Medical Management of Spasticity in Children with Cerebral Palsy

Maura McManus

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Abstract

Spasticity is the most common motor disorder in cerebral palsy (CP). It is a component of the upper motor neuron syndrome. Spasticity is probably due to an imbalance between inhibitory and excitatory impulses in the spinal cord. In CP, there is believed to be a deficiency of descending impulses that typically stimulate the release of the inhibitory neurotransmitter gamma-aminobutyric acid (GABA). Although some spasticity may be necessary for function in children with neurologic impairment, it is often a problem that can be difficult to treat. Multiple approaches are available for treatment

M. McManus (✉)
Nemours Alfred I Dupont Hospital for Children, Wilmington, DE, USA
e-mail: mmcmanus@nemours.org; Maura.McManus@nemours.org

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of spasticity in patients with CP including therapies, oral medications, chemodenervation, and intrathecal baclofen therapy. Orthopedic and neurosurgical procedures are also available. A multidisciplinary team should be involved in defining reasonable treatment goals including the patient, and family, physical and occupational therapists, nurses, physiatrist, neurologist, orthopedist, and neurosurgeon.

Keywords
Spasticity · Cerebral palsy · Function · Chemodenervation

Introduction

In patients with cerebral palsy, spasticity is the most common motor disorder and is seen in approximately two thirds of the population (Gans and Glenn 1990). It is part of the upper motor neuron syndrome and is often defined as a velocity-dependent increase in resistance to passive stretch associated with increased deep tendon reflexes (Lance 1980). Spasticity is noted in children with other neurologic impairment as well including brain injury and spinal cord injury. It is probably due to an imbalance between inhibitory and excitatory impulses in the spinal cord. In CP, there is believed to be a deficiency of descending impulses that typically stimulate the release of the inhibitory neurotransmitter gamma-aminobutyric acid (GABA). GABA acts presynaptically to inhibit the release of excitatory neurotransmitters such as glutamate and aspartate, resulting in relative excess of excitatory impulses and resultant hypertonia (Gormley 1999). Some spasticity is necessary for function in children with neurologic impairment, but it can also be a difficult problem to treat. Multiple approaches are available for treatment of spasticity in patients with CP including therapies, oral medications, chemodenervation, and intrathecal baclofen therapy. Orthopedic and neurosurgical procedures are also available (Ried et al. 1998).

Pathophysiology

The physiology of spasticity includes the stretch reflex which is the basic neural mechanism responsible for maintaining muscle tone. It is mediated in large part by monosynaptic connections in the spinal cord. Excitability of stretch reflex depends critically on descending control from higher brain centers. This reflex can be tested by tapping the patella tendon (Rymer and Katz 1994). The basic circuitry of the stretch reflex includes a skeletal muscle being stretched which leads to stretch of the muscle spindle that stimulates sensory ending Ia which monosynaptically excites alpha motor neurons to same muscle from which they arise and to motor neuron of synergistic muscles. This also inhibits the motor neurons to antagonist muscle inhibitory interneuron. Gamma motor neuron functions to adjust the length of the intrafusal fibers to allow it to be ready for the next stretch (Gilman and Newman 1987) (Fig. 1).

Several supraspinal pathways are felt to be involved in the control of muscle tone. Cortex, basal ganglia, and cerebellum all provide important modulation of brainstem structures in normal motor control. Selective destruction of corticospinal tracts does not result in spastic hypertonia but rather hypotonia. Interruption of extrapyramidal fibers is likely needed before spastic hypertonia develops (Katz and Campagnolo 1994). Possible mechanisms for spasticity include increased neuronal excitation because of loss of inhibitory effects of descending tracts. Also alterations in segmental interneural responsiveness and changes in segmental reflex function are likely play a role in these changes in tone (Sheean and McGuire 2009). The pontine reticulospinal tract and vestibulospinal tract are excitatory tracts, while medullary reticulospinal tract is an inhibitory tract. Pyramidal traits are involved in muscle strength. The synaptic effects of descending pathways may be categorized as mediating changes in the excitability of spinal motoneurons and/or as mediating changes in the responses of segmental reflex pathways (Katz and Campagnolo 1994).

