Spastic Paralysis of the Elbow and Forearm

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Abstract

Keywords

- ▶ spasticity
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As the physiologic recovery period concludes, the patient is evaluated for surgical procedures that may rebalance muscle function and correct deformity. Upper extremity function is the product of complex and highly sophisticated mechanisms working in unison, and a careful, systematic preoperative evaluation is critical. A good function of the hand cannot be achieved without adequate position of the shoulder, elbow, forearm, and wrist. The goals of surgery must be practical and clearly understood by the patient and the family.

Spasticity presents as muscular hypertonia and hyperexcitability of the stretch reflex, which are typical of upper motor neuron syndrome.^{1,2} Physiopathologically, spasticity is due to the medullar and supramedullar alteration of the afferent and efferent pathways. Treatment is multidisciplinary and involves the collaboration of rehabilitators, neurophysiologists, neurologists, pediatricians, orthopaedic surgeons, and psychologists, who all contribute with their different therapeutic aspects and characteristics (which can be pharmacological, peripheral neurological blockages, surgical, etc.).^{2,3} The characteristic posture of the upper extremities in spastic cerebral palsy is the inward rotation of the shoulder, flexion of the elbow and pronated forearm, flexion of the wrist with ulnar deviation, and the deformity of the fingers (flexion contracture, swan-neck, and thumbs-in-palm).^{4–6} The primary objectives in these patients will be to improve communication with their surroundings, to perform activities of daily living, and to increase mobility and walking.

Control of Spasticity

Nonsurgical Management Options

The treatment of spasticity is highly dependent on the time since injury and the prognosis for recovery. In the period of neurologic recovery, temporizing interventions are most appropriate because permanent changes may result in the chronic imbalance of forces across joints. The prevention of additional complications, such as disuse muscle atrophy, joint

contractures, heterotopic ossification, and peripheral neuropathies, is critical in optimizing functional outcome.^{2,4,7} Several treatment choices are available, including cast and splint use, oral drug therapy, intrathecal baclofen, chemodenervation, neurolytic blocks, phenol blocks, and botulinum toxin (BoNT) blocks.^{2,6,7}

Cast and Splint Use

In addition to a variety of medical regimens to be discussed, various casting or splinting techniques can provide temporary relief of spasticity. Casting helps maintain muscle fiber length and diminishes muscle tone by decreasing sensory input and providing a structural impediment to spastic contracture. Changed on a weekly basis, serial casts can be used to correct joint contractures and is successful for contractures present < 6 months. Although there is some effect on spasticity, this is generally not a practical treatment modality. Dynamic splinting has a very limited role in treating spastic contractures and may even exacerbate the situation by triggering increased muscle tone. Both dynamic splints and serial casting may be very helpful after surgical release to correct the residual static component. As an adjunct, local anesthetic nerve blocks are helpful prior to cast application as they relieve spasticity and improve limb positioning.^{6–9}

Oral Drug Therapy

Several oral antispastic agents may be used during this period; however, it is critical to understand the strengths

and weaknesses of each prior to initiating therapy. Antispastic agents that have sedating properties such as baclofen (Lioresal, Novartis Pharmaceuticals), diazepam (Valium, Hoffmann-La Roche), and clonidine may impair patients with attention deficits or memory disorders. In addition to its general central nervous system depressant actions, baclofen may inhibit both polysynaptic and monosynaptic reflexes at the spinal cord level. Dantrolene (Dantrium, Pfizer), the drug of choice for treating clonus, is another drug that can be used to control spasticity. Dantrolene produces relaxation by directly affecting the contractile response of skeletal muscle at the myoneural junction. It interferes with the release of calcium from the sarcoplasmic reticulum, thus causing dissociation of the excitation-contraction coupling mechanism. Although it does not affect the central nervous system directly, it may cause drowsiness, dizziness, and generalized weakness. The most substantial issue encountered with the use of dantrolene is hepatotoxicity. The risk appears greatest in those older than 35 years, females, and those taking other medications. The lowest effective dose of dantrolene should be used; serial liver enzyme functions should be monitored closely. If no beneficial effect is noted after 45 days, the drug should be discontinued.^{7,9,10}

