Summary

Clubfoot or congenital talipes equinovarus is a common congenital abnormality of uncertain aetiology. It presents with fixed cavus, adductus, varus and equinus of the foot. Serial manipulation and casting using the Ponseti technique can produce a plantigrade, pain-free, functional foot in the majority of cases in the long term. Most patients treated this way will require only minimal surgery, such as Achilles tenotomy, dramatically reducing the need for extensive, open releases. Recurrent and complex clubfoot can also be treated by the Ponseti technique but some challenging cases still require surgical correction. Those that resist primary or revision operations can be salvaged by arthrodesis but the long term results are less predictable. This review will summarise the current theories on aetiology and pathogenesis, assessment and management according to the Ponseti regime, surgical options for primary clubfoot and recurrences and possible future directions.

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Introduction

Clubfoot or congenital talipes equinovarus is one of the most common congenital orthopaedic abnormalities. It can be isolated or associated with other serious congenital abnormalities, especially if bilateral and severe. The ideal aim of treatment is to achieve a functional, pain-free, plantigrade foot in the long term. Over recent years, and since this topic was last reviewed in this journal,¹ there has been a general trend away from extensive open releases towards serial manipulative techniques coupled with minimal surgery, such as the Ponseti technique. A small number of resistant cases may benefit from primary surgery and complex recurrences still present considerable management difficulty.

Aetiology

As with many medical conditions, clubfoot has a multifactorial aetiology and probably represents an end-point of several disease processes.

Genetics

Clubfoot occurs in approximately 1 per 1000 live births in the UK (0.1%), with Polynesians being affected six times more commonly. The male to female ratio is 3 to 1 and up to 50% are bilateral. There is a monozygotic twin concordance...
of 33%² and if one sibling has clubfoot the chance of a second being similarly affected rises nearly 30 times to 3%.³ Theories vary between involvement of a single major gene, possibly on the long arm of chromosome 2,⁴ and polygenic inheritance patterns. These observations imply a genetic component, but other factor(s) may be more important.

**Embryology**

There are three main phases of foot development in utero. In the initial phase (between weeks 5 and 6) the foot begins to develop in line with the leg. The embryonic stage (from 6 to 7 weeks) is characterised by a phase of fibular growth. The lateral part elongates relative to the medial and the foot adopts a “clubfoot-like” posture. During the final foetal phase (8 to 9 weeks), the tibial side of the leg and foot develops, correcting the position to that observed in the normal newborn.⁵ It has been suggested that an interruption during the foetal phase of growth results in a clubfoot deformity. The time of onset and duration of that interruption affects the overall severity. Several agents have been implicated such as a chemical teratogen, viral infection, radiation and hormonal imbalance but no definite cause has yet been identified. Observations of the seasonal variation in the incidence of clubfoot have lent some support to an environmental or viral aetiology, but other studies have found no correlation.⁶

**Soft tissues**

There have been many studies looking at the macroscopic, microscopic and ultrastructural appearance of the soft tissues involved in the clubfoot. The muscles, tendons, ligaments, nerves and blood vessels all demonstrate abnormalities compared to normal tissue. These changes are undoubtedly linked and it is not yet fully understood which, if any, are driving forces in the development of clubfoot and which are secondary.

Some anatomical studies have shown anomalous muscles (eg accessory soleus) and/or tendons, but these findings are inconsistent and certainly do not occur in all cases. A more common finding is an absent or small anterior tibial artery and dorsalis pedis. This abnormality is also found in association with fibular hemimelia and may represent growth arrest during the late embryonic phase of growth mentioned above. It is also possible that this vascular insufficiency is the primary cause of clubfoot⁷ and studies of foetal anatomy have noted that these vascular abnormalities are more prominent in early foetal life around the time of foot development.⁸

The leg muscles, especially the posterior and medial groups, in a child with clubfoot tend to be smaller in girth and shorter in length when compared with normal and the tendon of tibialis posterior in particular is often severely thickened.⁹ The degree of shortening is proportional to the severity of clubfoot. The muscles have a higher proportion of connective tissue and the ratio of type I to type II fibers is increased, both of which are signs of neurogenic atrophy.¹⁰ Ponseti has linked the degree of in vitro protein synthesis with the severity of clubfoot. Muscle cells with very high levels of collagen synthesis and low levels of non-collagen protein synthesis occur in children with severe clubfoot. This gradually returns to normal by age 6 to 7, the age after which clubfoot is very unlikely to recur.

