

Classification of gait patterns in spastic hemiplegia and spastic diplegia: a basis for a management algorithm

J. Rodda^a and H. K. Graham^b

^aHugh Williamson Gait Laboratory, Royal Children's Hospital; and ^bOrthopaedic Surgery, University of Melbourne, Orthopaedic Surgery, Royal Children's Hospital, Hugh Williamson Gait Laboratory, Murdoch Children's Research Institute, Royal Children's Hospital, Parkville, Victoria, Australia

Keywords:

ankle-foot orthoses,
contractures,
gait classification,
gait patterns, spasticity,
spastic diplegia, spastic
gait, spastic hemiplegia

Classifications of gait and postural patterns in spastic hemiplegia and spastic diplegia are presented, based on the work of previous authors. The classifications are used as a biomechanical basis, linking spasticity, musculoskeletal pathology in the lower limbs, and the appropriate intervention strategies. The choice of target muscles for spasticity management, the muscle contractures requiring lengthening and the choice of orthotics are then linked to the underlying gait pattern.

Introduction

Gait patterns in spastic motor disorders have been described by a number of authors, but only two classifications are widely used. Winters *et al.* described four gait patterns in spastic hemiplegia and Sutherland and Davids described four patterns of knee motion in spastic diplegia (Winters *et al.*, 1987; Sutherland and Davids, 1993). The widespread use of these classifications and the frequency with which they are quoted in the literature is evidence for their utility and the need felt by many researchers to describe the complexities of spastic gait disorders in a manageable format. The classification of gait patterns in this article is based on the above studies. Management recommendations are made on the basis of the classification, drawing from many sources but essentially reflecting the author's biases and experiences. The majority, are not supported by randomized trials and there is ample scope for further clinical trials in this challenging area.

Management options are for convenience grouped as 'spasticity management' or 'contracture management'. This is overly simplistic but is based on the simple premise that there is a reversible type of muscle stiffness and a more fixed type of stiffness. The former is sometimes referred to as spasticity or dynamic contracture and may respond to oral medications, injections of phenol or botulinum toxin type A, selective dorsal rhizotomy or administration of intrathecal baclofen. The latter is referred to as fixed or myostatic contracture

and is traditionally addressed by lengthening of contracted muscle tendon units. Management strategies are complex and, almost invariably, more than one method is required to achieve optimal outcomes. For example, injection of the gastrocnemius muscle with botulinum toxin type A may improve the range of ankle dorsiflexion in the younger child with spastic hemiplegia but on its own may have little impact on gait or function. Gait is more likely to improve if an ankle foot orthosis is used to manage the deficiency in ankle dorsiflexor function.

Focal vs. generalized spasticity

In considering spasticity management options, a primary issue is whether the spasticity is focal, regional or generalized (Boyd and Graham, 1997; Graham *et al.*, 2000; Gormley *et al.*, 2001). Oral medications are absorbed and act systemically and equally on all muscle groups. Selective dorsal rhizotomy (SDR) affects mainly the lower limbs although beneficial changes are sometimes found in the upper limbs (Loewen *et al.*, 1998). Intrathecal baclofen (ITB) acts mainly on the lower limbs, although the effects on the upper limbs and trunk can be varied according to the siting of the catheter tip and the concentration and rate of delivery of the drug (Albright *et al.*, 1995; Albright, 1996; Muhonen, 2001). Intramuscular injection of botulinum toxin type A (BTX-A) to the gastrocnemius is a focal intervention. The BTX-A heavy chains bind quickly and irreversibly to the cholinergic nerve terminals, so that there is little spread to neighbouring muscles (Dolly *et al.*, 1984; Graham *et al.*, 2000). Fine wire EMG studies confirm changes in muscles remote to the injection site, but these effects are usually of little consequence.

Correspondence: Professor H. K. Graham, Royal Children's Hospital, Flemington Road, Parkville, 3052 Victoria, Australia (tel.: +61 (03) 9345 5446; fax: +61 (03) 9345 5447).

There is no precise definition as to what constitutes focal, regional and generalized spasticity. We have previously suggested that focal intervention with BTX-A should be restricted to four large muscle groups at one time because of concerns about systemic spread and toxicity (Boyd and Graham, 1997; Graham *et al.*, 2000). Others have used higher doses and injected more muscles with benefit and few side-effects (Molenaers *et al.*, 1999). It is strongly recommended that multilevel injections and high doses be used only by experienced clinicians. Recommended doses and dilutions for the management of spasticity in children with cerebral palsy have been previously published (Graham *et al.*, 2000).

Selective dorsal rhizotomy is usually reserved for a highly selected group of children with spastic diplegia who have significant lower limb spasticity and good underlying strength and selective motor control (Peacock and Arens, 1982). Ideally, SDR should be undertaken before there are fixed contractures or the child may require an extensive deformity correction programme, in addition to spasticity management. SDR requires an intensive rehabilitation programme and some children may be unable to cope because of cognitive or behavioural problems. Functional outcomes of SDR were studied in three randomized trials with the control group receiving physical therapy (PT) only. Two of the three studies confirmed functional benefits from the addition of rhizotomy to a programme of physical therapy, but the third and largest found an equal improvement in the PT only compared to the SDR and PT group (Steinbok *et al.*, 1997; McLaughlin *et al.*, 1998; Wright *et al.*, 1998). A recent meta-analysis of the three trials confirmed a functional benefit, as judged by the Gross Motor Function Measure (McLaughlin *et al.*, 2000). In all three studies and in numerous uncontrolled studies, SDR has been shown to permanently reduce spasticity and to improve gait (Vaughan *et al.*, 1991, 1998; Park and Owen, 1992; Thomas *et al.*, 1996). There are some reports that indicate increased SDR-associated musculoskeletal deformities including scoliosis, lordosis, hip migration and foot deformities (Green, 1991).

Intrathecal baclofen is an increasingly widely used option for spasticity management in cerebral palsy and the criteria are less strict than for SDR. Because the dose of baclofen can be adjusted or even discontinued, ITB can be offered as a fully reversible intervention, to more severely involved children, including those with severe spastic quadriplegia or generalized dystonia (Krach, 1999; Gormley *et al.*, 2001). After ITB, there is good evidence for reduced spasticity, improved joint range of motion and

improved comfort, and less robust evidence for improved function (Albright *et al.*, 1995; Albright, 1996). Improvements in gait have been reported on subjective criteria, but have not been confirmed using motion analysis (Gerszten *et al.*, 1998; Piccinini *et al.*, 1999; Motta *et al.*, 2000).

