Current Concept Review

Biomechanical Basis for Treatment of Pediatric Foot Deformities Part II: Pathomechanics of Common Foot Deformities

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Abstract:
The biomechanical basis for causation and treatment of pes planus, convex pes valgus (vertical talus), pes cavus, paralytic deformities, and foot deformities in cerebral palsy are presented. In each instance, the altered alignment of the hindfoot and forefoot, the configuration of the medial longitudinal arch and the instability of the talonavicular joint differ. The patterns of muscle imbalance across the axes of movement in paralytic deformities and the concept of treatment aimed at muscle rebalancing across these axes are graphically illustrated. The common deformities of the foot encountered in cerebral palsy are described and, in particular, the different deformity patterns resulting from spasticity of the gastrocnemius. The importance of preserving the function of the soleus and avoiding over-lengthening of the gastroc-soleus is emphasized.

Key Concept:
- Common deformities of the foot are complex and have deformity components involving the hindfoot, the forefoot, the longitudinal arch, the talonavicular joint and the alignment of the talus.

PES PLANUS (FLATFOOT)

Pes planus or pes planovalgus is characterized by loss of the medial longitudinal arch, hindfoot valgus, and forefoot abduction (Figure 1).

Factors that Contribute to Collapse of the Arch

Body Weight

The frequency of flatfoot increases as the Body Mass Index (BMI) increases.[1]

Figure 1. *Pes planus of the right foot in an adolescent following isolated paralysis of the tibialis posterior muscle due to poliomyelitis. The hindfoot valgus (A) and the forefoot abduction are evident (B, C).*

Extrinsic Muscles

Contractures of muscles attached to either end of the arch (tibialis anterior and the gastrocnemius-soleus) can flatten the arch (Figure 2). In clinical practice, a contracted gastrocnemius-soleus muscle is frequently encountered in children with flatfeet but may be overlooked unless passive dorsiflexion of the ankle is tested with the knee extended and the foot in inversion (Figure 3).

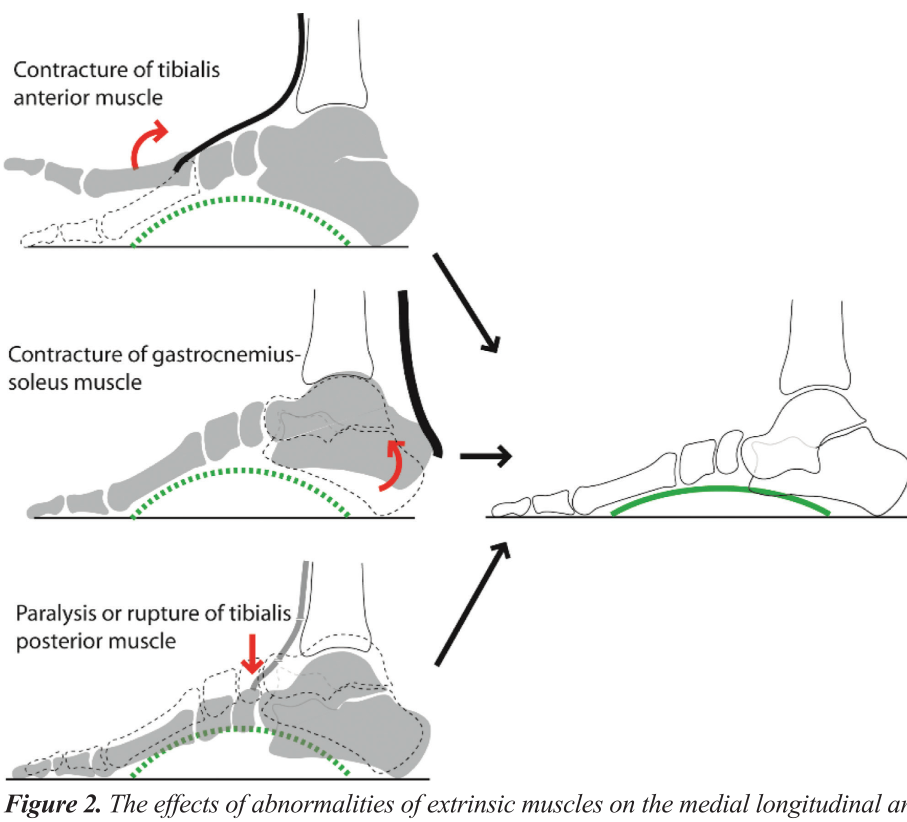


Figure 2. *The effects of abnormalities of extrinsic muscles on the medial longitudinal arch.*

Figure 3. *Technique of testing for contracture of the gastrocnemius-soleus with the knee extended and foot inverted.*

If the gastrocnemius-soleus is short, the hindfoot rolls into valgus and the arch collapses. Compensatory hindfoot valgus helps avoid an equinus posture and enables the heel to rest on the ground. This reduces the lever arm of the gastrocnemius-soleus with weakness of push-off and a typical flatfooted gait (Figure 4).

Active contraction of the tibialis posterior accentuates the arch and the arch collapses when the tibialis posterior is paralyzed (Figures 1 and 2).

Ligaments of the Foot

Flatfeet are common in syndromes with generalized ligament laxity. The plantar calcaneonavicular or spring ligament, stretching from the sustentaculum tali on the calcaneum to the navicular, is crucial in maintaining the arch. It forms an integral part of the acetabulum pedis and supports the head of the talus while weight-bearing. The support of the head of the talus by the spring ligament may be lost when the hindfoot is in valgus (Figure 5), and if the forefoot is abducted, the talus will then lie in complete plantarflexion (Figure 6).

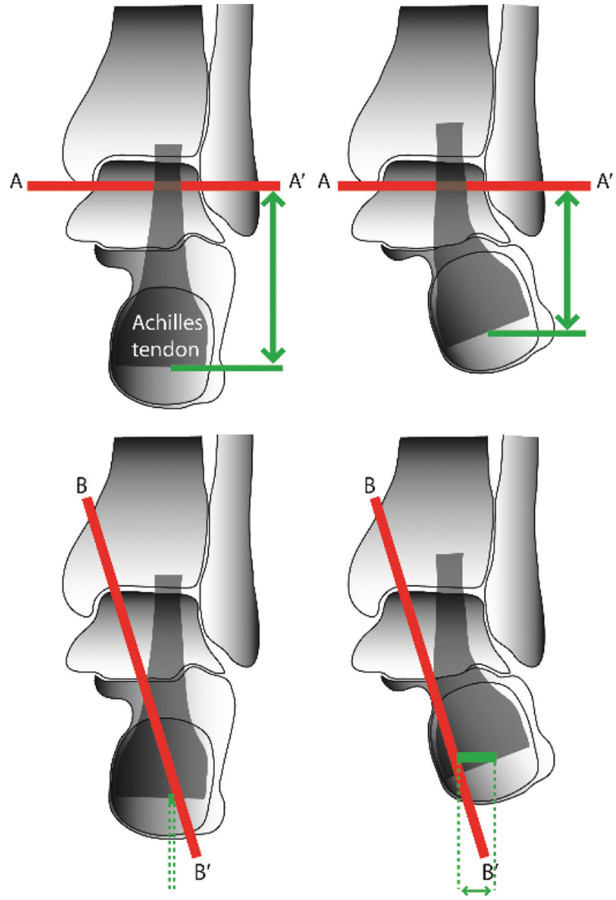


Figure 4. *A short gastrocnemius-soleus leads to a valgus hindfoot and short lever arm of the muscle between the axis of the ankle joint (AA') and the insertion of the Achilles tendon (green double arrow top right). The valgus moment of the muscle across the subtalar axis (BB') is also increased (bottom right).*

Biomechanical Basis and Rationale for Surgical Treatment of Flatfoot

Calcaneal Lengthening

Lengthening the lateral column of the foot to treat pes planus initially suggested by Dillwyn-Evans[2] and promoted by Mosca,[3] entails an open wedge osteotomy of the anterior end of the calcaneum. The anterior end of the calcaneum adducts along with the rest of the CPU restoring the support of the talar head by the spring ligament (Figure 7A, B, C). Restoration of the normal alignment of the talocalcaneonavicular joint results in a concomitant and obligatory reduction of the hindfoot valgus.