The ligaments and joint capsules on the posterior and medial side of the foot and ankle are thickened and those laterally tend to be thin and weak. There is an increase of collagen fibers in the thickened tissues with high numbers of mast cells, fibroblasts and myofibroblasts,¹¹ appearances similar to scar tissue. This has led to the retraction fibrosis theory of clubfoot aetiology¹² analogous to cicatrisation of scar tissue. It also explains the continuing deformity seen in recurrences.

**Anatomy**

The four main anatomical abnormalities can easily be remembered by the mnemonic “CAVE” — Cavus, Adduction, Varus and Equinus (Fig. 1). Cavus is an increased height of the vault of the foot and in clubfoot is due to pronation of the forefoot in relation to the hindfoot, with plantar flexion of the first ray. Although the whole foot appears to be in a supinated position, this forefoot/hindfoot relationship is integral to understanding the initial corrective step required by the Ponseti technique (see below). The cuneiforms and metatarsals are adducted but of normal shape. The midfoot is adducted, primarily at the talo-navicular joint. The talus and navicular are wedge shaped and the navicular is medially displaced (in severe cases it nearly abuts the medial malleolus) and inverted such that it is in an almost vertical position. The calcaneus is severely plan-tarf lexed, medially displaced and inverted below the talus such that it lies below and almost in line with the talus. This accounts for the equinus and varus deformities and for the reduced AP and lateral talo-calcaneal angles seen on x-ray (Fig. 2). The ankle, although in a plantarf lexed position, is relatively normal.

As mentioned above, the posterior and medial structures are short and thick. The calcaneo-navicular (spring), deltoid and talo-navicular ligaments along with the tibialis posterior tendon hold the foot in an adducted position. The posterior talo-fibular, posterior calcaneo-fibular, posterior and medial talo-calcaneal and posterior tibio-talar ligaments along with tightness of the gastro-soleus complex contribute to the equinus and varus. Since the insertion of the tendons has medialised, there is medial displacement of tibialis anterior and the long toe extensors. These can now act as deforming forces, pulling the midfoot and forefoot into further adduction and inversion.

The motion of the calcaneus under the talus has been described as a mitered hinge, a hinge at 45° to the horizontal rotating around a fixed axis. This is somewhat simplistic in that the subtalar joint has no fixed axis but rather is a kinematic chain whose movements depend upon the inclination and curvature of the involved joint surfaces coupled with dynamic capsular, ligamentous and musculo-tendinous forces. The entire tarsus moves as a unit and movement in anatomical directions (adduction, abduction, flexion, extension, inversion and eversion) cannot be separated. Thus movement of one part of the foot invariably causes movement elsewhere, which may be in a different plane or direction. Varus movement of the
calcaneus, for example, actually comprises medial rotation, flexion and inversion of the calcaneus. The cuboid and navicular follow into adduction and inversion in front of the talus. Reversal of this movement by reducing the navicular into its normal position, forces the calcaneus to derotate, abduct and evert below the talus. This understanding forms the basis for the Ponseti method of manipulation and casting described below.

Assessment

The assessment of an infant with clubfoot falls into 4 parts, antenatal diagnosis, examination, assessment of severity and investigations.

Antenatal diagnosis

Though commonly diagnosed post-natally, prenatal ultrasound diagnosis is possible and can help with parental counselling and organising tertiary referral such that treatment can commence as soon as possible after birth. The positive predictive value of antenatal ultrasound is over 80% with almost no false negatives and even the severity of clubfoot can be estimated, although less accurately. Studies have identified that in many patients the foot deformity is not isolated. As many as 2/3 may be associated with at least one other abnormality, although this is often minor and may be unrelated to the clubfoot. In bilateral and/or severe cases of clubfoot, there is a higher incidence and severity of associated anomalies. Urogenital abnormalities, neural tube defects, cardiac defects, arthrogryposis and other musculoskeletal anomalies account for the majority. Abnormal karyotypes are present in 5–10% (XXY, XXX, trisomies 18 and 21) and although not routinely used in this country, some specialists recommend amniocentesis if clubfoot is diagnosed. Clubfoot can be seen as early as 12 weeks of gestation and up to 2/3 have an associated polyhydramnios. This contradicts the historical belief that clubfoot is due to intrauterine moulding which occurs well after 20 weeks. There is no association between clubfoot and hip dysplasia; in fact, there may be a negative association.

