

Movement Disorders in Cerebral Palsy

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Abstract

Keywords

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- dystonia
- athetosis
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Children with cerebral palsy often have a mixture of multiple disorders of movement that interact with each other and with the developmental process. While this complicates the process of symptomatic diagnosis, there is nevertheless a close link between clinical impairments and the underlying etiology and distribution of injury. I describe the major categories of impairment, including hypertonic symptoms, hyperkinetic symptoms, and negative signs. Within each category, there are specific features that are helpful for distinguishing between multiple impairments that affect motor function. Identification of the particular impairments affecting each child is essential to guide appropriate medical and rehabilitation interventions.

Introduction

Cerebral palsy (CP) has always been considered to be a poorly defined or “wastebasket” diagnosis. In fact, it is a very precise syndrome with a set of impairments in movement that can be closely linked to the underlying injury. This is analogous to the precision with which stroke syndromes can be defined in adults. Correct diagnosis of subtypes of CP as well as the impairments that lead to disability can be essential for identification of the most appropriate treatment. The current definition of CP has been established by an interdisciplinary group: “CP describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of CP are often accompanied by disturbances of sensation, perception, cognition, communication, and behavior; by epilepsy; and by secondary musculoskeletal problems.”¹ The importance of this definition is that it stresses the evolving clinical picture despite a static and nonprogressive injury. Therefore, appropriate diagnosis and treatment requires a careful understanding of the interaction between injury and development.

The subtypes of CP are classified by the distribution and nature of impairments. The most common classification includes diplegic, hemiplegic, tetraplegic, dyskinetic, and

ataxic.² Although these types are generally easily distinguishable, in children there are often mixed symptoms that make classification more difficult.³ The importance of these subtypes is the relatively close link between the anatomic distribution of impairment and the underlying etiology. For example, diplegic CP is most often caused by injury to periventricular white matter as is frequently seen in prematurity or in placental insufficiency during the third trimester.⁴ Hemiplegic CP is most often caused by unilateral ischemic injury due to presumed embolic events in the perinatal period, and this most closely mimics adult stroke. The tetraplegic form is often associated with widespread gray matter injury as might occur with profound perinatal acute hypoxic injury or bacterial meningitis. The dyskinetic form is associated with acute partial hypoxic injury often at the time of term birth.⁵ Dyskinetic CP can also be associated with several metabolic abnormalities including kernicterus that are known to affect basal ganglia, and possibly with vasculitis of lenticulostriate vessels in response to perinatal inflammatory processes.^{6,7} The ataxic form of CP is often caused by developmental anomalies of the parietal lobes or posterior fossa, including malformations of the cerebellum.⁸ During the first year of life, such anomalies may present with hypotonia that only develops into ataxia several years later.⁹

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Because of the close link between etiology and clinical impairment, there is a close link between the neuroimaging findings and the clinical findings similar to what can be seen in adult stroke. Unlike adult stroke where the categories are primarily defined by anatomic localization of injury, in children the localization is more often defined by cell type such that certain disorders may affect neuronal cellular elements and are often referred to as “gray matter injury,” whereas others may have preferential injury of glial cells including oligodendrocytes, and are thus referred to as “white matter injury.”⁴ Changes in the relative susceptibility of neurons and glial cells to hypoxic injury during the third trimester of pregnancy account for the relative rates of injury to white matter or gray matter as a function of the timing of injury.^{4,10,11} For example, injury early in the third trimester either due to premature birth or placental insufficiency more frequently affects oligodendrocyte maturation and leads to periventricular white matter disorders,^{11,12} whereas injury late in the third trimester or at term birth typically causes more selective injury to neuronal elements.⁴ The pattern of gray matter injuries seen in acute hypoxic events near term has been referred to as “selective neuronal necrosis” and can affect basal ganglia, periorolandic cortex, substantia nigra, and the subiculum of the hippocampus.⁴

A detailed understanding of the relationship between timing of injury, mechanism of injury, distribution of injury, and the consequent symptoms and body distribution of impairment is essential for understanding the nature of the movement disorders in CP and for making appropriate diagnoses and selecting the most appropriate treatments. In particular, it is important to be able to distinguish CP from other treatable diagnoses that may mimic it during certain phases of the illness.¹³ Because CP is a disorder characterized by static pathology, there are no biochemical markers and when it is caused by acquired injury there are no genetic markers. Therefore, it is important to look for and exclude similar-appearing metabolic disorders and neurotransmitter disorders that may have specific treatments.

Classification of Impairments

The NIH Taskforce on Childhood Motor Disorders was established to classify the different motor disorders that cause impairments in childhood. Here, we will discuss those that are commonly seen in CP. The intent of the taskforce was to define words at the impairment level rather than at the etiology or syndromic level. For example, it was important that “spasticity” be defined in terms that could be identified at a single joint, rather than as part of a “spasticity syndrome” or “upper motor neuron syndrome,” which implies particular patterns or particular etiologies that in some cases are not well supported by scientific evidence.³ It was also important that impairments be defined in such a way that they can be recognized when multiple impairments coexist in the same child. Therefore, the definitions are based on clinically observable phenomena that could be identified by a trained examiner. Definitions based on impairments provide a link between the underlying pathophysiology and the resulting

disability at a level of description that may be particularly suitable for choosing medical or surgical intervention.^{3,14,15}

The different types of impairments can be roughly classified into positive and negative signs. The positive signs are generally related to excess unwanted activation of muscles, whereas the negative signs are generally characterized by lack of appropriate activation of muscles.¹⁴ These definitions are not always consistent and sometimes are overlapping. For example, hypertonia that is caused by excess muscle activation can be a cause of slow movement that would ordinarily be considered a negative sign. One particular reason why the positive/negative distinction is helpful is that there may be an approximate link to categories of treatment. Many of the available medical and surgical interventions are more effective at reducing positive signs than improving negative signs. For example, increased tone about a joint can be reduced by medication or surgery, but most medical or surgical interventions do not directly increase strength. Conversely, interventions such as rehabilitation and movement practice often improve negative signs including strength and coordination.

Movement disorders in adults are often classified by the distinction between “pyramidal” and “extrapyramidal” causes. This distinction does not correspond well to currently known neuroanatomy; therefore, although these terms remain commonly used in the adult literature, we will avoid them. The movement disorders in CP are usually mixed disorders and in some cases may partially compensate for each other. For example, spasticity can compensate for weakness. We prefer the term “motor disorder” to distinguish it from the classical use of the term “movement disorder” as seen in adults. The most important consideration is that treatment needs to address the multiple factors that affect each child’s movement and that will affect their development of movement and motor skill. In the following sections, we will describe individual motor disorders based on the definitions of the taskforce.

