# Orthopaedic Management of Spasticity

TYLER S. PIDGEON, MD; JOSE M. RAMIREZ, MD; JONATHAN R. SCHILLER, MD


## ABSTRACT

Spasticity is a common manifestation of many neurological conditions including multiple sclerosis, stroke, cerebral palsy, traumatic brain injury, and spinal cord injuries. Management of spasticity seeks to reduce its burden on patients and to limit secondary complications. Non-operative interventions including stretching/splinting, postural management, physical therapy/strengthening, anti-spasticity medications, and botulinum toxin injections may help patients with spasticity. Surgical management of these conditions, however, is often necessary to improve quality of life and prevent complications. Orthopaedic surgeons manage numerous sequelae of spasticity, including joint contractures, hip dislocations, scoliosis, and deformed extremities. When combined with the efforts of rehabilitation specialists, neurologists, and physical/occupational therapists, the orthopaedic management of spasticity can help patients maintain and regain function and independence as well as reduce the risk of long-tem complications.


**KEYWORDS:** Spasticity, Orthopaedics, Multiple sclerosis, Stroke, Cerebral palsy, Traumatic brain injury, Spinal cord injuries (SCI)

## INTRODUCTION

Spasticity is a common manifestation of many neurological conditions including multiple sclerosis (MS), stroke, cerebral palsy (CP), traumatic brain injury (TBI), and spinal cord injury (SCI).[1–4] Spasticity can lead to severe physical, psychological, and social impairments.[4–6] While numerous non-operative treatments are available, surgical management is often necessary to improve quality of life and prevent complications.

## EPIDEMIOLOGY

Spasticity affects up to one-third of all stroke survivors. Six months after stroke, as many as 50% of patients have developed contractures.[5,7,8] Up to 90% of patients with MS experience spasticity and as many as one-third modify their activity as a result.[5,6] Spasticity has been reported in 25% to 89% of patients with TBI and is a common feature in SCI (65% to 78% of patients) and CP (72% to 91%).[9–14]

## DIAGNOSIS

"Spasticity" refers to "disordered sensorimotor control resulting from an upper motor neuron (UMN) lesion, presenting as intermittent or sustained involuntary activation of muscles."[1,2,4,6,15] Patients commonly exhibit increased tone, hyperreflexia, clonus, a Babinski sign, reduced velocity of movement, reduced motor control, weakness, and loss of dexterity.[1,2,4,16] Increased tone manifests as a resistance to passive motion that is mediated by exaggerated spinal motor neuron responses to muscle stretch.[2–4] Over time, sarcomeres in underutilized muscles are replaced by fat and connective tissue, resulting in contractures.[2] Upper extremity spasticity often presents with hypertonia in the shoulder adductors; elbow, wrist, and finger flexors; and forearm pronators.[1,2] Lower extremity spasticity usually presents with high tone in the hip adductors, knee flexors, ankle plantarflexors and invertors, and great toe extensors.[1,2,4]

## NON-OPERATIVE MANAGEMENT

### Stretching/Exercise/Posture

Passive stretching is a mainstay of spasticity treatment, as it decreases the excitability of motor neurons and maintains flexibility.[2] Exercise improves motor control, strength, and overall function in addition to helping trunk, pelvic, and shoulder girdle muscles to control distal movements.[2,3] Exercise may not directly reduce spasticity, but it does not worsen hypertonia, as was previously thought.[2,3,17] Additionally, weight bearing reduces spasticity, improves bone mineral density, enhances psychological health, and aids lung, bowel, and bladder function; all of these benefits are especially important for people with disabilities.[2,3]

### Medications

**Table 1** summarizes the oral and injectable medications commonly used in the management of spasticity. Oral agents may unmask weakness and should be started at low doses and titrated up as needed.[2,3] Targeted Botulinum toxin (Botox) injections cause selective weakening of spastic muscles, obviating the generalized weakness associated with oral agents.[2] Similarly, neurolysis with phenol injections can achieve targeted muscle weakening, but should be performed only with pure motor nerves to avoid the risk of chronic neuropathic pain.[2–4]

**Table 1. Medications for the Treatment of Spasticity[2]**

Table 1 illustrates many of the commonly used oral medications for the treatment of spasticity.