Fig. 1 Stretch reflex

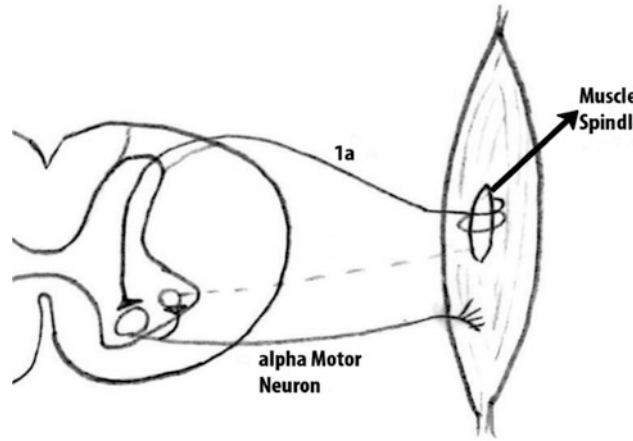
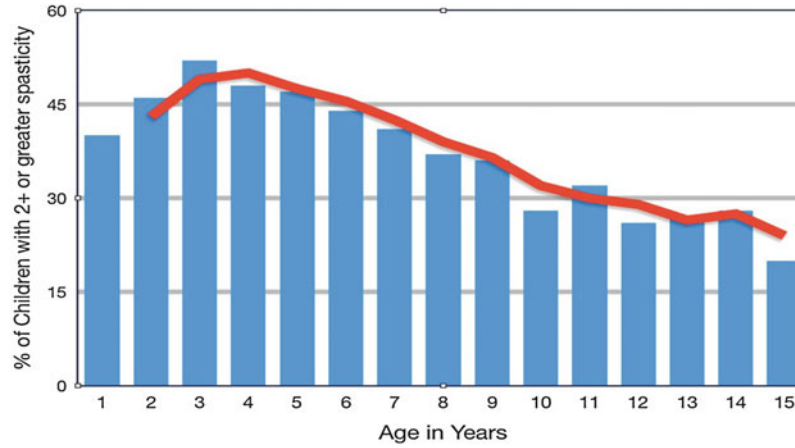


Fig. 2 Percentage of children with CP with spasticity in gastroc-soleus muscle group as grade 2 or more on Modified Ashworth Scale related to age (Hagglund and Wagner 2008)



There are felt to be differences in spinal and cerebral spasticity. Spinal spasticity is thought to be due to removal of inhibition on segmental polysynaptic pathways which lead to a slow, progressive rise of excitatory state. Afferent activity from one segment may lead to muscle response many segments away. Flexor and extensor muscles may be excited. Cerebral spasticity is thought to be due to enhanced excitability of monosynaptic pathways which lead to a rapid buildup of reflex activity. There is a bias toward over activity in antigravity muscles (Katz 1988).

Spasticity is felt to be part of the upper motor neuron syndrome. This is an abnormal motor function secondary to lesions in cerebral cortex, subcortex, or spinal cord. There are positive signs including increased muscle tone, hyperreflexia, primitive reflexes, and clonus. Negative signs include weakness, fatigue, and poor dexterity (Gormley 1999). It is important to be able to recognize all of these signs, not just spasticity, as they all impact function. Spasticity may cause pain, limit sleep, lead to joint deformity, and interfere with function. It may also interfere with care including transfers, toileting, bathing, and dressing (Krach 2001). In children with cerebral palsy, spasticity is typically not a problem during the first 6 months of life. It can be noted between 6 and 24 months. A baby with cerebral palsy may actually be quite floppy during first 6 months (Miller and Bachrach 2017). Spasticity may become more obvious as their nervous system matures, such as with increased myelination. In 2008, data from Hagglund and Wagner's CP group in Sweden described the natural history of spasticity. They reported that in children with CP, muscle tone increases up to 4 years of age and then decreases up to 12 years of age (Fig. 2).