Positive Signs

We can divide the positive signs into those that primarily affect posture and those that primarily result in movement. The three signs that primarily affect posture are spasticity, hypertonic dystonia, and rigidity, all of which are characterized by hypertonia. The signs commonly seen in CP that primarily result in movement are hyperkinetic dystonia, chorea, choreoathetosis, and athetosis. Myoclonus, tremor, tics, and stereotypies can occur in CP but are not commonly associated with it.

Spasticity

Spasticity is thought to be caused by changes in the excitability thresholds of spinal motor neuron pools.¹⁶ This may be mediated by neurotransmitters including serotonin, and possibly it is associated with persistent inward calcium currents.^{17,18} Whatever the mechanism, spasticity is characterized by a threshold for activation of motor units that depends on both position and velocity.^{19,20} Although in very severe forms of spasticity it may not be possible to

detect the threshold owing to inability to move the joint, one of the defining features is the “spastic catch.”³ Spasticity tends to cause either complete flexion or complete extension of a joint, maintaining it either at the joint limit or tending to apply force such that it moves toward a joint limit. It can thus be distinguished from normal postural mechanisms that tend to stabilize at intermediate ranges of joint angle. Normal postural mechanisms do not have thresholds and tend to have a much more graded response to perturbation.

The treatment of spasticity is complicated by the fact that in some cases it may compensate for weakness. Spasticity is frequently seen in children with significant weakness, and when it affects antigravity muscles it may facilitate maintenance of posture against gravity. Spasticity that leads to extensor tone in the hips and knees may allow a child to use a stander, whereas their own ability to voluntarily extend their hips and knees may be significantly less. Spasticity affecting trunk musculature, while difficult to evaluate because of the impossibility of detecting a spastic catch in the trunk, may be an important compensation for truncal weakness. The effects of spasticity on gait can be complex. Often it creates abnormal postures and in some cases it is thought that triggering of spastic reflexes may be responsible for the “jump” or “bounce gait” pattern.^{21–23}

Because of the persistence of spastic postures, and probably because of associated abnormal recruitment and activation patterns of muscles, there are permanent changes that include muscle and tendon shortening,^{24,25} joint breakdown, or joint dislocation that can lead to permanent orthopedic deformities.²² This may be worsened when spasticity persists during sleep. Although the mechanisms of normal sleep paralysis are not completely known,²⁶ it is possible that abnormalities of spinal motoneuron excitability in spasticity may interfere with those mechanisms and prevent full relaxation of muscles during sleep.

Spasticity is associated with the function of particular descending spinal tracts. The vestibulospinal and reticulospinal tracts may play a role, and the pattern of extension or flexion of muscles may be partly linked to vestibular inputs,^{27,28} although this is not certain.²⁹ In CP, the bulbospinal tracts including vestibulospinal, reticulospinal, and rubrospinal tracts remain intact, although perhaps poorly modulated by cortical control. The particular patterns of flexion and extension in the upper and lower extremities that are often seen in spasticity and are sometimes referred to as “pyramidal tract weakness” probably represent the relative contributions of the vestibulospinal, reticulospinal, and rubrospinal tracts to patterns of extension and flexion.³⁰

Less severe periventricular white matter injury (periventricular leukomalacia) is confined to the fibers closest to the ventricles that carry signals for the legs. More severe cases also affect the more laterally placed arm fibers.³¹ Thus, symmetric injury to these regions tends to produce spastic diplegia in milder cases, and spastic tetraplegia in more severe cases.^{32,33} Because involvement of bilateral upper extremities is associated with more severe global injury, there may also be significant effects on gray matter and therefore a more mixed picture that includes dystonia, seiz-

ures, and impaired cognitive function. When symptoms are unilateral (often due to stroke or intraventricular hemorrhage), then it is possible to have involvement of both arm and leg (on one side) with relative sparing of gray matter structures. Thus, upper extremity spasticity is most commonly seen in hemiplegic CP, because in tetraplegic CP dystonia will often obscure the presence of a spastic catch at the wrist or elbow.

Dystonia

The mechanisms of dystonia are not well known and it is difficult to quantify. Dystonia is almost always detrimental, and unlike spasticity it does not compensate for other deficits. It is often associated with injury to the basal ganglia, but more recent investigations have suggested that injury to cerebellum, thalamus, and some regions of the cortex may also be responsible.^{34,35} While in dyskinetic CP we frequently see dystonia associated with injury to the basal ganglia, close inspection of diffusion-weighted imaging in acute hypoxic-ischemic injury suggests that other areas including substantia nigra and precentral gyrus may be affected and therefore it is difficult to be certain of the anatomic localization. Dystonia seems to be associated with increased long latency reflexes that may be a mixture of spinal and transcortical in origin.^{36,37} The most characteristic feature of dystonia is a set of stereotyped involuntary postures that are inserted into or superimposed on attempts at voluntary movement. These postures may be associated with hypertonia, but hypertonia is not necessarily a component of dystonia.³

Typical postures in dystonia involve joints at a midposition of joint angle, suggesting a very precise balance of activity in flexors and extensors and the ability to maintain that balance against perturbation. This is a feature that is also characteristic of voluntary control of posture and is not expected to arise solely from spinal mechanisms. There is a graded response to perturbation such that large perturbations away from the involuntary posture typically lead to a larger muscle response. Unlike spasticity, there is not a threshold for response.

Dystonic postures are typically triggered by attempts at voluntary movement. Dystonia almost always resolves during sleep, probably due to the supraspinal and suprabulbar origins of the disorder. Dystonia is much less commonly associated with permanent musculoskeletal injury than spasticity. Although in very severe forms of dystonia there can be changes in bony structures or dislocation of joints, in moderate to severe cases often there is no permanent orthopedic injury and the muscle and tendon shortening that is commonly seen in spasticity does not seem to occur in dystonia. Whether there are permanent changes in muscle morphology due to dystonia similar to those seen in spasticity is unknown.

The taskforce distinguished the possibility of dystonia causing movement or hyperkinetic features as well as hyper-tonic features.¹⁵ We will discuss separately the definition of chorea and choreoathetosis, but it is not known whether hyperkinetic forms of dystonia represent the combination of postural (hypertonic) dystonia with choreoathetosis or whether they represent a different phenomenon. Certain

forms of dystonic tremor are seen in genetic and metabolic dystonias but less commonly in CP.

