Management of Spasticity in Children With Cerebral Palsy

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Spasticity and other forms of muscle overactivity caused by cerebral palsy may impair function or ease of care or may cause discomfort or poor body image. The treatment program for a child with spasticity may include allied health therapy, exercise, casting, constraint-induced therapy, oral medications, chemodenervation, intrathecal baclofen, selective dorsal rhizotomy, and orthopedic surgery. Techniques may be combined for greater efficacy and better tailoring to the needs of the child. This article provides an overview of each approach, with a review of significant research findings in support of each.

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Properly defined, spasticity is "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome." Along with the other positive motor phenomena of the upper motor neuron syndrome—including phasic stretch reflexes (clonus and hyperreflexia), flexor and extensor spasms, cocontraction, dystonia, and associated phenomena—spasticity can have a significant functional impact on a child with cerebral palsy (CP). These various forms of muscle overactivity (for which spasticity is often a convenient, although overly simplified, "shorthand" term) represent important therapeutic targets in the attempt to provide functional gains for the child. Nonetheless, it is important to realize that the negative symptoms—weakness and lack of dexterity—may be far more functionally disabling and are far less amenable to treatment. Even so, tone reduction in the properly selected patient can lead to important gains, and acceptance of the limits is not a reason to avoid trying therapy.

The range of treatments for excess tone is large, from simple stretching exercises to oral and injectable therapies to surgeries. In this article, we review treatment planning for spasticity in CP and provide an overview of treatment options.

Evaluation and Treatment Planning

The treatment program begins with a careful and thorough evaluation to determine whether muscle overactivity is interfering with some aspect of function, comfort, cosmesis, or care. If it is not, no treatment is necessary, and it should not be undertaken. Additionally, whether the patient’s spasticity is aiding function—for instance, whether lower extremity stiffness actually improves transfer ability in the face of underlying leg muscle weakness—should be determined. Reduction of such “useful” spasticity may possibly be counterproductive; on the other hand, when combined with muscle strengthening and appropriate orthotics, reduction of spasticity may lead to overall functional benefit and therefore can be contemplated. Thorough evaluation requires the input of the entire spasticity management team, including the patient and caregivers, physicians, physical and occupational therapists, nurses, orthopedists, and orthotists, as well as surgeons and other professionals in some cases. Psychologists, social workers, and educators may round out the team.

Spasticity in CP can be classified by affected body region:

- Spastic diplegia (both legs involved, more than arms)
- Hemiplegia (involves an arm and a leg on the same side of the body)
- Double hemiplegia (both arms involved, more than legs)
- Tetraplegia and/or quadriplegia (all 4 limbs involved, usually severely)

Within this broad scheme, it is important to document the particular pattern of spastic involvement (eg, scissoring thighs, adducted shoulder) and the degree of severity.

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Tone is typically measured with the Ashworth Scale, a subjective but nonetheless useful and easily administered clinical scale that rates tone from 1 (no hypertonia) to 4 (rigid in flexion or extension).

Evaluation includes not only measurement of tone with the Ashworth Scale, but also one or more measures of motor performance and functional ability, such as the Gross Motor Function Measure (GMFM), the Functional Independence Measure, or the Barthel Index. Such measures should be chosen not only for their relevance to the clinical picture and treatment plan but also for their ease of use, keeping in mind that a measure is only as good as the consistency with which it is used during follow-up.

In the broadest terms, the goals of any spasticity treatment plan are to maximize active function, ease care, and prevent secondary problems such as pain, subluxation, and contracture. Compromises are often required to navigate among multiple potentially conflicting goals. Because the patient may have goals and dreams and because the caregiver plays the central role in the day-to-day management of the child, each should be a central figure in setting treatment goals.

When setting treatment goals, it is often vital to temper the caregiver’s overly ambitious hopes and to focus on realistic, attainable goals. This may become even more important after a successful round of treatment, when excitement over improved function leads to unrealistic expectations for further improvement. At each stage of goal setting, treatment planning, and plan modification, it is vital to obtain full information from the caregiver regarding the effects of previous treatments and the details of the child’s current situation. To develop the best treatment plan, these details must go beyond a strictly clinical picture to include how the child is functioning in the home and school, interests of the child (eg, sports or other activities) that may be affected by treatment, and other factors important to the child’s life. Equally important, the needs of the caregiver must be considered in formulating treatment. For instance, the tone reduction that will improve ease of hygiene is likely to be greater than that which will improve transfers.

