair balance and stability. Furthermore, the attitude of the foot means that the talus is not locked in the mortise of the ankle joint, which can give further problems with stability. During swing, equinus causes difficulties with foot clearance in swing, thus the toes drag along the ground. Shoe fitting can cause especial difficulty. Equinus deformity of the ankle is often associated with varus and valgus deformities of the foot. The tightness of the Achilles’ tendon acting across the subtalar joint will tend to exacerbate any instability already present. In diplegia the most common deformity is valgus and in hemiplegia varus. Although either deformity can occur in any pattern of cerebral palsy and indeed both deformities can occur in the opposite feet of a bilaterally involved individual.

**Calcaneus deformity**

Although equinus is often a very striking deformity it is important to remember Mercer Rang’s\(^7\) adage that ‘a little equinus is better than any calcaneus’. If the Achilles tendon becomes overly long it allows the tibia to fall forwards resulting in a requirement to flex both the hips and knees, i.e. crouch. This requires continual activity of the quadriceps and hip extensors to resist gravity. (see below).

**Treatment of equinus**

There is limited published information on the natural history of deformities in cerebral palsy. In diplegia many young children have an equinus pattern gait. However, in older non-surgically treated patients this pattern is less frequent with many adopting a crouch pattern of gait with deteriorating function. In such cases the foot becomes excessively dorsiflexed either by true dorsiflexion at the ankle or effective dorsiflexion by the subtalar joint tipping into valgus, probably due to the inability of the ankle/subtalar joint complex to withstand the increasing loads placed on it by increasing body weight. Treatment must take account of such future developments as well as the presenting clinical problem.

A toe-walking child does not necessarily need surgical treatment. Many children with cerebral palsy are quite functional. Particularly during the early years, when equinus is due to calf spasticity and not calf contracture, it may be managed by a variety of conservative means.

**Casting**

Serial casts to correct and maintain the plantigrade position of the foot in the presence of calf spasticity have been used for many years. Plaster of Paris is cheap and easy to apply. Children readily adapt to walking in plaster casts, but the cast cannot be removed for bathing, etc., and this is not a long-term treatment for equinus. Corry et al.\(^8\) failed to produce continuing improvement in a neutral plantigrade position. However, Cottalorda\(^9\) recently showed that serial casting, in a dorsiflexed rather than a neutral position, achieved a good range of passive dorsiflexion. While this was maintained for 3 years in 50% of children, two-thirds of them resumed toe walking. Additionally there are concerns that casting may stress the STJs and there is a significant incidence of plaster sores.

**Orthotics**

The aim of an orthotic is to control the joint(s) it crosses. Polypropylene ankle foot orthoses (AFOs) are commonly used to control equinus. It is often useful to cast such AFOs in a few degrees of dorsiflexion, particularly if the equinus has been accommodated by ‘back kneeing’ (hyperextension). Ground reaction ankle foot orthoses (GRAFOs) may be used to control excessive dorsiflexion of the ankle through stance but are of most value where this is due to weakness of the calf, rather than tightness of the hamstrings. Articulated (hinged) AFOs are helpful for climbing stairs and for getting up off the floor. Some believe\(^10\) that an articulated AFO will permit the diplegic child a more normal gait pattern by allowing a little dorsiflexion through the gait cycle, but still not allowing crouch. This is not the consensus view and many practitioners do not recommend the use of hinged AFOs in diplegia because of the risk of crouch gait but they are useful in hemiplegia.

**Alcohol and phenol**

These neurotoxic agents have been used to block motor end plates chemically. They are injected into the motor
points of muscle using EMG guidance. Injection can be uncomfortable. The duration of action is variable. Although these agents are inexpensive and effective their use has been largely supplanted by botulinum toxin.