Triple C Procedure

The triple C procedure described by Rathjen and Mubarak[4] entails a medial displacement osteotomy of the calcaneum, an open wedge osteotomy of the cuboid, and a closed wedge osteotomy of the medial cuneiform (Figure 7D, E, F) which corrects the forefoot abduction and the hindfoot valgus.[5]

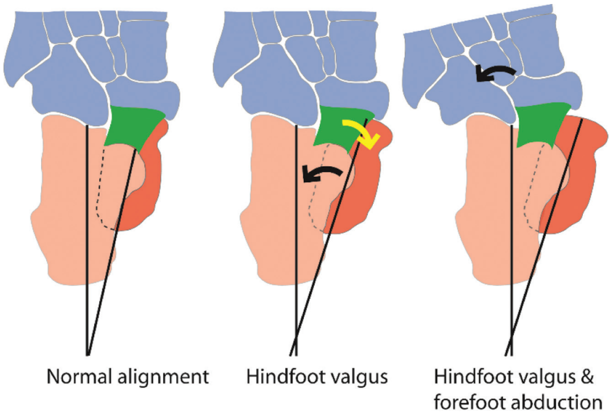


Figure 5. *The normal relationship of the tarsal bones viewed from the sole (left). A major portion of the head of the talus (dark orange) is not visualized, as it is resting on the plantar calcaneonavicular ligament (spring ligament) shown in green. When the hindfoot is in valgus, the calcaneo-pedal unit (CPU) abducts (black arrow middle figure). The spring ligament moves laterally along with the CPU, exposing the head of the talus (yellow arrow). In addition, if there is an abduction deformity of the forefoot (black arrow right), the greater part of the head of the talus is exposed and the support of the head of the talus by the spring ligament becomes tenuous.*

Arthroereisis

While not commonly performed by pediatric orthopaedists, the operation corrects hindfoot valgus by preventing the sinus tarsi from closing (Figure 8).

The factors that contribute to causation of pes planus and the characteristics of the deformity are summarized in Tables 1 and 2.

CONGENITAL VERTICAL TALUS (CONGENITAL CONVEX PES VALGUS)

Congenital vertical talus is a complex deformity involving the hindfoot and forefoot (Figure 9).

Deformities of the Hindfoot

The severe hindfoot valgus, hindfoot equinus, and forefoot abduction in congenital vertical talus are an exaggeration of that seen in pes planus. This results in complete loss of support of the talar head by the spring ligament, and the talus lies vertically in plantarflexion (Figures 5 and 6).[6,7]

Deformities of Forefoot

The tibialis anterior and the toe extensors are contracted, which is a feature not seen in pes planus. This results in dorsiflexion of the forefoot in relation to the hindfoot. The navicular moves dorsally and dislocates dorsally. Contracture of the peronei contributes to the abduction deformity of the forefoot and lateral dislocation of the navicular.

The combination of deformities leads to the rocker-bottom appearance (Figure 10).

The factors that contribute to causation of vertical talus and their consequences are summarized in Tables 1 and 2.

Congenital vertical talus may occur as an isolated anomaly, as part of arthrogryposis, or in association with

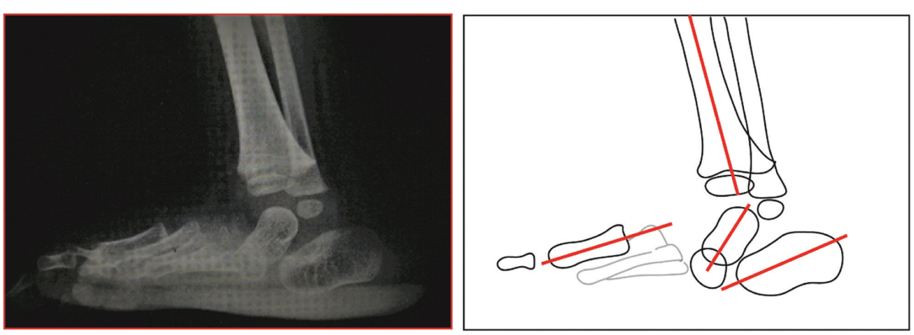


Figure 6. *Lateral view radiograph of the foot of a child with severe flatfoot. The talus is markedly plantarflexed, the talonavicular joint is dislocated, and the hindfoot is in valgus.*