Examination

The examination of the child with clubfoot should be head to toe, systematically looking for associated anomalies. In particular, the spine should be inspected along with a neurological examination, and all the joints assessed for stiffness characteristic of arthrogryposis.
The important factor is to distinguish flexible, positional talipes from true clubfoot. In the former, the deformity is fully correctible with no fixed equinus. Severe metatarsus adductus can be confused with clubfoot, but again, there is no fixed equinus and no hindfoot deformity. These conditions are associated with moulding and care should be taken to examine the infant’s hips.

Assessment of severity

There is a definite relationship between the severity of clubfoot and the number and severity of associated abnormalities. Severe or complex clubfeet are more likely to require extensive surgery, are more prone to recurrence following treatment and have a somewhat poorer outcome. For these reasons it is important to have a method of assessing and documenting clubfoot severity. It is also desirable to monitor the effect of interventions. Several scoring systems are in use, for example, the Dimeglio score, the Carroll severity scale and the Pirani score. All of these systems have been independently validated. Inter- and intra-observer reliability is very good and they correlate well with patient-based assessments of outcome.17

The Pirani score is commonly used, and for simplicity we have only included a detailed description of this system.18,19 It consists of 6 parts, each of which can have a score of 0, 0.5 or 1, giving a total score from 0–6. The more severe the clubfoot, the higher the score. Examination can be divided into ‘look, feel, and move’ and is separated into hindfoot and midfoot components. Each consists of 3 of the 6 components to give a hindfoot score and a forefoot score of 0–3 (Tables 1a and 1b).

Investigations

Though many radiological modalities have been used, in routine practice no formal investigations are required. The bones of the newborn are mostly cartilaginous with only small ovoid ossification centers present in the calcaneum and talus. This makes assessment of the axes and thus angular relationships difficult to determine. The position of the infant’s foot and x-ray plate is crucial and can be difficult to replicate accurately. This is illustrated by a poor correlation between angles measured by plain radiography and those measured by 3D CT.20 The correlation between radiological and clinical outcomes is variable and certainly surgery is not indicated to correct radiological abnormality. Decisions on initial treatment are therefore made purely on clinical grounds. Ultrasound and MRI have been used to assess clubfeet and monitor the response to treatment. MRI in particular has elegantly shown the gradual correction of deformity seen when treating clubfoot conservatively.21,22

It is important to remember that even in well treated clubfoot some radiological abnormalities persist long term.23 The tarsal bones tend to be smaller in volume and characteristic flattening of the talar dome is common. Research-based investigations, such as pedobarography and electromyography reinforce this point.

Table 1a  Pirani score, (hindfoot)

<table>
<thead>
<tr>
<th>‘LOOK’</th>
<th>0</th>
<th>No heel crease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posterior</td>
<td>0.5</td>
<td>Mild heel crease</td>
</tr>
<tr>
<td>crease</td>
<td>1</td>
<td>Deep heel crease</td>
</tr>
<tr>
<td>‘FEEL’</td>
<td>0</td>
<td>Hard heel (calcaneum in normal position)</td>
</tr>
<tr>
<td>Empty</td>
<td>0.5</td>
<td>Mild softness</td>
</tr>
<tr>
<td>heel sign</td>
<td>1</td>
<td>Very soft heel (calcaneum not palpable)</td>
</tr>
<tr>
<td>‘MOVE’</td>
<td>0</td>
<td>Normal dorsiflexion</td>
</tr>
<tr>
<td>Rigidity</td>
<td>0.5</td>
<td>Foot reaches plantigrade with knee extended</td>
</tr>
<tr>
<td>of equinus</td>
<td>1</td>
<td>Fixed equinus</td>
</tr>
</tbody>
</table>

Figure 2  Plain AP and lateral radiographs of the immature foot. The talo-calcaneal and talar-1st MT angles are shown. On the AP view (A), the normal talo-calcaneal and talar-1st MT angles are approximately 25° and 0° respectively. On the lateral view (B), the normal talo-calcaneal is approximately 45°.
The foot should be moved to the position of maximum correction when assessing the medial crease.