Rigidity

Pure rigidity in children is rare, and because rigidity is commonly associated with dystonia it may not be easily distinguishable in CP. The “lead pipe” type of rigidity seen in Parkinson disease is usually associated either with neuroleptic treatment or with rare disorders such as juvenile Parkinson disease or neurotransmitter disorders. Dopamine deficiency in childhood seems to primarily produce dystonia, sometimes combined with bradykinesia. This is best known from dopa-responsive dystonia (Segawa disease), in which there is a partial failure of production of dopamine. It is important to be aware of the existence of this disorder because it can be a mimic of CP, yet usually quite treatable. The most common cause of dopaminergic failure in CP may be due to the selective neuronal necrosis pattern of injury associated with acute hypoxic injury at term.⁴ This pattern can affect substantia nigra pars compacta and thus the origins of dopaminergic projections to striatum. There may also be injury in striatum itself and so it is likely that there is a combination of both presynaptic and postsynaptic dopaminergic failure. The injury to presynaptic cells may partly explain the response of some children with CP to treatment with levodopa, but the injury to postsynaptic cells may explain why response is only partial and why many children do not respond to levodopa or dopamine agonists.

Lead pipe rigidity suggests resistance to movement without a preferred posture. This would be characteristic of viscosity and therefore velocity dependent (but without a threshold as in spasticity). It has been shown that even in children with dystonia, there is a continuum of position dependence and velocity dependence with most children showing a mixture of position- and velocity-dependent responses to passive movement.³⁶ Therefore, rigidity may represent either one extreme of this spectrum or a separate disorder. The importance of recognizing rigidity in CP is because relatively pure rigidity may be an important clue to the need for further investigation of other possible etiologies.

Hyperkinetic Disorders

Positive symptoms that lead to movement are hyperkinetic disorders. The hyperkinetic disorders that occur in CP are hyperkinetic dystonia, choreoathetosis, chorea, and athetosis. It is not known whether choreoathetosis represents the combination of chorea and dystonia, whether it is the same as hyperkinetic dystonia, or whether it is a completely different phenomenon.¹⁵

Hyperkinetic Dystonia and Choreoathetosis

Choreoathetosis is named for the combination of chorea and athetosis, but athetosis is rarely seen on its own and it is not known if this is a separate phenomenon. Athetosis is defined to be a slow writing movement, whereas chorea is a faster movement but the distinction can be subtle. Careful observa-

tion of video recordings of children with choreoathetosis frequently reveals inserted dystonic postures, suggesting that these two phenomena may be closely linked. Both dystonia and choreoathetosis are exacerbated by attempts at voluntary movement or posture.

Hyperkinetic dystonia and choreoathetosis are associated with injury to basal ganglia. When injury is due to perinatal hypoxic injury, subsequent magnetic resonance imaging scans often show bilateral regions of injury in the medial globus pallidus and the lateral thalamus. When injury is due to kernicterus, the magnetic resonance imaging scans are usually normal.

Chorea

Chorea is characterized by stereotyped inserted movement fragments as opposed to the stereotyped inserted postures seen in dystonia. These fragments are often not easily visible to the examiner but can be discerned by close examination of video recordings. Chorea is not usually associated with CP and should prompt an investigation for other underlying metabolic or inflammatory causes. One of the features that may be helpful in distinguishing the choreoathetosis commonly seen in dyskinetic CP from chorea seen in other disorders is that in chorea there is usually movement at attempted rest, giving the impression of “fidgetiness.” Although it can be exacerbated by the state of alertness and overall activity, chorea is often not significantly exacerbated by movement and appears to interfere with movement much less than would otherwise be expected. In contrast, choreoathetosis seems to worsen significantly with attempts at movement very similar to what is seen in hypertonic dystonia. Children with chorea compensate well and are often surprisingly accurate in attempts to reach to a target. They do not seem to fall while walking despite significant dancing movements. In contrast, children with choreoathetosis caused by CP are often severely impaired by their movements, and they have difficulty walking or using their arms.

Other Hyperkinetic Disorders

Other hyperkinetic disorders that can be seen in childhood include myoclonus, tremor, tics, and stereotypies. These are probably no more common in CP than in otherwise unaffected children. Myoclonus is very concerning and suggests gray matter irritability often associated with genetic, metabolic, inflammatory, or degenerative disorders, and it should prompt a thorough search for such causes. True rhythmic tremor is not considered a feature of CP and is rare in children. The most common form of tremor in childhood is familial essential tremor. Tic disorders and stereotypies are not thought to occur with higher frequency in CP than in otherwise unaffected children.

Negative Signs

The next category of motor disorders is the negative signs.¹⁴ These include weakness, hypotonia, bradykinesia, reduced selective motor control, ataxia, balance disorders, and dyspraxia. Not all of these were defined by the taskforce. The

negative signs are the ones that are most amenable to rehabilitation, practice, and skill acquisition and in many cases do not respond to medical or surgical interventions. These signs may be more impairing than the positive signs and may prevent compensation for other motor deficits.

Weakness

Weakness is relatively difficult to define because the ability to generate voluntary force and involuntary force need to be separated. The taskforce defined weakness as the inability to generate voluntary force.¹⁴ Therefore, a joint with very actively contracting or co-contracting muscles that may be quite hypertonic and generate very high resistance to passive force applied by the examiner would still be considered weak if the child is not able to move that joint or modulate the force voluntarily. It is quite typical for clinicians to test force by asking the child to resist passive movement, but this would confound weakness with hypertonia and therefore it is important to ensure that the child can generate force both isometrically and during active movement.

There are multiple etiologies of weakness including both peripheral and central causes. In CP, probably the most common cause is periventricular white matter injury leading to inability to modulate strength in the lower extremities. Injury to the cellular origins of the corticospinal tract in frontal cortex would be another possible cause. In many cases, injury to corticospinal tract is associated with lack of ability to generate fine modulation of force, but the overall range of force that can be generated may not be decreased. In other words, corticospinal injury can be manifested more as a deficit of the fine control of force rather than a deficit of the ability to produce force.

Hypotonia

The task force did not provide a definition of hypotonia, but this is often seen in children younger than 1 year. Hypotonia does not usually persist in CP and it often subsequently evolves into ataxia or spasticity.^{38,39} Hypotonia is decreased resistance to passive stretch. Hypotonia is difficult to measure reliably⁴⁰ and it must be distinguished from weakness. One might imagine that in pure hypotonia, voluntary force and speed of movement would be normal, yet the resistance to perturbation would be decreased. This could lead to an appearance of floppiness and during attempts at locomotion might lead to instability on uneven surfaces. It is important to realize that there are significant differences in baseline tone even between different healthy children: some children maintain relatively high tone, while others have relatively low tone during normal behavior. Only when hypotonia does not modulate appropriately with voluntary movement can we identify it as pathological.

