Clinical Article

The orthopaedic management of myelomeningocoele

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Abstract

Myelomeningocoele is the commonest congenital birth defect.[1] The Cape Town prevalence has been reported as 2.5 per 1 000 births in the white population and 1 per 1 000 births in the black and coloured population.[2] The incidence at Red Cross Children's Hospital has remained static at a mean of 12 new patients a year since 1987. Although this is a relative decrease, it remains significantly high. Part of the reason is that the foetal anomaly ultrasound (and elective termination) available to all antenatal patients in the Western Cape is not utilised by unbooked patients. The incidence of elective termination in the USA is 23%.[3]

Myelomeningocoele requires a multidisciplinary approach. At Red Cross Children's Hospital the weekly spinal defects clinic involves neurosurgeons, urologists and stomatherapists (who teach the patients bladder and bowel care), orthopaedic surgeons and orthoptists. Neurosurgeons do the primary closure and insert and maintain a ventriculo-peritoneal shunt in 95% of the patients who have hydrocephalus due to an Arnold-Chiari malformation. Urologists treat the ninety per cent of patients who are incontinent. Orthopaedics has protean applications in myelomeningocoele.

Key words:
Myelomeningocoele, orthopaedic management

Goals of treatment

The patient's needs in order of importance are:[4] a stable self-image, an adult sexual role, independent communication, daily living activities, a career and mobility. It is important to note that walking ability is last on this list and that orthopaedic management should be realistic, keeping in mind the patient's walking potential.

Walking potential

The **ambulatory status** was defined by Hoffer[5] *et al*. A functional walker implies a community or household walker, while a non-walker is permanently in a wheelchair or walks with assistance during exercise only.

The patient's **walking or ambulatory potential** is mainly determined by the **neurological level**.[6,7] Spasticity, obesity and deformity can also influence walking ability. Babies should be assessed several times before the parents are given a prognosis for walking potential.

Level is defined as the lowest level of voluntary power equal to or more than MRC 3/5. Spasticity and skip lesions can rarely occur below this level, but are rare.[8] *Table I* gives the incidence per level as seen in our unit.

The walking potential for each neurological level is shown in *Table II*. The presence or absence of quadriceps determines whether the patient will walk or not. Without quadriceps the patient is paraplegic.

Table I: Incidence of patients per neurological level

Neurological level	Patients (%)
Paraplegic (thoracic, lumbar 1 and 2)	23
Midlumbar (lumbar 3 and 4)	30
Calcaneus (lumbar 5)	17
'Normal' (sacral 1)	30

Midlumbar (L3 and L4) patients walk with ankle-foot orthoses (AFOs) as they have flail feet, and require crutches as they have no hip abductors (gluteus medius, L5) and no hip extensors (gluteus maximus, S1). Midlumbar (L4) patients will walk into adulthood because they have medial hamstrings which contribute one third of hip extension power.[9] In my experience patients with L3 level seldom walk as adults. Midlumbar patients learn to 'backset' their hips with a baby walker and then graduate to AFOs and crutches.

Table II. Walking potential for each neurological level

Level	Function	Muscle	Walking potential
Thoracic	Paraplegic		Non-walker
Lumbar 1, L2	Paraplegic, hip flexion	Psoas	Non-walker
Lumbar 3	Knee extension	Quadriceps	33% walk at 4-5 years
Lumbar 4	Knee extension	Quadriceps	100% walk at 3-4 years
	Knee flexion	Medial hamstrings	
Lumbar 5	Ankle dorsiflexion	Tibialis anterior	100% walk at 2-3 years
Sacral 1	Ankle plantarflexion	Gastrosoleus	100% walk at 1-2 years

Orthopaedic applications

The orthopaedic problems encountered for each level of involvement are given in *Table III*.

Table III. Orthopaedic problems for each neurological level

Level	Deformity	Treatment	Age (years)
Paraplegic	Scoliosis	Instrumentation and fusion	>10
	Kyphosis	Kyphectomy, instrumentation, fusion	>10
	Equinus	Achilles tenotomy or posterior release	1
Midlumbar L3, 4	Hip dislocation	Nil	
	Internal tibial torsion	Supramalleolar derotation osteotomy	> 5
	Club foot	Ponseti, posterior release or PMR	< 1
	Cong. vertical talus	Anterior and posterior release	1
	Ankle valgus	Medial tibial stapling or varus osteotomy	≥10
Calcaneus L5	Calcaneus	Tibialis ant. transfer or anterior release	≤ 4
	Ankle valgus	Stapling or osteotomy	≥10
	Hindfoot valgus	Calcaneal lengthening	≥ 10
'Normal' S1	Cavovarus	Plantar release ± first metatarsal osteotomy ± calcaneal osteotomy	> 5

Spine

Scoliosis and kyphosis occur almost exclusively in the paraplegic group.