### Table 1b Pirani score, (midfoot)

<table>
<thead>
<tr>
<th>'LOOK'</th>
<th>0</th>
<th>No deviation from straight line</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lateral border of foot</td>
<td>0.5</td>
<td>Medial deviation distally</td>
</tr>
<tr>
<td>'FEEL'</td>
<td>1</td>
<td>Severe deviation proximally</td>
</tr>
<tr>
<td>Talar head</td>
<td>0.5</td>
<td>Reduced talo-navicular joint</td>
</tr>
<tr>
<td>'MOVE'</td>
<td>1</td>
<td>Irreducible talo-navicular joint</td>
</tr>
<tr>
<td>Medial crease</td>
<td>0.5</td>
<td>Mild medial crease</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>Deep medial crease</td>
</tr>
</tbody>
</table>

*a* The foot should be moved to the position of maximum correction when assessing the medial crease.

### Management

The goals of treatment are to achieve a plantigrade, pain-free, functional foot for the life of the patient. The foot should be cosmetically acceptable, have good mobility and require no specialised footwear or orthoses once treatment is complete. Parents should be informed that in the vast majority of cases this is achievable but the foot will never be entirely normal.

The two main methods of management in use today are the Ponseti technique and the French physical therapy technique. When done well, both have excellent long term results and avoid major surgery in the majority of patients. In this country the Ponseti regime is being increasingly used and this is described in detail below.

### The Ponseti technique

At 40 years follow-up, 78% of patients treated by this method can be expected to have a good or excellent functioning foot in the long term. This compares with 85% of subjects without clubfoot, assessed using the same outcome measures. 93% will require an additional procedure, usually Achilles tenotomy alone or combined with tibialis anterior transfer.

Treatment should begin as early as possible and consists of manipulation and serial plaster casts changed at weekly intervals. At each cast change, the foot is examined and the Pirani score noted. The manipulations follow a specific protocol which aims to correct all deformities simultaneously except equinus (Fig. 3).

The forefoot must be brought into the correct relationship with the midfoot. There is pronation of the forefoot with plantarflexion of the first ray. The forefoot is therefore supinated by lifting the first ray (which makes the overall deformity appear worse initially) and gently abducted using counter pressure against the lateral border of the talar head. No pressure must be placed around the heel or in the region of the calcaneo-cuboid joint as this will block calcaneal movement (Fig. 4A). With subsequent plasters the forefoot, now in its correct relationship with the midfoot, is gradually brought from supination to neutral whilst increasing the amount of abduction. The talo-navicular, calcaneo-cuboid and subtalar joints are simultaneously reduced. The calcaneum will evert, abduct and rotate under the talus to assume its normal position without any direct manipulation of the hindfoot whatsoever. By the last plaster, the foot should be in approximately 70° of abduction. Each plaster is initially placed below the knee and the moulding performed. It must then be completed to the groin with the knee in 90° flexion. The ligaments are gradually stretched in plaster and the immature, mostly cartilaginous, tarsal bones begin to remodel.

Once the foot is in a position of abduction and the heel is in valgus, the equinus can then be addressed. It is important to dorsiflex the entire foot as pressure under the forefoot can cause dorsiflexion at the midtarsal joints and a rocker-bottom deformity (Fig. 4B). The majority of patients will require percutaneous Achilles tenotomy. The more severe the initial Pirani score the more likely the need for tenotomy if the foot cannot be brought to at least neutral with the knee flexed then a tenotomy is performed. This can be done under local anaesthesia but in our center a general anaesthetic is preferred. A blade is passed anterior to the tendon, turned posteriorly and the tendon divided from deep to superficial. The foot should be able to reach 15° of dorsiflexion. A further cast is applied and left for 3 weeks.