Intrathecal Baclofen

Continuous infusion of intrathecal baclofen may be useful in managing spasticity secondary to spinal cord injury with diminished cognitive function.¹¹⁻¹³ Baclofen pump use also has an advantage over oral drug therapy because it automatically applies small concentrations of medication directly to the intrathecal space. The smaller doses effectively control spasticity, while minimizing side effects. The pump is placed in a subfascial pocket in the abdominal wall, and a catheter is routed subcutaneously into the intrathecal space. The pump is refilled by injection into the reservoir chamber. The dosage and rate of administration can be easily adjusted by using a laptop computer that signals commands to the pump.^{7,9,10,14}

Chemodenervation and Neurolytic Blocks

Focal injection with neurolytic or chemodenervating agents is the most suitable approach for treating focally restricted, spasticity-related motion. Chemodenervation agents such as botulinum toxin A (BoNT-A) as well as neurolytic agents such as phenol or alcohol are used during the period of neurologic recovery; their effects typically last 3 to 5 months. When the effects of these agents subside, a clinical evaluation of motor function should assess whether recovery has taken place and whether further injections are necessary. For these agents to be optimally effective, it is critical that the patient's functional problems be accurately ascribed to specific spastic muscles. If necessary, this can be assessed using multichannel dynamic electromyography (EMG). If multiple muscles are found to contribute, multiple targeted injections can be performed.⁷

Phenol Blocks

When used in aqueous concentrations of 5% or more, phenol, a derivative of benzene, denatures the protein membrane of peripheral nerves. When injected into or near a nerve, phenol

reduces neural traffic along the nerve, making it useful as a temporary treatment of spasticity. The destructive process may begin to show effects several days after injection, but phenol also has a local anesthetic quality that allows a clinician and the patient to see "partial results" shortly after the phenol block is performed. The denaturing process induced by phenol continues for several weeks with regeneration typically occurring within 3 to 5 months. Known to induce an incomplete nerve block, phenol blocks allow relatively normal volitional control of muscles, while more specifically combating spastic movement.¹⁵ During the injection, a surface stimulator is briefly used to approximate the percutaneous stimulation site in advance. A 25-gauge Teflon-coated hypodermic is advanced toward the motor nerve and electrical stimulation is adjusted by noting whether contraction of the desired muscle takes place. As one gets closer to the motor nerve, less intense current is required to produce a contractile response. The motor nerve is injected when minimal current produces a visible or palpable contraction of the muscle. Generally, 4 to 7 mL of 5% to 7% aqueous phenol is injected at each site. Injection into a blood vessel must be avoided by first aspirating the syringe to ensure there is no blood return.^{7,9}

Botulinum Toxin Blocks

The injection of BoNT-A has become another popular approach to functional problems associated with spasticity. Ordinarily, an action potential propagated down a motor nerve triggers the presynaptic release of acetylcholine. The released acetylcholine traverses the synapse and activates postsynaptic receptors and ultimately causes depolarization of the muscle membrane. Botulinum toxin type A, produced by *Clostridium botulinum*, inhibits release of acetylcholine at the neuromuscular junction.¹⁶ Botulinum toxin has long been used to treat a variety of dystonias and is currently approved by the U.S. Food and Drug Administration for the treatment of strabismus, blepharospasm, and facial spasm. In addition, multiple recent studies describe its successful use in the treatment of spasticity in individuals with cerebral palsy, stroke, head trauma, and multiple sclerosis.¹⁷⁻²³ Following direct injection of BoNT into an affected muscle, clinical benefit typically lasts 3 to 5 months. Depending on the size of the muscle, dosing ranges between 10 and 200 U. Most physicians wait at least 12 weeks before reinjection and do not administer more than 400 U in a single treatment session. A different strategy is needed for a limb that requires several injections because this upper limit of 400 U can be reached rather quickly when injecting large proximal muscles or several smaller distal muscles. In this case, BoNT-A and phenol may be combined. While BoNT-A is injected into smaller distal muscles, phenol can be injected into larger proximal muscles. Injection technique varies.²⁴ Electromyography can also be used to localize the injection. When a Teflon-coated hypodermic needle electrode is inserted into the affected limb, the uncoated distal tip of the needle acts as the electrode to localize the specific targeted muscle using electrical stimulation. When the desired muscle contracts, the needle advance is stopped, and the BoNT or phenol is