Scoliosis

The incidence of scoliosis in our unit is 8% of paraplegic patients but the literature reports up to 80%.[10] It occurs by the age of 10 years. Scoliosis results in pelvic obliquity with subsequent loss of sitting balance or loss of functional paraplegia as the patients require their arms to keep themselves upright. Pelvic obliquity can also cause an ischial trophic ulcer which can be very recalcitrant to treatment. Severe scoliosis decreases lung capacity.

The treatment is surgical if the curve exceeds 50°. A pre-operative MRI is indicated to rule out a neurosurgical cause such as syringomyelia or tethering of the cord. The current policy is to fuse the entire segment from T2 to the pelvis.[11] This corrects the pelvic obliquity and prevents non-union in the area of dysraphism but decreases the lumbosacral mobility which has been reported to result in decreased ability for wheelchair transfer and an increase in trophic ulceration.[12]

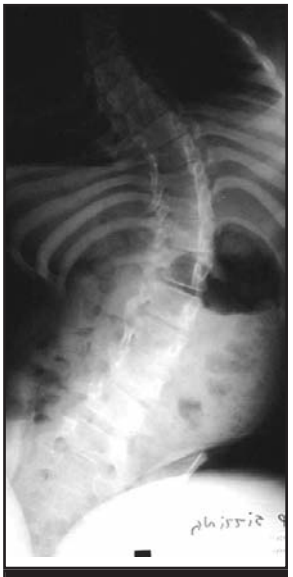


Figure 1 A.
Pre-operative A-P radiograph of a 12-year-old paraplegic patient with scoliosis affecting sitting balance

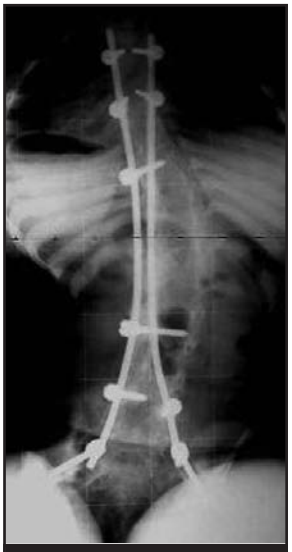


Figure 1 B.
Radiograph after anterior interbody release and fusion, followed by posterior fusion and instrumentation with pedicle screws and rods

Scoliosis results in pelvic obliquity with subsequent loss of sitting balance or loss of functional paraplegia

Posterior fusion has evolved from Harrington rods to Luque instrumentation to the current pedicle screws and rods. The high incidence of non-union because of the absent posterior elements necessitated anterior instrumentation such as Dwyer or Zielke. Currently anterior interbody release and fusion precedes posterior pedicle screws and rods (*Figures 1 A and B*).

Kyphosis

Kyphosis occurs in 8% of paraplegic patients in our unit. Due to the severe lateral displacement of the posterior elements, the erector spinae become perverted flexors. The severe kyphosis leads to loss of sitting balance but the main morbidity is the recurrent ulceration due to the underlying bony prominence.

To obviate this significant and costly problem, the best treatment would be to excise the vertebrae involved in the kyphus and fix the spine with tension band wiring at the primary closure soon after birth. However, due to lack of neurosurgical and orthopaedic communication, this does not seem to occur. The patient is left to around 10 years of age in order to obtain as much body height as possible from lumbar spine growth. A kyphectomy and posterior rodding is then performed (*Figures 2 A and B*).