The total number of plasters varies from 4 to 10, more severely affected feet generally requiring longer treatment. The severity as assessed by the Pirani score should gradually reduce at each examination (Fig. 5). Usually the forefoot score reduces first, followed by the hindfoot score. If the deformity is not improving or if more than 10 casts are required then conservative measures can be said to have failed.

Following successful serial casting the infant is placed in to a ‘boots and bar’ splint. This consists of a soft sandal holding both feet in 70° of abduction and 15° of dorsiflexion connected by a bar of approximately shoulder width. This must be worn at all times, except bath time, for 10 weeks followed by night and nap time use until 4 years of age.

### The French technique

This method, described by Bensahel, involves regular gentle manipulations of the foot in a relaxed child and addresses the deformities in a very similar manner to Ponseti. Individual treatments last approximately 30 minutes and are done daily for 2 weeks then twice weekly until correction is achieved. This takes approximately 6 to 8 weeks for the cavus, varus and adductus and up to several months to correct the residual equinus. A flexible splint is worn in between sessions. In his hands, Bensahel reports a 93% good or excellent outcome at skeletal maturity with only a 23% operation rate, although surgery tended to be more extensive than simple Achilles tenotomy. More severe cases of clubfoot were associated with longer treatment times, a higher rate of surgical intervention and a poorer outcome.

### The complex clubfoot

A minority of feet, approximately 5%, do not respond to the Ponseti or French techniques described above. The
Figure 3  Sequence of pictures showing the Ponseti method of clubfoot correction. The 1st ray is elevated correcting the alignment of the forefoot (A). In this position, the heel remains in varus (D). The forefoot is then brought in to progressively greater degrees of abduction with counter pressure against the talar head (B, C). The calcaneus corrects to neutral (E) and finally valgus (F) without any direct manipulation of the heel itself.

Figure 4  Pressure on the calcaneocuboid joint rather than the talar head leads to a midfoot break (A). Although the foot may appear corrected, the talo-navicular joint is not reduced and the hindfoot is not corrected. Forced dorsiflexion of the forefoot can lead to a rocker-bottom deformity (B).
deformities are very severe at presentation, scoring 5.5 or 6 on the Pirani scale. All of the metatarsals are in plantaris, signified by a deep transverse plantar crease in the sole of the foot, and the great toe appears to be short and hyperextended. The equinus is severe and fixed, with a short, thickened, medially displaced Achilles tendon. Primary surgery in this group is technically difficult and often has poor results. Ponseti has published a modification to his standard regimen which reports good results after an average of 2 years follow up instead of just dorsiflexing the first ray, the whole foot is grasped and by pushing up on all the rays with both thumbs, the plantaris can be serially corrected. Nearly all patients in this series required at least one Achilles tenotomy with 6% needing a second. Complications occurred in 22% but were mostly minor and related to cast problems. Due to the short follow-up, it is not yet known whether these feet will do well in the long term or go on to require more extensive surgery.

Recurrent clubfoot

Roughly 1/3 of patients will suffer a relapse and the main predictor of recurrence is non-compliance with the splint. Interestingly, the severity of deformity at presentation does not seem to be a factor. 80% of recurrences occur in the first 2 years of life with the majority of the rest occurring up to age 6. Approximately 5% occur after this time, a third of which may have a previously undiagnosed neuromuscular condition such as Charcot-Marie-Tooth disease or myotonic dystrophy. Stiff, recurrent clubfeet may be the only sign of a distal arthrogryposis. The child with recurrent talipes should therefore be thoroughly re-examined. Ponseti casting should be restarted straight away and continued until correction is re-achieved. Further Achilles tenotomy or tibialis anterior transfer should be considered as indicated. Parents must be educated regarding the
importance of splintage and the vast majority of recurrent clubfeet can be successfully corrected. Tibialis anterior transfer, whereby the tendon is moved from its insertion into the medial cuneiform and first metatarsal to the lateral cuneiform, is indicated for recurrent clubfoot as the tibialis anterior has a supinating and varising action. The foot must initially be corrected by repeat Ponseti plastering and the child must be over approximately 2.5 years of age, when the lateral cuneiform is ossified. A straight incision is made from the medial cuneiform 5 cm proximally along the line of the tendon. The tendon is divided distally and mobilised back to the extensor retinaculum, which is left intact. A second small incision is made overlying the lateral cuneiform and dissection carried out medial to extensor digitorum longus, through extensor digitorum brevis down to bone. The tendon is passed subcutaneously and inserted through a hole drilled in the lateral cuneiform. It can be secured over a button in a manner similar to flexor tendon repair to the terminal phalanx of the finger. The leg is placed in an above knee plaster in the corrected position for 4 weeks and then the suture and button are removed. There is no need for a splint if this procedure is successful, making it a good option in the cases when boots and bar cannot be tolerated.