GABA: "Gamma-Aminobutyric acid." QHS: "At bedtime;" 1XD: "One time daily;" 2XD: "Two times daily;" 3XD: "Three times daily."

| Drug Name | Administration | Mechanism | Dosage | Side Effects |
|---|---|---|---|---|
| Baclofen | Oral, Intrathecal | GABA agonist | Starting dose: 5mg 3XD. Increase 5-10mg weekly until desired effect. Max dose: 90-120mg/day | Weakness, drowsiness, dizziness, sexual dysfunction, urinary incontinence, reduction of seizure threshold, withdrawal |
| Benzodiazepines | Oral, Intravenous | Potentiates GABA system | Starting dose (clonazepam): 500µg QHS. Max dose: 1mg QHS | Drowsiness, dizziness, fatigue, respiratory depression, dependency, withdrawal, seizures, hypotension, tachycardia |
| Gabapentin | Oral | Stimulates GABA biosynthesis | Starting dose: 300mg 1XD day 1; 300mg 2XD day 2; 300mg 3XD day 3. Increase by 300mg every 2-3 days until desired effect. Max dose: 3600mg daily | Weight gain, gastro-intestinal disturbances, confusion, depression, hostility, sleep disturbance |
| Pregabalin | Oral | GABA agonist | Starting dose: 75mg 2XD. Max dose: 300mg 2XD | Weight gain, gastrointestinal disturbances, confusion, depression, hostility, sleep disturbance |
| Tizanidine | Oral | Central α-2 adrenergic system agonist | Starting dose: 2mg QHS. Increase by 2mg weekly as needed. Max dose: 36mg (divided into 3-4 daily doses) | Dry mouth, gastrointestinal disturbance, hypotension, acute hepatitis, withdrawal: hyperadrenergic syndrome |
| Dantrolene | Oral, Intravenous | Blocks calcium release from sarcoplasmic reticulum, blocking contraction of muscle cells | Starting dose: 25mg 1XD. Increase by 25mg per week as needed. Max dose: 100mg 3-4 times daily | Hepatotoxicity and rare fatalities (need regular liver function tests) |

## OPERATIVE MANAGEMENT

### Intrathecal Baclofen

Baclofen inhibits the spinal cord's reflex arc, which reduces resting muscle tone. It can be delivered via intrathecal pumps in small doses of high concentrations that can be titrated to a desirable level of inhibition.[2–4,16,18–21] Patients who rely on some tone to maintain posture will benefit from such a reduction without elimination of their spasticity.[2,3,16] As with any implantable device, infection is a concern.[16,22–24] Furthermore, errors in surgical implantation or catheter-related problems can cause baclofen overdose or withdrawal.[4,16,25–27]

### Selective Doral Rhizotomy

Selective dorsal rhizotomy (SDR) reduces afferent input to the spinal reflex arc, dampening the heightened response to muscle elongation seen in spasticity.[2,16] SDR is a relatively permanent and cost-effective solution when compared to baclofen pumps, which require regular maintenance and refills.[16,28] SDR in children reduces the need for future orthopaedic procedures.[16,29] The procedure has also been shown to improve motion throughout the gait cycle.[30,31] Because SDR affects all afferent signals at the dorsal root, decreased proprioception and sensory function may impair walking and standing.[16,32]

## ORTHOPAEDIC MANAGEMENT

### Upper Extremity Management

Reconstructive surgery of the upper extremity in patients with spasticity can improve range of motion (ROM), strength, functional grasp, dexterity, two-point discrimination, stereognosis, and limb positioning.[33–37] Surgical options include tendon transfers, muscle/tendon lengthening, and joint stabilization.[35,37,38] **Table 2** summarizes numerous reconstructive options for the management of common spastic problems of the upper extremity.

Forearm pronation deformity can be addressed by rerouting or releasing the pronator teres (PT) muscle.[38] Tenotomy is preferred in patients who are able to supinate and have

**Table 2.** Common Soft Tissue Reconstructive Procedures for Treatment of Spasticity of the Upper Extremitya

| Location | Deformity | Procedures |
|---|---|---|
| Elbow | Flexion | • Lengthening: Biceps, brachialis |
| Forearm | Pronation | • Releases: Pronator teres, pronator quadratus<br>• Rerouting: Pronator teres |
| Wrist | Flexion +/- ulnar deviation | • Lenthening: FCR/FCU<br>• Flexor pronator slides<br>• Tendon Transfers: PT, BR, FCU, ECU, FCR to ECRB/ECRL |
| Fingers | Flexion | • Lengthening: FDS<br>• Tendon Transfers: FCU/BR to EDC, FDS tenodesis, lateral band rerouting |
| Thumb | Thumb-in-palm | • Releases: Adductor pollicis, 1st DI<br>• 1st web space Z-plasty<br>• Lengthening: FPL<br>• Tendon Transfers: FCR/PL/BR to APL, FCR/PL/BR to EPB, PL/BR to EPL<br>• Rerouting: EPL |

a: Adapted from Upper extremity Surgical Treatment of Cerebral Palsy. Van Heest et al., 1999

continuous PT spasticity.[38] Tendon transfer is favored for phasic PT contractions during supination.