The frequent association of infantile hypotonia with cerebellar malformations and subsequent ataxia suggests that the cerebellum may be an important mediator of the control of tone.⁴¹ Hypotonia seen in older children is more typical of peripheral neuromuscular disorders. While parents and clinicians may be acutely aware of hypotonia, this is rarely if ever a

complaint of the children themselves unless there is associated weakness.

Bradykinesia

Another negative sign that was not defined by the taskforce is bradykinesia. Bradykinesia is most commonly seen in parkinsonian syndromes and may be associated with dystonia in several different disorders.^{42–45} Slow movement in dystonia is usually attributed to cocontraction, but in fact there is no evidence nor biomechanical mechanism to suggest that cocontraction would produce slow movement.^{36,37,46} Bradykinesia may represent a combination of a slow rate of movement, slowed rate of increase or decrease of force, and delay in initiation of movement. In extreme forms of bradykinesia, there can be up to a 30-second pause between the request for a voluntary movement and the actual initiation of that movement. The examiner must be aware of this and allow the child adequate time. Similarly if the child has a very slow rate of increase in force, they may appear weak unless given enough time to allow the force to increase to the desired level.

In adults, bradykinesia is typically associated with dopaminergic dysfunction,⁴⁷ and the fact that dystonia in childhood is often associated with either pre- or postsynaptic dopaminergic dysfunction provides a possible mechanism for the coincidence of these two disorders. However, the actual origins of bradykinesia have not been well studied in children, and dopamine receptors may have different distributions with age.⁴⁸ Furthermore, quantification of bradykinesia separately from weakness or dystonia is difficult.

Reduced Selective Motor Control

A very important negative sign that was defined by the taskforce is reduced selective motor control.⁴⁹ Reduced selective motor control is the inability to activate muscles except in particular patterns or synergies.¹⁴ For example, in the lower extremities there appears to be an extensor synergy that involves simultaneous extension of the hip, knee, and ankle.^{50,51} A child with reduced selective motor control will have difficulty simultaneously flexing the ankle and extending the knee or flexing the knee and extending the ankle.^{52,53} This provides the common diagnostic method which can be performed in a gait laboratory in which a surface electromyography electrode will be placed on the thigh muscle and the child asked to maintain the thigh relaxed while extending the ankle.

It is particularly important to recognize the presence of reduced selective motor control because the extensor pattern mimics spasticity. The way in which this can be distinguished from spasticity is that full range of motion of the joints can be easily achieved so long as the synergy pattern is respected. For example, full dorsiflexion of the ankle may be possible with the knee and hip flexed, whereas full extension of the ankle may only be possible with the knee and hip extended. If this is the case, it suggests that surgical interventions designed to reduce involuntary ankle extension will not be effective as the problem is not constrained by the biomechanics. Reduced selective motor control can mimic weakness because activation of muscles out of synergy will be very

difficult and will appear to represent a decreased voluntary ability to generate force.

In adults, there appears to be an upper extremity synergy that involves adduction of the shoulder and extension of the elbow.^{54–56} It is not known whether or not a similar synergy exists in children with CP.

While scales have been developed to quantify reduced selective motor control,⁵³ treatment of this disorder is frustrating and attempts at retraining patterns of spinal activity have limited success. Discovery of new treatment methods will be particularly important because this impairment is a major contributor to inefficient and poorly balanced gait.

Ataxia

Ataxia is the inability to coordinate muscles throughout a movement.¹⁴ Active movement requires complex patterns of muscle activity that not only achieve the movement but also compensate for the dynamic forces generated by the musculoskeletal system and inertia of the limb. It can be very difficult to identify ataxia clinically in the presence of weakness or dystonia. Furthermore, movement in ataxia may be abnormal due to compensatory strategies adopted by the child. Clinical evaluation is facilitated by noting that in ataxia multijoint movement is more difficult than single-joint movement,^{57,58} and rapid movement is more difficult than slow movement.⁵⁹ For example, dysmetria on a finger-to-target task may be improved if the elbow is stabilized or the child is asked to move more slowly. Children may have difficulty compensating for perturbations,⁶⁰ and they may not learn to compensate even with practice on a known perturbation.^{61,62} Since different regions of the cerebellum are responsible for control of limbs, axial posture and balance, and eye movements, it is important to assess all three types of function individually.^{63,64}

Ataxic CP is rare and usually is associated with malformations of the hindbrain.^{9,65} It is particularly notable that ataxia is only rarely seen in children with other acquired injuries such as partial resection of the cerebellum,⁶⁶ with ataxia occurring more frequently only when deep cerebellar nuclei are involved.⁶⁷ Like reduced selective motor control, ataxia can be significantly impairing to many aspects of motor function. The cerebellum is also involved with oculomotor function and associated disorders including oculomotor apraxia can occur.⁶⁸

Balance Disorders

Balance disorders are critical impediments to unassisted ambulation.^{69,70} These were not defined specifically by the taskforce and ongoing research is needed to appropriately classify and quantify balance disorders in children. Nevertheless, it is clear that many children with CP including relatively mild forms have difficulty compensating for perturbations during gait,⁷¹ and standing or ambulating over uneven terrain is particularly challenging for many of these children.⁷² Balance disorders in adults are often due to multiple factors including sensory deficits, impairment of multijoint reflexes, impairment of coordination, weakness, vestibular or visual impairments, and deficits of sensory integration. It is likely

that in children, similar multifactorial ideologies must be considered.⁶⁹ Balance does appear to be a trainable skill,⁷⁰ but the origins of the balance disorder in an individual child must first be precisely assessed.

Dyspraxia and Apraxia

The taskforce distinguished between apraxia and dyspraxia.¹⁴ Apraxia occurs more commonly in adults and is the loss of skilled movements associated with specific tasks or gestures. A common adult classification originally by Liepmann separates adult apraxia into ideational, ideomotor, and limb-kinetic forms.^{73,74} Whether this taxonomy is helpful in children is not known. In children, we define dyspraxia as the inability to acquire age-appropriate skill despite sufficient exposure and practice. As dyspraxia represents lack of acquisition of skills, it is a learning disability for movement. There may be sensory, motor, cognitive, or sensory-motor integration deficits that can contribute to dyspraxia. Dyspraxia is the major motor deficit seen in developmental coordination disorder,^{75–77} and autistic spectrum disorders, but its prevalence in CP is unknown.

One of the most important consequences of dyspraxia is the inability to learn to compensate for other movement disorders. In particular, a child with primary dystonia who does not have dyspraxia may learn very interesting and creative ways to compensate for their dystonic postures so that they can still accomplish the necessary skill. A child with dyspraxia will be unable to compensate. As another example, a child with ataxia but without dyspraxia will have a wide-based gait to compensate for the gait instability due to their ataxia. A child with concomitant dyspraxia may not widen the base of their gait and therefore may be more likely to fall. In adults, apraxia is associated with injury to premotor regions. The localization of dyspraxia in children is unknown.