Figure 2 A.
Pre-operative lateral radiograph of a 10-year-old paraplegic patient with a kyphus which caused recurrent episodes of trophic ulceration

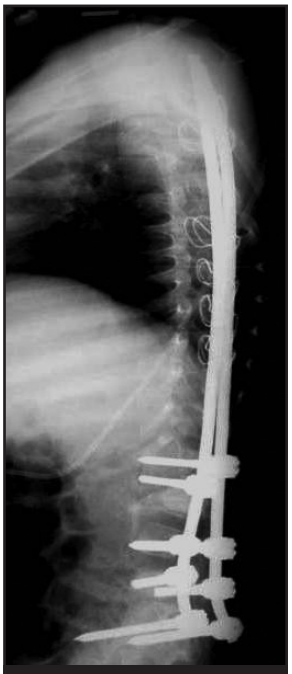


Figure 2 B.
Radiograph after kyphectomy, posterior fusion and instrumentation with rods, pedicle screws and sublaminar wiring

Hip

In the midlumbar (L3 and 4) myelomeningocoele paralytic sub- or dislocation of the hip occurs due to muscle imbalance. Absent abductors (gluteus medius L5) and extensors (gluteus maximus SI) with unopposed adductors and flexors result in forces pushing the hip laterally and superiorly. Secondary bony changes (increased valgus and anteversion of the femoral neck and acetabular dysplasia) result in hip sub- and dislocation. This mechanism is also responsible for the hip instability in cerebral palsy, Charcot-Marie-Tooth neuropathy and polio. Sharrard described the posterolateral iliopsoas transfer in 1964.[13] He and other authors felt that this transfer with or without bony surgery (varus derotation osteotomy of the femoral neck and acetabuloplasty) would result in more hip stability and better walking abilility.[14,15]

We reviewed 35 midlumbar patients and showed that 86% of L3 and 46% of L4 hips become unstable. Only 33% of the L3 patients walked but l00% of L4 patients walked. Walking ability was not influenced by hip stability.[16] We are in accord with other authors who believe that walking ability is determined by the neurological level and not whether the hip is located or not.[17-19] The better walking potential of L4 patients is due to the presence of medial hamstrings that account for one-third of hip extensor strength.[9]

Unlike the unstable hip in cerebral palsy, long-term studies of up to 40 years have shown that the unstable hip in myelomeningocoele does not result in painful arthritis.[20] We have therefore not attempted to stabilise the midlumbar hip since 1992. The only rare exception would be a patient with asymmetrical involvement, where the one side is normal and the other side is midlumbar level.

Knee

In our experience the knee rarely requires surgery in ambulant myelomeningocoele patients.

Hyperextension deformity mostly responds to treatment with serial plasters in patients under 1 year of age. If persistent, V-Y quadricepsplasty with or without capsular release can be done.

Flexion deformity of the knee has a similar treatment plan to that in cerebral palsy and arthrogryposis. A flexion deformity of <20° does not usually adversely affect the gait in the ambulant patient but can be treated with serial plasters. Between 20° to 40° a posterior capsular release is performed via a posteromedial and a posterolateral incision. If indicated (the popliteal angle with straight leg raising exceeds the static flexion deformity angle) the hamstrings are lengthened through the same incisions.[21] If the flexion deformity exceeds 40°, gradual correction with an external fixator is currently the treatment of choice.[22]

Tibial torsion

Midlumbar patients may have persistent internal tibial torsion after 5 years of age. We have no experience with the transfer of semitendinosus to biceps femoris as described by Dias *et al*.[23]

External tibial torsion is seen in older children and aggravates valgus at the knee and ankle.

We have treated tibial torsion with a supramalleolar derotation osteotomy, crossed K-wires and plaster, but have become disillusioned due to a high incidence of delayed union. T-plate fixation, which we have used for the last two years, has eliminated this problem.[24] The malleable transverse limb of a small AO T-plate is applied distal to the osteotomy. The plaster is changed at 3 weeks and the patient measured for AFOs, which are fitted when the plaster is removed at 6 weeks.

The foot

The aims of treatment of foot deformities are to obtain a plantigrade, mobile and braceable foot, and hopefully avoid trophic ulceration which is a significant morbidity and can end in amputation. A very important study by Maynard *et al*, found that 94% of feet in myelomeningocoele require surgery. Post-operatively the incidence of trophic ulceration in flexible plantigrade feet was 0%, in flexible non-plantigrade feet 25%, in rigid plantigrade feet 36% and in rigid non-plantigrade feet 100%.[25] It is therefore imperative to obtain a plantigrade foot and avoid operations that decrease hindfoot and midfoot motion, such as arthrodeses and posteromedial release.