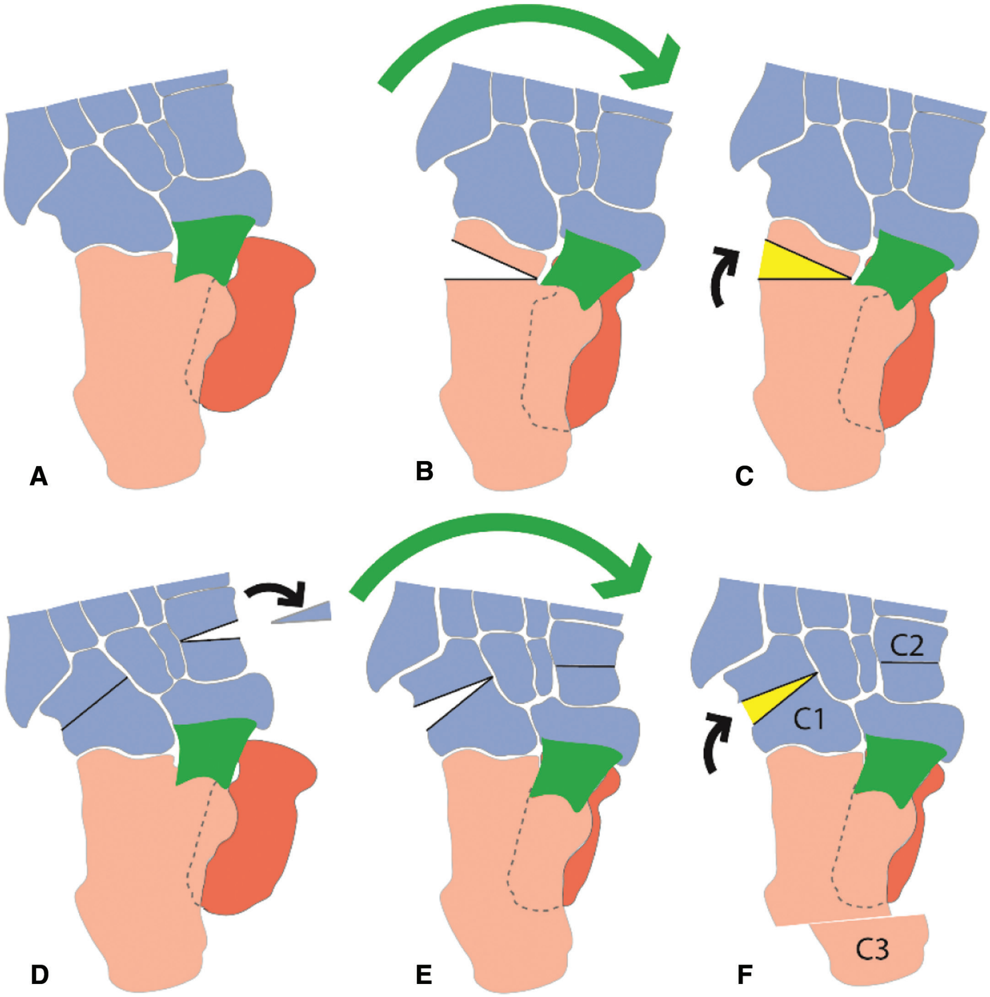


Figure 7. *Diagrammatic representation of calcaneal lengthening (A, B, C) and the triple C procedure (D, E, F) as viewed from the sole. Calcaneal lengthening involves an open wedge osteotomy of the calcaneum (B, C) held open with a bone graft (yellow). The triple C procedure involves an open wedge osteotomy of the cuboid, a closed wedge osteotomy of the medial cuneiform, and a medial displacement osteotomy of the calcaneum (D, E, F). By adducting the forefoot, the support of the talar head by the spring ligament (green) is restored.*

other syndromes. Paralytic congenital vertical talus may be seen in children with spina bifida.[8] The underlying cause will have a bearing on treatment; isolated congenital vertical talus is likely to be more amenable to nonoperative treatment such as the Dobbs method. Arthrogrypotic vertical talus would need surgical release while tendon transfers may be needed for paralytic vertical talus.

Treatment

Serial manipulation of congenital vertical talus is diametrically opposite to that of clubfoot. The forefoot is adducted to correct the forefoot abduction and concomitantly minimize the hindfoot valgus through the reorientation of the CPU.[9] The forefoot is also plantarflexed in an attempt to restore the arch of the foot. The talar head is used as a fulcrum while manipulating the forefoot.[9] Once the forefoot deformities improve, reduction of the talonavicular dislocation is attempted. Tenotomy of the Achilles tendon is almost always needed to correct the equinus deformity. In the more recalcitrant cases, transfer of the tibialis anterior to the neck of the talus has been recommended.[10] This removes

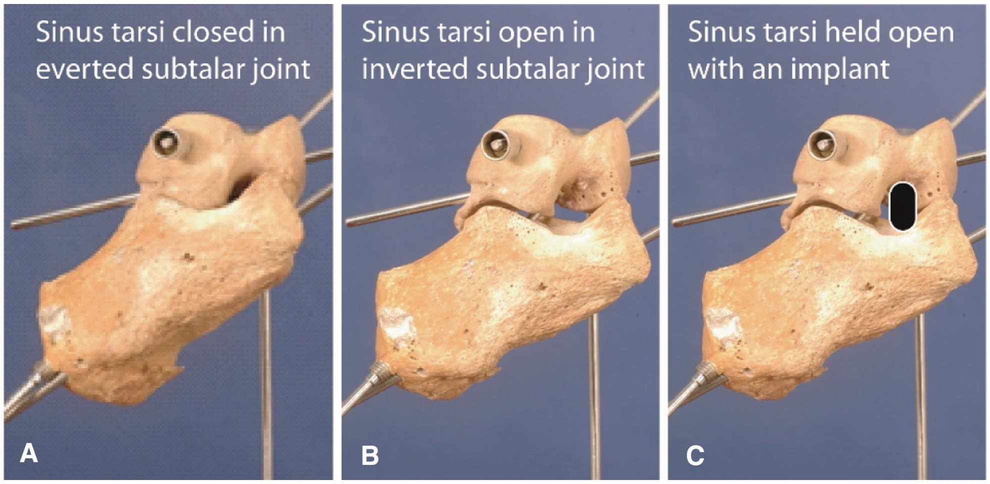


Figure 8. *Articulated model of the hindfoot bones shows that the sinus tarsi is closed when the subtalar joint is everted (A) and open when the joint is inverted (B). In arthroereisis, an implant placed in the sinus tarsi prevents the subtalar joint from everting (C).*

Table 1. Factors That Contribute to Deformity

Contributory Factors	Deformity	Structure Affected
Soft tissue contractures	Pes planus	Gastrocnemius-soleus muscle
	Vertical talus	Gastrocnemius-soleus muscle Ankle and toe dorsiflexors Peronei Capsular contractures
	Pes cavus	Tibialis posterior Plantar fascia
	Spastic equinovarus	Gastrocnemius-soleus Tibialis posterior
	Spastic equinovalgus	Gastrocnemius-soleus Peronei
Laxity of soft tissue	Pes planus	Spring ligament
	Vertical talus	Spring ligament Tibialis posterior
Muscle imbalance across joints	Pes planus	Paralysis / weakness of tibialis posterior
	Neurogenic vertical talus	Paralysis / weakness of tibialis posterior
	Pes cavus	Weakness of gastroc-soleus or tibialis anterior or intrinsic muscles
	Spastic equinovarus	Spasticity of gastrocnemius-soleus, tibialis posterior, tibialis anterior Weakness of peronei
	Spastic equinovalgus	Spasticity of gastroc-soleus, peronei Weakness of tibialis anterior & posterior

Table 2. Characteristics of Common Foot Deformities

Deformity	Hindfoot Alignment	Forefoot Alignment	Position of the Talus	Talocalcaneo-navicular Joint	Medial Longitudinal Arch	Spring Ligament
Clubfoot & Spastic equinovarus	Varus	Adduction	Plantar-flexed	Navicular subluxed medially	Accentuated	Contracted
Flatfoot & Spastic planovalgus	Valgus	Abduction	Plantar-flexed	Navicular subluxed dorsally & laterally	Collapsed	Stretched
Vertical talus	Valgus	Abduction	Severely plantarflexed	Navicular dislocated dorsally & laterally	Reversed	Severely stretched
Pes cavus	Neutral / Varus / Valgus	Neutral / Adduction	Normal / dorsiflexed	Navicular subluxed plantarward	Accentuated	Normal / contracted

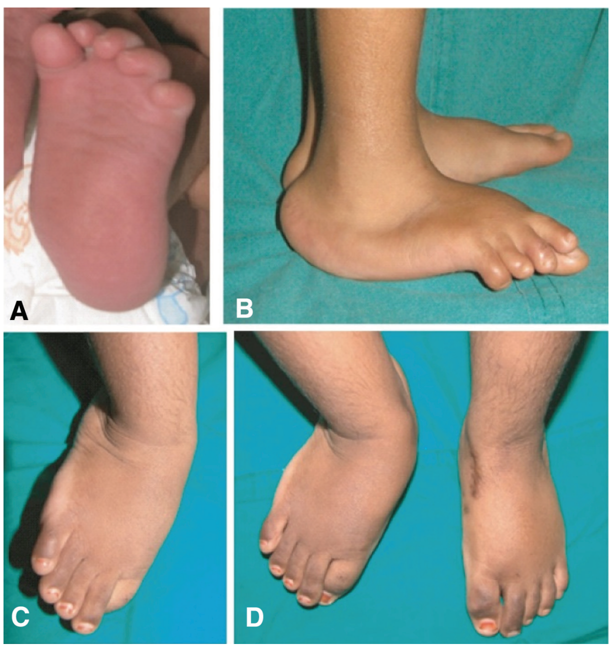


Figure 9. The deformities in congenital vertical talus include forefoot abduction (A) hindfoot equinus, forefoot dorsiflexion with reversal of the arch of the foot (B), hindfoot valgus and eversion of the foot (C). The medial border of the untreated right foot is convex while both borders of the left foot are straight following surgical correction (D).