Assessment

Multiple approaches are available for the evaluation and treatment of spasticity in patients with CP. The goals for treatment should be realistic and individualized, and they need to be agreed upon by patient, family/caregiver, and medical team. Ideally, a multidisciplinary team should be involved in the decision-making. Such a team should include patient, family, physical and occupational therapists, nurse, physiatrist, neurologist, orthopedist, and neurosurgeon (Massagli 1991). Clinical assessment requires an understanding of motor patterns such as co-contractions, synergy patterns, and static vs. dynamic tone. One needs to appreciate the chronicity and severity of the spasticity. The distribution and location are important. You must understand what muscles are involved. The approach to treatment is different for generalized versus localized spasticity. It is very important to understand what noxious stimuli can increase spasticity such as joint pain or a urinary tract infection. You need to treat what is causing the noxious stimuli before trying to reduce the tone directly.

Since spasticity is part of the upper motor neuron syndrome, we need to remember that there are often other functional issues that a patient is dealing with at the same time including weakness, fatigue, and poor dexterity. Impaired balance and motor planning are also common issues in this population. It is important to be able to recognize all of these, not just spasticity, as they all impact function. Treating one of these issues does not necessarily improve another. A family needs to know that treating spasticity will not improve motor planning, for example. You, as a provider, need to be able to describe and define these different elements of function to a patient and family. One of the most important issues to understand when a patient comes to you for treatment of spasticity is how much weakness is present in these spastic muscles. Many children with CP who have spasticity and functionally stand and/or take steps are using some of their spasticity to maintain their upright functional position. Taking too much tone away from such a patient may significantly decrease their overall function.

There are scales that have been developed to measure tone. The Ashworth Scale or the Modified Ashworth Scale is often used to quantify spasticity but are not always able to distinguish between spasticity and soft tissue tightness (Damiano et al. 2002). The Tardieu Scale or Modified Tardieu Scale is better at this distinction but is not as good at quantifying in a clinical setting (Sheean and McGuire 2009) (Table 1).

Treating Spasticity

Consider treating spasticity if it is interfering with some level of function, care, or comfort. This may include a need to alleviate pain, improve hygiene, or decrease caregiver burden. It is appropriate to treat spasticity to prevent complications such as pressure ulcers and joint contractures. Working through spasticity to enhance function for a patient with CP can be challenging. Treatments that are available include physical and occupational therapy, orthoses, oral medications, chemodenervation, intrathecal baclofen, orthopedic surgery, and neurosurgery. Many children with spastic cerebral palsy receive multiple interventions especially throughout their growing years. Since stretching, splinting, and positioning are considered the first-line approach to treatment, physical and occupational therapists typically meet patients very early. These other medical and surgical interventions can be used in conjunction with these therapies when further tone reduction is felt to be necessary (McMahon et al. 2015). Noting a patient's medical comorbidities is necessary so as not to complicate other medical issues while treating spasticity. For example, when used in combination with anticonvulsants, muscle relaxants can significantly increase risk of sedation.

Therapy Services

Physical and occupational therapies are essential to patients dealing with spasticity. Many children with cerebral palsy have had the support of therapists since they were in the NICU. These services can fortunately extend into a patient's home at discharge from the nursery in the form of Early

Table 1 Modified Ashworth scale and modified Tardieu scale

Modified Ashworth scale:	
0	No increase in tone
1	Slight increase in tone with catch or minimal resistance at end of ROM
1+	Slight increase in tone with minimal resistance through less than half of ROM
2	Marked increase through most of ROM
3	Considerable increase, PROM difficult
4	Rigid in flexion and extension
Modified Tardieu scale:	
R2 – The angle of full ROM when velocity of stretch is as slow as possible	
R1 – The angle of muscle reaction when velocity of stretch is as fast as possible	
Spasticity angle = R2–R1	
Large spasticity angle indicates a greater degree of spasticity (dynamic component)	
Smaller spasticity angle indicates a greater degree of contracture	