Late presentation

Late presenting or neglected clubfoot is fortunately uncommon in the UK. There are studies showing that clubfoot treatment can start up to 6 months following birth with no change in the overall outcome and a group in Brazil have successfully treated neglected clubfeet in children as old as 9 years using the Ponseti method. In this study, the length of time required in plaster was longer, averaging 4 months. A third of patients required a posterior release to allow full correction of the hindfoot deformity. A 3 year follow-up of these patients showed excellent early results but long term function is not yet known.

Primary surgery

For this condition, primary surgery refers to procedures more extensive than Achilles tenotomy or tibialis anterior transfer. Due to the success of manipulative and plastering regimens the number of surgeons performing major primary clubfoot surgery has reduced dramatically. That said, surgery is indicated where conservative measures have failed or in syndromic feet. There are very many procedures described for clubfoot correction involving either the soft tissues, the bones or both, making objective evaluation of their relative long term efficacy extremely difficult. Results are varied and although no randomised controlled trial exists, the outcomes may be poorer in those undergoing extensive surgery compared with conservative management. Short term results can be very good. However 50% have demonstrable osteoarthritis of the subtalar joint by 30 years with a similar percentage having a poor overall result. Stiffness is very common and some patients also complain of muscle weakness, pain and residual deformity. Others have found that long term results are much better and the more severely deformed feet have an improved prognosis if operated on before 3 months, when there is a greater potential for remodelling. This contradicts another study suggesting the outcome is improved if surgery is delayed until 12 months as early surgery is associated with severe scarring and high chance of recurrence. Heterogeneity between studies makes direct comparison difficult.

Once the decision has been made to operate, the type of surgery required must be ascertained. Bensahel has popularised the 'à la carte' approach to clubfoot surgery, similar to the 'progressive surgical approach' coined by George Simons i.e. release or lengthen only those structures which are tight and contributing to the deformity. There are three main approaches in use, the posteromedial, the Cincinnati hemi-circumferential, and the two-incision approach. The three main releases are posteromedial, plantar and lateral.

Once the soft tissues have been addressed, the joints should be able to be held in their normal or near normal relationships. Intraoperative x-rays will help determine if this is the case. The bones are not of a normal shape and therefore acute correction in this manner will be associated with joint incongruity. Temporary fixation with k-wires is sometimes necessary to hold the bones whilst remodelling occurs and the foot should be placed in plaster in the corrected position, although this may endanger the blood supply. The time in plaster will depend on the age of the patient and the extent of surgery performed but will usually be for 4 to 6 weeks. If present, the wires are then removed and splintage commenced.

Posteromedial soft-tissue release

This release is used for residual equinus and hindfoot varus. The tendo-achillis is divided (age < 1 year) or lengthened (age ≥ 1 year). If the equinus has been present for a long time then capsulotomy of the ankle and subtalar joints along with release of the posterior inferior tibio-fibular, the posterior talofibular and the calcaneo-fibular ligaments may be required. The superficial deltoid ligament is divided along with the talo-navicular joint capsule. The tibialis posterior tendon is lengthened and the flexor tendons released from their sheaths, or lengthened if very short and large enough to repair.

Plantar release

For cavus deformity, a plantar release is done. The abductor hallucis is released from its origin on the calcaneus; provided dissection is carried out superior to this muscle, the medial plantar, lateral plantar and calcaneal neurovascular bundles will be protected. The plantar fascia, plantar calcaneo-navicular ligament and short toe flexors are divided at their origins and the calcaneo-cuboid and talonavicular joint capsules are released medially and inferiorly.