a deforming force and converts it to a corrective force that pulls up the plantarflexed talus. Another salutary effect of this transfer is that it rebalances muscle forces acting on the first ray. Releasing the tibialis anterior from the first metatarsal results in unopposed action of the

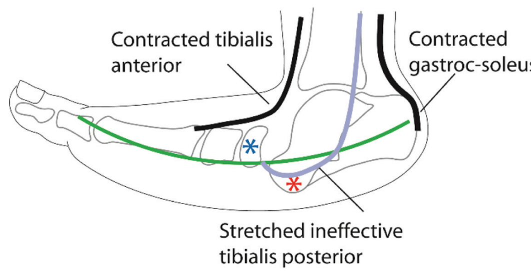


Figure 10. Contracture of both the ankle dorsiflexors and plantarflexor along with an ineffective tibialis posterior result in reversal of the longitudinal arch of the foot (green). The talus is severely plantarflexed with the head of the talus in the sole (red asterisk), and the navicular is dislocated dorsally (blue asterisk).

peroneus longus leading to plantarflexion of the first ray and accentuation of the longitudinal arch (Figure 11). This dynamic plantarflexion of the first ray will only occur if the peroneus longus is intact. Hence, if release of the peronei is deemed necessary, the peroneus longus can be divided proximally just above the ankle while the peroneus brevis is divided close to is insertion and the proximal stump of the peroneus brevis attached to the distal stump of the peroneus longus tendon.

PES CAVUS

Pes cavus is characterized by a high medial longitudinal arch that fails to reduce in height on weight-bearing. Pes cavus may be a congenital anomaly without any underlying neuro-muscular disorder; this form does not

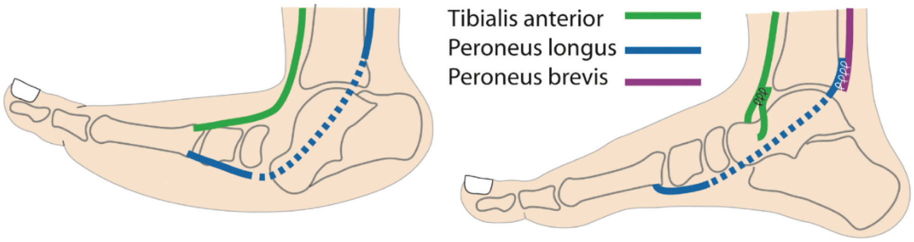


Figure 11. Transferring the tibialis anterior to the neck of the talus facilitates restoration of the medial longitudinal arch. The peronei are sectioned at different levels and anchored to each other ensuring continuity of the peroneus longus tendon so that it can exert a plantarflexor force on the first metatarsal.

progress and is usually asymptomatic. Neuromuscular pes cavus is, by far, more common and can occur in Charcot Marie Tooth disease (Hereditary Motor Sensory Neuropathy [HMSN]), poliomyelitis, spinal dysraphism. spina bifida, cerebral palsy, and Friedreich's ataxia.[11]

Neuromuscular Pes Cavus

Neuromuscular pes cavus is associated muscle imbalance across the ankle, subtalar or metatarsophalangeal joints.

Patterns of Deformity

The longitudinal arch gets accentuated due to plantarflexion of the metatarsals or dorsiflexion of the calcaneum (Figure 12).

Apart from accentuation of the medial longitudinal arch, other deformities of the hindfoot and forefoot may be present. The pattern of these deformities is dictated by the pattern of muscle imbalance (Table 3).

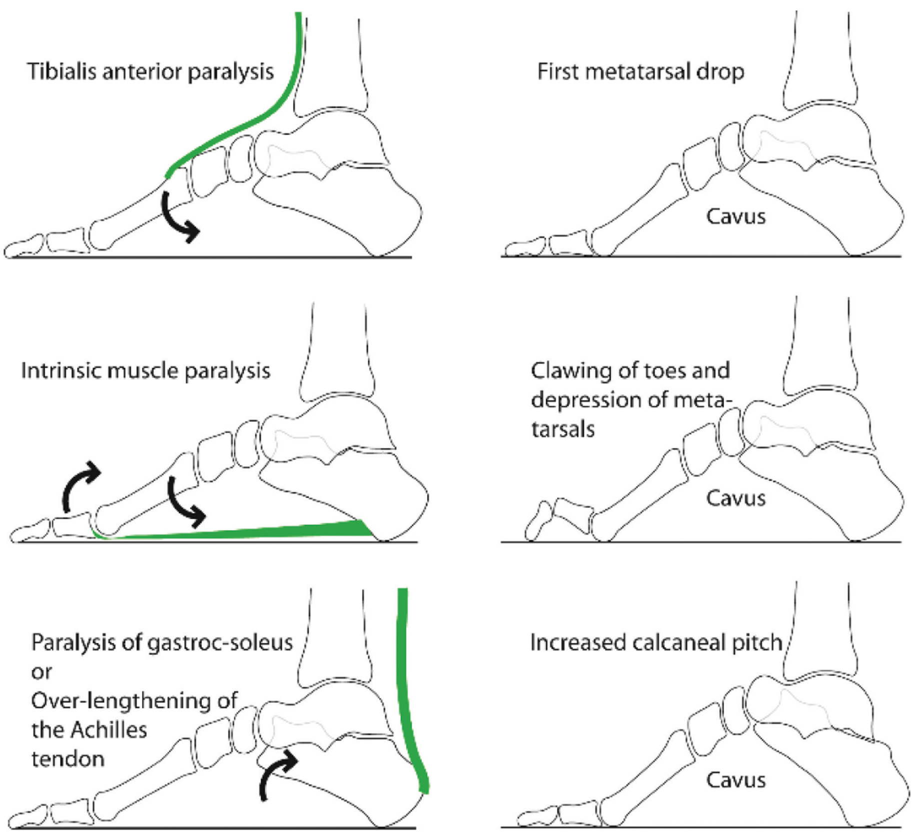


Figure 12. Mechanisms by which muscle paralysis can lead to a pes cavus deformity.

Table 3. Patterns of Paralysis and Paralytic Deformities Associated with Pes Cavus

Joint	Muscle Paralyzed	Deformity
Ankle	Tibialis anterior	Equinocavus
	Gastrocnemius-soleus	Calcaneocavus
Subtalar joint	Peronei	Cavovarus
	Tibialis posterior	Cavovalgus
Ankle & subtalar joint	Tibialis anterior & peronei	Equinocavovarus
MTP joints	Intrinsic muscles	Claw toes with secondary cavus

In addition to muscle imbalance causing deformity, a forefoot deformity may induce a compensatory hindfoot deformity (Figures 8 and 9). Forefoot pronation secondary to a dropped first metatarsal seen in association with a compensatory hindfoot varus in HMSN is an example of this phenomenon. Primary and compensatory deformities are supple to begin with but tend to become rigid. The treatment strategies will vary depending on whether the deformity is supple or rigid. Differentiating a supple hindfoot varus from a rigid varus is possible by performing the Coleman block test (Figure 13).[12]

Sensory Loss on the Sole in Association with a Cavus Deformity

Loss of sensation associated with a cavus deformity may occur when there is a lesion involving the first sacral segment of the spinal cord (e.g., as in low spina bifida). Loss of pain sensation alone with a cavus foot may be encountered in syringomyelia. A cavus deformity *per se* can result in high plantar pressures, increasing the risk of neuropathic ulceration. In addition, if there is a hindfoot varus or valgus deformity, the risk of developing plantar ulcers is much higher. In children with paralysis of the gastroc-soleus, the normal rockers of the foot in the stance phase are not seen, instead, there is uncontrolled dorsiflexion of the ankle after heel-strike. This results in shearing forces on the plantar surface of the heel which can result in ulceration (Figure 14).