Multiple other factors influence treatment plan and timing. The age of the child, presence of comorbidities such as seizures or cognitive impairment, the ability of the family to carry out home treatments or return for regular follow-ups, financial concerns, and other issues are all considered in determining the best treatment.

Although every case is unique, several broad parameters can help shape decision making. Orthopedic surgery should be delayed until the gait is mature. Meanwhile, stretching, physical therapy (PT), and orthotics are used to maintain range of motion. Oral medications, botulinum toxin injections, intrathecal baclofen, and/or rhizotomy may also be appropriate. When gait is mature (between ages 6 and 10), gait analysis and clinical examination can be used to determine whether surgery is necessary. To avoid multiple surgeries and periods of immobility, one should try to perform all surgery at one time and remobilize the child as soon as possible. Continued stretching and medical treatment are used as needed to maintain range of motion and mobility.

**PT and Orthoses**

The importance of regular stretching in maintaining full range of motion and preventing contracture cannot be overstated. Luckily, children want to move, and much of the need for range-of-motion exercises can be met by the daily activities of a reasonably active child. However, when impairments are dealt with by compensatory strategies that minimize movement of the affected joint, the potential for contracture increases. Thus, regular stretching of all affected limbs is generally prescribed. Stretching can reduce severity of tone for several hours, providing a short-term, but not long-term, antispasticity action.

PT for the child with CP may encompass a regular exercise program,4 horseback riding,5 and a variety of modalities, including biofeedback6 and electrical stimulation.7-10

Spastic muscles are often weak. The efficacy of lower limb strength training has been examined in several controlled clinical trials.11-14 The results of these trials suggest that when added to a well-designed PT program, strength training can improve gait parameters without worsening spasticity.

The intensity and constancy of PT needed to maximize gains have been the subject of several studies. Bower et al compared the effects of the usual amount of PT to intensive therapy (1 hour per day, 5 days per week) over 6 months in children between ages 3 and 12.15 They found no significant difference between the groups in either function (as measured by the GMFM or performance (measured by the Gross Motor Performance Measure) at the end of the 6-month treatment, though inclusion of age and severity covariates suggested a trend toward significance. Six months after the end of the trial, there was no significant difference between groups, even with inclusion of these covariates. The authors concluded that such intensive therapy was not superior to the normal therapy that children were already receiving, at least in its long-term effects. A similar result was shown by Weindling et al, who compared 6 months of standard PT, standard PT plus extra PT delivered by a trained assistant for 1 hour per week, and standard PT plus a home visit from a family support worker. They found no evidence that additional PT affected motor function, developmental status, or adaptive function. Moreover, there was no benefit seen from intervention from the family support worker on parental stress or family needs. The authors concluded that extra intervention is not necessarily beneficial, although they recognized that research is still needed to determine what constitutes adequate intervention for children of different needs.16 Christiansen et al compared 2 regimens of PT—intermittent (4 times a week for 4 weeks, alternating with a 6-week hiatus) or continuous (once or twice a week, every week)—both for 30 weeks.17 They found no difference in GMFM between the 2 regimes and noted that compliance was higher in the intermittent group.

Constraint-induced therapy (also called forced-use therapy) has received much attention during the past decade. In this protocol, a hemiparetic child’s better-functioning upper extremity is constrained to force use of the more poorly functioning one. This is believed to lead to plastic changes in the
brain that improve the function of the less able limb. Randomized trials have shown the potential of this therapy for increased acquisition of new motor skills and increased dexterity that may last for at least 6 months. Compliance may be a significant issue, one addressed by Charles et al, who tested a “child friendly” schedule of 6 hours per day of constraint for 10 of 12 consecutive days. They found this regimen was effective in ways similar to a more restrictive one. These results were retained over the long term and could be improved upon by a further course of treatment.

Ankle-foot orthoses (AFOs) are commonly used to treat dynamic equinus in CP. Carlson et al have shown with gait analysis that AFOs could significantly reduce ankle excursion and increase dorsiflexion angle at foot strike, as well as provide other benefits, although neither stride length nor walking speed were improved. AFOs can also improve the sit-to-stand transition in preambulatory children whose standing is impaired by equinus. Bjornson has suggested that dynamic AFOs are most effective in younger children.

One cautionary note is in order: the patient or caregiver may expect use of any of the new modalities, such as botulinum toxin, intrathecal baclofen, or selective dorsal rhizotomy, to “substitute” for an individualized PT program. Experience has shown that patients who do not continue with a program of strengthening and stretching do not realize the potential benefits of the intervention.