The most widely used interventions in spastic cerebral palsy are therefore injections of BTX-A for focal spasticity and orthopaedic surgery for fixed deformities. A small number of children who satisfy stringent criteria may benefit from SDR. The indications for ITB therapy are evolving and may well expand with further study (Krach, 1999; Gormley *et al.*, 2001).

Postural and gait patterns: management algorithm (Figures 1 and 2)

Although gait and posture are variable in children who have cerebral palsy, there are certain patterns that can be identified and recognized by clinicians using a variety of tools. In general, spastic motor patterns are reasonably consistent from stride to stride and from day to day (Gage, 1991). However, over the longer term, there are often changes with age and as the result of intervention. The most common change with age is from a pattern of 'toe walking' (because the gastrocnemius is dominant) to a pattern of increasing hip and knee flexion and eventually, 'crouch gait' with hip and knee flexion and ankle dorsiflexion (Rab, 1991). The transition from equinus gait to crouch gait is seen in many children with severe spastic diplegia or spastic quadriplegia as a normal progression, but it may be accelerated by an injudicious isolated lengthening of the heel cord (Sutherland and Cooper, 1978; Borton *et al.*, 2001). Variations in gait patterns related to the topographical type of cerebral palsy are best seen in the contrast between unilateral spastic cerebral palsy (spastic hemiplegia) and bilateral spastic cerebral palsy (spastic diplegia and spastic quadriplegia). Although there are at least four types of gait patterns seen in hemiplegia, in general, there is more involvement distally and therefore true equinus is the basis of the most common patterns (Winters *et al.*, 1987). In diplegia and quadriplegia, more proximal involvement is typical and therefore apparent equinus and crouch gait are frequently seen.

Gait patterns can only be precisely identified and accurately categorized by the use of instrumented motion analysis (Gage, 1991). The purpose of this article is to draw attention to common postural patterns that can be recognized by a combination of careful inspection of gait and clinical examination. Observational gait analysis can be greatly enhanced by the use of two-dimensional video recording, especially if

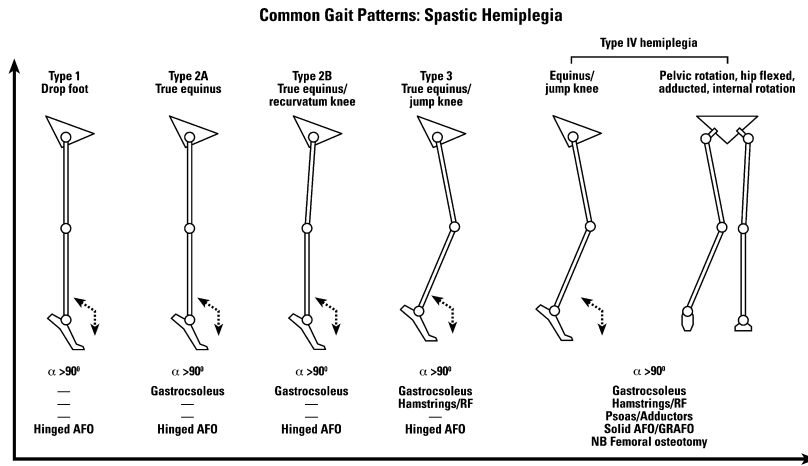


Figure 1 Gait patterns and management algorithm spastic hemiplegia. © Rodda and Graham, Royal Children’s Hospital, Melbourne, Australia.

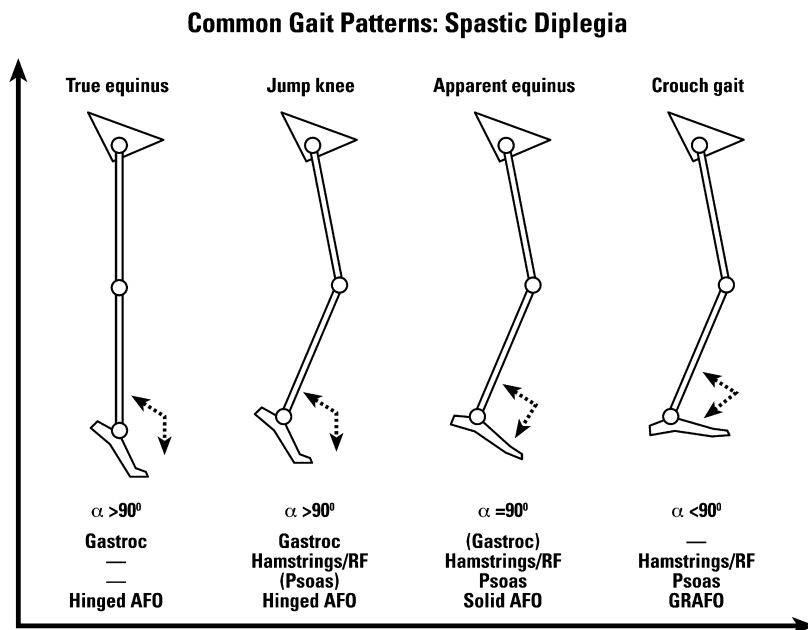


Figure 2 Gait patterns and management algorithm spastic diplegia. © Rodda and Graham, Royal Children’s Hospital, Melbourne, Australia.

there is the facility for slow motion replay (Boyd and Graham, 1999b). These common patterns are more accurately referred to as postural patterns rather than gait patterns. Although the pattern will vary according to the precise part of the gait cycle, the postural patterns referred to here are usually most clearly seen during the middle and end of the stance phase of gait. Given that the focus of this classification are postural patterns caused by spasticity or contracture of the principal sagittal plane motors, the classification is largely based on sagittal plane observation of gait. However, reference to the coronal and transverse planes are necessary in Type 4 hemiplegia and also in diplegia/quadruplegia.

Common postural/gait patterns spastic hemiplegia (Figure 1)

Introduction

The most widely accepted classification of gait in spastic hemiplegia is that reported by Winters *et al.* (1987). They subdivided hemiplegia into four gait patterns based on sagittal plane kinematics. The classification has direct relevance to understanding the gait pattern and management. Given that spasticity in hemiplegia is unilateral, management with SDR and ITB is not appropriate, but BTX-A is very useful for spasticity, and orthopaedic surgery for fixed deformity.