Lateral release

It is seemingly counterintuitive to consider lateral release given the anatomy of clubfoot deformity. The lateral
structures are usually attenuated and thin compared to the thickened, contracted medial tissues. However a lateral approach, in addition to the releases described above, allows capsulotomy of the lateral parts of the talo-navicular and calcaneo-cuboid joints. This provides further mobility of the subtalar and midfoot joints where posteromedial and plantar releases have been inadequate.

Complications

Serious complications are uncommon and depend very much on the type of surgery performed. Avascular necrosis (AVN), infection, overcorrection, pain, stiffness and recurrence have been reported. It must be noted however that all of these, with the exception of AVN, have occurred when using the Ponseti method.

Overcorrection may be the result of overzealous primary surgery and is more likely to happen in children who have evidence of generalised ligamentous laxity. The hindfoot is in valgus with a flattened medial arch. Management of this condition should follow the normal sequence with orthotics being first line and surgical options considered later as symptoms dictate. The added complication of a scarred and possibly multiply-operated foot must be taken into account when counselling patients prior to major corrective surgery.

Revision surgery

Despite best efforts, some clubfeet resist primary treatment, whether it is conservative or surgical, and these cases present considerable management difficulty. The numbers are small and long term comparative studies lacking. The child may have an associated neuromuscular condition and therefore the soft tissues do not respond to surgery in the normal manner. Another cause may be inadequate release at primary surgery. If the child is still less than 2 years old, repeat soft tissue release can be done as described above. Over the age of 2, it is likely that bony surgery will be needed in addition. Major osteotomies should be reserved until the foot is more mature, after 4 years of age. All children and parents must be warned that the long term outcome is variable with many patients requiring further corrective surgery or arthrodesis in the future.

The osteotomies described in the literature are numerous but can be broadly classified in terms of which deformity they aim to correct.

Cavus

Residual cavus can be treated by midtarsal osteotomy. This can be an inferior opening wedge, superior closing wedge or dome type (Akron osteotomy), which also allows forefoot adduction to be corrected. It is usual to perform the osteotomy through the midfoot bones but more distal osteotomies at the level of the tarso-metatarsal joints have been described.

Adductus

Adduction occurs primarily at the talo-navicular and calcaneo-cuboid joints, but also at the tarso-metatarsal joints. Combinations involving shortening of the lateral column (Dillwyn-Evans procedure) and/or lengthening of the medial column address the former and metatarsal osteotomies can be performed to address the latter. The lateral column can be shortened by excising a wedge of bone and fusing the calcaneo-cuboid joint. As the child grows, the medial column will lengthen relative to the lateral and thus further correction achieved.

An opening wedge osteotomy of the medial cuneiform, thus lengthening the medial column, has the advantage of not shortening a foot which is already small as part of the clubfoot deformity. Only limited correction can be attained by this method alone.

Varus

Residual hindfoot varus can be corrected by calcaneal osteotomy. The Dwyer osteotomy is an oblique, lateral, closing wedge osteotomy that will correct varus and some equinus. More recently, a curvilinear calcaneal osteotomy has been described which brings the centre of rotation (about which the correction is done) closer to the true centre of deformity. This has achieved good results in terms of deformity correction as measured radiologically but it is too early to say whether this will be reflected in long term clinical results.

External fixation

All of the deformities can be potentially corrected by the use of external fixators. Both the Ilizarov system and the Taylor Spatial Frame have been used for clubfoot correction. It is usually combined with either a midfoot or calcaneal osteotomy, although purely soft-tissue Ilizarov correction has been performed in children up to 8 years old with good early results. During correction it is vital to obtain lateral radiographs of the ankle to ensure that the distal tibial physis is not being distracted. Early complications are frequent, often relating to pin site problems. Late complications include spontaneous ankylosis, recurrence of deformity and requirement for surgical arthrodesis. A recent paper with almost 5 year follow up of recurrent clubfeet treated using Ilizarov frames showed a 45% rate of spontaneous ankylosis, 31% had recurrence of deformity and 37% of feet went on to require arthrodesis. Approximately 75% had an acceptable result clinically after an average of 56 months. Another study with 6½ year follow up reported 85% of patients had fair or poor results with over 50% requiring revision surgery.