Equinus

Equinus deformity often occurs in paraplegic and midlumbar patients. Our policy is to treat this at one year of age to facilitate transfer in paraplegic patients and ambulation in midlumbar patients. An Achilles tenotomy, or if required a posterior release (Achilles tenotomy, posterior capsulotomy and calcaneofibular ligament release) results in a plantigrade, mobile foot.

Club foot

Club foot occurs almost exclusively in midlumbar patients.

Early treatment with the Ponseti method does not cause subtalar stiffness. However, some feet are very stiff and resistant to Ponseti manipulation, and a posteromedial release may have to be done. We assessed subtalar mobility after club foot surgery and found that subtalar motion was never decreased after posterior release in normal or myelomeningocoele patients. After posteromedial release in normal patients the subtalar joint was almost always stiff, but not always in myelomeningocoele patients. We postulated the stiff subtalar joint in normal patients was due to the Z-lengthened tibialis posterior tendon that became stuck down, while in myelomeningocoele patients all tendons are cut.[26] However, the mobility of the subtalar joint after posteromedial release in myelomeningocoele patients is not predictable; therefore a plantigrade foot is imperative to avoid trophic ulceration.

Some feet are very rigid and require a talectomy as a primary procedure or after a recurrence.[27] The standard talectomy should be preceded by a limited posterior and medial release via a hemi-Cincinnati incision through which the tibialis anterior, tibialis posterior, toe flexors and Achilles tendon are cut. This prevents a residual forefoot deformity after the talectomy.

Congenital vertical talus

The aetiology in our unit of congenital vertical talus is one-third idiopathic, one-third associated with other congenital abnormalities (e.g. arthrogryposis) and one-third neurological of which half are due to midlumbar myelomeningocoele.[28]

The treatment is similar to normal patients, except that in myelomeningocoele the involved tendons are cut. At 1 year of age a simultaneous anterior release (tibialis anterior, extensor hallucis longus and extensor digitorum communis tenotomy with open reduction of the talo-navicular ± the calcaneo-cuboid joint) and posterior release (Achilles tenotomy ± posterior capsulotomy) are done.

Valgus

A valgus foot occurs in the midlumbar and L5 level myelomeningocoele. In ankle valgus the callosity is under the medial malleolus and in hindfoot valgus the callosity is under the talar head.

Ankle valgus is due to shortening of the fibula with secondary lateral wedging of the distal tibial epiphysis and lateral tilt of the talus. Normally the distal fibular growth plate is at the level of the talar plateau; if it is between the top of the talus and the distal tibial growth plate the shortening is mild; if it is in line with the distal tibial growth plate there is moderate shortening; and if it is proximal to the distal tibial growth plate there is severe shortening.[29]

In the immature patient (with at least 2 years of growth remaining), stapling of the distal medial tibial growth plate gives good results.[30] Three small Blount staples are used and the staples are left in situ until there is 10° overcorrection into varus.

In the mature patient a varus supramalleolar osteotomy described by Wiltse is the best option.[31] It centralises the mechanical axis of the tibia over the talus and does not have the prominent medial malleolus found after medial wedge removal (*Figures 3 A and B*).

We have no experience with Achilles tenodesis of the fibula, which prevents a short fibula.[32]

The workhorse for the treatment of **hindfoot valgus** has been the Fulford modification[33] of the Grice subtalar arthrodesis.[34] Although it remains a good option for the valgus foot in diplegic cerebral palsy, it results in a stiff hindfoot (*Figure 4*). The calcaneal lengthening procedure first described by Evans[35] and revived by Mosca *et al*[36] is a better option as it does not affect the mobility of the subtalar joint (*Figure 5*).