Type 1 hemiplegia

In Type 1 hemiplegia there is a 'drop foot' which is noted most clearly in the swing phase of gait, due to inability to selectively control the ankle dorsiflexors during this part of the gait cycle. There is no calf contracture and therefore during stance phase, ankle dorsiflexion is relatively normal. In our experience, this gait pattern is rare, unless there has already been a calf lengthening procedure. The only management required is a leaf spring ankle foot orthosis (AFO). Spasticity management and contracture surgery are clearly not required.

Management summary

- Spasticity management: Not applicable.
- Contracture management: Not applicable.
- Orthotic management: Leaf spring or hinged AFO.

Type 2 hemiplegia

- 2a Equinus plus neutral knee and extended hip.
- 2b Equinus plus recurvatum knee and extended hip.

Type 2 hemiplegia is by far the most common type in clinical practice. True equinus is noted in the stance phase of gait because of spasticity and/or contracture of the gastroc-soleus muscles. There is a variable degree of drop foot in swing because of impaired function in tibialis anterior and the ankle dorsiflexors. A pattern of true equinus is seen, with the ankle in the plantar flexion range through most of the stance phase. The plantar flexion-knee extension couple is over active and the knee may adopt a position of extension or recurvatum (Boyd and Graham, 1997).

Gastroc-soleus spasticity can be managed by intramuscular botulinum toxin type A injections, especially in the younger child (Cosgrove *et al.*, 1994; Koman *et al.*, 1994, 2000; Corry *et al.*, 1998; Flett *et al.*, 1999). If there is a mild contracture, supplemental casting can be very effective (Boyd and Graham, 1997; Molenaers *et al.*, 1999). The majority of children will also require orthotic support, both to control the tendency to 'drop foot' during the swing phase of gait but also to augment and prolong the response to botulinum toxin type A chemodenervation.

Once a significant fixed contracture develops, lengthening of the gastrocnemius and soleus may be indicated. Type 2 hemiplegia with a fixed contracture of the gastroc-soleus constitutes the only indication for isolated lengthening of the tendo achillis (Graham and Fixsen, 1988; Borton *et al.*, 2001). If the knee is fully extended or in recurvatum, then a hinged AFO with an

appropriate plantar flexion stop is the most appropriate choice of orthosis.

Management summary

- *Spasticity management*: BTX-A dose 4–8 U/kg, divided between 2 and 4 sites.
- *Contracture management*: Tendo Achilles lengthening, Strayer calf lengthening, if the contracture is confined to the gastrocnemius.
- *Orthotic management*: Hinged AFO or leaf spring AFO.

Equinovarus deformities can be managed by concomitant injection of tibialis posterior at the time of calf injection or by split transfer of tibialis posterior (Green, 1991; Boyd and Graham, 1997; O'Byrne *et al.*, 1997). Equinovalgus deformities can be managed initially by calf injection and an AFO. Calf injection often improves AFO tolerance and efficacy. Older children with progressive valgus deformities are likely to become brace intolerant and require bony surgery such as os calcis lengthening or subtalar fusion (Miller *et al.*, 1995).

Type 3 hemiplegia

Type 3 hemiplegia is characterized by gastroc-soleus spasticity or contracture, impaired ankle dorsiflexion in swing and a flexed, 'stiff knee gait' as the result of hamstring/quadriceps cocontraction.

Management may consist of botulinum toxin type A injections for gastroc-soleus spasticity and at a later stage, muscle tendon lengthening for gastroc-soleus contracture. In addition, lengthening of the medial hamstrings with transfer of rectus femoris to the semitendinosus is the most effective long-term solution for the stiff knee (Sutherland *et al.*, 1990; Chambers *et al.*, 1998). A solid or hinged AFO may also be helpful; the choice should be according to the integrity of the 'plantar-flexion, knee-extension couple'.

Management summary

- *Spasticity management*: BTX-A injections to the calf and hamstrings, dose 4 U/kg per muscle, 8 U/kg body weight. BTX-A injections to rectus femoris have been tried with little clinical or motion analysis evidence of success.
- *Contracture management*: tendo achillis lengthening combined with lengthening of the medial hamstrings and transfer of the rectus femoris to the gracilis or semitendinosus.
- *Orthotic management*: solid or hinged AFO, according to the pre and post intervention integrity of the plantar-flexion, knee-extension (PF-KE) couple.

Type 4 hemiplegia (Figure 3)

In Type 4 hemiplegia there is much more marked proximal involvement and the pattern is similar to that seen in spastic diplegia. However, because involvement is unilateral, there will be marked asymmetry, including pelvic retraction. In the sagittal plane there is equinus, a flexed stiff knee, a flexed hip and an anterior pelvic tilt. In the coronal plane, there is hip adduction and in the transverse plane, internal rotation. Management is similar to Type 2 and Type 3 hemiplegia, in respect of

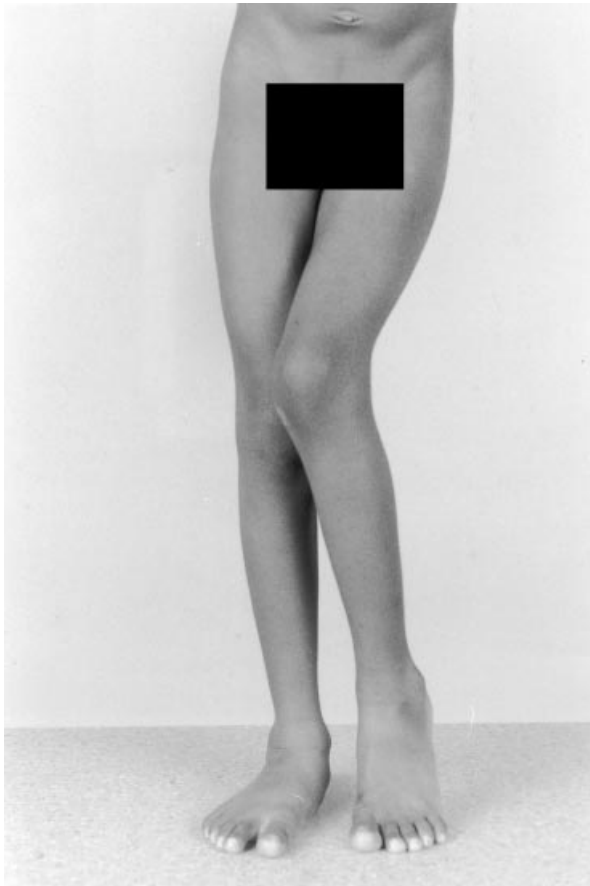


Figure 3 Gage Type IV hemiplegia: this boy has a severe left-sided spastic hemiplegia, Winters and Gage Type IV. He has a fixed equinus at the ankle, a flexed knee, a flexed adducted and internally rotated hip. His motion analysis showed that all of these deformities had the expected effect on gait and that the knee was both flexed and very stiff throughout the gait cycle. His management consisted of lengthening of the tendo Achillis, lengthening of the medial hamstrings and transfer of rectus femoris to semitendinosus, lengthening of adductor longus, lengthening of psoas at the brim of the pelvis and a left proximal femoral derotation osteotomy. Postoperatively he was managed in a solid AFO for the first 6 months and after his gastroc-soleus regained strength, a hinged AFO allowed a more normal gait pattern.