Angles measured on weight-bearing lateral radiographs will show the abnormal alignment of the hindfoot (Figure 15).[13]

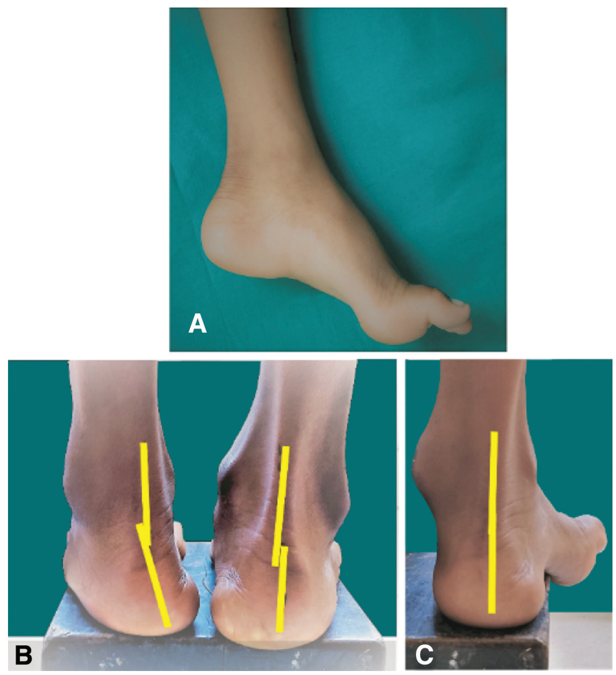


Figure 13. *Appearance of left foot of a boy with a paralyzed tibialis anterior muscle (A). The first metatarsal is plantarflexed, the medial longitudinal arch is accentuated. Coleman Block test (B & C): On standing with both feet on the block, the left heel rolls into varus to enable the pronated forefoot to rest on the ground (B). When the plantarflexed first ray is not supported by the block and hangs free off the edge of the block, the normal alignment of the hindfoot is restored (C), indicating that the hindfoot deformity is supple.*

Treatment of Pes Cavus

The deformity may be corrected by releasing soft tissue contractures (e.g., plantar fascia and the intrinsic muscles). Bony surgery will be needed in long-standing cases (dorsal wedge osteotomy of the base of the first

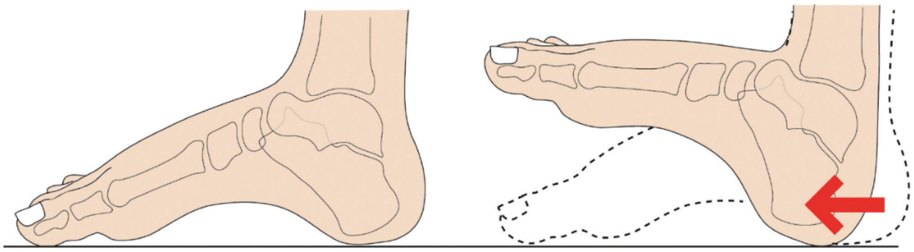


Figure 14. *In children with paralysis of the gastroc-soleus, the normal rockers in the stance phase are not seen. Uncontrolled dorsiflexion of the ankle produces shear forces under the heel (red arrow).*

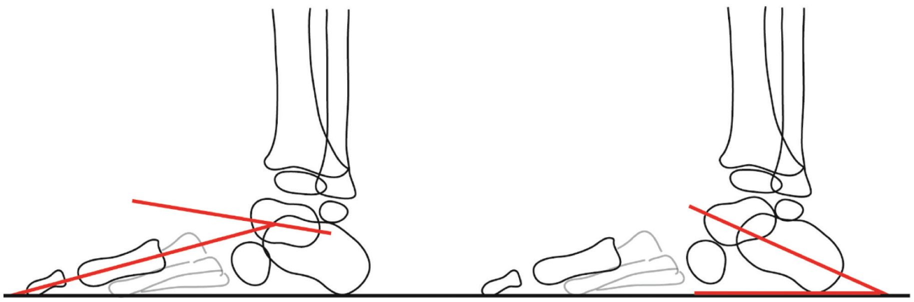


Figure 15. *Meary's angle (left) of 15-30 degrees is considered moderate cavus, and an angle greater than 30 degrees is regarded as severe cavus. Calcaneal pitch (right) > 30 degrees is indicative of pes cavus.*

metatarsal or the medial cuneiform and medial and proximal displacement osteotomy of the calcaneum).[14] The tendency for progression or recurrence of deformity can be minimized by restoring muscle balance (Table 4).

Neuropathic ulceration in children with sensory loss may be prevented by correcting deformities and making the foot plantigrade and restoring power of plantarflexion with an early tendon transfer to reduce shear forces under the heel.

Table 4. Rationale of Tendon Transfers for Pes Cavus[15,16]

Tendon Transfer	Rationale
Transfer of peroneus longus to the peroneus brevis	Removes the muscle causing the first metatarsal to drop when the tibialis anterior is weak
	Retains evertor power
Jones transfer (transfer of the extensor hallucis longus (EHL) tendon to the neck of the first metatarsal)	The transferred EHL elevates the metatarsal countering the metatarsal drop in the presence of tibialis anterior weakness
Hibbs transfer of the extensor digitorum longus (EDL) tendons to the 2nd to 5th metatarsal necks	The transferred EDL elevates the metatarsals
Transfer of the flexor hallucis longus (FHL) from the distal phalanx to the proximal phalanx	The transfer reduces clawing of the hallux
Tibialis posterior muscle transfer to the dorsum of the foot	The transfer augments the power of ankle dorsiflexion in children with a weak tibialis anterior muscle
	The transfer removes a deforming force if the hindfoot is in varus

PARALYTIC DEFORMITIES OF THE FOOT

Paralytic deformities of the foot develop if there is muscle imbalance across the axis of the ankle joint or the subtalar joint. The patterns of deformities are quite predictable when a particular muscle is paralyzed and depends on the relationship of tendons to the axes of the ankle and subtalar joints. The further a tendon runs from the axis of the joint, the greater the force moment that is generated by the muscle (Figure 16). A clear understanding of this concept is very useful for understanding why a deformity has developed and it is also vital for planning treatment.