**Oral Medications**

Oral antispasticity agents have the advantage of ease of use but the disadvantages of systemic effect and significant side effects. Thus, they are most appropriate in children for whom only mild tone reduction is needed or in whom spasticity is widespread. Unfortunately, most studies on the efficacy of these agents are old, did not address measures of function, and employed trial designs less rigorous than the best practices of today. In addition, most antispasticity trials have been carried out in adults with spastic disorders, and relatively few trials have been conducted in children. Thus, the choice of agent is more a matter of personal experience and trial and error than of rigorous, evidence-based medicine. Continued use of any particular agent must be justified by clear benefit to the patient.

**Baclofen**

Baclofen is a GABA-B agonist, and oral baclofen is often the drug of choice for spasticity of spinal cord origin in adults. It may be useful in selected pediatric patients, although little research has been conducted to support use in this population, and it does not have Food and Drug Administration approval for use in CP. Milla et al showed in a double-blind crossover trial that baclofen reduced spasticity significantly better than placebo and improved both passive and active movement. More recently, Scheinberg et al showed that whereas there was no effect on spasticity as measured by the Modified Tardieu scale or on the Pediatric Evaluation of Disability Inventory, there was improvement vs placebo on the Goal Attainment Scale, a functional assessment tool. The tendency of Baclofen tendency to cause confusion and sedation limits the dose, although these effects may improve over several weeks of treatment. A typical starting dose in a child is 2.5 mg/d, which can be gradually titrated up to a maximum of 20-60 mg/d, depending on body size. Weaning off of Baclofen must be gradual to avoid a withdrawal syndrome.

**Tizanidine**

Tizanidine is a centrally acting alpha-2 noradrenergic agonist that has been shown to reduce tonic stretch reflexes and to enhance presynaptic inhibition in animals. A Russian study in 30 diplegic children of tizanidine dosed at 6 mg/d reported improved motor ability, which was confirmed with electromyography. Side effects were reported to be well tolerated. To date, no clinical trials of this agent in children have been published in the English-language literature. Sedation and the requirement for frequent dosing have been limiting factors in tizanidine use. Nevertheless, the sedative quality can be an advantage when the medication is delivered at night. Improved initiation of sleep and reduced tone are potential benefits.

**Diazepam**

Diazepam is a benzodiazepine that facilitates the postsynaptic action of GABA. A series of trials in the 1960s demonstrated its ability to reduce spasticity in children with CP. More recently, Mathew et al showed the ability of diazepam to reduce muscle overactivity in comparison to placebo in a randomized trial of 180 children. Diazepam has also been compared with and used in conjunction with dantrolene sodium in the same small trial, the 2 agents appeared to be equally effective, and the combination was more effective than either alone. The daily dose of diazepam is usually 0.12-0.8 mg/kg, divided into 3-4 doses. The known sedation profile of this class of medications must be considered. Diazepam at bedtime may aid sleep without carry-over daytime sedation.

**Dantrolene Sodium**

Unlike other oral antispasticity agents, dantrolene sodium works at the muscle level, inhibiting calcium release from the sarcoplasmic reticulum, causing muscle weakness. In double-blind crossover studies, it has been shown to reduce spasticity in children with CP. In double-blind crossover studies, it has been shown to reduce spasticity in children with CP. It can cause global weakness and, despite its peripheral mode of action, sedation. It also has the potential for hepatotoxicity in approximately 1% of patients. It does not have long-term stability in liquid form, which limits its administration primarily to children able to take capsules.

In general, the cognitive and sedative side effects of oral medications can be said often to overshadow any improvement in spasticity, leaving the patient with minimal global gain in function. Thus, other types of treatments often play a more important role in tone management in the child with CP. Oral agents may be most useful as adjuncts in children with seizures, sleep disturbance, or other conditions in which
the other effects of these medications, besides the antispastic effects, are useful.

**Botulinum Toxin, Phenol, and Alcohol**

Chemodenervation (also called neurolysis or neuromuscular blockade) refers to the use of an injectable therapy to prevent nerve-muscle transmission. Two strategies are in current use: perineural injection of phenol or ethyl alcohol and intramuscular injection of botulinum toxin (BoNT). All 3 are appropriate for focal spasticity or for targeting specific problem muscles in more generalized spasticity.