Dyspraxia interferes with rehabilitation because one of the characteristic features of dyspraxia is the inability to copy complex gestures or tasks when demonstrated.⁷³ This interferes with the ability to be taught new skills. It is therefore very difficult for children with dyspraxia to make use of therapists, coaches, or successful examples of performance by other children. They have difficulty with tool use, hand grip, and writing. Because of the increased processing demands for complex movements, there may be a delay in processing that may mimic bradykinesia. Unusual postures adopted during tasks and compensatory muscle activity or increased cocontraction due to relatively increased cognitive demands may cause dyspraxia to mimic dystonia. It is very important to recognize that dyspraxia commonly co-occurs with other motor disorders and to treat it separately if possible. The optimal treatment regimen is unknown,⁷⁸ but one method is to break down complex movements into much simpler ones that can be practiced individually and later recombined.⁷⁵

Treatment

Although it is usually impossible to treat the underlying brain injury that caused the CP, there are nevertheless many

opportunities for treatment of the resulting impairments. Optimal treatment generally requires an approach to all of the impairments, although the type of intervention and the priorities for intervention must be determined by careful consultation with the child and parents to determine their goals for functional ability. In many cases, successful treatment will require consideration of factors not just at the impairment level but also the effect of these impairments on specific skills, the effect of those skills on participation in school and social activities, and the effect of the environment on facilitating or impeding adequate performance.⁷⁹ I will very briefly summarize some of the ways in which treatment categories can be associated with specific impairments.

Perhaps most important is treatment of mechanical and orthopedic issues. Children will not learn or progress if they are in pain, and joint deformities, dislocations, or other neurologically mediated biomechanical deficits that lead to pain must be rectified as soon as possible. Careful attention to seating posture, the use of orthotics, and bracing systems will all have potential for significant benefit and improvement of overall function.

Medical treatment is usually confined to positive signs, but it is important to remember that spasticity is often useful and complete reduction of spasticity may reveal underlying weakness that prevents the child from progressing. Perhaps the most common oral medication for treatment of spasticity is baclofen,⁸⁰ and other medications include tizanidine, diazepam, and injection of botulinum toxin.^{79,81,82} The mechanism of botulinum toxin function in CP is not well understood. Since the goal of botulinum injection is to titrate so that spasticity can be reduced without causing significant weakness, it is likely that the primary effect in most cases is reduced excitation of intrafusal fibers, thereby reducing length and velocity-dependent stretch reflexes.

Botulinum toxin may have a similar mechanism in dystonia mediated by long-latency stretch reflexes.³⁶ Reduction in these reflexes may be helpful in reducing inserted postures or reducing the tone associated with the inserted postures. Other medications frequently used for dystonia in CP include anticholinergic medications such as trihexyphenidyl (benzhexol) and levodopa for possible presynaptic dopaminergic deficits.^{83–85} Carbamazepine has been helpful in some children with hyperkinetic dystonia, but other antidyskinetic medications commonly used in adults including amantadine or tetrabenazine have been less helpful in CP.^{86,87}

Most negative signs are not amenable to medical intervention with the exception of bradykinesia which is treated primarily with dopaminergic or stimulant medications. The mainstay of treatment for negative signs remains guided practice which can be performed in a rehabilitation setting. For older children, particularly teenagers, therapy can be transitioned to a sports and fitness model rather than a medical model. This will be much more interesting to the child and likely to reinforce positive self-esteem if they feel that they are accomplishing skills that are of interest to them. In such cases, the role of rehabilitation clinicians remains important to monitor for complications of abnormal biomechanics and movement.

Children with CP are at risk of late worsening particularly associated with unusual postures or unusual repetitive movements and this needs to be carefully evaluated over time and corrected as soon as problems are identified.⁸⁸ Late worsening can include progressive joint disease due to disuse, or spinal abnormalities from unusual postures or dyskinetic movements.⁸⁹ The long-term effect of specific therapy interventions including medical and surgical interventions is not known. It is important to remember that CP is a developmental disorder and therefore changes are expected over time, and while hopefully many of these changes involve improvements, it is also important to look for worsening that can occur due to skeletal growth, associated changes in body habitus, associated medical issues, or abnormal patterns of movement.⁹⁰

Prosthetics and orthotics are of significant benefit for a large number of children. It is important to distinguish between devices that are intended to assist children or substitute for lost function, and devices whose purpose is to train the child for better function of their own body. Typical assistive devices include assistive communication devices, adapted keyboards, and mobility devices such as a wheelchair. Concerns are frequently raised that increasing dependence on assistive devices will impede the child's ability to develop their own natural skills in these areas. While this is certainly a risk, it is also important to provide children with devices that allow them adequate participation in school and social function so that those skills may be learned even before their motor function improves.

Devices that are designed for retraining include virtual reality,^{91–93} and robotic training environments,^{94–96} as well as other environments with altered dynamics including the use of spring-like attachments to the child⁹⁷ or assisted treadmill training with harness support against gravity.^{98–100} For some children, the use of elastic or neoprene garments¹⁰¹ or elastic tape applied to the skin^{102–104} may be helpful. In these cases, the goal is for the movement to feel as close to the intended movement as possible and to give the child the experience of more natural movement while accentuating attention to the proprioceptive consequences of movement. Therefore, the therapist and the devices can apply graded resistance to movement rather than assistance because normal movement is usually against at least mild levels of resistance. The use of altered dynamic environments is a topic of ongoing research.

In conclusion, the movement disorders in CP are complex and most children have multiple disorders simultaneously leading to multifactorial effects on function. Many of the disorders will interact with each other and will also interact with ongoing development, skill learning, and school performance. The effect of injury is compounded by the child's need to learn new skills during a period of motor development. Careful attention to improvement in the most important impairments for each child may have long-lasting consequences in terms of their overall level of function and participation. The very close link between etiology, impairment, and treatment options for children with CP makes accurate diagnosis, categorization, and selection of targeted treatment essential.