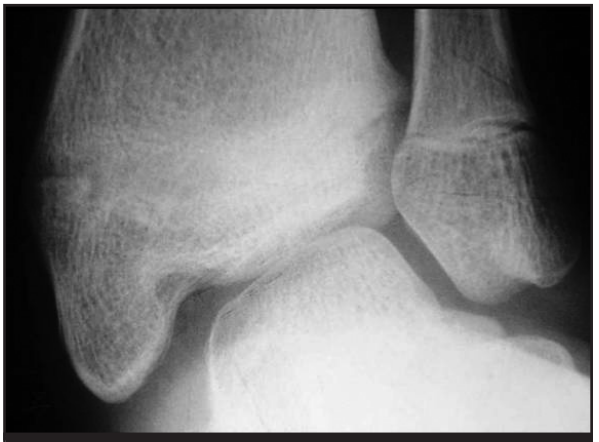


Figure 3 A.
A 14-year-old midlumbar boy with ankle valgus

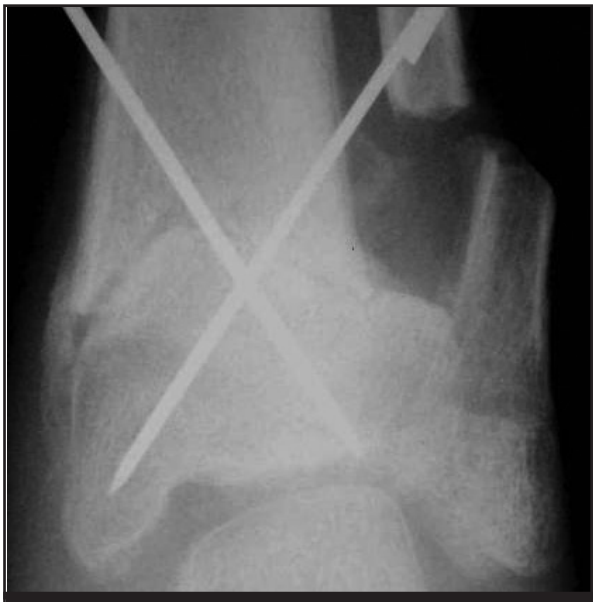


Figure 3 B.
Postoperative radiograph showing Wiltse type varus osteotomy

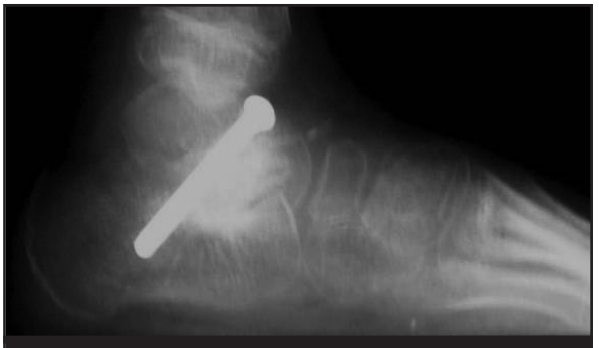


Figure 4.
Lateral radiograph showing correction of hindfoot valgus after Fulford subtalar arthrodesis

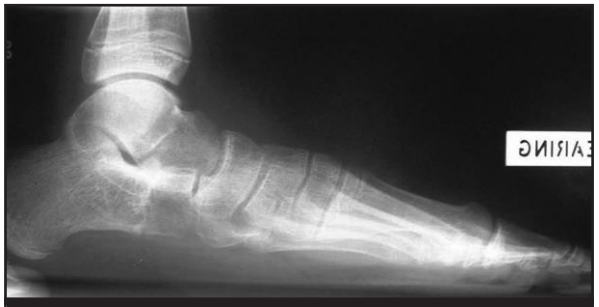


Figure 5.
One year after calcaneal lengthening there is correction of the hindfoot valgus and incorporation of graft

Calcaneus

A calcaneus gait and a subsequent calcaneal deformity occur in patients with L5 level (plantarflexion without dorsiflexion) and weak S1 level (plantarflexion weaker than dorsiflexion). The weak push-off results in a crouch gait, but more importantly a trophic ulcer of the heel which can lead to calcaneal osteitis and eventually destruction of the calcaneus (*Figures 6 A and B*). An algorithm for the treatment of calcaneus is given in *Table IV*.

Weak plantarflexion of the flexor muscles in the sole of the foot without gastrosoleus results in cavus of the forefoot and a 'pistol-grip' bony deformity of the calcaneus. The cavus entity of this **calcaneocavus** deformity is measured by the Meary's angle (normal = 0°) and the bony deformity of the calcaneus by the calcaneal pitch (normal = 15°) and the Coleman ratio[37] (*Figure 7*). If the Coleman ratio is <0.5, a superior displacement osteotomy of the calcaneus is indicated.[38] This should be preceded at the same sitting by a plantar release to correct the cavus.

Peabody described the tibialis anterior transfer to the heel in polio to replace absent dorsiflexion in 1938.[39] The use of this transfer in myelomeningocoele has been controversial.[40-42] The main controversies were the correct age to perform the transfer, and whether an anterior release would not give similar results.