Both phenol and alcohol have been used in children with CP, although neither has been widely or rigorously tested. Phenol is typically injected at a concentration of 3%-6% aqueous solution, whereas absolute alcohol is diluted to 30%-50%. The target nerve is identified with electrical stimulation, a procedure that may be poorly tolerated in children, making sedation or anesthesia necessary. The agent is injected perineurally, where it promotes denervation via axonal degeneration. The effect is not permanent, with functional reinnervation occurring over months to years. Studies have shown the benefits for spasticity in CP of both alcohol and phenol. Benefit ranges from a few weeks to 2 years or more, with no factors conclusively identified in the literature as determining the duration. Adverse effects of both agents include a significant risk of pain or paresthesia when targeting a mixed nerve, which may persist. This, combined with the high degree of skill required to target the nerve and the availability of botulinum toxin as an alternative, has kept phenol and alcohol from assuming a larger role in focal spasticity management. Nonetheless, the absence of immunogenicity and the lower cost compared to BoNT make these agents more attractive in some settings.

Botulinum toxin is an exotoxin produced by *Clostridium botulinum*, the same organism responsible for tetanus. There are 7 naturally occurring serotypes of the toxin, A-G, all of which are zinc proteases that target the synaptic vesicle fusion machinery at the neuromuscular junction. Denervation occurs because vesicles cannot fuse with the synaptic membrane, and therefore, acetylcholine cannot be released. The 7 serotypes differ in the specific component of the fusion machinery they target, their duration of action, their unit potency, and their immunogenic potential. Two serotypes, A and B, are commercially available. BoNT-A is marketed under the trade names Botox (Allergan; throughout the world), Dysport ( Ipsen; in Europe and elsewhere), and Xeomin (Merz; in Europe and elsewhere). BoNT-B is marketed by Elan Biopharmaceuticals in the United States as Myobloc and in Europe as NeuroBloc. At effective antispasticity doses, all 4 types have roughly the same duration of clinical action, approximately 3 months. Potency is expressed in mouse units, the amount of toxin required to kill 50% of mice in a standardized assay. Because of differences in molecular formulation and other variables, the potency of a single unit varies greatly among the commercial types. Although a unit of Botulinum toxin is roughly as potent as 1 unit of Xeomin, 4 units of Dysport, and 40-50 units of Myobloc, it is important to recognize that there is not a simple ratio of dosing equivalencies. Because of these differences, it is critical to specify the commercial brand when discussing units for dosing recommendations.

Most published research in CP has been conducted with BoNT-A, although a small number of studies have examined the effect of BoNT-B in this population. Sanger et al injected BoNT-B into the biceps and brachioradialis or 1 or both arms in 7 children with CP and upper extremity dystonia. In this open-label trial, reaching speed improved in response to treatment, although no dose-dependent effect was observed. BoNT-B and BoNT-A have both been combined successfully with phenol injection, allowing an increase in the number of treated muscles per injection session. BoNT-B has a tendency to cause more autonomic side effects than BoNT-A, and should be used with this caution in mind.

Although earlier studies of BoNT-A concentrated mainly on demonstrating spasticity reduction via lowered Ashworth scores, more recent work has focused on determining the functional benefit from injections. Fehlings et al showed that upper extremity injections of BoNT-A plus occupational therapy were superior to occupational therapy alone on the Quality of Upper Extremity Skills Test, an effect they correlated with preinjection grip strength. Lower extremity function has also been shown to improve significantly, however, this may not correlate with an improvement in health-related quality of life.

Injections can also ease pain and have been shown by one group to be equivalent to fixed casting for improvement of dynamic calf tightness but inferior to casting by others. The 2 can be combined, which some studies have shown to produce better results than either alone. Casting is usually delayed until the peak effect of the toxin, about 2-4 weeks after injection, which produces superior results to immediate casting.

The functional improvements seen in randomized trials are somewhat modest, but this may be more attributable to limitations of study design than to the limitations of the therapy itself. Focal improvements in spasticity are difficult to capture with global measures of function such as the GMFM, and the individualized dosing and injection patterns that optimize therapy are usually not possible in rigorous trials. Practitioners and caregivers usually agree that the benefits they see from BoNT injection are greater than those revealed in clinical trials, although family satisfaction may not always accompany measurable functional gains. Even though there may be no progressive improvement over the course of repeated injection cycles, the benefits of a single injection seen early in treatment can persist for many injection cycles without waning.