References

- 1 Rosenbaum P, Paneth N, Leviton A, et al. A report: the definition and classification of cerebral palsy April 2006. *Dev Med Child Neurol Suppl* 2007;109:8–14
- 2 Prevalence and characteristics of children with cerebral palsy in Europe. *Dev Med Child Neurol* 2002;44(9):633–640
- 3 Sanger TD, Delgado MR, Gaebler-Spira D, Hallett M, Mink JW; Task Force on Childhood Motor Disorders. Classification and definition of disorders causing hypertonia in childhood. *Pediatrics* 2003;111(1):e89–e97
- 4 Volpe JJ. *Neurology of the Newborn*. Philadelphia, PA: WB Saunders; 2000
- 5 Sanger T. Hyperkinetic disorders in childhood. In: Suchowersky O, Comella C, eds. *Hyperkinetic Movement Disorders*. Humana Press; 2012:221–258
- 6 Mittendorf R, Kuban K, Pryde PG, Gianopoulos JG, Yousefzadeh D. Antenatal risk factors associated with the development of lenticulostriate vasculopathy (LSV) in neonates. *J Perinatol* 2005; 25(2):101–107
- 7 Sisman J, Logan JW, Westra SJ, Allred EN, Leviton A. Lenticulostriate vasculopathy in extremely low gestational age newborns: Inter-rater variability of cranial ultrasound readings, antecedents and postnatal characteristics. *J Pediatr Neurol* 2014;12(4): 183–193
- 8 Korzeniewski SJ, Birbeck G, DeLano MC, Potchen MJ, Paneth N. A systematic review of neuroimaging for cerebral palsy. *J Child Neurol* 2008;23(2):216–227
- 9 Miller G, Cala LA. Ataxic cerebral palsy—clinico-radiologic correlations. *Neuropediatrics* 1989;20(2):84–89
- 10 Back SA, Han BH, Luo NL, et al. Selective vulnerability of late oligodendrocyte progenitors to hypoxia-ischemia. *J Neurosci* 2002;22(2):455–463
- 11 Back SA, Gan X, Li Y, Rosenberg PA, Volpe JJ. Maturation-dependent vulnerability of oligodendrocytes to oxidative stress-induced death caused by glutathione depletion. *J Neurosci* 1998; 18(16):6241–6253
- 12 Buser JR, Maire J, Riddle A, et al. Arrested preoligodendrocyte maturation contributes to myelination failure in premature infants. *Ann Neurol* 2012;71(1):93–109
- 13 Sanger TD. Pathophysiology of pediatric movement disorders. *J Child Neurol* 2003;18(Suppl 1):S9–S24
- 14 Sanger TD, Chen D, Delgado MR, Gaebler-Spira D, Hallett M, Mink JW; Taskforce on Childhood Motor Disorders. Definition and classification of negative motor signs in childhood. *Pediatrics* 2006;118(5):2159–2167
- 15 Sanger TD, Chen D, Fehlings DL, et al. Definition and classification of hyperkinetic movements in childhood. *Mov Disord* 2010; 25(11):1538–1549
- 16 Rymer WZ, Katz RT. Mechanisms of spastic hypertonia. *Phys Med Rehabil* 1994;8(3):441–451
- 17 Heckmann CJ, Gorassini MA, Bennett DJ. Persistent inward currents in motoneuron dendrites: implications for motor output. *Muscle Nerve* 2005;31(2):135–156
- 18 Heckman CJ, Johnson M, Mottram C, Schuster J. Persistent inward currents in spinal motoneurons and their influence on human motoneuron firing patterns. *Neuroscientist* 2008;14(3):264–275
- 19 Levin MF, Feldman AG. The role of stretch reflex threshold regulation in normal and impaired motor control. *Brain Res* 1994;657(1–2):23–30
- 20 Jobin A, Levin MF. Regulation of stretch reflex threshold in elbow flexors in children with cerebral palsy: a new measure of spasticity. *Dev Med Child Neurol* 2000;42(8):531–540
- 21 Rodda J, Graham HK. Classification of gait patterns in spastic hemiplegia and spastic diplegia: a basis for a management algorithm. *Eur J Neurol* 2001;8(Suppl 5):98–108
- 22 Wren TA, Rethlefsen S, Kay RM. Prevalence of specific gait abnormalities in children with cerebral palsy: influence of cerebral palsy subtype, age, and previous surgery. *J Pediatr Orthop* 2005;25(1):79–83
- 23 Rodda JM, Graham HK, Carson L, Galea MP, Wolfe R. Sagittal gait patterns in spastic diplegia. *J Bone Joint Surg Br* 2004;86(2): 251–258
- 24 Smith LR, Lee KS, Ward SR, Chambers HG, Lieber RL. Hamstring contractures in children with spastic cerebral palsy result from a stiffer extracellular matrix and increased in vivo sarcomere length. *J Physiol* 2011;589(Pt 10):2625–2639
- 25 Barrett RS, Lichtwark GA. Gross muscle morphology and structure in spastic cerebral palsy: a systematic review. *Dev Med Child Neurol* 2010;52(9):794–804
- 26 Brooks PL, Peever JH. Identification of the transmitter and receptor mechanisms responsible for REM sleep paralysis. *J Neurosci* 2012;32(29):9785–9795
- 27 Sheean G. The pathophysiology of spasticity. *Eur J Neurol* 2002;9 (Suppl 1):3–9, 53–61
- 28 Pandyan AD, Gregoric M, Barnes MP, et al. Spasticity: clinical perceptions, neurological realities and meaningful measurement. *Disabil Rehabil* 2005;27(1–2):2–6
- 29 Brown P. Pathophysiology of spasticity. *J Neurol Neurosurg Psychiatry* 1994;57(7):773–777
- 30 Orlovsky GN. The effect of different descending systems on flexor and extensor activity during locomotion. *Brain Res* 1972;40(2): 359–371
- 31 Melhem ER, Hoon AH Jr, Ferrucci JT Jr, et al. Periventricular leukomalacia: relationship between lateral ventricular volume on brain MR images and severity of cognitive and motor impairment. *Radiology* 2000;214(1):199–204
- 32 Volpe JJ. Neurobiology of periventricular leukomalacia in the premature infant. *Pediatr Res* 2001;50(5):553–562
- 33 Rogers B, Msall M, Owens T, et al. Cystic periventricular leukomalacia and type of cerebral palsy in preterm infants. *J Pediatr* 1994;125(1):S1–S8
- 34 Neychev VK, Fan X, Mitev VI, Hess EJ, Jinnah HA. The basal ganglia and cerebellum interact in the expression of dystonic movement. *Brain* 2008;131(Pt 9):2499–2509
- 35 Neychev VK, Gross RE, Lehericy S, Hess EJ, Jinnah HA. The functional neuroanatomy of dystonia. *Neurobiol Dis* 2011; 42(2):185–201
- 36 van Doornik J, Kukke S, Sanger TD. Hypertonia in childhood secondary dystonia due to cerebral palsy is associated with reflex muscle activation. *Mov Disord* 2009;24(7):965–971
- 37 Kukke SN, Sanger TD. Contributors to excess antagonist activity during movement in children with secondary dystonia due to cerebral palsy. *J Neurophysiol* 2011;105(5):2100–2107
- 38 Jones MW, Morgan E, Shelton JE, Thorogood C. Cerebral palsy: introduction and diagnosis (part I). *J Pediatr Health Care* 2007; 21(3):146–152
- 39 Rosenbloom L. Diagnosis and management of cerebral palsy. *Arch Dis Child* 1995;72(4):350–354
- 40 Pomeroy VM, Dean D, Sykes L, et al. The unreliability of clinical measures of muscle tone: implications for stroke therapy. *Age Ageing* 2000;29(3):229–233
- 41 Bodensteiner JB. The evaluation of the hypotonic infant. *Semin Pediatr Neurol* 2008;15(1):10–20
- 42 Brashear A, Butler LJ, Ozelius LJ, et al. Rapid-onset dystonia-parkinsonism: a report of clinical, biochemical, and genetic studies in two families. *Adv Neurol* 1998;78:335–339
- 43 Young AB, Shoulson I, Penney JB, et al. Huntington's disease in Venezuela: neurologic features and functional decline. *Neurology* 1986;36(2):244–249
- 44 Nygaard TG, Trugman JM, de Yebenes JG, Fahn S. Dopa-responsive dystonia: the spectrum of clinical manifestations in a large North American family. *Neurology* 1990;40(1):66–69
- 45 Cook DG, Fahn S, Brait KA. Chronic manganese intoxication. *Arch Neurol* 1974;30(1):59–64