A spastic flexor carpi ulnaris (FCU) can lead to wrist flexion and ulnar deviation, which impairs grasp and release. Patients with voluntary control of the FCU may benefit from transfer to the extensor carpi radilias brevis (ECRB), as described by Green.[39] Patients lacking finger extension may be best served with transfer to the finger extensors or combining the Green procedure with lengthening of the finger flexors.[37] Transfer of the FCU to the extensor carpi radialis longus (ECRL) can correct ulnar deviation. Additionally, transfers of the pronator teres, brachioradialis, or extensor carpi ulnaris (ECU); flexor carpi radialis (FCR) transfer to the ECRB; lengthening of the wrist flexors; or wrist fusion can be used to address the flexion deformity.[37]

Adduction of the thumb due to a spastic adductor pollicis (AP) muscle characterizes thumb-in-palm deformities.[38] Spasticity of the flexor pollicis muscles, metacarpophalangeal joint instability, and interphalangeal joint flexion or hyperextension can also contribute to the deformity.[38] In patients with voluntary control of the extensor pollicis longus (EPL), a radial transfer of the tendon from the third to first dorsal compartment on the radial side of the thumb can help restore thumb extension when combined with a release of the AP.[40,41] Associated first web space contractures can be addressed via z-plasty.[38] The deformity can also be addressed by tendon transfer to the thumb abductors and extensors and/or lengthening of thumb flexors.[38]

## Hip Abnormalities

Hip deformities such as coxa valga, femoral anteversion, and acetabular dysplasia are common in patients with spasticity.[42] Up to 75% of patients with CP may experience hip subluxation, with more severely affected patients experiencing higher rates of subluxation and dislocation.[42–45] Hip abnormalities should be identified early in at-risk patients to prevent long-term complications.[42]

Radiographically, hip subluxation can be quantified by the Reimer migration index (RMI) and acetabular dysplasia can be evaluated by the acetabular index (AI), as illustrated in **Table 3**.[42] Independent ambulators should obtain a baseline AP pelvis film between age two and four, and should be followed clinically unless the exam suggests that further imaging is needed.[42] Patients who ambulate with assistive devices or not at all and who have an RMI of less than 30% should obtain yearly radiographs until age eight, and then biannual films until skeletal maturity.[42] Patients with an RMI of more than 30% require serial radiographs every six months.[42]

Numerous soft tissue operations for spastic hips in skeletally immature patients can prevent or address deformity and/or dislocation.[42] Adductor and iliopsoas lengthening or tenotomy have been shown to improve ROM, prevent dislocation, and reduce the need for bony reconstruction in children with hip muscle spasticity.[42,46,47]

Bony procedures about the hip may address acetabular and femoral deformities in skeletally immature patients with promising results.[48,49] Patients without substantial acetabular dysplasia benefit from proximal femoral osteotomy alone or combined with adductor/iliopsoas soft tissue procedures (**Figure 1**).[42] Such osteotomies often produce varus angulation to address coxa valga and rotation to address femoral anteversion. Patients with an abnormal AI may also require pelvic osteotomy.[42] Children under eight years of age with an RMI of 30%-60% may be treated operatively with adductor and iliopsoas lengthening/release; however, children over eight with an RMI greater than 40% and all children with an RMI greater than 60% should undergo a pelvic osteotomy combined with proximal femoral shortening/varus osteotomy and soft tissue releases.[42]

Skeletally mature patients with spastic hip deformities are considerably more challenging to treat. Nevertheless, periacetabular osteotomies combined with varus and de-rotational proximal femoral osteotomies can be successful.[50,51] In addition, proximal femoral resection procedures have been used to reduce pain and improve sitting in non-ambulatory adult patients with spastic hip deformities.[52,53] Using the resected femoral head to cap the resection arthroplasty reduces the risk of postoperative heterotopic ossification[54] Alternatively, a valgus-producing proximal femoral osteotomy pointing the femoral head away from the acetabulum allows indirect load transferring, which prevents proximal migration of the femur seen with resection arthroplasty.[55–57] Finally, some authors have demonstrated success with total joint arthroplasty in skeletally mature patients with spastic hip deformities.[42,58,59]