We retrospectively reviewed 46 procedures (36 transfers and 10 anterior releases) in 26 patients.[43] We concluded that the best age for surgery was between 3 and 4 years. After 4 years the incidence of trophic ulceration increased from 0 to 30%. We will however observe a patient carefully after 4 years of age if we are not sure about the neurological level or voluntary power.

To obtain effective plantarflexion after the transfer, the tibialis anterior must have 4 or 5 power.

To maintain dorsiflexion of the foot post transfer, the toe extensors should have at least 3 power. If not, the patient will have a dropfoot gait, and one might as well do an anterior release (excise tib. ant., EHL and EDC) ± a lateral release if valgus (excise peronei).[44]

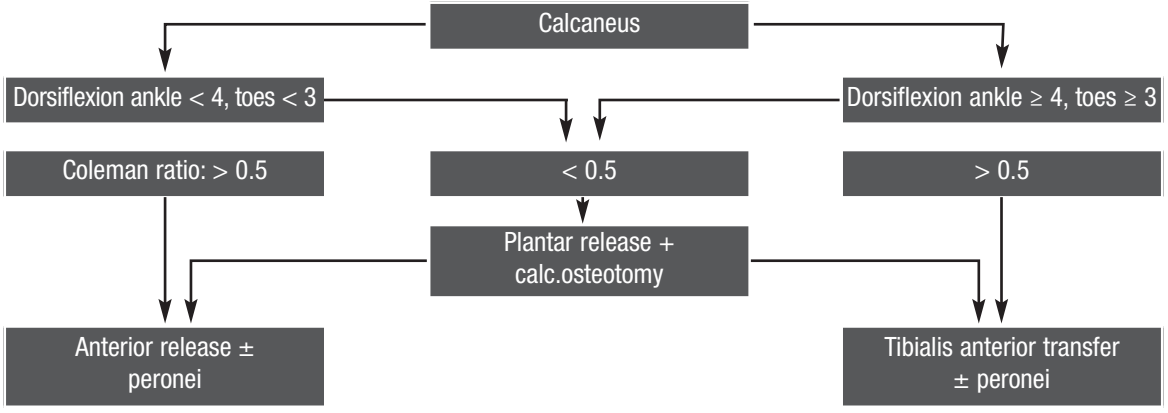
Cavovarus

Cavovarus occurs in S1 level myelomeningocoele patients due to imbalance between the intrinsic and extrinsic foot muscles. In our paediatric orthopaedic practice two-thirds of cavovarus is due to hereditary sensory motor neuropathy (HSMN I and II) and one-third due to spinal dysraphism. Of the spinal dysraphism, 50% are due to S1 myelomeningocoele, and the other half due to lipoma, tethered cord, diastomatomyelia and sacral agenesis.[45]

If the deformity is mobile on the Coleman test, a plantar release should suffice. In our experience these patients are seldom symptomatic before the age of 8 years. The deformity is then not convincingly mobile and the patient invariably requires, besides a plantar release, a first metatarsal osteotomy and a valgus osteotomy of the calcaneus as described by Dwyer.[46]

In our paediatric orthopaedic practice two-thirds of cavovarus is due to hereditary sensory motor neuropathy (HSMN I and II) and one-third due to spinal dysraphism

Table IV. Algorithm for the treatment of calcaneus deformity



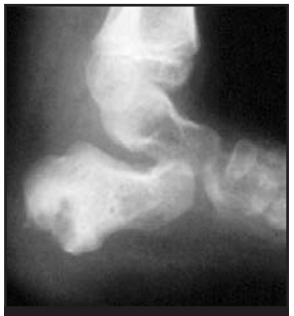


Figure 6 A.
Lateral radiograph of the heel in a 6-year-old L5 level patient showing calcaneal osteitis underlying a trophic ulcer