injected. Alternatively, patients may be asked to try to contract the targeted muscle, or the muscle may contract involuntarily. For deep or small spastic muscles (e.g., long toe flexors, tibialis posterior, finger flexors), electrical stimulation is the preferred means of localizing the muscle prior to injection. In contrast to the onset of muscle relaxation with phenol, which occurs gradually over 24 hours, the effectiveness of BoNT takes place over a 2-week period. Physical therapy utilizing both active and passive techniques is initiated to increase shoulder range of motion (ROM). Blocks can be repeated as needed during the period of neurologic recovery.^{7,9,16,25,26}

Management of Elbow Flexor Spasticity

During the physiologic recovery phase following brain injury, the control of spastic elbow flexion requires the decrease of excessive tone in each of the flexor muscles. Dynamic EMG has shown that the brachioradialis muscle is the most spastic of these flexors.²⁷ Muscle tone may be decreased by chemodenervation of the brachioradialis muscle employing BoNT or phenol blocks. Spasticity of the biceps and brachialis muscles may also interfere with elbow extension, and injection of these muscles with BoNT is helpful. Physical therapy utilizing active and passive ROM of the elbow is begun immediately following the block; every attempt is made to incorporate functional activities of the upper extremity into the therapy program.^{26,28}

Evaluation of Residual Limb Deformities

As the physiologic recovery period concludes, the patient is evaluated for surgical procedures that may rebalance muscle function and correct deformity. Upper extremity function is the product of complex and highly sophisticated mechanisms working in unison; a careful, systematic preoperative evaluation is critical. Furthermore, the goals of surgery must be practical and clearly understood by the surgical team, associated therapists, the patient, and the patient's family.

Assessment of Cognition and Communication

During a thorough physical examination, assessment must include an evaluation of cognition and communication skills.^{29,30} The patient should be capable of following simple commands; this is critical to any potential postoperative therapy program. In addition, the patient should have sufficient cognition to incorporate any change in motor function into functional use. Adequate memory is also required to retain what is taught and learned during postoperative therapy.²⁹

Surgical Management Options

It is often difficult to organize the clinical signs and symptoms because many different aspects of the motor control system may be affected by head injury. The key is to focus on the impact of the movement disorder on the patient's function. Surgical treatment of spasticity is most effective when functional problems are approached from an isolated standpoint. Surgical lengthening/release as well as tendon transfers can

provide effective solutions to focal functional problems.¹⁰ In a neurologically impaired patient, it may be difficult to distinguish among the many potential causes of limited joint motion. One must keep in mind the multitude of possibilities such as increased muscle tone, myostatic contracture, joint deformity, periarticular heterotopic ossification, undetected fracture or dislocation, joint subluxation, pain, or diminished cognition and cooperation on the part of the patient. Although surgical options in the treatment of elbow and forearm spastic paralysis can be highly effective, it is critical that the clinician successfully identifies the focal functional problem and develops an approach encompassing all factors impacting that particular dysfunction.^{9,10,31,32}