**Table 3. Radiographic Measurement of Hip Dysplasia[42]**

Table III illustrates two techniques for the radiographic evaluation of hip dysplasia. The Reimer migration index (RMI) is designed to evaluate subluxation of the femoral head while the acetabular index (AI) calculates dysplasia of the acetabulum. Increased AI suggests that a pelvic osteotomy may be necessary. AP: Anterior-Posterior.

| Classification | Measurement | Normal Values | Abnormal Values |
|---|---|---|---|
| Reimer Migration Index | Calculates width of uncovered femoral head compared to whole head | < 30% | > 40%: Hip at risk<br>> 60%: Dislocated hip |
| Acetabular Index | Measured on AP pelvis by calculating angle between acetabular roof and Hilgenreiner's line | > 5 yo: 20o<br>< 5 yo: 25o | > 5 yo: > 20o<br>< 5 yo: > 25o |

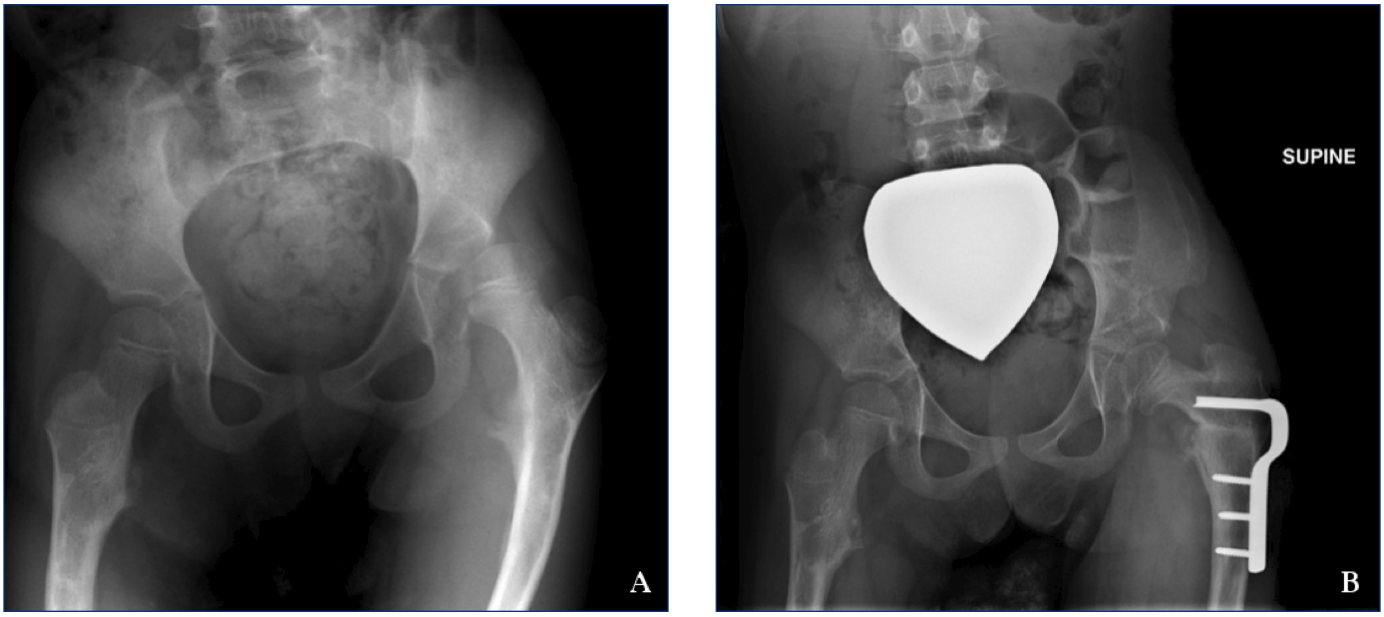
## Foot Abnormalities

Foot abnormalities are common in patients with spastic conditions and can lead to pain and difficulty with ambulation, shoe wear, and bracing.[42] In patients with CP, planovalgus and equinovarus deformities are most common.[42] Surgical correction of such deformities is reserved for patients aged ten or older, while younger children can often be managed with orthotics.