Dosing guidelines for BoNT-A as Botox, for adults and children, have been developed by consensus. With time, the recommended ceiling doses used by experienced practitioners have increased from 4 to 16 U/kg (Botox) in some cases. In children, maximum dosing should take into consid-
eration the child’s weight, muscle bulk, and degree of spasticity. The lowest effective dose with an injection interval of at least 3 months or more should be used to minimize the risk of antibody development. Children may need a topical anesthetic or light sedation during injection. Electromyography (EMG) or electrical stimulation is sometimes used to help target difficult-to-localize muscles, such as in the upper extremities, although clinical examination is sufficient for determining affected muscles in many situations. Injection of the psoas requires visualization by ultrasound or other means. Some injectors potentiate the effects of injection with neuromuscular electrical stimulation applied over the injection site for the first 3 days after injection. The family can be instructed to perform this.

Adverse effects of injection are usually mild and transient, consisting of pain on injection, occasionally a mild flulike syndrome, and excess weakness. The botulinum neurotoxin complex is immunogenic, and repeated exposure can lead to immunoresistance. Rates of immunoresistance in spasticity treatment have not been published, but the accepted rate in adults with dystonia is approximately 3%. Recently, Allergan introduced a new batch of Botox to replace the original commercial batch that had been the source of all commercial Botox until then. Initial reports indicate that the new batch may be less immunogenic. If immunoresistance develops, it is possible to switch serotypes, although immunoresistance to the second may develop more quickly than to the first.

**Rhizotomy**

Selective dorsal rhizotomy (SDR) was introduced in North America in the early 1980s, and has since become a commonly used treatment for reduction of moderate to severe lower-extremity spasticity. During the procedure, the patient is positioned prone and the dural sac is exposed so that nerve roots from L2 to S2 can be identified. Individual nerve rootlets are identified and stimulated independently. Those afferent rootlets that elicit excessive activity, monitored via EMG and visual observation, are cut. Typically 25%-50% of rootlets are cut. Controversy exists about how accurately EMG identifies the appropriate rootlets. Hays et al found no consistent relationship between the proportion of abnormally responding rootlets and the degree of spasticity and gross motor abnormality. They concluded that EMG monitoring as it is typically performed “is unlikely to identify accurately those neural elements that contribute to spasticity in children with CP.”

Results from clinical trials of SDR have indicated that it reduces spasticity, although the magnitude of the effect may be variable. Engberg et al showed that ankle spasticity could be reduced almost to normal by SDR, although the standard deviations were large, indicating high response in some patients and minimal response in others. The same group obtained similar results in a measure of hip spasticity conducted later. In a prospective study, Buckon et al demonstrated that SDR and orthopedic surgery can each contribute significantly to improvements in movement over the long term.

The relationship of spasticity reduction to functional improvement has also been controversial with this treatment, as with others, and 2 investigator-masked trials of similar design have reached opposite conclusions. Both groups randomized candidates to receive surgery plus PT or PT alone. Both groups quantified functional changes with the GMFM and used a variety of measures to quantify spasticity changes. McLaughlin et al included 21 surgical and 17 PT-only patients, with a 24-month follow-up, whereas Wright et al included 12 surgical and 12 PT-only patients, with a 12-month follow-up. Evaluators in both studies were blinded to surgical status. They differed in one significant aspect: McLaughlin et al sectioned a mean of 25% of rootlets vs approximately 50% for Wright. In addition, patients in the Wright study were significantly more functionally impaired at baseline than those in the McLaughlin study. Both groups showed significant postoperative reduction in spasticity with SDR plus PT compared to that with PT alone, which, in the words of McLaughlin, was judged by blinded investigators to be “clinically obvious and meaningful.” However, in that study, no long-term functional differences were seen between the 2 groups, despite a 24-month follow-up in which spasticity reduction might have been expected to afford the SDR group an advantage. In contrast, Wright et al showed a 7.7 point difference in GMFM scores after 12 months that favored the rhizotomy group. Further studies will be needed to determine whether the different outcomes in these 2 studies were attributable to differences in patient population, surgical technique, or some other variable.

Reduction in strength following SDR has historically been a concern, although more recent studies have not found objective loss of strength. A retrospective study of 158 children who underwent SDR in a single center showed that postoperative complications occurred in up to 30% of patients. Approximately 10% of patients had back pain starting 6 months or more after surgery, sensory changes, and neurogenic bladder or bowel problems. In our experience, the best candidates for SDR are children from ages 4–8 for whom spasticity is more significant in the legs than in the arms, with reasonably well-preserved leg strength and mobility, and whose spasticity is interfering with that mobility. SDR is followed by intensive PT to remobilize the child and improve strength. Despite best management, most children who require SDR will also eventually require orthopedic surgery to correct spasticity-induced deformities. However, SDR may reduce the number of such surgeries required. Overall, the use of SDR has declined with the expanding indications for intrathecal baclofen.