Spastic Elbow Pathology

Spastic Flexion Contracture of the Elbow

Hypertonia in the "antigravity" elbow flexors leads many patients to complain of an extension deficit, or at least, that the elbows persistently "ride up" when they stand or walk. They may also complain that the flexed elbow hooks other people and/or door frames as well as complicates putting on a shirt or jacket. In addition, an elbow flexion contracture prevents adequate positioning of the hand in space, hence limiting its function. A temporizing improvement with BoNT or phenol motor block may be obtained during the physiologic recovery phase of injury. When neurologic recovery has plateaued, surgical correction of an elbow flexion deformity may be performed. Although elbow flexion motion is relatively normal, the usual clinical picture is one of cogwheel motion on attempted elbow extension. Dynamic EMG helps confirm the presence of volitional capacity as well as abnormal synergy of each of the elbow flexors.²⁷ Dynamic recordings are normally obtained from the biceps, brachialis, brachioradialis, the flexor-pronator mass, and the three heads of the triceps. The brachioradialis muscle most commonly shows continuous spastic activity as well as one or both heads of the biceps muscle. Directed by this information, the surgeon can devise a rational plan to improve elbow control.^{26,27,33,34}

Techniques of Selective Elbow Flexor Lengthening

Two different techniques are most commonly used to lengthen the elbow flexors in patients with active function; the technique chosen is dependent on the amount of static contracture present.³⁵ When the deformity is primarily dynamic, the biceps should be lengthened at its proximal musculotendinous junction. This allows immediate postoperative mobilization of the elbow. If the static component of the deformity is substantial, a Z-plasty of the distal biceps tendon is advisable. When a Z-lengthening is performed, the elbow should be immobilized for 4 weeks or more to avoid reconstructive failure.³⁶⁻³⁸

Elbow with Dynamic Deformity

To lengthen the biceps proximally, a 4-cm longitudinal incision is made over the deltopectoral interval starting at the lower edge of the pectoralis major tendon. The musculotendinous junctions of

both the long and short heads of the biceps are exposed. The tendinous fibers are sharply transected directly overlying the muscle belly, which allows the muscle-tendon unit to lengthen. Next, to lengthen the brachialis distally, the anterolateral approach to the elbow is used. At ~5 cm proximal to the elbow flexion crease, an incision is made over the lateral border of the biceps with a slight curve into the cubital crease. The radial nerve is identified and protected in between the brachialis and brachioradialis, and the brachialis muscle is exposed and lengthened over its broad muscle-tendon junction. The distal myotendinous junction of the brachioradialis is identified. The muscle is rolled, exposing its deep surface. The radial nerve is carefully protected, and the tendinous fibers are transected sharply, allowing the brachioradialis to lengthen. No permanent immobilization is usually necessary when the muscles have been lengthened with this method. Active ROM exercises are often started on the first day after surgery, associated with nighttime splinting. No resistive exercises are allowed for 3 weeks to prevent overlengthening of the muscles and resultant weakness. Functional elbow flexor lengthening significantly enhances the fluid control of elbow motion and improves hand placement in properly selected patients.^{38,39}

Elbow with Static Deformity

Under tourniquet control, a curvilinear incision is made on the volar aspect of the elbow, beginning laterally over the origin of the brachioradialis muscle. The incision passes lateral to the antecubital crease and gently curves anteriorly to the anterolateral aspect of the forearm. Dissection develops the interval between the brachioradialis and brachialis musculature, which allows identification and protection of the radial nerve. If the brachioradialis has been demonstrated by dynamic EMG to be spastic and without any functional capacity, it is transected through its muscle belly, or elevated off its humeral insertion. If the brachioradialis has volitional control, it can be lengthened at its distal myotendinous junction through a separate incision on the dorsal radial aspect of the mid-forearm. Proximally, a release of the fascia surrounding the muscle can also be very helpful. The lacertus fibrosus is divided and the entire length of the biceps tendon exposed. A Z cut is made for the entire length of the biceps tendon. The biceps is retracted, and the underlying brachialis muscle is exposed. The broad band of tendinous fibers on the anterior surface of the brachialis muscle is sharply transected, leaving the underlying muscle tissue intact. The elbow is then extended, fractionally lengthening the brachialis by ~1 cm. The ends of the biceps tendon are sutured in a lengthened position using a nonabsorbable suture. If preoperative nerve conduction studies demonstrated an ulnar neuropathy at the level of the elbow, anterior transposition of the ulnar nerve is performed. Postoperatively, the patient is placed in a posterior splint. The splint is maintained for 4 weeks; then a program of active therapy is begun. Night splints are used for an additional 3 weeks to protect the biceps tendon repair.^{38,39}