- 46 Malfait N, Sanger TD. Does dystonia always include co-contraction? A study of unconstrained reaching in children with primary and secondary dystonia. *Exp Brain Res* 2007;176(2):206–216
- 47 Tabaddor K, Wolfson LI, Sharpless NS. Diminished ventricular fluid dopamine metabolites in adult-onset dystonia. *Neurology* 1978;28(12):1254–1258
- 48 Segawa M. Development of the nigrostriatal dopamine neuron and the pathways in the basal ganglia. *Brain Dev* 2000;22 (Suppl 1):S1–S4
- 49 Löwing K, Brogren Carlberg E. Reliability of the selective motor control scale in children with cerebral palsy. *Adv Physiother* 2009;11(2):58–63
- 50 Zwaan E, Becher JG, Harlaar J. Synergy of EMG patterns in gait as an objective measure of muscle selectivity in children with spastic cerebral palsy. *Gait Posture* 2012;35(1):111–115
- 51 Cahill-Rowley K, Rose J. Etiology of impaired selective motor control: emerging evidence and its implications for research and treatment in cerebral palsy. *Dev Med Child Neurol* 2014;56(6): 522–528
- 52 Fowler EG, Goldberg EJ. The effect of lower extremity selective voluntary motor control on interjoint coordination during gait in children with spastic diplegic cerebral palsy. *Gait Posture* 2009; 29(1):102–107
- 53 Fowler EG, Staudt LA, Greenberg MB, Oppenheim WL. Selective Control Assessment of the Lower Extremity (SCALE): development, validation, and interrater reliability of a clinical tool for patients with cerebral palsy. *Dev Med Child Neurol* 2009;51(8): 607–614
- 54 Krabben T, Prange GB, Molier BI, Rietman JS, Buurke JH. Objective measurement of synergistic movement patterns of the upper extremity following stroke: an explorative study. *IEEE Int Conf Rehabil Robot* 2011;2011:5975430
- 55 Sukal TM, Ellis MD, Dewald JP. Shoulder abduction-induced reductions in reaching work area following hemiparetic stroke: neuroscientific implications. *Exp Brain Res* 2007;183(2):215–223
- 56 Ellis MD, Holubar BG, Acosta AM, Beer RF, Dewald JP. Modifiability of abnormal isometric elbow and shoulder joint torque coupling after stroke. *Muscle Nerve* 2005;32(2):170–178
- 57 Topka H, Konczak J, Schneider K, Boose A, Dichgans J. Multijoint arm movements in cerebellar ataxia: abnormal control of movement dynamics. *Exp Brain Res* 1998;119(4):493–503
- 58 Bastian AJ, Martin TA, Keating JG, Thach WT. Cerebellar ataxia: abnormal control of interaction torques across multiple joints. *J Neurophysiol* 1996;76(1):492–509
- 59 Bastian AJ. Mechanisms of ataxia. *Phys Ther* 1997;77(6):672–675
- 60 Bakker M, Allum JH, Visser JE, et al. Postural responses to multidirectional stance perturbations in cerebellar ataxia. *Exp Neurol* 2006;202(1):21–35
- 61 Bastian AJ. Cerebellar limb ataxia: abnormal control of self-generated and external forces. *Ann N Y Acad Sci* 2002;978:16–27
- 62 Shimansky Y, Wang JJ, Bauer RA, Bracha V, Bloedel JR. On-line compensation for perturbations of a reaching movement is cerebellar dependent: support for the task dependency hypothesis. *Exp Brain Res* 2004;155(2):156–172
- 63 Dichgans J, Diener H. Clinical evidence for functional compartmentalization of the cerebellum. In: Bloedel JR, Dichgans J, Wolfgang Precht W, eds. *Cerebellar Functions*. Berlin, Heidelberg: Springer; 1985:126–147
- 64 Dietrichs E. Clinical manifestation of focal cerebellar disease as related to the organization of neural pathways. *Acta Neurol Scand Suppl* 2008;188:6–11
- 65 Miller G. Minor congenital anomalies and ataxic cerebral palsy. *Arch Dis Child* 1989;64(4):557–562
- 66 Aarsen FK, Van Dongen HR, Paquier PF, Van Mourik M, Catsman-Berrevoets CE. Long-term sequelae in children after cerebellar astrocytoma surgery. *Neurology* 2004;62(8): 1311–1316
- 67 Konczak J, Schoch B, Dimitrova A, Gizewski E, Timmann D. Functional recovery of children and adolescents after cerebellar tumour resection. *Brain* 2005;128(Pt 6):1428–1441
- 68 Guzzetta A, Mercuri E, Cioni G. Visual disorders in children with brain lesions: 2. Visual impairment associated with cerebral palsy. *Eur J Paediatr Neurol* 2001;5(3):115–119
- 69 Woollacott MH, Shumway-Cook A. Postural dysfunction during standing and walking in children with cerebral palsy: what are the underlying problems and what new therapies might improve balance? *Neural Plast* 2005;12(2-3):211–219, discussion 263–272
- 70 Gage JR, Novacheck TF. An update on the treatment of gait problems in cerebral palsy. *J Pediatr Orthop B* 2001;10(4):265–274
- 71 Hsue BJ, Miller F, Su FC. The dynamic balance of the children with cerebral palsy and typical developing during gait. Part I: Spatial relationship between COM and COP trajectories. *Gait Posture* 2009;29(3):465–470
- 72 Rose J, Wolff DR, Jones VK, Bloch DA, Oehlert JW, Gamble JG. Postural balance in children with cerebral palsy. *Dev Med Child Neurol* 2002;44(1):58–63
- 73 Goldenberg G. Apraxia and beyond: life and work of Hugo Liepmann. *Cortex* 2003;39(3):509–524
- 74 Goldenberg G. Apraxia - the cognitive side of motor control. *Cortex* 2014;57:270–274
- 75 Polatajko HJ, Cantin N. Developmental coordination disorder (dyspraxia): an overview of the state of the art. *Semin Pediatr Neurol* 2005;12(4):250–258
- 76 Polatajko H, Fox M, Missiuna C. An international consensus on children with developmental coordination disorder. *Can J Occup Ther* 1995;62(1):3–6
- 77 Gibbs J, Appleton J, Appleton R. Dyspraxia or developmental coordination disorder? Unravelling the enigma. *Arch Dis Child* 2007;92(6):534–539
- 78 Mandich AD, Polatajko HJ, Macnab JJ, Miller LT. Treatment of children with Developmental Coordination Disorder: what is the evidence? *Phys Occup Ther Pediatr* 2001;20(2-3): 51–68
- 79 Russman BS, Tilton A, Gormley ME Jr. Cerebral palsy: a rational approach to a treatment protocol, and the role of botulinum toxin in treatment. *Muscle Nerve Suppl* 1997;6:S181–S193
- 80 Albright AL. Baclofen in the treatment of cerebral palsy. *J Child Neurol* 1996;11(2):77–83
- 81 Koman LA, Mooney JF III, Smith BP, Walker F, Leon JM; BOTOX Study Group. Botulinum toxin type A neuromuscular blockade in the treatment of lower extremity spasticity in cerebral palsy: a randomized, double-blind, placebo-controlled trial. *J Pediatr Orthop* 2000;20(1):108–115
- 82 Graham HK, Aoki KR, Autti-Rämö I, et al. Recommendations for the use of botulinum toxin type A in the management of cerebral palsy. *Gait Posture* 2000;11(1):67–79
- 83 Jankovic J. Treatment of dystonia. *Lancet Neurol* 2006;5(10): 864–872
- 84 Sanger TD. Pediatric movement disorders. *Curr Opin Neurol* 2003;16(4):529–535
- 85 Hoon AH Jr, Freese PO, Reinhardt EM, et al. Age-dependent effects of trihexyphenidyl in extrapyramidal cerebral palsy. *Pediatr Neurol* 2001;25(1):55–58
- 86 Pranzatelli MR. Oral pharmacotherapy for the movement disorders of cerebral palsy. *J Child Neurol* 1996;11(Suppl 1): S13–S22
- 87 Heggarty H, Wright T. Tetrabenazine in athetoid cerebral palsy. *Dev Med Child Neurol* 1974;16(2):137–142
- 88 Ando N, Ueda S. Functional deterioration in adults with cerebral palsy. *Clin Rehabil* 2000;14(3):300–306
- 89 Fuji T, Yonenobu K, Fujiwara K, et al. Cervical radiculopathy or myelopathy secondary to athetoid cerebral palsy. *J Bone Joint Surg Am* 1987;69(6):815–821