The flexible planovalgus foot is first addressed

**Figure 1. Spastic Hip**

Figure 1A illustrates a child with cerebral palsy suffering from a hip dislocation on the left side secondary to spasticity. This child was successfully treated with a proximal femoral osteotomy. The post-operative radiograph is displayed in Figure 1B. *Radiographs courtesy of Dr. Jonathan Schiller.*




with lateral column lengthening through the calcaneus (in patients who ambulate without assistive devices) or via reduction of the talus and calcaneus and a subtalar fusion (in patients with more severe gait problems).[42,60–62] The medial column can then be assessed; if there is residual forefoot supination a tibialis posterior advancement (in mild cases) or plantarflexion osteotomy of the first ray is performed.[42] Particularly severe cases may require a talonavicular arthrodesis.[42,63] Rigid planovalgus foot is treated with lateral column lengthening and triple arthrodesis.[64]

The equinovarus foot is common in hemiplegic patients and may be treated with split tendon transfers in mild, flexible cases.[42,65,66] Patients with forefoot/midfoot inversion may benefit from split anterior tibial tendon transfers while patients with hindfoot varus are treated with split posterior tibial tendon transfers (but some patients require both procedures).[65] In more severe but flexible cases, an additional wedge or sliding calcaneal osteotomy is used to address residual hindfoot varus.[42] In cases of rigid equinovarus foot, triple arthrodesis is often required.[67]

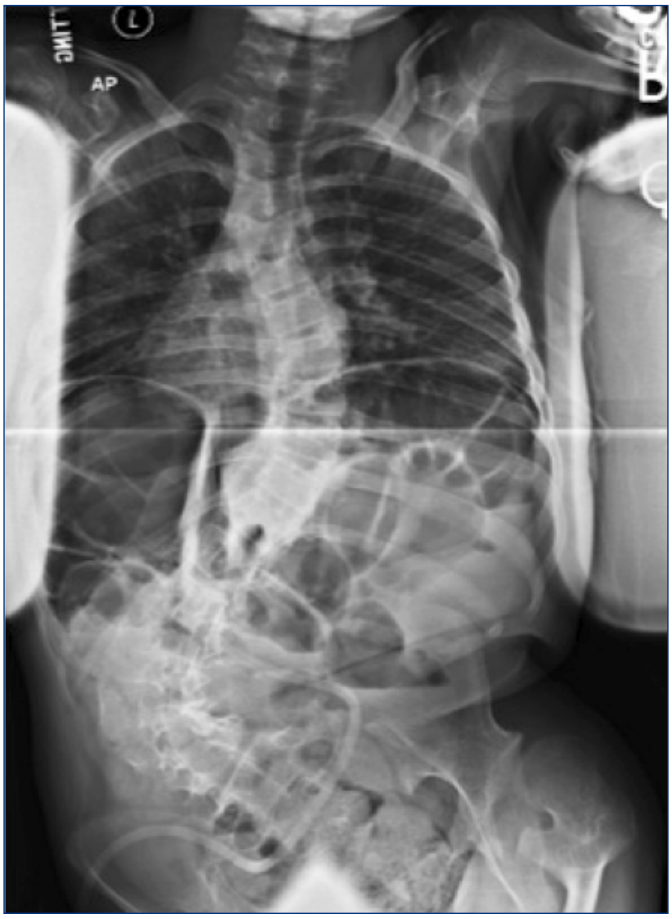
Both planovalgus and equinovarus deformities can be associated with and exacerbated by Achilles contractures related to gastrocnemius tone.[42] Lengthening of the gastrocsoleus complex may be necessary to achieve appropriate correction of these deformities; however, great caution must be observed in diplegic patients with a crouching gait, which may be worsened by plantarflexion weakness.[42,68]

## Spine Abnormalities

Spine deformity is common in patients with spasticity (**Figure 2**). Scoliosis has been reported in up to 77% of children with CP and is common in more severely affected patients.[42,69–73]

**Figure 2. Neuromuscular Scoliosis**

Figure 2 illustrates a patient with cerebral palsy with severe neuromuscular scoliosis. *Radiograph courtesy of Dr. Jonathan Schiller.*