Elbow without Volitional Control

In patients with no volitional control of the elbow, persistent spasticity of the elbow flexors may induce a myostatic

contracture and flexion deformity. This can result in skin maceration and a breakdown of the antecubital space. This position of severe elbow flexion also predisposes the ulnar nerve to compression neuropathy. In this case, release of the brachioradialis from its origin may be performed as previously described. If the deformity is not severe, the brachialis muscle is lengthened at its myotendinous junction by transecting the tendinous fibers on the anterior surface of the muscle, leaving the underlying muscle intact. The remainder is left to counterbalance the triceps muscle. If the elbow flexion contracture is severe and chronic, the brachialis muscle should be completely transected. An anterior capsulectomy should be avoided because of postoperative stiffness and intra-articular adhesions. In very severe contractures, it may be necessary to perform multiple Z-plasties of the antecubital skin or even sometimes flap coverage. If preoperative nerve conduction studies demonstrated an ulnar neuropathy at the level of the elbow, anterior transposition of the ulnar nerve should be performed. Postoperatively, the elbow is immobilized in extension at the point where there is no tension on the neurovascular structures or skin. Physical therapy, serial casting, or dynamic splinting can be used later to improve extension range. Approximately 50% correction of the deformity can usually be expected at surgery, without causing excessive tension on the contracted neurovascular structures. Gradual extension of the elbow with serial casting, dynamic splinting, or physical therapy may further help correct the residual deformity.^{39,40}

Spastic Extension

Spastic elbow extension is much less common than spastic flexion; these patients often demonstrate a brainstem infarct or injury. As a result, they complain of difficulty reaching their face to perform activities of daily living. Although experience is limited, good results have been reported with a fractional lengthening of the triceps tendon, which allows improved flexion ROM at the cost of decreased extension power and an extensor lag. This procedure should be used with caution in patients who rely on their arms to assist with ambulation.

Spastic Forearm Pathology

Spasticity

Supination and pronation spastic deformities are commonly associated with elbow spasticity, wrist spasticity, or both. In this setting, pronation deformities of the forearm are far more common than supination deformities. Many activities of daily living depend on active supination, such as handling utensils, grooming, and dressing. Physical examination reveals a fully pronated resting position of the forearm. When passive supination ROM exceeds the range of active supination, pronator muscle dyssynergy during active supination should be suspected. Both the pronator teres and pronator quadratus may contribute, showing varying degrees of volition and spasticity. Dynamic EMG of the pronator teres, pronator quadratus, and biceps can further delineate the exact contributions from adjacent muscle.²⁷ During the period of functional recovery, phenol or BoNT can be injected into

either or both pronators, depending on clinical and laboratory analyses. In the case of residual deficits, surgical lengthening of the pronator teres and pronator quadratus may be performed, depending on their individual voluntary capacities. The clinical goal is to improve active supination function by reducing pronator dyssynergy. Fractional lengthening of the pronator teres is an accepted treatment. When an excessive amount of the pronator teres origin is released or when there is no function in the pronator quadratus muscle, an iatrogenic and less functional supination deformity may occur. When the pronators are contracted and not volitionally active, individual muscle releases may be considered.³⁹

Spastic Pronation

Approaching the pronator teres may be most prudent in the interval between the mobile wad and the flexor carpi radialis in the mid-forearm. Care must be taken to preserve the superficial radial nerve and radial artery within this interval. The pronator teres is identified as it inserts on the radius. Myotendinous lengthening is performed by cutting the tendinous fibers of the musculotendinous junction and allowing the tendon fibers to slide on the muscle belly, thereby lengthening the musculotendinous unit. When dynamic EMG has demonstrated that the pronator is spastic, but does not have any volitional activity, the pronator can be entirely released from its insertion on the radius.