the distal problems. However, there is a high incidence of hip subluxation and careful radiographic examination of the hip is important (Simmons *et al.*, 1998). The adducted and internally rotated hip will usually require lengthening of the adductors and an external rotation osteotomy of the femur. Failure to address the hip adduction and hip internal rotation will usually mean that any distally focused intervention will fail and the overall outcome will be poor.

Management summary

- **Spasticity management:** multilevel injections of BTX-A, including the calf and hamstrings, sometimes the hip adductors and hip flexors, according to the clinical indications and experience of the injector. Dose 4 U/kg/muscle, total dose 12–16 U/kg body weight.
- **Contracture/deformity management:** lengthening of the calf, medial hamstrings (with rectus femoris transfer when indicated), hip adductors and iliopsoas. External rotation osteotomy of the femur;
- **Orthotic management:** ground reaction (Saltiel) AFO, solid AFO or hinged AFO, according to the integrity of the PF–KE couple.

Common postural/gait patterns spastic diplegia/quadriplegia: a management algorithm (Figure 2)

The patterns of knee involvement in spastic diplegia described by Sutherland and Davids are an excellent basis for classifying postural and gait patterns in spastic diplegia (Sutherland and Davids, 1993). The following classification draws heavily on their work but has some important differences:

- The sagittal plane is considered as a whole; i.e. pelvis, hip, knee, and ankle;
- The stiff-knee pattern may be present as part of the other sagittal plane patterns and is therefore not included as a separate entity;
- The classification starts with equinus/calf dominance and ends with crouch/hamstring/hip flexor dominance; and;
- The classification follows the commonly observed changes with age and intervention.

Torsional deformities of the long bones and foot deformities are frequently found in spastic diplegia, in association with musculo-tendinous contractures. These are collectively referred to as ‘lever arm disease’ (Gage, 1991). Muscles work most effectively on rigid bony levers, which are in the line of gait progression. Maldirected or bent levers reduce the effectiveness of muscle action. The most common bony problems are medial femoral torsion, lateral tibial torsion and mid

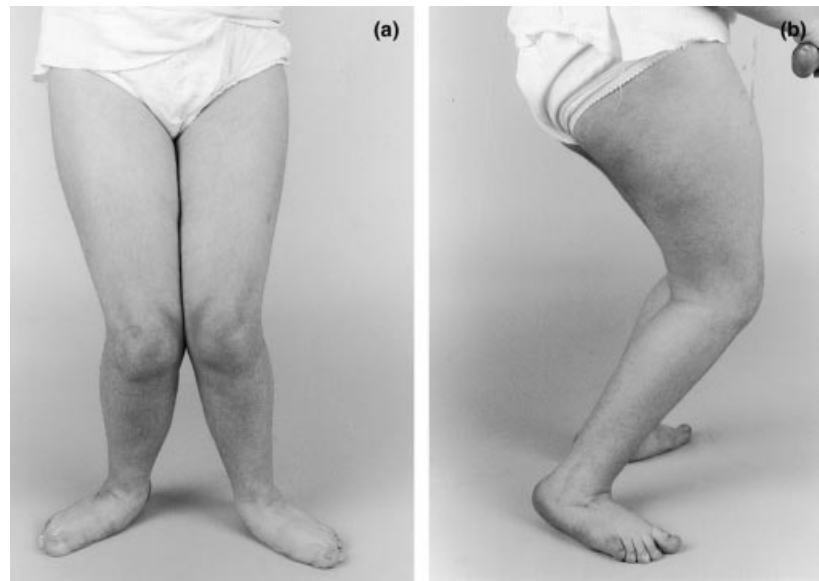


Figure 4 (a) This shows the features of 'lever arm disease'. There is an out toed stance and gait pattern because of mid foot breaching and lateral tibial torsion; (b) shows the sagittal view shows a crouch gait pattern. When the bony lever (the foot) is both bent and maldirected, the already weakened gastroc-soleus is unable to control the progression of the tibia over the planted foot and a crouch gait results.

foot breaching, with foot valgus and abduction. Rotational osteotomies and foot stabilization surgery are often required, in association with the spasticity and contracture management.

The PF-KE couple is a critical biomechanical concept in understanding interrelationships between the foot-ankle and knee levels. A stable foot in the line of progression, when acted on by a competent gastroc-soleus, controls the progression of the tibia over the planted foot during the stance phase of gait. The ground reaction vector is directed anterior to the knee joint, providing an extensor moment at the knee and reducing demands on the quadriceps. A weak or overlengthened gastroc-soleus and a maldirected, breached foot, may contribute to crouch gait (Figure 4a,b). The integrity of the PF-KE couple can be improved by correcting foot deformity, correcting external tibial torsion and using a ground reaction ankle foot orthosis (Gage, 1991).

1. True Equinus

When the younger child with diplegia begins to walk with or without assistance, calf spasticity is frequently dominant resulting in a 'true equinus' gait with the ankle in plantar flexion throughout stance and the hips and knees extended. True equinus may be hidden by the development of recurvatum at the knee. The patient can stand with the foot flat and the knee in recurvatum (Miller *et al.*, 1995). The equinus is real, but hidden. Botulinum toxin type A can be very effective for gastrocnemius spasticity, to improve stability in stance. A more stable base of support can be enhanced by the use of hinged AFO's (Buckon *et al.*, 2001). A few

children with diplegia remain with a true equinus pattern throughout childhood and, if they develop fixed contracture, may eventually benefit from isolated gastrocnemius lengthening. The persistence of this pattern is unusual and seen in only a small minority of children with diplegia. It is more common in children with spasticity, secondary to spinal lesions (e.g. hereditary spastic paraparesis).