Botulinum toxin A

This toxin is produced by the bacterium Clostridium botulinum. It acts by blocking the transmission of acetylcholine across the motor end plates of skeletal muscle. It has been shown to be effective in reducing the spasticity of muscles into which it is injected and is currently licensed in the United Kingdom for use in the calf in children with cerebral palsy. It has also been used with reported success in the adductors, hamstrings and upper limb. The indication for its use is dynamic contracture of muscle secondary to spasticity. Some authors contend that it is of most use in the child aged 1–5 years, the time of greatest motor development, when it is most likely that the course of the disease will be modified. Others, however, have found no correlation between age and efficacy of botulinum, as long as there were no fixed contractures. The pharmacological response generally lasts for 6 months; however, biomechanical and functional benefits may persist for longer periods, and its use may be repeated.

Botulinum toxin is particularly useful in controlling equinus in the younger child in whom calf surgery would have an unacceptable risk of over-correction. To maximize its effect botulinum injections may be combined with short periods of casting or with an intensive physiotherapy and orthotics regimen to maximize muscle stretching.

Eames et al. showed that 1 year following botulinum toxin injection to the calf 30% of muscles were still longer than their baseline measurement.

Surgical

Achilles tendon lengthening is rarely indicated in spastic diplegia, as often only gastrocnemius is tight and soleus is not. If the whole tendon is lengthened there is a risk of losing the retarding effect of soleus on the tibia during stance, which would allow it to slide into crouch gait. Only 50% of diplegic children undergoing isolated Achilles tendon lengthening, either by a Baker’s slide or a Z-lengthening of the Achilles tendon as isolated procedures are likely to do well. Percutaneous lengthening of the Achilles tendon is even less reliable. In order to divide only the gastrocnemius tendon, its fascial layer can be separated from the underlying soleus, as described by Strayer, and divided singly. This is our preferred option for diplegia. Good results have been shown for gastrocnemius recession, in children with spastic diplegia, in relation to improved stride length and passive range of ankle movement and some decrease in plantarflexion at the terminal stance phase of the gait cycle.

For diplegic children, it must be considered that equinus in gait may be caused by tightness of the hamstrings, and that the gastrocnemius may not be tight at all. Steinweander et al. found awake examination and gait analysis unreliable and advocated examination under general anaesthesia (EUA) as the only way to distinguish between fixed and dynamic equinus, and further that dynamic equinus needs no surgery. In their series of patients undergoing multilevel surgery, they determined by EUA whether or not to address the calf. All had shown evidence of equinus in pre-operative gait analysis. If the calf was found not to be tight, no surgery was done at that level, if it was tight an intramuscular gastrocnemius lengthening was performed. All showed improvement in post-operative ankle kinetics and kinematics. For those having no calf contracture, the equinus noted preoperatively was attributed mainly to tight hamstrings.

Equinus in hemiplegia is more likely to be due to contracture of the whole calf, and not just gastrocnemius, as in diplegia. It is usually ‘real’ and not ‘apparent’. Thus, it usually is appropriate to lengthen the whole tendon. A variety of techniques have been described but there is little in the literature to suggest any of these methods of calf lengthening in hemiplegia is superior to any other.

The authors preferred technique is a formal open Z-lengthening. Other published techniques include the White slide technique of lengthening which relies on the rotational properties of the fibres of the Achilles tendon. The tendon is cut anteriorly in its distal portion, and medially in its proximal portion. Dorsiflexion of the ankle then produces a sliding effect and gaps open at each partial tenotomy site, but the tendon remains in continuity. This may be performed as an open or closed procedure. The triple cut, wherein the tendon is cut three times, laterally proximally and distally and once medially between these two, may also be done as an open or closed procedure. Once again, dorsiflexion of the foot produces sliding of the fibres and in-continuity lengthening of the tendon. Baker’s slide, where the aponeuroses of both the gastrocnemius and soleus are divided in a triangular fashion just below the muscular insertion, may be appropriate for hemiplegic equinus.

Children with hemiplegia are more likely to relapse while those with diplegia are more likely to sink into crouch following surgery for equinus. The more severely affected the child, regardless of whether they are hemiplegic or diplegic, the less likely calf lengthening is to be effective. The optimal age to lengthen the calf is 8 years and over when there is less chance of calcaneus, particularly for diplegic children.