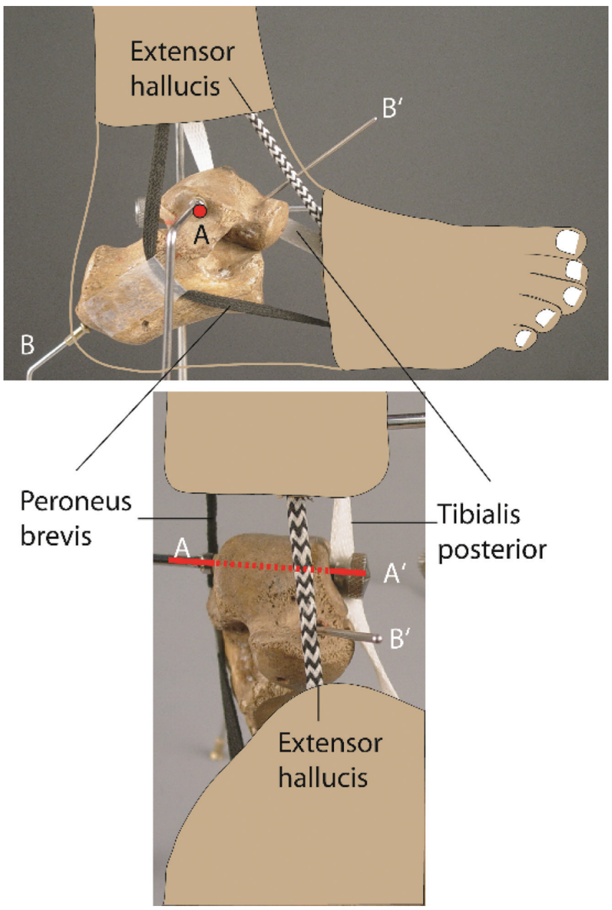


Figure 16. Articulated model of the hindfoot showing the relationship of three tendons to the axes of the ankle (AA') and subtalar joints (BB'). The extensor hallucis has a good dorsiflexor moment as it runs anterior to the axis of the ankle joint (AA' above), but it has virtually no inversion or eversion moment as it runs almost in line with the subtalar axis (B' below). The peroneus brevis and the tibialis posterior have strong evertor and invertor moments, respectively, as they run at a distance from the subtalar axis.

The normal relationship of the tendons crossing the ankle and subtalar joint can be depicted diagrammatically as shown in Figure 17.

Tendons that run anterior to the axis of the ankle are dorsiflexors while those that run posterior to this axis are plantarflexors of the ankle. Similarly, all tendons running medial to the subtalar axis are invertors and tendons running lateral to the subtalar joint are evertors (Figure 18).

The same template is used to plan tendon transfers to retore muscle balance across these axes and correct paralytic deformities (Figures 19 and 20).

The two examples of planning tendon transfer around the foot and ankle in Figures 19 and 20 show how the

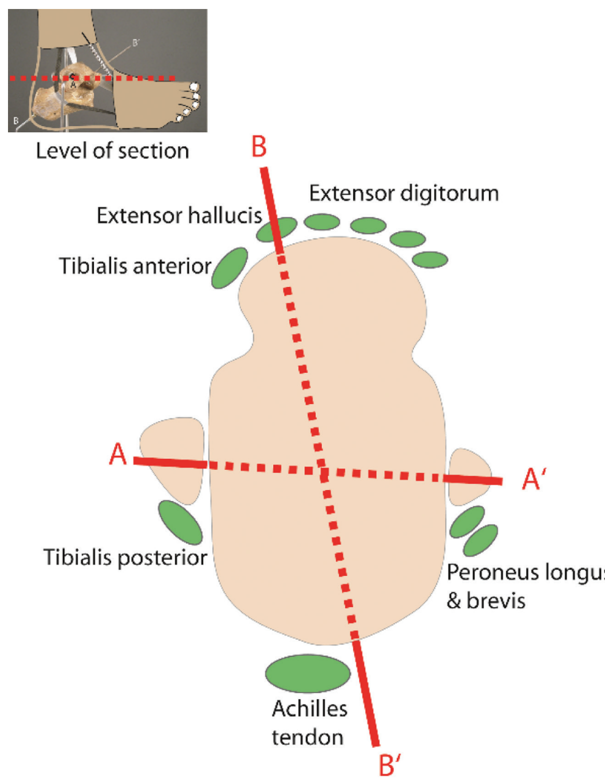


Figure 17. *Diagrammatic representation of a horizontal section through the ankle at the level of the malleoli. The axis of the ankle joint (AA'), the axis of the subtalar joint (BB'), and the position of tendons crossing the ankle are shown.*

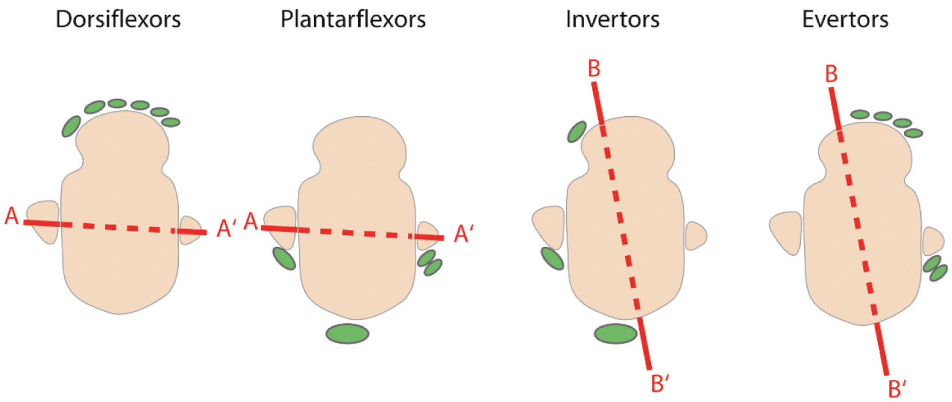
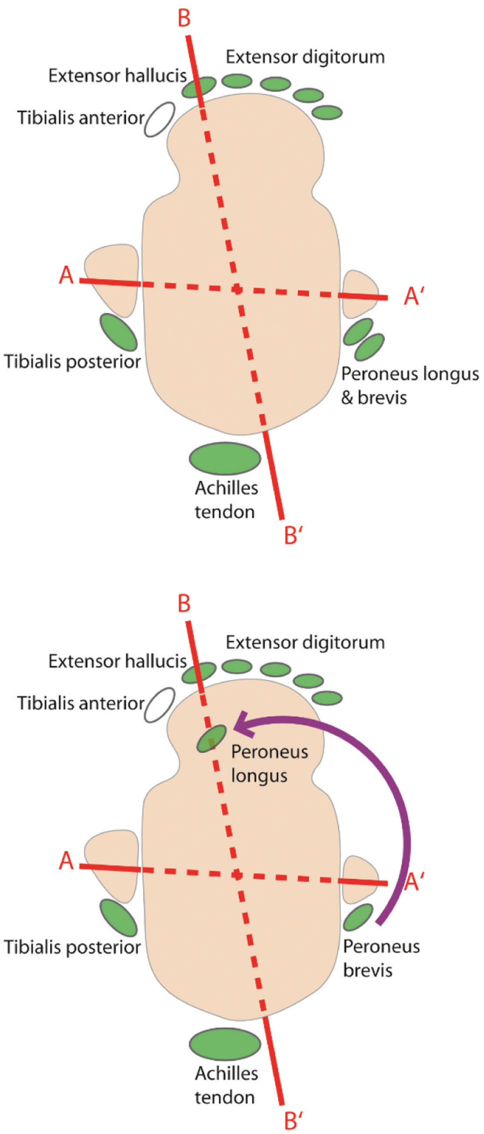


Figure 18. Action of muscles and their relationship to the axes of the ankle and subtalar joints.