Management summary

- *Spasticity management:* BTX-A injections to the calf; 4 U/kg each side, 8 U/kg total.
- *Contracture management:* lengthening of the gastrocnemius.
- *Orthotic management:* solid or hinged AFO, according to the integrity of the PF-KE couple.

2. Jump gait (with or without stiff knee)

(Figure 5a,b,c,d)

The jump gait pattern is very commonly seen in children with diplegia, who have more proximal involvement, with spasticity of the hamstrings and hip flexors in addition to calf spasticity. The ankle is in equinus, the knee and hip are in flexion, there is an anterior pelvic tilt and an increased lumbar lordosis. There is often a stiff knee, because of rectus femoris activity in the swing phase of gait. This is the description given by Sutherland and Davids, although Miller *et al.* describe the ankle as being neutral in jump gait (Sutherland and Davids, 1993; Miller *et al.*, 1995). We prefer the former description and to use the term 'apparent equinus' for flexed hip, flexed knee and plantigrade ankle/foot (Boyd *et al.*, 1999a).

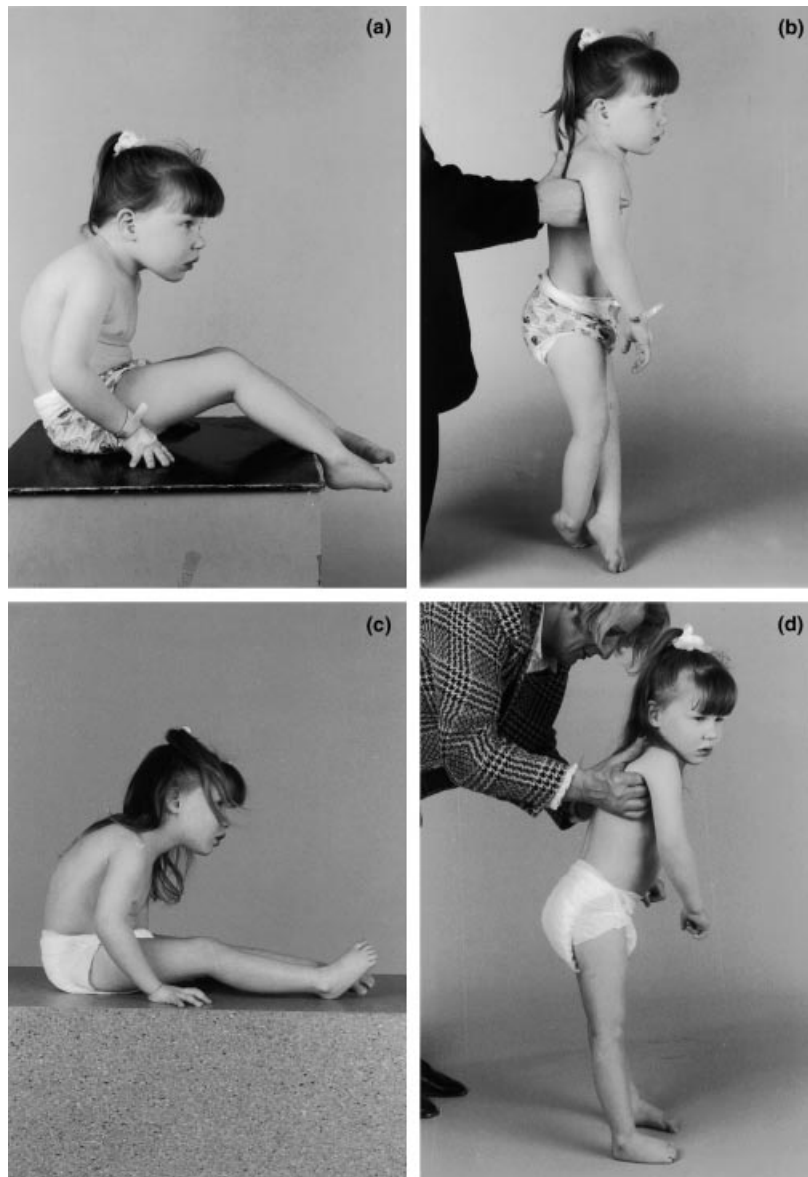


Figure 5 This 3-year-old-girl has severe spastic diplegia and is delayed in standing and walking. In (a) she has difficulty in long sitting, she sits on a posteriorly tilted pelvis with flexed knees. When supported in standing, she has a 'jump gait' pattern with equinus at the ankle, flexion at the knee and flexion at the hip. Clinical examination confirmed that her problems were mostly the result of spasticity in these muscle groups, not contracture. She was managed by injection of botulinum toxin type A at a dose of 4 U/kg BW to the hamstrings bilaterally and to the gastrocsoleus bilaterally. The total dose of botulinum toxin used was 16 U/kg BW. In (c) it can be seen that she can now 'long sit' more comfortably and in (d) her standing posture is improved. Her feet are plantigrade, her knees extend fully but her hip flexion has increased as has her anterior pelvic tilt and lumbar lordosis. She would have benefited from injections to the iliopsoas by the technique described by Molenaers *et al.* (1999).

In younger children, this pattern can be managed effectively by botulinum toxin type A injections to the gastrocnemius and hamstrings and the provision of an AFO. A more extensive multilevel approach, with BTX-A injections may also be appropriate, but is not for the inexperienced practitioner (Molenaers *et al.*, 1999). SDR may be the best solution to achieve a permanent reduction in tone, provided the appropriate indications are observed (Peacock and Arens, 1982; Gormley *et al.*, 2001).

In older children musculotendinous lengthening of the gastrocnemius, hamstrings and iliopsoas may be indicated with transfer of the rectus femoris to semitendinosus for co-contraction at the knee (Gage, 1991;

Molenaers *et al.*, 2001). The jump knee pattern and stiff knee pattern described by Sutherland and Davids, in our experience, frequently coexist.

Management summary

- **Spasticity management:** in younger/less involved children BTX-A injections to the calf and hamstrings. Multilevel injections of BTX-A may be useful. Selective dorsal rhizotomy may be the optimum choice for small group of children, following previously reported selection criteria.

- **Contracture/deformity management:** single event multilevel surgery, addressing all contractures and lever arm dysfunction.