The pronator quadratus is approached via an incision over the volar aspect of the forearm just proximal to the wrist crease and ulnar to the palmaris longus (PL). The finger flexor tendons are retracted radially to expose the broad myotendinous junction of the pronator quadratus. The tendon fibers are transected, leaving the underlying muscle fibers intact. The arm is then supinated, separating the ends of the tendon fibers and lengthening the pronator quadratus. When the pronator quadratus is contracted and does not have any functional capacity, it may be completely transected. Active forearm supination is reliably improved by pronator lengthening. A program of physical therapy to improve passive forearm rotation and strengthen the supinator muscles can further enhance the outcome. When active supination needs to be restored, a rerouting of the pronator teres can be performed. Using the same approach previously described, the tendon of the pronator teres is first Z-lengthened. The distal end of the tendon, usually short, is often extended with periosteum. It is then rerouted around the radial shaft, through the interosseous membrane, from radial to ulnar, then sutured to the proximal end of the tendon. This procedure must be carefully indicated, particularly when other supination procedures are performed (pronator quadrator release, transfer of the flexor carpi ulnaris to the extensor carpi radialis brevis) to avoid a less-functional supination contracture. Other supination producing procedures, such as rerouting of the brachioradialis, are rarely used in this indication due to the usual spasticity of this muscle.^{39,41}

Spastic Supination

Spastic supination is associated with elbow flexion deformities, but is relatively uncommon. A supination deformity may be caused by the biceps, the supinator, or both. Physical

examination supplemented by dynamic EMG will assist in determination of the relative contribution of each.²⁷ Most commonly, a supination deformity can be treated by rerouting the direction of biceps pull.⁴² On rare occasions, supinator release is required.

Technique of Biceps Rerouting

Spastic supination may be corrected by rerouting the biceps tendon distally around the radius to reposition the forearm in neutral rotation. In case of fixed contracture, a concomitant release of the interosseous membrane may be added. In case of severe contracture, pronation-producing osteotomies of the proximal ulna and distal radius can be necessary. Following completion of a curvilinear incision on the volar aspect of the elbow, the lacertus fibrosus is released, and the full length of the biceps tendon—as for a Z-plasty—taking care to maintain as much length as possible. A Krakow stitch is then placed in both ends of the tendon. Under direct visualization, the distal end of the tendon is then passed around the neck of the radius, from radial to ulnar, to rotate the forearm to a neutral rotation position. During this portion of the procedure, it is critical to avoid injury to the posterior interosseous nerve. The tendon is then repaired to itself using nonabsorbable sutures. The repair must be protected with a long-arm cast for 4 to 6 weeks, with the forearm resting in neutral rotation.^{38,42}

Conclusion

The surgical treatment applied by orthopaedic surgeons in the upper extremities is aimed at achieving an enhanced adaptive functionality rather than morphological normality. Factors to be taken into account include age, voluntary control over muscles and joints, level of severity of the spasticity (Ashworth Scale), and stereognostic sensitivity. Correction of shoulder, elbow, and forearm spasticity and deformities helps with a better positioning of the hand in space. They have to be part of a comprehensive plan involving the entire extremity.