Intervention Services. The first-line treatment includes therapeutic exercises that focus on stretching, strengthening, splinting, and positioning. By 12–24 months the spasticity in CP is usually much more obvious, and therapy intervention may need to become more intensive. Various physical modalities such as heat/cold and functional electrical stimulation (FES) have been noted to be helpful. Therapists use tools, as described above, such as Modified Ashworth Scale and Modified Tardieu Scale to measure tone (Sheean and McGuire 2009). They also use many functional assessment scales such as Gross Motor Function Measure (GMFM) (Miller 1998) and Pediatric Functional Independence Measure (WeeFIM) (Krach et al. 2015) as well as Shriner's Hospital Upper Extremity Evaluation (SHUEE) and Quality of Upper Extremity Skills Test (QUEST) (King 2005). These tools are also helpful in measuring the effectiveness of certain interventions. It can be helpful for families to learn a home exercise program to allow for carryover. As children with spastic cerebral palsy get older and various surgical interventions are performed, the therapeutic intervention increases in the community and sometimes in the school setting.

Bracing and Positioning

In a patient with spasticity, orthoses/splints can help to improve function, optimize joint alignment, control abnormal tone, prevent soft tissue deformity, and protect tissues postoperatively. Ankle-foot orthoses (AFO) can be solid, articulating, static, or dynamic. Improvement in joint position and function are often seen with these. Knee-ankle-foot orthoses (KAFO), although less commonly used in CP, can be very helpful for standing and transfers. Upper extremity splints include wrist/hand resting splint, cock-up splint, and opponens splint. Upper extremity static splints are more common than dynamic splints as the latter often decrease sensory feedback during functional activities and children can be reluctant to use them (Miller 1998). Appropriate seating systems that help to reduce tone are essential including wheelchairs and feeding chairs (Gans and Glenn 1990).

Chemodenervation

Botulinum toxin (BoNT) injections, motor point blocks, and nerve blocks are used to treat localized spasticity. Botulinum toxin type A or B is a neurotoxin released by clostridium botulinum. In the USA, three toxins are available on the market, Botox, Myobloc, and Dysport. Botox and Dysport are BoNT type A and Myobloc is BoNT type B (Sheean and McGuire 2009). BoNT acts presynaptically at the neuromuscular junction to block the release of acetylcholine. These nerve terminals do not regain function. In 3–6 months, new nerve terminals grow secondary to collateral axonal sprouting. Limit treatment to no more frequently then every 3 months, secondary to risk of antibody formation. All muscle identification techniques

begin with anatomical localization, but some providers also use supplemental techniques to maximize the accuracy of needle placement. These techniques include electromyography (EMG) guidance, EMG auditory signal amplifiers (EMG-SA) (Walker et al. 2015), electrical stimulation, and ultrasound guidance (Henzel et al. 2010).

Alcohol motor point blocks and nerve blocks involve a chemical neurolysis that denatures protein in the myelin sheath of a nerve. 3% or 5% phenol are most commonly used. It is generally performed with conscious sedation. There is a risk of painful dysesthesias. You need to use electrical stimulation for guidance to get as close as possible to motor point (Gormley 1999; Elovic et al. 2009).

Oral Medications

Several oral medications have been used to reduce spasticity including diazepam, baclofen, dantrolene sodium, tizanidine, and clonidine. Although they can decrease spasticity, their sedating side effects are not well tolerated in children (Massagli 1991). None of these medications have been universally effective in treating spasticity. Baclofen has been noted to be moderately helpful when taken orally for spasticity of spinal origin in adults. It has been relatively unhelpful in treating spasticity of cerebral origin, especially in children with CP. Intrathecal baclofen has been shown to reduce spasticity with fewer side effects (Watanabe 2009).