- **Orthotic management:** ground reaction (Saltiel) AFO, solid AFO or hinged AFO according to the integrity of the PF-KE couple.

Apparent equinus (with or without stiff knee) (Figure 6a,b)

As the child gets older and heavier, a number of changes may occur which may render the calf muscle and the PE-KE couple less competent. Equinus may gradually decrease as hip and knee flexion increase (Rab, 1991; Sutherland and Davids, 1993; Molenaers *et al.*, 2001). There is frequently a stage of 'apparent equinus' where the child is still noted to be walking on the toes and simple observational gait analysis may mistakenly conclude that the equinus is real, when it is in fact apparent (Miller *et al.*, 1995; Boyd and Graham, 1997). Sagittal plane kinematics will show that the ankle has a normal range of dorsiflexion but the hip and knee are in excessive flexion throughout the stance phase of gait. At this stage, weakening the gastrocnemius by injections of botulinum toxin type A or lengthening of the gastrocnemius muscle will only provoke a crouch gait and result in impaired function (Sutherland and Cooper, 1978; Borton *et al.*, 2001). Management should be focused on the proximal levels, where the hamstrings and iliopsoas may benefit from spasticity treatment or musculotendinous lengthening (Corry *et al.*, 1999). Redirection of the ground reaction vector in front of the knee can best be achieved by the use of a solid or a ground reaction AFO.

Management summary

- **Spasticity management:** in younger/less involved children, BTX-A injections to the hamstrings and iliopsoas.
- **Contracture/deformity management:** single event multilevel surgery, addressing all contractures and lever arm problems.
- **Orthotic management:** ground reaction (Saltiel) AFO, solid AFO or hinged AFO according to the integrity of the PF-KE couple.

Crouch gait (with or without stiff knee gait) (Figure 7)

Crouch gait is defined as excessive dorsiflexion or calcaneus at the ankle in combination with excessive flexion at the knee and hip. This pattern is part of the natural history of the gait disorder in children with more severe diplegia and in the majority of children with spastic quadriplegia. Regrettably, the commonest cause of crouch gait in children with spastic diplegia is isolated lengthening of the heel cord in the younger child (Sutherland and Cooper, 1978; Borton *et al.*, 2001). Once the heel cord has been lengthened, if the spasticity/contracture of the hamstrings and iliopsoas has not been recognized and is not managed adequately, there will be a rapid increase in hip and knee flexion (Miller *et al.*, 1995). The result is an unattractive, energy expensive gait pattern, followed by anterior knee pain and patellar pathology in adolescence. Frequent BTX-A injections to the gastroc-soleus, without addressing the hamstrings/iliopsoas or providing adequate orthotic support can also lead to

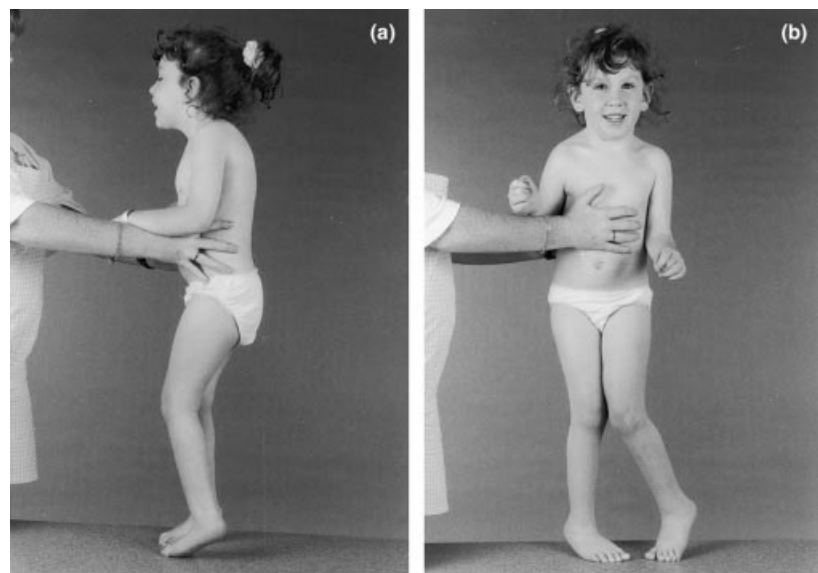


Figure 6 (a) This 4-year-old girl stands on her toes with her heels off the ground. There is also increased hip and knee flexion. This is apparent equinus. The foot is at 90° to the leg, the heel is off the ground because of the hip and knee flexion. (b) In addition to the contractures of the iliopsoas and hamstrings she has an intoed stance and gait because of bilateral medial femoral torsion, another type of lever arm dysfunction.



Figure 7 This is the classic sagittal profile of crouch gait. There is excessive ankle dorsiflexion, increased knee flexion and increased hip flexion. The plantar flexion knee extension couple is incompetent with the ground reaction vector directed behind the knee. This boy was managed by lengthening of the hamstrings and iliopsoas with ground reaction AFOs to redirect the ground reaction vector in front of the knee.

progressive crouch gait. Crouch gait is always difficult to manage and usually requires lengthening of the hamstrings and iliopsoas, a ground reaction AFO and adequate correction of bony problems such as medial femoral torsion, lateral tibial torsion and stabilization of the foot. By the time it is recognized, the musculo-skeletal pathology is usually too advanced to respond to intramuscular BTX-A.

Management summary

- **Spasticity management:** in younger/less involved children BTX-A injections to the hamstrings and hip flexors.
- **Contracture/deformity management:** single event multilevel surgery, addressing all contractures, bony torsional abnormalities and joint instability.

- **Orthotic management:** long-term use of a ground reaction (Saltiel) AFO until the integrity of the PF-KE couple is clearly re-established.

Coronal and transverse plane issues

The simple classification above concentrates on observation and examination of the sagittal plane motors, the gastrocnemius, hamstrings-rectus femoris and iliopsoas. However, the majority of children with spastic diplegia have also coronal plane and transverse plane problems. In the coronal plane, spasticity or contracture of hip adductors may be evident, as well as such issues as limb length discrepancy and hip subluxation.

The transverse plane is the most difficult to appreciate on visual inspection and three-dimensional gait analysis is always required for a full evaluation (Gage, 1991). In the transverse plane the common problems are pelvic rotation, medial femoral torsion, lateral tibial torsion, and foot deformity. Management of sagittal plane problems such as spasticity or contracture will usually fail if there are significant transverse plane problems that are not dealt with. The use of twister cables attached to an AFO may help the younger child for a short period of time but these orthoses are neither particularly effective nor well tolerated. The definitive answer to lever arm disease is bony surgery.