**Intrathecal Baclofen**

Intrathecal baclofen (ITB) is delivered to the intrathecal space via a catheter attached to an implanted pump. Because of direct delivery to the central nervous system, the required dose is less than 1% of that delivered orally, thus limiting the side effect of lethargy, which is especially of concern in this
Population. ITB has been shown to reduce spasticity in CP in several large trials.\textsuperscript{81-83} Some functional improvement has been demonstrated as well.\textsuperscript{83,84} ITB is expensive, but a recent modeling of cost-effectiveness indicated that over a 5-year period, ITB, despite increasing cost of care by $49,000 compared to alternative treatment, added 1.2 quality-adjusted life-years, which is considered “good value for the money.”\textsuperscript{85}

ITB is typically indicated for patients with lower-extremity spasticity, although studies have shown significant benefit on upper-extremity spasticity as well. The dimensions of the pump—about the size and shape of a hockey puck—limit the use of ITB in the very young children, although a smaller pump is available and has been implanted in children as young as age 3. Typically, candidates are screened with a bolus injection of baclofen delivered via lumbar puncture. A catheter trial with incremental dosing over several days is beneficial in certain patients, especially those with dystonia or other movement disorders. A negative response is an indication that further clinical evaluation may be necessary, for instance, to rule out fixed contracture as the source of muscle tightness.

The pump is implanted subcutaneously or subfacially in the abdomen, and the catheter is advanced to the high thoracic region. The pump contains a refillable reservoir, and an alarm sounds when the reservoir is low. Refills, delivered transtranscutaneously, are required every 2–3 months. The pump is programmable via a telemetry wand, and the dose may be adjusted to serve different functions over the course of the day, for instance, increasing at night to aid in comfort for sleep and decreasing in the morning to aid transfers.

Chronic ITB use carries a small but significant risk of serious complications. Overdose from a programming error is possible, and may lead to respiratory depression and coma. Abrupt withdrawal caused by emptying of the reservoir, pump failure, or catheter withdrawal, kinking, or breakage is more common. More than a dozen cases of a withdrawal syndrome and 6 deaths have been reported in the literature.\textsuperscript{86} Symptoms develop over 1-3 days and include prodomal itching or paresthesias, rebound spasticity, priapism in men, tachycardia, hypotension or labile blood pressure, dysphoria evolving to decreased consciousness, and the potential for seizures. The most effective treatment is the prompt restoration of ITB therapy. Intravenous benzodiazepines and high-dose oral or enteral baclofen, along with life-support measures, may be needed until restoration is possible.

**Orthopedic Surgery**

Despite best medical and rehabilitation management, children with spastic CP will often eventually require orthopedic surgery to correct deformities induced by muscle overactivity. Uncorrected deformities can cause pain, interfere with mobility or care, and lead to subluxation. It is estimated that hip subluxation or dislocation occurs in up to 25% of children with CP.\textsuperscript{87} Correction typically involves multiple tenotomies and transfers of the thigh adductors and, in severe cases, femoral osteotomy. Equinovarus foot is the most common deformity seen in CP, resulting in a flexed ankle and turned-in foot, sometimes with toe curling. Treatment often involves tendon lengthening. An advance is the SPLATT—split anterior tibial transfer—in which the tendon is split along its length and the distal end of the lateral half is transferred to the cuneiform and cuboid bones, where it can exert a corrective pull across the joint. This procedure is typically combined with Achilles tendon lengthening and toe flexor release. Treatment may also be prescribed for the knee and the upper extremities, depending upon the degree of deformity in these joints.

Orthopedic procedures are best performed sometime after gait has matured, usually between the ages of 6 and 10. As far as possible, the multilevel procedures are performed all at once to minimize postsurgical recovery times, and the child is mobilized as quickly as possible. PT remains a central part of treatment to strengthen and improve range of motion in newly realigned limbs.

**Conclusions**

Muscle overactivity can be a significant source of functional disability in a child with CP. Treatment planning is centered on improving function, comfort, and care; reducing pain; and preventing or correcting deformity. Oral medications, chemodenervation, rhizotomy, intrathecal baclofen, and orthopedic surgery may all play a role in treatment of the properly selected child. Physical and occupational therapy are central to any treatment plan.

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**References**