- 90 Gajdosik CG, Cicirello N. Secondary conditions of the musculoskeletal system in adolescents and adults with cerebral palsy. *Phys Occup Ther Pediatr* 2001;21(4):49–68
- 91 Bryanton C, Bossé J, Brien M, McLean J, McCormick A, Sveistrup H. Feasibility, motivation, and selective motor control: virtual reality compared to conventional home exercise in children with cerebral palsy. *Cyberpsychol Behav* 2006;9(2):123–128
- 92 Deutsch JE, Borbely M, Filler J, Huhn K, Guarrera-Bowlby P. Use of a low-cost, commercially available gaming console (Wii) for rehabilitation of an adolescent with cerebral palsy. *Phys Ther* 2008;88(10):1196–1207
- 93 Snider L, Majnemer A, Darsaklis V. Virtual reality as a therapeutic modality for children with cerebral palsy. *Dev Neurorehabil* 2010;13(2):120–128
- 94 Meyer-Heim A, Ammann-Reiffer C, Schmartz A, et al. Improvement of walking abilities after robotic-assisted locomotion training in children with cerebral palsy. *Arch Dis Child* 2009;94(8):615–620
- 95 Fluet GG, Qiu Q, Kelly D, et al. Interfacing a haptic robotic system with complex virtual environments to treat impaired upper extremity motor function in children with cerebral palsy. *Dev Neurorehabil* 2010;13(5):335–345
- 96 Smania N, Bonetti P, Gandolfi M, et al. Improved gait after repetitive locomotor training in children with cerebral palsy. *Am J Phys Med Rehabil* 2011;90(2):137–149
- 97 Bar-Haim S, Harries N, Belokopytov M, et al. Comparison of efficacy of Adeli suit and neurodevelopmental treatments in children with cerebral palsy. *Dev Med Child Neurol* 2006; 48(5):325–330
- 98 Cherng RJ, Liu CF, Lau TW, Hong RB. Effect of treadmill training with body weight support on gait and gross motor function in children with spastic cerebral palsy. *Am J Phys Med Rehabil* 2007; 86(7):548–555
- 99 Krebs HI, Ladenheim B, Hippolyte C, Monterroso L, Mast J. Robot-assisted task-specific training in cerebral palsy. *Dev Med Child Neurol* 2009;51(Suppl 4):140–145
- 100 Begnoche DM, Pitetti KH. Effects of traditional treatment and partial body weight treadmill training on the motor skills of children with spastic cerebral palsy. A pilot study. *Pediatr Phys Ther* 2007;19(1):11–19
- 101 Siracusa C, Taynor M, Geletka B, Overby A, Willan M. Effectiveness of a biomechanical intervention in children with spastic diplegia. *Pediatr Phys Ther* 2005;17(1):83–84
- 102 Yasukawa A, Patel P, Sisung C. Pilot study: investigating the effects of Kinesio Taping in an acute pediatric rehabilitation setting. *Am J Occup Ther* 2006;60(1):104–110
- 103 Iosa M, Morelli D, Nanni MV, et al. Functional taping: a promising technique for children with cerebral palsy. *Dev Med Child Neurol* 2010;52(6):587–589
- 104 da Costa CS, Rodrigues FS, Leal FM, Rocha NA. Pilot study: Investigating the effects of Kinesio Taping® on functional activities in children with cerebral palsy. *Dev Neurorehabil* 2013; 16(2):121–128