Valgus

Valgus is a feature of diplegic cerebral palsy (Fig. 4). When a tight Achilles tendon pulls on the hind foot, the foot
will ‘break’ through its mid-portion in an attempt to accommodate the plantarflexion and still maintain the foot flat on the floor during the stance phase of gait. In diplegia the foot breaks into valgus. This is not so much because of any over-activity of the peroneals, but more because of poor support from a weak tibialis posterior. The foot drifts out into abduction and is inclined to be externally rotated.

The normal foot relies on the movement of the subtalar joint to ‘lock’ the midfoot during stance so that it functions like a rigid lever. In the equino-valgus foot of diplegia, the subtalar joint becomes incompetent so that the midtarsal joints do not ‘lock’. The foot cannot act as a lever, against which the body can ‘push off’ the ground, causing instability. The foot no longer follows the line of progression of the body and the ground reaction forces are not properly directed in relation to the proximal joints (Gage’s ‘lever arm disease’). Ordinarily the ground reaction force passes anterior and medial to the knee in stance, but in equino-valgus it passes posterior and lateral to it and the passive stabilizing effect of the ground reaction force passing anterior to the knee (Fig. 1) is lost. Simultaneously, the ground reaction force passing lateral to the knee leads to genu valgum. In plano-valgus, the forefoot supinates, i.e. rotates in the opposite direction to the hind foot. If this is a structural deformity, it may result in early recurrence of hind foot valgus if not corrected at the same time.

Apart from gait dysfunction, equino-valgus also causes problems with excessive weight bearing on the medial border of the foot, causing skin problems and difficulties with shoes.

**Treatment of valgus**

Before attempting any treatment of the equino-valgus foot in diplegia, weight-bearing X-rays should be taken to ensure that the valgus is not originating in the ankle joint. The temptation to treat children too young should also be avoided as pes valgus can be found in normal children up to the age of about 4–7 years, simply due to ligamentous laxity.

Mild degrees of equino-valgus may be treated conservatively using orthotics. If the subtalar joint alone is unstable a University of California Biomechanics Laboratory (UC-BL) orthosis may be adequate to control undesirable inversion or eversion at the subtalar joint. However, if equinus or calcaneus accompanies it, it will be necessary to include the ankle joint using an AFO. Although empirically it seems reasonable that protecting the shape of the foot may reduce the risk of progressive deformity there is as yet no evidence to support this view.

More severe valgus will require a surgical treatment.

**Achilles tendon (tendo achilles) lengthening (TAL)**

Some form of TAL is almost always required (see above for the various techniques) with or without other procedures.

**Calcaneal lengthening**

This is a modification of the technique originally described by Dilwyn Evans and was originally thought not to be suitable for neuromuscular flatfeet. However, Mosca successfully treated a selected group of patients, including some with cerebral palsy, for flat foot using this technique. This is probably most suitable for younger patients and those with less severe deformity. The osteotomy is made approximately 1 cm proximal to the calcaneo-cuboid joint into which is inserted a trapezoidal, tricortical iliac crest graft. The intention is to lengthen the calcaneus which dorsiflexes the talus, inverting the subtalar joint and aligning the midtarsal joints, to provide a rigid lever. The rationale is that as the midfoot sags into pes valgus with lowering of the longitudinal arch, the lateral column becomes relatively short.

**Subtalar arthrodesis**

This is indicated when the foot can be brought to the neutral position in equinus. It should only be undertaken
if the tarsal bones are sufficiently ossified for successful arthrodesis to take place. Either an intra- or an extra-articular arthrodesis may be used. A necessary pre-requisite of extra-articular arthrodesis is that the subtalar joint is passively correctable, allowing the heel to lie directly beneath the mechanical axis through the talus and ankle joint. If this is not possible an intra-articular arthrodesis should be performed.

If extra-articular, we are in favour of using Dennyson and Fullford’s modification of the Grice procedure. The walls of the sinus tarsi are denuded of cortical bone, which can be turned back as flaps into the base of the sinus. A tri-cortical wedge of iliac crest is inserted into the sinus to hold it open and prevent the hind foot from collapsing back into valgus. A cannulated screw is then placed across the talo-navicular joint to hold it reduced while the graft incorporates.