References

- 1 Pandyan AD, Gregoric M, Barnes MP, et al. Spasticity: clinical perceptions, neurological realities and meaningful measurement. *Disabil Rehabil* 2005;27(1–2):2–6
- 2 Nair KP, Marsden J. The management of spasticity in adults. *BMJ* 2014;349:g4737
- 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 Narayanan UG. Management of children with ambulatory cerebral palsy: an evidence-based review. *J Pediatr Orthop* 2012;32 (Suppl 2):S172–S181
- 5 Carlson EJ, Carlson MG. Treatment of swan neck deformity in cerebral palsy. *J Hand Surg Am* 2014;39(4):768–772
- 6 Kent RM. Cerebral palsy. *Handb Clin Neurol* 2013;110:443–459
- 7 Vadivelu S, Stratton A, Pierce W. Pediatric tone management. *Phys Med Rehabil Clin N Am* 2015;26(1):69–78
- 8 Parziale JR, Akelman E, Herz DA. Spasticity: pathophysiology and management. *Orthopedics* 1993;16(7):801–811

- 9 Tilton A. Management of spasticity in children with cerebral palsy. *Semin Pediatr Neurol* 2009;16(2):82–89
- 10 Deon LL, Gaebler-Spira D. Assessment and treatment of movement disorders in children with cerebral palsy. *Orthop Clin North Am* 2010;41(4):507–517
- 11 Loubser PG, Narayan RK, Sandin KJ, Donovan WH, Russell KD. Continuous infusion of intrathecal baclofen: long-term effects on spasticity in spinal cord injury. *Paraplegia* 1991;29(1):48–64
- 12 Korenkov AI, Niendorf WR, Darwish N, Glaeser E, Gaab MR. Continuous intrathecal infusion of baclofen in patients with spasticity caused by spinal cord injuries. *Neurosurg Rev* 2002;25(4):228–230
- 13 Ordia JI, Fischer E, Adamski E, Chagnon KG, Spatz EL. Continuous intrathecal baclofen infusion by a programmable pump in 131 consecutive patients with severe spasticity of spinal origin. *Neuro-modulation* 2002;5(1):16–24
- 14 Butler C, Campbell S; AACPDm Treatment Outcomes Committee Review Panel. Evidence of the effects of intrathecal baclofen for spastic and dystonic cerebral palsy. *Dev Med Child Neurol* 2000;42(9):634–645
- 15 Dimitrijevic MR, Sherwood AM. Spasticity: medical and surgical treatment. *Neurology* 1980;30(7 Pt 2):19–27
- 16 Esquenazi A, Albanese A, Chancellor MB, et al. Evidence-based review and assessment of botulinum neurotoxin for the treatment of adult spasticity in the upper motor neuron syndrome. *Toxicon* 2013;67:115–128
- 17 Slawek J, Bogucki A, Reclawowicz D. Botulinum toxin type A for upper limb spasticity following stroke: an open-label study with individualised, flexible injection regimens. *Neurol Sci* 2005;26(1):32–39
- 18 Borg-Stein J, Pine ZM, Miller JR, Brin MF. Botulinum toxin for the treatment of spasticity in multiple sclerosis. *New observations. Am J Phys Med Rehabil* 1993;72(6):364–368
- 19 Cameron MH, Bethoux F, Davis N, Frederick M. Botulinum toxin for symptomatic therapy in multiple sclerosis. *Curr Neurol Neurosci Rep* 2014;14(8):463
- 20 Cardoso E, Rodrigues B, Lucena R, Oliveira IR, Pedreira G, Melo A. Botulinum toxin type A for the treatment of the upper limb spasticity after stroke: a meta-analysis. *Arq Neuropsiquiatr* 2005;63(1):30–33
- 21 Kabus C, Hecht M, Japp G, et al. Botulinum toxin in patients with multiple sclerosis. *J Neurol* 2006;253(Suppl 1):I26–I28
- 22 Wasiak J, Hoare B, Wallen M. Botulinum toxin A as an adjunct to treatment in the management of the upper limb in children with spastic cerebral palsy. *Cochrane Database Syst Rev* 2004;(4):CD003469