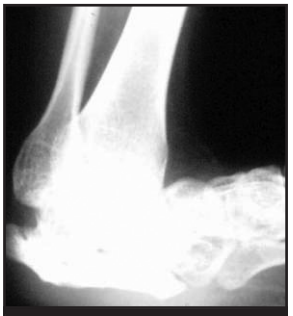


Figure 6 B.
Three years later there is complete destruction of the calcaneus

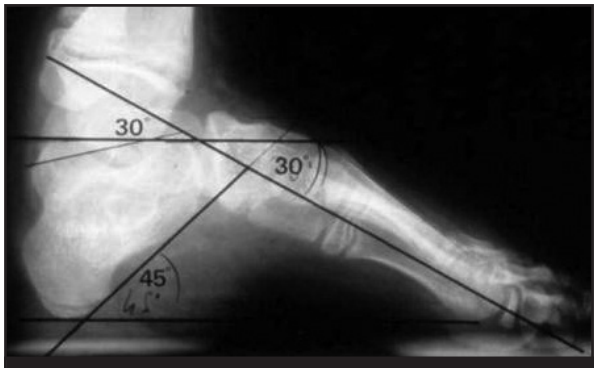


Figure 7.
Lateral radiograph of the foot of an 11-year-old girl with weak S1 level showing a significant calcaneocavus deformity. Note that the Coleman ratio, i.e. the horizontal distance from the centre of the tibia to the posterior end of the calcaneus, divided by the vertical distance from the ankle joint to the bottom of the calcaneus, is < 0.5

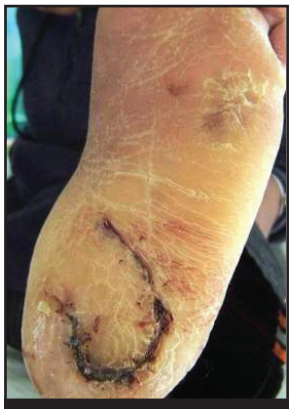


Figure 8 A.
Trophic ulcer of the heel in a 8-year-old L5 level patient

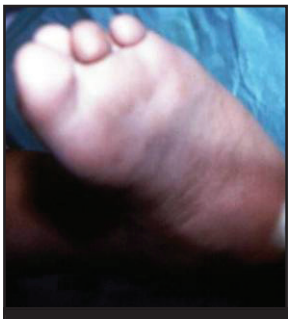


Figure 8 B.
Ulcer remained healed after a superior displacement osteotomy of the calcaneus and a medially based rotation flap

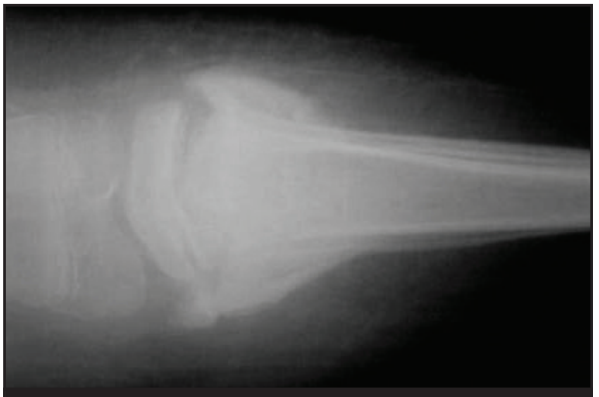


Figure 9.
Abundant callus formation in a stress fracture of the proximal tibial physis of a L3 level patient

Trophic ulcers

Trophic ulcers of the foot are due to a stiff, non-plantigrade foot or to a neglected injury in an insensitive foot. The surgeon should always endeavour to obtain a plantigrade, mobile foot.[25] In a neglected injury the normal subcutaneous fat pad of the sole of the foot has been destroyed, and if the ulcer heals by secondary intent with scar tissue, it invariably breaks down again.

The treatment of a trophic ulcer is to reconstitute a normal skin and fat pad by means of a medially based rotation flap (*Figures 8 A and B*). This should be preceded by correcting a non-plantigrade foot and removing bony prominences under the ulcer. If the ulcer is on the heel, the posterior calcaneus can be displaced superiorly with the osteotomy described for the 'pistol grip' heel, even in the absence of a calcaneal deformity.

Charcot arthropathy and fractures

Both Charcot arthropathy and fractures can present with an exaggerated clinical response of swelling, warmth and erythema. The wrong diagnosis of cellulitis, septic arthritis or osteitis is usually made.

In Charcot arthropathy the initial radiographs are normal and the joint should be protected (initially in a cast for 6 weeks and then with an orthosis) to prevent further joint destruction and characteristic radiographic features.

Fractures develop exuberant callus. In diaphyseal and metaphyseal fractures there is usually a history of trauma and appropriate immobilisation is required. Physeal fractures are due to repetitive stress fractures in a poorly sensate limb and, similar to Charcot arthropathy, require long-term orthotic splinting after the initial cast immobilisation (*Figure 9*).[47] Physeal bar formation with growth arrest can occur.

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