ISOLATED TIBIALIS ANTERIOR PARALYSIS

Deformity:
First metatarsal drop
Medial cavus
Forefoot pronation

Muscle imbalance:
Plantarflexors stronger than dorsiflexors
Evertors stronger than invertors

Tendon transfer:
Peroneus longus transfer to the dorsum of the foot

Site of attachment:
In line with the subtalar axis

Effect of the transfer:
First metatarsal drop corrected
(by removing plantarflexor force of peroneus longus on first metatarsal)

Muscle balance restored across axis of ankle
Dorsiflexor power augmented
Plantarflexor power reduced

Muscle balance restored across subtalar axis
Evertor power reduced

***Figure 19.** Rationale of peroneus tendon transfer for isolated tibialis anterior paralysis.*

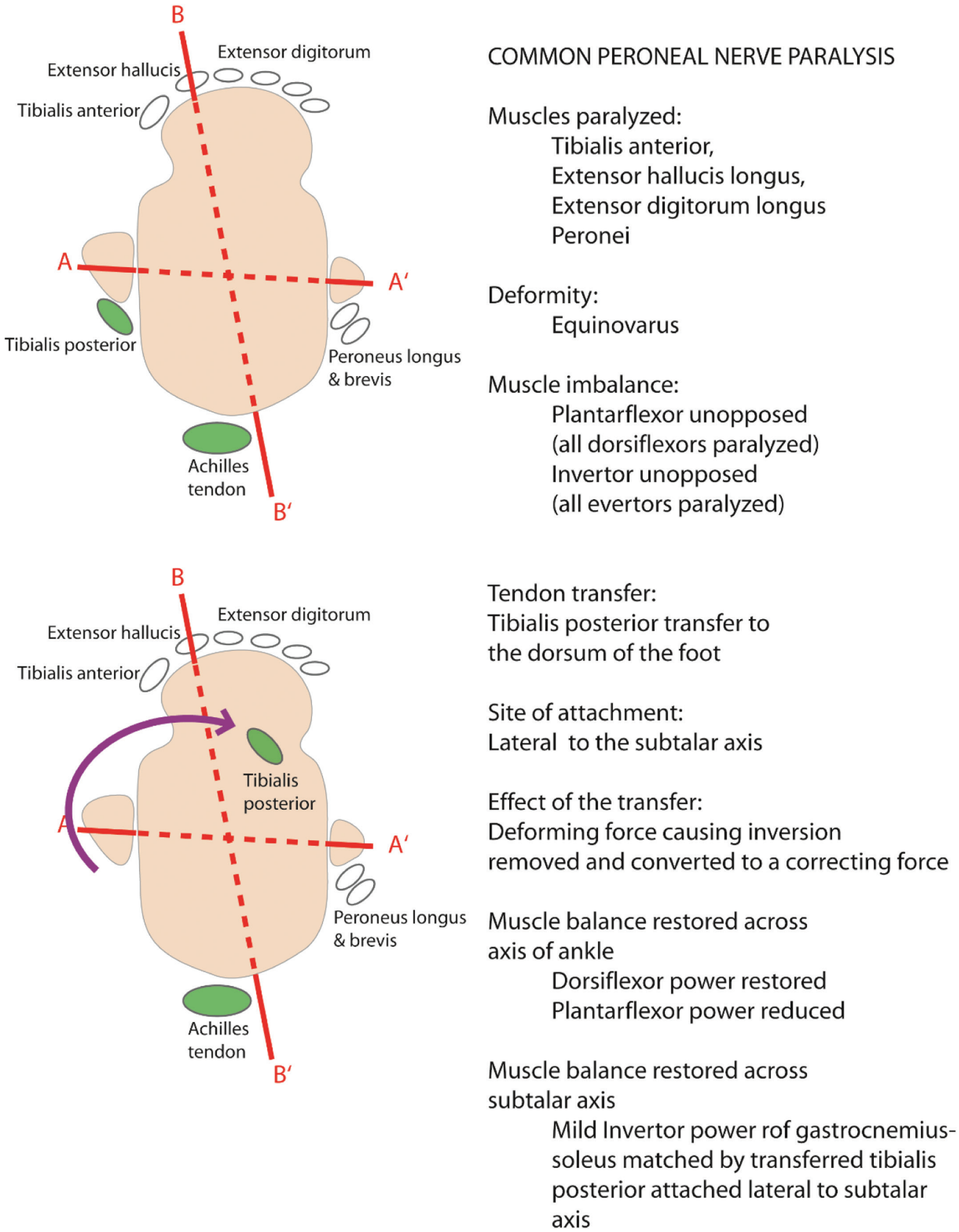


Figure 20. *Rationale of performing a tibialis posterior transfer for peroneal nerve palsy.*

underlying muscle imbalance can be corrected with appropriate tendon transfers.

Intrinsic Muscle Paralysis

Paralysis of intrinsic muscles of the foot will result in clawing of the toes where the metatarsophalangeal joint (MTP) is hyperextended and the interphalangeal joints (IP) is flexed.[17] Clawing can be reversed by preventing hyperextension of the MTP joint by pulling up the metatarsal or flexing the proximal phalanx by performing simple tendon transfers (Figure 21).

FOOT DEFORMITIES IN CEREBRAL PALSY

Foot deformities are very common in children with cerebral palsy, and they arise due to muscle imbalance across the joints of the foot secondary to spasticity and muscle weakness.[18] The deformities tend to be sequential and progressive with growth. Initially, the deformities

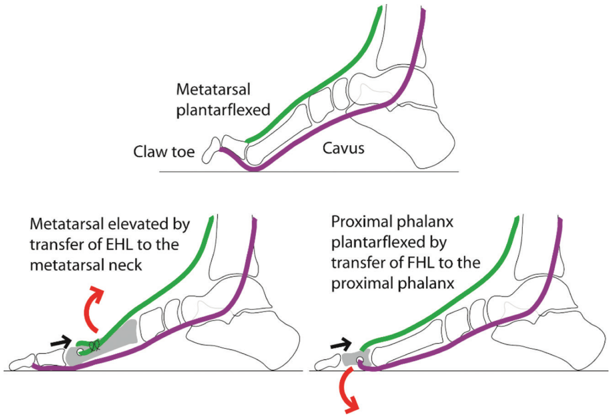


Figure 21. *Tendon transfers for correcting clawing of the great toe.*

are flexible and dynamic, and over time, myostatic contractures develop in the spastic muscles. The deformities then become more rigid and if left untreated, structural skeletal abnormalities develop.

The consequences of foot deformities in ambulatory children with cerebral palsy include lever arm dysfunction, poor power generation, and increase in energy expenditure during gait. The energy inefficiency of walking, in turn, leads to reduction in the ambulatory capacity of the children.[18]

The three common foot deformities noted in cerebral palsy are equinus, equino planovalgus, and equino-cavovarus.

Equinus & Equino Planovalgus Deformities

Equinus arises due to spasticity of the gastroc-soleus muscle. The relative contributions of the gastrocnemius and soleus differ based on the type of cerebral palsy. Gastrocnemius is the main contributor in spastic diplegic cerebral palsy while both gastrocnemius and soleus are spastic in hemiplegic cerebral palsy. Spasticity of either component of the gastroc-soleus can initially result in dynamic equinus, where the child walks on his or her toes but stands with the heel resting on the ground (Figure 22A-C).

The effect of a contracture of the gastrocnemius can manifest in different ways. Firstly, the child may stand and walk throughout the stance phase of gait with the foot in equinus with only the toes resting on the ground. Secondly, a mid-foot break may develop. The forefoot dorsiflexes while the hindfoot remains plantarflexed (Figure 22D-F), the medial longitudinal arch is reversed and only the forefoot rests on the ground. Thirdly, the hindfoot may develop a valgus deformity secondary to the contracted gastrocnemius and this enables the entire sole to rest on the ground (Figure 22G-22I). Often, a mid-foot break and hindfoot valgus develop concomitantly in older children with spastic diplegia (Figure 23). This equino planovalgus deformity of cerebral palsy has features similar in many ways to a severe planovalgus deformity with a tight Achilles tendon in otherwise normal children.