Diazepam is the most frequently used benzodiazepine and is one of the oldest antispasticity medications in use today. It has an inhibitory effect at spinal cord level and at the supraspinal level. It acts postsynaptically, and while it doesn't bind directly to GABA receptors, it increases GABA's affinity to bind to GABAa receptors. The greatest clinical efficacy of benzodiazepines has been in patients with spinal cord injury and multiple sclerosis. Sedation is the most disabling side effect especially for someone with spasticity of cerebral origin. These can have a very long half-life due to active metabolites. The use of benzodiazepines may lead to physical dependence (Katz and Campagnolo 1994).

Baclofen is a GABA analog that acts at the presynaptic level in the spinal cord and interferes with the release of excitatory neurotransmitters. It is lipophilic and crosses the blood-brain barrier poorly; as a result large doses may be necessary to see an effect. It has been noted to be most effective in patients who have spasticity of spinal origin including spinal cord injury. There is limited evidence that oral baclofen improves function and even some evidence that some GABAergic medications may impair function. Overdose may lead to respiratory suppression, hypotension, and bradycardia. Abrupt withdrawal could lead to mental status changes or seizures (Krach 2001).

Dantrolene sodium acts directly on muscle, decreasing release of calcium from sarcoplasmic reticulum. It is rarely used to decrease spasticity in patients with cerebral palsy as there is limited evidence in literature that it is effective in this population. The potential for the serious side effect of hepatotoxicity can also limit its use. Liver function tests should be done before starting treatment and periodically while taking the medication (Katz and Campagnolo 1994).

Clonidine is a centrally acting alpha 2-adrenergic agonist that is felt to decrease sympathetic outflow from the brain. At the spinal cord level, its effects are thought to be related to both presynaptic inhibition of sensory afferents and inhibition of the release of glutamate, which is an excitatory amino acid. It is lipophilic and crosses the blood-brain barrier poorly. While an enteral form is available, it is also very effective in the form of a transdermal patch that is changed every 5–7 days. Side effects are hypotension, bradycardia, and sedation (Watanabe 2009).

Tizanidine is a newer alpha 2-agonist that is chemically related to clonidine. It binds to receptors at spinal and supraspinal levels. At the spinal level, it increases presynaptic inhibition of motor by decreasing the release of excitatory amino acids. It is more commonly used in spasticity of spinal origin rather than spasticity of cerebral origin. Improvements in tone have been noted, but there is limited evidence on improvements in function (Watanabe 2009).

One controversial treatment is cannabis or tetrahydrocannabinol (THC) which is the main

Table 2 Medications used to treat spasticity in children

Drug	Mechanism of action	Side effects/precautions
Diazepam	Has an inhibitory effect at spinal cord and at supraspinal level. It acts postsynaptically to increase GABA's affinity to bind to GABAa receptors	CNS depression with sedation, impaired cognition, long half-life
Baclofen	GABA analog that acts at the presynaptic level in the spinal cord and interferes with the release of excitatory neurotransmitters	Sedation, overdose may lead to respiratory suppression, hypotension, and bradycardia. Abrupt withdrawal can cause seizures
Dantrolene sodium	Acts directly on muscle decreasing release of calcium from sarcoplasmic reticulum	Risk of hepatotoxicity Must monitor LFTs
Clonidine	Centrally acting alpha 2-adrenergic agonist that acts both in brain and spinal cord by enhancing presynaptic inhibition and decreasing excitatory output. Transdermal patch form available	Sedation, hypotension, bradycardia
Tizanidine	Alpha 2 agonist that acts both in the brain and spinal cord by enhancing presynaptic inhibition and decreasing excitatory output	Sedation, hypotension

active ingredient in cannabis. Cannabinoids have been shown to have efficacy in treating spasticity of spinal origin and are currently being studied. Some literature supports the hypothesis that the relaxing effect of marijuana on muscles in patients with spasticity of spinal origin is due to an antispastic effect, perhaps inhibition of polysynaptic reflexes, rather than simply due to a general relaxation response. It is not routinely used in children with CP at this time (Adams and Hicks 2005).

As mentioned earlier, understanding a patient's medical comorbidities is necessary so as not to complicate other medical issues while treating spasticity. For example, many muscle relaxants can cause constipation and urinary retention. When used in combination with anticonvulsants, muscle relaxants can significantly increase risk of sedation (Table 2).