With optimal management of spasticity and muscle length in early childhood, the requirement for musculotendon surgery is decreasing but the requirement for bony surgery remains unchanged. Fortunately, the outcome of bony surgery in cerebral palsy is in general more predictable, more durable and more effective than soft tissue surgery.

Summary and conclusions

The postural and gait patterns in this algorithm can be most accurately identified by using a combination of clinical examination, video recording of gait and instrumented gait analysis. The critical role of clinical examination in the assessment of dynamic and fixed contracture is outside the scope of this article but has been covered comprehensively elsewhere (Boyd *et al.*, 1998).

When instrumented gait analysis is unavailable, pattern recognition may help inform clinicians where to look for associated spasticity or contracture and not to focus on a single problem or anatomic level. The doses of botulinum toxin used by the author are found in detail in previous publications and need not be repeated here (Cosgrove *et al.*, 1994; Corry *et al.*, 1998; Graham *et al.*, 2000). The orthotic recommendations

are necessarily general rather than specific. This should not be misconstrued to imply that orthotic prescription is unimportant. Appropriate orthotic use is critical to the magnitude and duration of response to spasticity and contracture management. The principal issue is that the degree of orthotic support is predicated on the integrity of the PE-KE couple before intervention. The PF-KE couple is invariably altered and is frequently unstable after intervention. Thus, orthoses must be regularly evaluated as an integral part of spasticity management in children with cerebral palsy. Despite some excellent recent studies, the precise indications for the posterior leaf spring AFO, hinged AFO, solid AFO and ground reaction or Saltiel AFO, have not been fully established (Harrington *et al.*, 1984; Ounpuu *et al.*, 1996; Abel *et al.*, 1998; Buckon *et al.*, 2001).

Many questions remain unanswered and there is ample scope for more clinical trials with objective outcome measures. Many of the existing studies have small numbers, short-term follow up and the outcome measures are neither comprehensive nor sufficiently objective.

References

- Abel MF, Juhl GA, Vaughan CL, Damiano DL (1998). Gait assessment of fixed ankle-foot orthoses in children with spastic diplegia. *Arch Phys Med Rehab* **79**:126–132.
- Albright AL (1996). Intrathecal baclofen in cerebral palsy movement disorders. *J Child Neurol* **11**:S29–S35.
- Albright AL, Barry MJ, Fasick MP, Janosky J (1995). Effects of continuous intrathecal baclofen infusion and selective posterior rhizotomy on upper extremity spasticity. *Pediatr Neurosurg* **23**:82–85.
- Borton DC, Walker K, Pirpiris M, Nattrass GR, Graham HK (2001). Isolated calf lengthening in cerebral palsy – outcome analysis of risk factors. *J Bone Joint Surg (Br)* **83**:364–370.
- Boyd R, Barwood SA, Baillieu CE, Graham HK (1998). Validity of a clinical measure of spasticity in children with cerebral palsy in a randomized clinical trial. *Dev Med Child Neurol* **40**:S7 [Abstract].
- Boyd R, Graham HK (1997). Botulinum toxin A in the management of children with cerebral palsy: indications and outcome. *Eur J Neurol* **4**:S15–S22.
- Boyd RN, Graham HK (1999b). Objective measurement of clinical findings in the use of botulinum toxin type A for the management of children with cerebral palsy. *Eur J Neurol* **6**(Suppl. 4):23–35.
- Boyd RN, Graham JEA, Nattrass GR, Graham HK (1999a). Medium-term response characterisation and risk factor analysis of botulinum toxin type A in the management of spasticity in children with cerebral palsy. *Eur J Neurol* **6**:S37–S46.
- Buckon CE, Thomas SS, Jacobson-Huston S, Sussman M, Aiona M (2001). Comparison of three ankle-foot orthosis configurations for children with spastic hemiplegia. *Dev Med Child Neurol* **43**:371–378.
- Chambers H, Lauer A, Kaufman K *et al.* (1998). Prediction of outcome after rectus femoris surgery in cerebral palsy: The role of co-contraction of the rectus femoris and vastus lateralis. *J Pediatr Orthoped* **18**:703–711.
- Corry IS, Cosgrove AP, Duffy CM, McNeill S, Taylor TC, Graham HK (1998). Botulinum toxin A compared with stretching casts in the treatment of spastic equinus: a randomised prospective trial. *J Pediatr Orthoped* **18**:304–311.
- Corry IS, Cosgrove AP, Duffy CM, Taylor TC, Graham HK (1999). Botulinum toxin A in hamstring spasticity. *Gait Posture* **10**:206–210.
- Cosgrove AP, Corry IS, Graham HK (1994). Botulinum toxin in the management of the lower limb in cerebral palsy. *Dev Med Child Neurol* **36**:386–396.
- Dolly JO, Black J, Williams RS, Melling J (1984). Acceptors for botulinum neurotoxin reside on motor nerve terminals and mediate its internalization. *Nature* **307**:457–460.
- Flett PJ, Stem LM, Waddy H, Connell TM, Seeger JD, Gibson SK (1999). Botulinum toxin A versus fixed cast stretching for dynamic calf tightness in cerebral palsy. *J Paediatric Children's Health* **35**:71–77.
- Gage JR (1991). *Gait Analysis in Cerebral Palsy*. London: MacKeith Press.
- Gerszten PC, Albright AL, Johnstone GF (1998). Intrathecal baclofen infusion and subsequent orthopaedic surgery in patients with spastic cerebral palsy. *J Neurosurg* **88**:1009–1013.
- Gormley ME, Krach LE, Piccini L (2001). Spasticity management in the child with spastic quadriplegia. *Eur J Neurol* **8**(Suppl. 5):127–135.
- Graham HK, Aoki KR, Autti-Ramo I *et al.* (2000). Recommendations for the use of botulinum toxin type A in the management of cerebral palsy. *Gait Posture* **11**:67–79.
- Graham HK, Fixsen JA (1988). Lengthening of the calcaneal tendon in spastic hemiplegia by the White Slide technique. *J Bone Joint Surg (Br)* **70**:472–475.
- Green NE (1991). Split posterior tibial tendon transfer: The universal procedure. In: Sussman, MD, ed. *The Diplegic Child*. Rosemont, Illinois: American Academy of Orthopaedic Surgeons.
- Harrington ED, Lin RS, Gage JR (1984). Use of the anterior floor reaction orthosis in patients with cerebral palsy. *Bull Orthotics Prosthetics* **37**:34–42.
- Houltram J, Noble I, Boyd RN, Corry I, Flett P, Graham HK (2001). Botulinum toxin type A in the management of equinus in children with cerebral palsy: an evidence-based economic evaluation. *Eur J Neurol* **8** (Suppl. 5):194–202.
- Koman LA, Mooney JF, 3rd Smith BP, Goodman A, Mulvaney T (1994). Management of spasticity in cerebral palsy with botulinum-A toxin: report of a preliminary, randomized, double-blind trial. *J Pediatr Orthoped* **14**:299–303.
- Koman LA, Mooney JF, Smith BP, Walker F, Leon JM and the Botox Study Group (2000). Botulinum toxin A neuromuscular blockade in the treatment of lower extremity spasticity in cerebral palsy: a randomised, double blind, placebo-controlled trial. *J Pediatr Orthoped* **20**: 108–115.
- Krach LE (1999). Management of intrathecal baclofen withdrawal: a case series. *Dev Med Child Neurol* **41**:S11.
- Loewen P, Steinbok P, Holsti L, MacKay M (1998). Upper extremity performance and self-care skill changes in children with spastic cerebral palsy following selective posterior rhizotomy. *Pediatr Neurosurg* **29**:191–198.
- McLaughlin JF, Bjornson KF, Astley SJ *et al.* (1998). Selective dorsal rhizotomy: efficacy and safety in an