The valgus of the hindfoot and forefoot abduction lead to loss of support of the talar head and subluxation of the talonavicular joint. The hindfoot valgus leads to a reduction of the lever arm of the gastroc-soleus resulting in weakness of push off. The factors that contribute to development of equino-plano-valgus deformity in cerebral palsy are shown in Table 1 and 2.

Treatment

For young children 8 and below with flexible dynamic equino-plano-valgus:

Bracing using an ankle foot orthosis and physical therapy are the primary treatment options. If the response to these measures is poor, chemo-denervation of the gastrocnemius, serial casting followed by physiotherapy and bracing are tried.

For children over 8 years with a gastrocnemius contracture and fixed equino-plano-valgus:

Lengthening of the gastroc-soleus needs to be undertaken with caution as overcorrection (Figure 24) or undercorrection may occur.[19]

In spastic diplegia, the gastrocnemius is contracted and usually the soleus is not. Consequently, unnecessary lengthening of the soleus, an important power generator, should be avoided as it can lead to iatrogenic crouch gait. Gastrocnemius recession is usually the procedure of choice. In patients with hemiplegia, both the

Figure 22. *Consequences of spasticity and contracture of the gastrocnemius muscle. Dynamic equinus in a three year old (A-C), mid-foot break in a seven year old (D-F), and valgus deformity of the hindfoot (G-I) in a fourteen year old.*

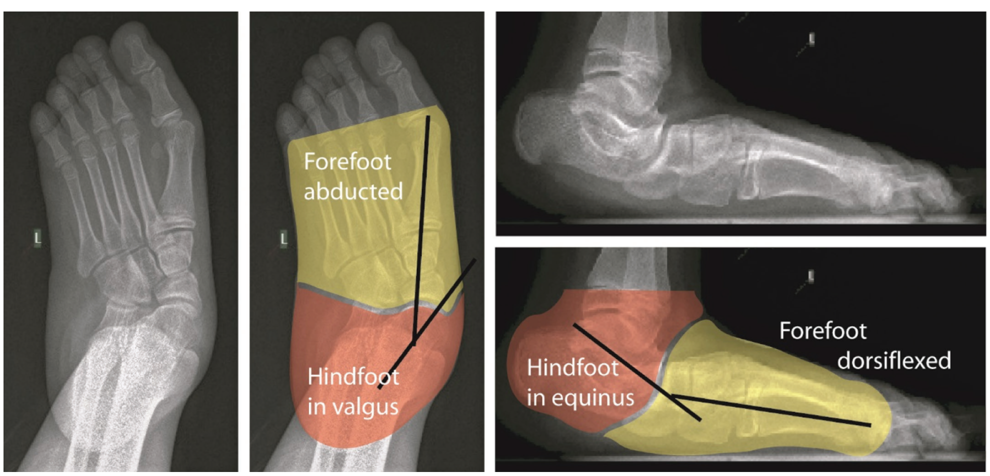


Figure 23. *Forefoot abduction and forefoot dorsiflexion associated with equinus and valgus of the hindfoot and a mid-foot break seen on weight-bearing radiographs of an adolescent with cerebral palsy.*

Figure 24. *Severe calcaneus deformity in an adolescent who had undergone lengthening of the Achilles tendon.*

gastrocnemius and the soleus are often contracted and, hence, lengthening of the Achilles tendon is justified.

For children over 12 years with rigid deformity:
Soft issue surgery as above and bony surgery as outlined in Table 5.

There are some reports of satisfactory outcomes of subtalar arthroreisis, but the procedure has not been shown to be successful in other studies.[24] Consequently, the procedure is not widely used for spastic equino-plano-valgus.

Spastic Equinovarus

Spastic equinovarus deformity is most frequently seen in hemiplegic cerebral palsy (Figure 25).

Table 5. Indications and Rationale of Operative Procedures for Equino-Plano-Valgus

Indications	Procedure	Biomechanical Rationale	Comment
Mild or moderate spastic plano-valgus Ambulatory child	Calcaneal lengthening[20,21]	Corrects forefoot abduction and hindfoot valgus	Controversy as to whether it is effective if deformity is severe[22,23]
Moderate spastic plano-valgus Ambulatory child	Calcaneal lengthening + Talonavicular capsular reefing + Advancement of tibialis posterior	Addresses soft tissue laxity in the medial column	Useful if forefoot abduction is not fully corrected following calcaneal lengthening
Persistence of forefoot abduction after calcaneal lengthening Poor ambulatory capacity	Calcaneal lengthening + Talonavicular arthrodesis	Corrects forefoot abduction and provides stable medial column	Minimizes risk of recurrence of forefoot adduction
Severe spastic equino-plano-valgus	Triple C procedure	Individual osteotomies can be adjusted to deal with varying severity of hindfoot valgus, planus and forefoot abduction	Preserves joint mobility
Severe deformity and limited ambulatory capacity	Subtalar arthrodesis	The hindfoot deformities can be well corrected	Loss of flexibility Low chance of recurrence
Limited ambulatory capacity Severe deformity not amenable to other procedures	Triple arthrodesis	All deformities can be addressed at the respective joints at which they occur	Stiffens the foot

Figure 25. *Severe spastic equinovarus in child with hemiplegic cerebral palsy.*

Table 6. Indications and Rationale of Operative Procedures for Spastic Equinovarus

Indications	Procedure	Biomechanical rationale	Comment
Young child Supple deformity (no contracture)	Physiotherapy Chemo-denervation of gastrocnemius, tibialis posterior and anterior Orthosis	Reduction of spasticity of deforming muscles & restore muscle balance	
Early contractures Deformity partially correctable	Split transfer of the tibialis anterior (TA) or tibialis posterior (TP) to the lateral border of the foot	Restores muscle balance	Choosing which tendon to transfer (TA or TP) is based on their relative contribution to the deformity.[25,*] Split tendon transfers are preferred to whole tendon transfers to avoid risk of overcorrection.
Equinovarus deformity associated with skeletal deformity	Muscle rebalancing + Mid-tarsal osteotomy + Calcaneal osteotomy	Corrects forefoot adduction and hindfoot varus	Unlike for pes plano-valgus no single osteotomy can achieve correction; multiple osteotomies may be required to improve alignment**
Severe, rigid deformity in adolescent	Muscle rebalancing + Triple arthrodesis	All the deformities can be addressed	Severe deformity can be corrected but foot is rendered stiff

**: This can be evaluated by a combination of clinical examination, observational and instrumented gait analysis, and EMG studies. TA overactivity is treated with a split tibialis anterior tendon transfer (SPLATT). TP overactivity is treated with a split tibialis posterior tendon transfer (SPLOTT). If both invertors show overactivity during gait on EMG, a SPLATT is combined with an intra-muscular lengthening of the TP.*[26]
***: Surgeries for hindfoot correction include lateral slide osteotomy or a lateral closing wedge osteotomy of the calcaneus. Surgeries for rigid forefoot adduction and supination include dorsolateral closing wedge cuboid osteotomy and medial opening wedge osteotomy of the medial cuneiform.*

It develops due to spasticity of the gastro-soleus and the invertors of the foot with weak dorsiflexors and evertors.

Treatment of spastic equinovarus has been summarized in Table 6.

Summary

In Part 1 we outline the normal foot mechanics and relationship between the hindfoot and the forefoot. As such, a basic understanding of the normal mechanics of the foot is needed to recognize the abnormal forces which lead to foot deformity. In the current Part 2 we identify common foot deformities which result from altered mechanics and present different options in order to correct. The factors that contribute to the causation and the cardinal clinical features of common deformities of the foot in children are summarized in Tables 1 and 2.

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