Intrathecal Baclofen

Intrathecal baclofen has been found to be very helpful in patients with cerebral palsy who have significant generalized spasticity (Albright et al.1993). It has also been noted to help to decrease dystonia in this population (Albright et al.1996). Careful assessment is needed to determine whether someone is a good candidate for intrathecal baclofen therapy. Baclofen is an analog of GABA, and when delivered intrathecally, it can diffuse to where $GABA_B$ receptors are believed to be located in the brain and spinal cord (Keenan et al. 2000). Intrathecal baclofen pump is implanted for delivering a continuous infusion of baclofen directly into the spinal fluid. This treatment works very well in quadriplegic CP patients and also diplegic CP patients (Armstrong 1992). It involves mcg dosing. The pump reservoir is refilled by percutaneous puncture through a septum in the pump at intervals of 1–6 months depending on the rate of delivery. Topical anesthetic cream can be used to numb the skin over the pump refill site to make the procedure more comfortable for the pediatric patient. Dosage adjustments are made via an external programmer and transmitted to the pump by a handheld radiofrequency wand. The pump can be programmed to deliver the baclofen in several modes including simple continuous infusion, complex continuous infusion (i.e., rate changes at set times during the day), and bolus infusion mode (Medtronic, Inc., Minneapolis, MN 2017).

Neurosurgery

Neurosurgical options for spasticity include selective dorsal rhizotomy (SDR). This involves sectioning a portion of sensory rootlets. It is typically restricted to lumbosacral plexus L1–L2 to S1–S2. Approximately 40% sensory nerves are sectioned.

The idea is to decrease the sensory input which then decreases the motor output. Extensive therapies after this procedure have been felt to be helpful in maximizing outcome. One meta-analysis described the ideal candidates as children between 3 and 8 years of age and GMFM level III or IV. For severe spastic quadriplegia, this procedure has been attempted for comfort and to decrease caregiver burden, with less success (Abbott 1996). Meta-analysis of SDR patients demonstrated that if there is any benefit, it is only in a few points of improvement and not dramatic functional improvements. Use of intrathecal baclofen in the pediatric patient having CP has yielded as good a reduction in tone as dorsal rhizotomy and does not represent an ablative procedure. This is important because, unlike rhizotomies, intrathecal baclofen therapy is entirely reversible (Albright et al. 1995).

Orthopedic Surgery

Spasticity in children with cerebral palsy often leads to muscle imbalance with joint deformities and bony changes. Surgical goals range from improved comfort to maximizing function. Orthopedic surgery for the management of spasticity can include tendon releases, muscle lengthenings, osteotomies, tendon and muscle transfers, arthrodesis, and spinal fusion (McMahon et al. 2015). Depending on goals of surgery, postoperative therapies might be recommended.

Summary Discussion

Managing spasticity can be challenging throughout the life of a child with CP. In a very young child, under the age of 3 years, we focus on physical and occupational therapy techniques. As mobility advances we often add AFO's to help improve functional gait. As tone increases over time, if it interferes with function, comfort, or care, we consider Botox injections. Sometimes these are in a series over a few years. In ambulatory patients, orthopedic surgery may be considered during middle childhood. If a child is noted to have significant generalized spasticity, intrathecal baclofen therapy may be considered. Depending on the needs over time, the interventions may change.

Conclusion

Treat spasticity only if it is a problem. While some spasticity may be necessary for function in children with neurologic impairment, it is often a problem for which patients and families seek treatment. Since spasticity is part of the upper motor neuron syndrome, we need to remember that there are often other issues that a patient is dealing with at the same time including weakness, fatigue, and poor dexterity. Impaired balance and motor planning are also common in CP. It is important to be able to recognize all of these issues not just spasticity, as they often all impact function. Treating one of these issues does not necessarily improve another. Multidisciplinary team approach is always the most helpful. This team includes the patient, family/caregiver, nurses, therapists, and physicians who work closely together to set appropriate goals for treatment.