Salvage surgery

Salvage surgery for clubfoot relies upon arthrodesis or takedown. Talonavicular, triple and pan-talar arthrodesis can be performed depending on the individual patient, the symptoms and the pathology. Wedges of bone can be removed at the same time thus correcting residual deformity and fusing the joints simultaneously. Talectomy can correct hindfoot but not midfoot deformity and allows some movement at the new joint between the tibia and calcaneum. It may be of particular benefit in children with
primary or recurrent clubfoot as a result of arthrogryposis or spinal dysraphism. A 20 year follow up of patients who underwent taelectomy for clubfoot has shown that 75% have a plantigrade foot that is pain-free whilst walking. However, 2/3 of these patients required additional surgery, 1/3 had radiological evidence of tibio-calcaneal arthritis and 29% had spontaneous fusion of the tibia to the calcaneum.\textsuperscript{45}

Future directions

There is much ongoing research into the actual causes of clubfoot, current theories of which have been discussed. So far, management is directed at treating the established deformity, but in the future it may be possible to influence clubfoot development and thus prevent its occurrence.

There have been trials using Botox as an adjunct to conservative management. Early results are promising and when combined with the Ponseti regimen may reduce the need for Achilles tenotomy.\textsuperscript{46} Further refinements to the Ponseti technique include a new custom-fit dynamic orthosis that has been shown to increase compliance, the most important factor in preventing recurrence.\textsuperscript{47}

The Bulgarian technique is a newly described procedure that helps to correct the hindfoot deformity at the time of percutaneous Achilles tenotomy. For very stiff, fixed equinus the Achilles tendon is divided in the anaeasthetised infant. A ‘cats-paw’ retractor is then inserted through the skin of the posterior heel into the calcaneum. Several minutes of traction pulls the calcaneum down into the correct position better than Achilles tenotomy alone. Early results are encouraging.\textsuperscript{48}

Conclusion

1. Clubfoot occurs in 1 per 1000 live births in the UK.
2. 50% are bilateral and 5% are ‘complex’. These cases are more often associated with other congenital anomalies, especially neural tube defects, arthrogryposis, abnormal karyotypes and urogenital abnormalities.
3. It is multifactorial in aetiology with a definite genetic component combined with intrauterine factors, but is not a result of fetal moulding.
4. Pathological tissues on the medial side are short and thick with high proportions of fibrous tissue and myofibroblasts — the ‘retraction fibrosis’ theory.
5. The deformities are cavus, adductus, varus and equinus. The forefoot is pronated in relation to the hindfoot but the foot is in supination as a whole.
6. Management consists of full history and examination followed by instigation of a conservative corrective regime starting as soon as practicable.
7. The aims are to achieve a pain free, functional, plantigrade foot in the long term.
8. The Ponseti technique corrects the cavus, adductus and varus simultaneously with serial plaster casts followed by ‘boots and bar’ splints 23 hours per day for 10 weeks then night time and naps for 4 years. Equinus correction requires achilles tenotomy in the majority of patients. Approximately 80% will have a good long term result.
9. Recurrence occurs in up to 1/3 patients and should be treated by re-examination for associated conditions, repetition of the conservative regimen plus repeat Achilles tenotomy or tibialis anterior transfer (after the age of 2.5) as indicated. Non-compliance with splints is the major risk for recurrence and their necessity should be reinforced. Recurrence should not be considered a failure of conservative management.
10. Complex clubfoot can still be treated conservatively using a modification to the Ponseti regimen.
11. Primary surgery should only be considered if conservative techniques fail to correct the clubfoot. It should be as minimal as possible and address those tendons, ligaments and joint capsules which are tight and contributing to the deformity — the ‘à la carte’ approach.
12. Revision surgery entails repeated or further soft tissue releases usually combined with one or more osteotomies (after the age of 4) to correct residual deformity. Gradual correction with external frames can be done. Results are variable with many going on to spontaneous ankylosis or requiring further salvage surgery in the future.
13. Salvage procedures are fusion or talcetomy. Roughly 3/4 can expect to have a foot that is pain-free at rest and for walking in the long term but most require more than 1 major operation to achieve this.

References