Ambulatory patients develop curves similar to that of idiopathic scoliosis patients; those with severe disease and/or tetraplegia develop a long C-shaped curve that involves the pelvis.[42] Progression is more common in younger, skeletally-immature children; however, patients with large curves (>50°) may progress after skeletal maturity.[42,71,72] Furthermore, ambulatory patients exhibit less curve progression compared to non-ambulatory patients.[42,72] SDR, laminectomy, laminoplasty, and baclofen pump placement (controversial) have been linked to scoliosis development in patients with spasticity.[42,74–79]

Progression of disease can lead to cosmetic deformity as well as functional, physiologic (respiratory and alimentary), and postural impairment.[42] Ambulatory patients may be treated like patients with idiopathic curves.[42] If patients have severe neuromuscular curves, the treatment focuses on improving spinal balance, posture, and sitting ability.[42,80] Non-operative intervention, such as seating modification, bracing, and serial casting may delay future surgical intervention such as fusion.[42,80] Caution must be observed in patients with chest wall deformities who may succumb to respiratory compromise with rigid bracing.[42]

Insertion of growing rods in anticipation of definitive fusion is an option in children with significant growth potential[42,81,82] Non-ambulatory children may be best served with a primary T2 to pelvis fusion, which minimizes the incidence of junctional kyphosis and addresses pelvic obliquity, respectively. [42,83] While most fusions are performed posteriorly, the presence of a rigid pelvis may justify additional anterior surgery or hip reconstruction.[84] Furthermore, anterior fusion may be indicated to avoid the crankshaft phenomenon in growing children.[42] Lastly, while some ambulatory patients may avoid fusion to the pelvis, ambulation can be maintained after such a procedure.[42,85]

## CONCLUSION

Spasticity is a devastating manifestation of numerous common neurologic conditions. A multi-disciplinary approach, with non-operative and surgical options, is required to adequately treat patients suffering from this condition. Orthopaedic surgeons can play an important role in alleviating symptoms, preventing complications, and improving function in patients with spasticity.