Cross-References

- Complications of Spine Surgery in Cerebral Palsy
- Infections and Late Complications of Spine Surgery in Cerebral Palsy
- Intrathecal Baclofen Therapy in Cerebral Palsy-Surgical Implantation and Problem Management
- Single-Event Multilevel Surgery for the Upper Extremity in Cerebral Palsy

References

Abbott R (1996) Selective rhizotomy for treatment of childhood spasticity. J Child Neurol 11:S36–S42

Adams MM, Hicks AL (2005) Spasticity after spinal cord injury. Spinal Cord 43:577–586

Albright AL, Barron WB, Fasick MP et al (1993) Continuous intrathecal baclofen infusion for spasticity of cerebral origin. JAMA 270:2475–2477

Albright AL, Barry MJ, Fasick MP et al (1995) Effects of continuous intrathecal baclofen infusion and selective dorsal rhizotomy on upper extremity spasticity. Pediatr Neurosurg 23:82–85

Albright AL, Barry MJ, Fasick MP et al (1996) Continuous intrathecal baclofen for symptomatic generalized dystonia. Neurosurgery 38:934–939

Armstrong RW (1992) Intrathecal baclofen and spasticity: what do we know and what do we need to know. Dev Med Child Neurol 34:739–745

Butler C, Campbell S (2000) Evidence of the effects of intrathecal baclofen for spastic and dystonic cerebral palsy. AACPDM Treatment Outcomes Committee Review Panel. Dev Med Child Neurol 42:634–645

Carr JH, Shepherd R (2001) Neurological rehabilitation: optimizing motor performance. Theoretical consideration in balance assessment. Aust J Physiother 47:89–100

Damiano DL, Abel MF (1998) Functional outcomes of strength training in spastic cerebral palsy. Arch Phys Med Rehabil 79:119–125

Damiano DL, Quinlivan JM, Owen BF et al (2002) What does the Ashworth scale really measure and are instrumented measures more valid and precise. Dev Med Child Psychol 44:112–118

Elovic EP, Esquenazi A, Alter KE et al (2009) Chemodenervation and nerve blocks in the diagnosis and management of spasticity and muscle overactivity. PMR 1:842–851

Gans B, Glenn M (1990) In: Glenn M, Whyte J (eds) The practical management of spasticity in children and adults. Lea & Febiger, Philadelphia, pp 1–7

Gerszten PC, Albright AL, Barry MJ (1997) Effect on ambulation of continuous intrathecal baclofen infusion. Pediatr Neurosurg 27:40–44

Gilman S, Newman SW (1987) Manter and Gatz's essentials of clinical neuroanatomy and neurophysiology. FA Davis Company, Philadelphia, pp 22–28

Gormley ME Jr (1999) Management of spasticity in children. Part 2: oral and intrathecal baclofen. J Head Trauma Rehabil 2:207–209

Hagglund G, Wagner P (2008) Development of spasticity with age in a total population of children with cerebral palsy. BMC Musculoskelet Disord 9:150

Henzel MK, Munin MC, Niyonkuru C et al (2010) Comparison of surface and ultrasound localization to identify forearm flexor muscles for botulinum toxin injections. PMR 2:642–646

Katz R (1988) Management of spasticity. Am J Phys Med Rehabil 67:108–116

Katz RT, Campagnolo DI (1994) Pharmacologic management of spasticity. Phys Med Rehabil State Art Rev 8:473–480

Keenan C, Alexander M, Sung I et al (2000) Intrathecal baclofen for treatment of spasticity in children. Phys Med Rehabil State Art Rev 12:275–283

King MM (2005) Cerebral palsy functional scoring levels, scoring scales. In: Miller F (ed) Cerebral palsy. Springer, New York, pp 834–838

Krach LE (2001) Pharmacotherapy of spasticity: oral medications and intrathecal baclofen. J Child Neurol 16:31–36