To perform the intra-articular procedure, the subtalar joint is exposed laterally. The articular surface is denuded and a bone graft inserted to distract the joint and place the weight-bearing surface of the calcaneus directly under the talus and ankle joint. Subtalar arthrodesis may be combined with a calcaneal lengthening.

Calcaneal osteotomy

A medially closing wedge of the calcaneus may be taken to correct hind-foot valgus. This procedure is seldom used, but may be suitable in hemiplegia with foot valgus.

Z lengthening of the peroneals

Valgus foot deformity in diplegia is unlikely to be due to peroneal spasticity and should not be used in isolation as it may result in weakness.

Plantar-medial plication of the talo-navicular joint capsule

This may be used in combination with subtalar arthrodesis or calcaneal lengthening. It is not likely to be effective if used alone.

Advancement of tibialis posterior tendons

This may improve the effectiveness of this tendon, but is most likely to be effective if combined with a bony procedure.

Triple arthrodesis

This is unsuitable for the younger patient, and really represents a salvage procedure for the skeletally mature, at least 13 years of age. It is more frequently employed for varus deformity rather than valgus deformity. Bleck has introduced the concept of using screw fixation of the triple arthrodesis; very much in the way described by Dennyson and Fullford for the subtalar arthrodesis.

Medial cuneiform osteotomy

A plantar-based osteotomy may be required to correct structural supination of the fore foot.

Varus

Many children with hemiplegia have an equino-varus foot deformity (Fig. 5). It is much less common in children with diplegia. Weakness of the evertors is not generally responsible for varus in cerebral palsy. While it may come about secondary to calf spasticity as the subtalar joint shifts in an attempt to dorsiflex against a tight Achilles tendon, most varus results from over-activity of the invertors, mainly because of spasticity of the tibialis posterior and anterior. If tibialis posterior alone is involved then only hind-foot varus is noted. The action of tibialis posterior in cerebral palsy, however, is not entirely predictable; it may be totally quiescent even in the presence of significant varus. If the more distally placed tibialis anterior is also involved then there is varus and supination of the forefoot as well.

Figure 5 Hemiplegic child with classical equino-varus deformity.
Bleck advises EMG to determine the relative contributions of tibialis anterior and posterior to the varus. Soleus is an invertor, particularly in terminal swing, and through to midstance, and may have some influence through the obliquity of the ankle mortise. Over-activity of flexors hallucis and digitorum longus can also contribute to varus, and if so will cause associated toe clawing. Varus will become more pronounced at midstance when the external moment of the ground reaction force attempts to dorsiflex the foot against the resistance of the contracted calf.

Varus causes abnormal loading of the lateral border of the foot with uncovering of the talar head causing callosities to develop here and at the base of the fifth metatarsal.

**Treatment of varus**

If flexible, equino-varus may be treated by redistributing or reducing the deforming forces producing it; if fixed, then bony surgery is probably necessary. The authors recommend that the first stage of treatment is to lengthen the Achilles tendon and then, if necessary, recessing the tibialis posterior above the medial malleolus. Split transfer of tibialis anterior may be used in selected cases, either at the time of initial or subsequent surgery, where it seems to be over-active.

Tibialis anterior transfer should be deferred until the tarsal bones are largely ossified to avoid the immature foot being moulded into valgus as a result of the strong pull of the transplanted tendon. The newly transplanted tendon should be protected with a cast and the corrected foot position maintained for a period thereafter by an AFO.

Attempts have been made to correct varus deformity by split transfer of the tibialis posterior, either alone or in combination with other procedures but indifferent results have been reported. Problems may have arisen because of failure to recognize fixed deformity resulting in under-correction. It is also likely that, even split, the tibialis posterior tendon could be too powerful for its transplanted site and result in over-correction. Bleck has cautioned that underneath every varus there is a valgus! He recommended Z-lengthening of the tibialis posterior tendon above the ankle.