- investigator-masked randomized clinical trial. *Dev Med Child Neurol* **40**:220–232.
- McLaughlin JF, Bjornson KF, Temkin N *et al.* (2000). Selective dorsal rhizotomy: meta-analysis of three randomized clinical trials. *Dev Med Child Neurol* **42**:S25.
- Miller F, Dabney K, Rang M (1995). Complications in cerebral palsy treatment. In: Epps, CH, Bowen, JR, eds., *Complications in Pediatric Orthopaedic Surgery*. Philadelphia: JB Lippincott, pp. 498–500.
- Molenaers G, Desloovere K, Eyssen M, Decat J, Jonkers I, De Cock P (1999). Botulinum toxin type A treatment of cerebral palsy: an integrated approach. *Eur J Neurol* **6**:S51–S57.
- Molenaers G, Desloovere K, Jonkers I *et al.* (2001). Single event multilevel botulinum toxin type A treatment and surgery: similarities and differences. *Eur J Neurol* **8**(Suppl. 5):88–97.
- Motta F, Galli M, Sibella F, Crivellini M (2000). The effect of intrathecal baclofen therapy on gait in children with cerebral palsy. *Dev Med Child Neurol* **49**:SP27(Abstract).
- Muhonen MG (2001). Spasticity and the baclofen pump. In: McGlone, DG, ed. *Pediatric Neurosurgery: Surgery of Developmental Neurological Systems*. Philadelphia: W.B. Saunders, pp. 1066–1074.
- O’Byrne JM, Kennedy A, Jenkinson A, O’Brien TM (1997). Split tibialis posterior tendon transfer in the treatment of spastic equinovarus foot. *J Pediatr Orthoped* **17**:481–485.
- Ounpuu S, Bell KJ, Davis RB, De Luca PA (1996). An evaluation of the posterior leaf spring orthosis using kinematics and kinetics *J Pediatr Orthoped* **16**:378–384.
- Park TS, Owen JH (1992). Surgical management of spastic diplegia in cerebral palsy. *N Engl J Med* **326**:745–749.
- Peacock WJ, Arens LJ (1982). Selective posterior rhizotomy for the relief of spasticity in cerebral palsy. *South African Med J* **62**:745–749.
- Piccinini L, Krach LE, Gage JR, Novacheck T (1999). Gait and functional evaluation pre- and postintra-thecal baclofen pump implantation. [Abstract]. *Dev Med Child Neurol* **41**:S12.
- Rab GT (1991). Diplegic Gait: Is there more than spasticity? In: Sussman, M, ed. *The Diplegic Child*. Rosemount, Illinois: American Academy of Orthopaedic Surgeons.
- Simmons T, Smithson F, Rodda J, Starr R, Boyd R, Graham HK (1998). The high incidence and clinical significance of hip dysplasia in Winters and Gage type 4 hemiplegia. [Abstract]. *Dev Med Child Neurol* **40**:S5.
- Steinbok P, Reiner AM, Beauchamp R, Armstrong RW, Cochrane DD (1997). A randomized clinical trial to compare selective posterior rhizotomy plus physiotherapy with physiotherapy alone in children with spastic diplegic cerebral palsy. *Dev Med Child Neurol* **39**:178–184.
- Sutherland DH, Cooper L (1978). The pathomechanics of progressive crouch gait. *Spastic Diplegia Orthopedic Clinics North Am* **9**:143–154.
- Sutherland DH, Davids JR (1993). Common gait abnormalities of the knee in cerebral palsy. *Clin Orthopaedics Related Res* **288**:139–147.
- Sutherland D, Santi M, Abel MF (1990). The treatment of stiff-knee gait in cerebral palsy: a comparison by gait analysis of distal rectus femoris transfer versus proximal rectus release. *J Pediatr Orthoped* **10**:433–441.
- Thomas SS, Aiona MD, Pierce R, Piatt JH (1996). Gait changes in children with spastic diplegia after selective dorsal rhizotomy. *J Pediatr Orthoped* **16**:747–752.
- Vaughan CL, Berman B, Peacock WJ (1991). Cerebral palsy and rhizotomy. A 3 year follow up evaluation with gait analysis. *J Neurosurg* **74**:178–184.
- Vaughan CJ, Subramanian N, Busse ME (1998). Selective dorsal rhizotomy as a treatment option for children with spastic cerebral palsy. *Gait Posture* **8**:43–59.
- Winters TF, Gage JR, Hicks R (1987). Gait patterns in spastic hemiplegia in children and young adults. *J Bone Joint Surg (Am)* **69**:437–441.
- Wright FV, Sheil EMH, Drake JM, Wedge JH, Naumann S (1998). Evaluation of selective dorsal rhizotomy for the reduction of spasticity in cerebral palsy: a randomized controlled trial. *Dev Med Child Neurol* **40**:239–247.