Krach LE, Gormley ME, Ward M (2015) Traumatic brain injury. In: Alexander MA, Matthews D (eds) Pediatric rehabilitation principles and practice. Demos Medical, New York, pp 429–453

Kroin JS, Ali A, York M et al (1993) The distribution of medication along the spinal cord after chronic intrathecal administration. Neurosurgery 33:226–230

Lance JW (1980) Symposium synopsis. In: Young RR, Feldman RG, Koella WP (eds) Spasticity: disordered motor control. Symposia Specialists, Miami, pp 485–495

Massagli TL (1991) Spasticity and its management in children. Phys Med Rehabil Clin N Am 2:867–889

Mathur SN, Chu SK, McCormick Z et al (2014) Long-term intrathecal baclofen: outcomes after more than 10 years of treatment. PMR 6:506–513

McCormick ZL, Chu SH, Binler D et al (2016) Intrathecal versus oral baclofen: a matched cohort study of spasticity, pain, sleep, fatigue, and quality of life. PMR 8:553–562

McMahon M, Pruitt D et al (2015) Cerebral palsy. In: Alexander MA, Matthews D (eds) Pediatric rehabilitation principles and practice. Demos Medical, New York, pp 336–364

Medtronic ITB Therapy product monograph (2017) Medtronic Inc, Minneapolis

Medtronic ITB Therapy technical note (2017) The effects of magnetic resonance imaging (MRI) on SyncroMed II drug infusion systems. Medtronic Inc, Minneapolis

Meythaler JM, Guin-Renfroe S, Law C et al (2001) Continuously infused intrathecal baclofen over 12 months for spastic hypertonia in adolescents and adults with cerebral palsy. Arch Phys Med Rehabil 82:155–161

Michaud LJ (2000) Electrical stimulation in children. In: Pediatric rehabilitation state of the art reviews. Physical medical rehabilitation, vol 14, no 2. Hanley and Belfus, Philadelphia, pp 347–362

Miller F (1998) Gait analysis in cerebral palsy. In: Dormans JP, Pellegrino L (eds) Caring for the child with cerebral palsy. Brookes, Baltimore, pp 169–191

Miller F, Bachrach SJ (2017) Cerebral palsy encyclopedia. In: Cerebral palsy- A complete guide for caregiving. Johns Hopkins University Press, Baltimore, p 442

Peacock W, Straudt L (1991) Functional outcomes following selective posterior rhizotomy in children with cerebral palsy. J Neurosurg 74:380–385

Penn R, Savoy SM, Corcos D et al (1989) Intrathecal baclofen for severe spinal spasticity. N Engl J Med 320:1517–1521

Phillips MM, Miljkovic N, Ramos-Lamboy M et al (2015) Clinical experience with continuous intrathecal

baclofen trials prior to pump implantation. PMR 7:1052–1058

Ried S, Pellegrino L, Albinson-Scull S et al (1998) The management of spasticity. In: Dormans JP, Pellegrino L (eds) Caring for the child with cerebral palsy. Brookes, Baltimore, pp 99–123

Rymer WZ, Katz RT (1994) Mechanisms of spastic hypertonia. Phys Med Rehabil State Art Rev 8:441–454

Sheean G, McGuire JR (2009) Spastic hypertonia and movement disorders: pathophysiology, clinical presentation and quantification. PMR 1:827–833

Simpson DM, Patel AT, Alfaro A et al (2017) OnabotulinumtoxinA injection for poststroke upper-limb spasticity: guidance for the injectors from a delphi panel process. PMR 9:136–148

Slominski AH (1984) Winthrop Phelps and the children's rehabilitation institute. Management of motor disorders of children with cerebral palsy. In: Scrutton D (ed) Management of the motor disorders of children with cerebral palsy. Spastics International Medical Publications, London, pp 59–74

Walker HW, Lee MY, Bahroo LB et al (2015) Botulinum toxin injection techniques for the management of adult spasticity. PMR 7:417–427

Watanabe TK (2009) Role of oral medications in spasticity management. PMR 1:839–841