### References


1. Nair, K. P. S. & Marsden, J. The management of spasticity in adults. *BMJ* **349,** g4737 (2014).
2. Kheder, A. & Nair, K. P. S. Spasticity: pathophysiology, evaluation and management. *Pract. Neurol.* **12,** 289–298 (2012).
3. Stevenson, V. L. Rehabilitation in practice: Spasticity management. *Clin. Rehabil.* **24,** 293–304 (2010).
4. Graham, L. A. Management of spasticity revisited. *Age Ageing* **42,** 435–441 (2013).
5. Martin, A., Abogunrin, S., Kurth, H. & Dinet, J. Epidemiological, humanistic, and economic burden of illness of lower limb spasticity in adults: a systematic review. *Neuropsychiatr. Dis. Treat.* **10,** 111–122 (2014).
6. Morley, A., Tod, A., Cramp, M. & Mawson, S. The meaning of spasticity to people with multiple sclerosis: what can health professionals learn? *Disabil. Rehabil.* **35,** 1284–1292 (2013).
7. Watkins, C. L. *et al.* Prevalence of spasticity post stroke. *Clin. Rehabil.* **16,** 515–522 (2002).
8. Kwah, L. K., Harvey, L. A., Diong, J. H. L. & Herbert, R. D. Half of the adults who present to hospital with stroke develop at least one contracture within six months: an observational study. *J. Physiother.* **58,** 41–47 (2012).
9. Schouten, Joost, W. & Andrew, I. R. in *Youmans Neurological Surgery, Sixth Edition* 3270–76 (Elsevier, 2011).
10. Elovic, E. & Zafonte, R. D. Hand Function in Children with Cerebral Palsy after Upper-Limb Tendon Transfer and Muscle Release. *Phys Med Rehabil State Art Rev* **15,** 327–48 (2001).
11. Thibaut, A. *et al.* Spasticity in disorders of consciousness: A behavioral study. *Eur. J. Phys. Rehabil. Med.* (2014).
12. Sköld, C., Levi, R. & Seiger, A. Spasticity after traumatic spinal cord injury: nature, severity, and location. *Arch. Phys. Med. Rehabil.* **80,** 1548–1557 (1999).
13. Maynard, F. M., Karunas, R. S. & Waring, W. P. Epidemiology of spasticity following traumatic spinal cord injury. *Arch. Phys. Med. Rehabil.* **71,** 566–569 (1990).
14. Odding, E., Roebroeck, M. E. & Stam, H. J. The epidemiology of cerebral palsy: incidence, impairments and risk factors. *Disabil. Rehabil.* **28,** 183–191 (2006).
15. Pandyan, A. D. *et al.* Spasticity: clinical perceptions, neurological realities and meaningful measurement. *Disabil. Rehabil.* **27,** 2–6 (2005).
16. Roberts, A. Surgical management of spasticity. *J. Child. Orthop.* **7,** 389–394 (2013).
17. Ada, L., Dorsch, S. & Canning, C. G. Strengthening interventions increase strength and improve activity after stroke: a systematic review. *Aust. J. Physiother.* **52,** 241–248 (2006).
18. Ben Smail, D. *et al.* Intrathecal baclofen for treatment of spasticity of multiple sclerosis patients. *Mult. Scler. Houndmills Basingstoke Engl.* **12,** 101–103 (2006).
19. Schiess, M. C. *et al.* Prospective 12-month study of intrathecal baclofen therapy for poststroke spastic upper and lower extremity motor control and functional improvement. *Neuromodulation J. Int. Neuromodulation Soc.* **14,** 38–45; discussion 45 (2011).
20. Zahavi, A., Geertzen, J. H. B., Middel, B., Staal, M. & Rietman, J. S. Long term effect (more than five years) of intrathecal baclofen on impairment, disability, and quality of life in patients with severe spasticity of spinal origin. *J. Neurol. Neurosurg. Psychiatry* **75,** 1553–1557 (2004).
21. Motta, F., Antonello, C. E. & Stignani, C. Intrathecal baclofen and motor function in cerebral palsy. *Dev. Med. Child Neurol.* **53,** 443–448 (2011).
22. Armstrong, R. W. *et al.* Intrathecally administered baclofen for treatment of children with spasticity of cerebral origin. *J. Neurosurg.* **87,** 409–414 (1997).
23. Albright, A. L. *et al.* Performance and complications associated with the synchromed 10-ml infusion pump for intrathecal baclofen administration in children. *J. Neurosurg.* **101,** 64–68 (2004).
24. Bennett, M. I., Tai, Y. M. & Symonds, J. M. Staphylococcal meningitis following Synchromed intrathecal pump implant: a case report. *Pain* **56,** 243–244 (1994).
25. Watve, S. V., Sivan, M., Raza, W. A. & Jamil, F. F. Management of acute overdose or withdrawal state in intrathecal baclofen therapy. *Spinal Cord* **50,** 107–111 (2012).
26. Meythaler, J. M., Roper, J. F. & Brunner, R. C. Cyproheptadine for intrathecal baclofen withdrawal. *Arch. Phys. Med. Rehabil.* **84,** 638–642 (2003).
27. Deer, T. R., Raso, L. J. & Garten, T. G. Inflammatory mass of

an intrathecal catheter in patients receiving baclofen as a sole agent: a report of two cases and a review of the identification and treatment of the complication. *Pain Med. Malden Mass* **8,** 259–262 (2007).

28. Langerak, N. G., Tam, N., Vaughan, C. L., Fieggen, A. G. & Schwartz, M. H. Gait status 17-26 years after selective dorsal rhizotomy. *Gait Posture* **35,** 244–249 (2012).
29. O'Brien, D. F., Park, T. S., Puglisi, J. A., Collins, D. R. & Leuthardt, E. C. Effect of selective dorsal rhizotomy on need for orthopedic surgery for spastic quadriplegic cerebral palsy: long-term outcome analysis in relation to age. *J. Neurosurg.* **101,** 59–63 (2004).
30. Nordmark, E. *et al.* Long-term outcomes five years after selective dorsal rhizotomy. *BMC Pediatr.* **8,** 54 (2008)

References 31–85

## Authors

Tyler S. Pidgeon, MD, Resident, Brown University Department of Orthopaedics.

Jose M. Ramirez, MD, Resident, Brown University Department of Orthopaedics.

Jonathan R. Schiller, MD, Assistant Professor of Orthopaedic Surgery, Brown University Department of Orthopaedics.

## Disclosures

This original work required no outside sources of funding. Dr. Schiller is an educational consultant for DePuy. Dr. Pidgeon and Dr. Ramirez have no conflicts of interest to declare.

## Correspondence

Tyler S. Pidgeon, MD
Division of Pediatric Orthopaedics and Scoliosis
Brown University Department of Orthopaedic Surgery
Rhode Island Hospital
593 Eddy Street, Providence, RI 02903
401-444-4030
Fax 401-444-6182
tyler_pidgeon@brown.edu