If tibialis anterior is very active, a split tibialis anterior transfer to peroneus brevis may be added. If the hind foot is rigidly in a position of varus, a lateral closing wedge osteotomy of the os calcis may be included with teno Achilles lengthening and the tendon transfers. In older children a triple arthrodesis may be required.

**Hallux valgus**

Hallux valgus is much more common in diplegic children and is a consequence of intrinsic muscle imbalance and abnormal loading of the pronated foot. It may arise from either over-activity of peroneus longus, whose sheath gives origin to the oblique head of adductor hallucis, which would thus be pulled laterally; or from medialization of forefoot loading due to forefoot pronation with rotation of the flexion/extension axis out of the sagittal plane. Alternatively, it may simply be that the repeated trauma to the pronated foot in pes valgus associated with diplegia weakens and stretches the medial support of the metatarso-phalangeal joint of the great toe, forcing it laterally. When hallux valgus does occur in hemiplegia it is likely to be due to spasticity of adductor hallucis. As in diplegia, it may occur as a consequence of pes valgus.

Malposition of the great toe has implications for gait, as it normally stabilizes the foot during third rocker and can lead to painful bunion and callosity over the first metatarsal head. Difficulty with shoes and ingrown toe nails can also be a problem.

Hallux valgus should be addressed by correcting foot malalignment first; in particular, the problems of equino-valgus, spasticity and any rotational problems proximally in the lower limb. This may suffice without direct treatment. If not, first metatarso-phalangeal joint arthrodesis in 10°–15° of dorsiflexion, first metatarsal osteotomy, capsulorraphy and adductor hallucis lengthening have all been suggested to treat hallux valgus in diplegia.

**Foot drop**

This is most common in diplegia. It occurs secondary to weakness of the tibialis anterior, which is unable to lift the foot into dorsiflexion in swing. If tibialis anterior is under voluntary control, this results in the foot contacting the ground with a ‘slapping’ gait, in which the heel does contact the ground first, but there is rapid plantarflexion through loading response. Often in cerebral palsy, tibialis anterior is not under voluntary control, in this situation, the foot meets the ground in the ‘foot flat’ position with the knee extended. This is best controlled by an orthosis.

**Crouch**

Rather than spasticity, there can be weakness of the calf muscles. If this involves the soleus then the tibia advances too rapidly, causing crouch gait. This is most commonly seen after injudicious calf lengthening in diplegic children. It may, however, occur as a primary pathology. If gastrocnemius is weak, there is delayed, or even lost, heel rise and loss of power for push-off at the end of the stance.
phase of the gait cycle. This results in loss of third rocker, since body weight cannot roll over the metatarsal heads, together with reduced velocity and step length. Inadequate push-off means that most of the power for foot clearance must come from the hip flexors.2

Dorsal bunion

Most common in hemiplegia, this occurs secondary to a combination of spasticities. The spastic tibialis anterior pulls the first metatarsal into dorsiflexion, while the spastic flexor hallucis longus pulls the proximal phalanx of the great toe into plantar flexion. The head of the first metatarsal is thus left uncovered and a bunion forms on its dorsal surface. This deformity again causes problems primarily by the development of painful callosities with shoe wear. Treatment for this is surgical, if the deformity cannot be accommodated in shoes, and may consist of transfer of tibialis anterior or flexor hallucis longus or osteotomy of the first metatarsal and bunionectomy. Arthrodesis of the first metatarso-phalangeal joint is also an option.

Metatarsus adductus

Most common in hemiplegia, this occurs secondary to spasticity in the adductor hallucis and may be more common following tendo Achilles lengthening.

This may be dynamic or fixed. If this requires correction, and is dynamic, soft tissue release should be adequate. If, however, it is a fixed deformity then more major bony procedures will be required which may include metatarsal osteotomies or medial cuneiform open wedge osteotomy. Theses larger procedures should only be contemplated if the deformity is the cause of significant difficulty with shoe wear.

REFERENCES