- 23 Yablon SA, Agana BT, Ivanhoe CB, Boake C. Botulinum toxin in severe upper extremity spasticity among patients with traumatic brain injury: an open-labeled trial. *Neurology* 1996;47(4):939–944
- 24 Kheder A, Nair KP. Spasticity: pathophysiology, evaluation and management. *Pract Neurol* 2012;12(5):289–298
- 25 Nalysnyk L, Papapetropoulos S, Rotella P, Simeone JC, Alter KE, Esquenazi A. OnabotulinumtoxinA muscle injection patterns in adult spasticity: a systematic literature review. *BMC Neurol* 2013;13:118
- 26 Sakzewski L, Ziviani J, Boyd R. Systematic review and meta-analysis of therapeutic management of upper-limb dysfunction in children with congenital hemiplegia. *Pediatrics* 2009;123(6):e1111–e1122
- 27 Keenan MA, Haider TT, Stone LR. Dynamic electromyography to assess elbow spasticity. *J Hand Surg Am* 1990;15(4):607–614
- 28 Gracies JM, Nance P, Elovic E, McGuire J, Simpson DM. Traditional pharmacological treatments for spasticity. Part II: General and regional treatments. *Muscle Nerve Suppl* 1997;6:S92–S120
- 29 Pennington L, Goldbart J, Marshall J. Speech and language therapy to improve the communication skills of children with cerebral palsy. *Cochrane Database Syst Rev* 2004;(2):CD003466
- 30 Aisen ML, Kerkovich D, Mast J, et al. Cerebral palsy: clinical care and neurological rehabilitation. *Lancet Neurol* 2011;10(9):844–852
- 31 Thomason P, Selber P, Graham HK. Single event multilevel surgery in children with bilateral spastic cerebral palsy: a 5 year prospective cohort study. *Gait Posture* 2013;37(1):23–28
- 32 Gough M, Eve LC, Robinson RO, Shortland AP. Short-term outcome of multilevel surgical intervention in spastic diplegic cerebral palsy compared with the natural history. *Dev Med Child Neurol* 2004;46(2):91–97
- 33 Caldwell C, Braun RM. Spasticity in the upper extremity. *Clin Orthop Relat Res* 1974;(104):80–91
- 34 Haefeli M, Calcagni M. [Hemiplegia and cerebral palsy - multidisciplinary treatment of the spastic upper extremity]. *Praxis (Bern 1994)* 2014;103(19):1133–1139
- 35 Mital MA. Lengthening of the elbow flexors in cerebral palsy. *J Bone Joint Surg Am* 1979;61(4):515–522
- 36 Manske PR, Langewisch KR, Strecker WB, Albrecht MM. Anterior elbow release of spastic elbow flexion deformity in children with cerebral palsy. *J Pediatr Orthop* 2001;21(6):772–777
- 37 Gong HS, Cho HE, Chung CY, Park MS, Lee HJ, Baek GH. Early results of anterior elbow release with and without biceps lengthening in patients with cerebral palsy. *J Hand Surg Am* 2014;39(5):902–909
- 38 Nath RK, Somasundaram C. Significant improvement in nerve conduction, arm length, and upper extremity function after intraoperative electrical stimulation, neurolysis, and biceps tendon lengthening in obstetric brachial plexus patients. *J Orthop Surg* 2015;10:51
- 39 Keenan MA. Management of the spastic upper extremity in the neurologically impaired adult. *Clin Orthop Relat Res* 1988;(233):116–125
- 40 Morrey BF. Surgical treatment of extraarticular elbow contracture. *Clin Orthop Relat Res* 2000;(370):57–64
- 41 Gschwind CR. Surgical management of forearm pronation. *Hand Clin* 2003;19(4):649–655
- 42 Metsaers WP, Nagels J, Pijls BG, Langenhoff JM, Nelissen RG. Treatment of supination deformity for obstetric brachial plexus injury: a systematic review and meta-analysis. *J Hand Surg Am* 2014;39(10):1948–1958.e2