An Orthopedic Perspective on the Management of Spasticity

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ABSTRACT

Neurologic conditions such as brain injuries, cerebral palsy, stroke and multiple sclerosis involve injury of upper motor neurons, which can manifest as spasticity. The resulting hypertonicity and imbalance of forces between muscle groups leads to deformities that impair patient function and can cause significant long-term complications. Symptoms of spasticity can be managed with nonoperative techniques such as physiotherapy, bracing, or medications. Other approaches such as intrathecal baclofen pumps and selective dorsal rhizotomy have also demonstrated efficacy in controlling spasticity. Spasticity that has failed nonoperative management can be treated with orthopedic surgeries that correct deformities by either restoring joint anatomy or re-balancing the forces of spastic muscle groups. Improved mobility and reduced pain after these procedures can help patients with their rehabilitation, function, and independence.

KEYWORDS: Orthopedics; spasticity; brain injuries; cerebral palsy; upper motor neuron injury; spinal cord injury (SCI); multiple sclerosis (MS)

INTRODUCTION

Spasticity is a common clinical sequela of neurologic conditions such as brain injuries, strokes, spinal cord injury (SCI), cerebral palsy (CP), and multiple sclerosis (MS).[1-3] Along with muscle weakness, hyperreflexia, clonus, hypertonicity, and a positive Babinski reflex, it is one of the characterizing signs of upper motor neuron (UMN) lesions.[1] Spasticity encompasses a broad range of presentations, ranging from reducible deformity to permanent contracture or joint instability, and thus, is a difficult condition to define.[2,3] Dressler et al recently revised the definition of spasticity as involuntary muscle hyperactivity – including one or more of rigidity, dystonia, spasms, spasticity, or spasms – in the context of central paresis.[4] The resulting deformities and motor impairments caused by spasticity generate significant physical, psychological, and social burdens for patients.[3] Treatment of the condition through nonoperative or surgical means is a critical component of neurorehabilitation, and can significantly improve quality of life, help patients regain independence, and prevent long-term complications.[5]

NONOPERATIVE MANAGEMENT

Physiotherapy

Rehabilitation through modalities such as exercise, cryotherapy, and stretching plays a significant role in managing spasticity, and research suggests that maximum benefit is achieved by early intervention. Physical therapy typically focuses on stretching of spastic muscle groups and strengthening of muscle antagonists to maximize passive and active range of motion.[6] The improved motor control and strength aids in controlling distal movements, thereby improving overall motor function. Exercises that involve weight-bearing have also been shown to increase bone mineral density, improve bowel function, and enhance mobility.[5,7] Physical therapists will also guide and support decision-making around adaptive and assistive devices, based on a patient's abilities and long-term goals for mobility. Furthermore, if a patient undergoes operative management, physical therapy plays a significant role in maximizing postoperative benefits.[8]

Casting and Bracing

Stretching consistently at home to reinforce physical therapy gains may be challenging for patients and families. Therefore, bracing can be a useful supplement to therapy, with positioning to correct contractures, improve flexibility, and increase range of motion. Bracing may also improve balance, transfers, and ambulation as well as help maintain a comfortable position in a wheelchair. Proper fitting of orthotics is necessary to maximize stretching and positioning benefits while avoiding skin breakdown and irritation.[9]

Extracorporeal Shockwave Therapy

Extracorporeal shock wave therapy (ESWT) is a procedure for the management of spasticity that can be done in outpatient clinics. While the exact mechanism is unknown, ESWT involves the application of focused, singular acoustic pulses to create pressure waves that induce cellular changes. For patients with spasticity, ESWT has been shown to reduce spasticity and pain while improving range of motion and function.[10] This technique is painless, safe, and non-invasive, with improvements seen as early as after one session. This technique can be combined with other treatments of spasticity, with evidence to suggest that ESWT may enhance the effects of Botulinum neurotoxin (Botox).[11]

Table 1. Common medications used for the treatment of spasticity.[12,14]

Drug Name	Administration	Mechanism	Dosage	Side Effects
Baclofen	Oral, Intrathecal	GABA-B Agonist	Maximum 40 mg/day if age less than 8 years Maximum 60 mg/day if age older than 8 years	Sedation, fatigue, constipation, hepatotoxicity, weakness, drowsiness, withdrawal, seizures, altered mental status
Benzodiazepines	Oral, intravenous	GABA-A Agonist	For Diazepam, 6 months and older 1–2.5 mg orally 3-4 times daily	Sedation, fatigue, constipation, confusion, respiratory depression, dependency, withdrawal
Clonidine/ Tizanidine	Oral	Alpha-2 Agonist	**Clonidine:** 0.02 ± 0.03 mg/kg/day **Tizanidine:** Children aged > 2 years, 0.3–0.5 mg/kg/day in 4 divided doses	Hypotension, bradycardia, muscle weakness, sedation, xerostomia, hallucinations, QT interval prolongation
Gabapentin	Oral	Alpha-$2_{\delta 1}$ subunit binding to inhibit Ca2+ currents	**Infants:** 5 mg/kg/day, titrate by adding up to 3x day or increasing dose **3–11 years:** 10–15 mg/kg/day in 3 divided doses **>11 years:** 300 mg 3 times daily, titrate up to 3600 mg/day	Somnolence, tremor, nystagmus, mood changes such as anxiety or aggression, fatigue, weakness, nausea/vomiting, headache, challenges with concentration
Dantrolene	Oral	Inhibition of Ca+ release from sarcoplasmic reticulum to prevent muscle contraction	0.5 mg/kg once daily for 7 days, then 0.5 mg/kg 3 times a day for 7 days, then 1 mg/kg 3 times a day for 7 days, then 2 mg/kg 3 times day Maximum 100 mg 4x per day	Liver failure, weakness, fatality associated with high dosing
Botulinum Toxin	Injection	Acetylcholine release inhibition	**Botox:** 8 u/kg or 300 u total for lower limbs 10 u/kg or 340 u for total body	Dry mouth, dysphagia with use in upper limbs/neck muscles, double vision, weakness, respiratory difficulties, spread to other muscles, resistance with increased usage
Phenol/Alcohol	Injection	Chemical Neurolysis	**Phenol:** concentration of 3–7% (50mg/mL-70 mg/mL), pediatric dosing 30mg/kg **Alcohol:** concentration typically 40–50%	Flushing, cardiac dysrhythmia neuropathic pain, paresthesia, bowel/ bladder incontinence, sexual dysfunction, muscle fibrosis, vascular sclerosis

Medications

Several oral medications can be used in the management of spasticity. These are usually GABA receptor agonists, though alpha-2 agonists, gabapentin, and dantrolene are also helpful (**Table 1**). Injectable medications such as Botox, alcohol, or phenol can also achieve a more targeted effect on spastic muscle groups. The effectiveness of these medications can decrease over time, so using them in combination with other medications and maximizing the time between injections may prolong their effectiveness.[12] Cannabis is also an increasingly popular treatment option for pain control in spasticity, particularly for those with MS.[13] The benefit of cannabis to treat spasticity in other neurologic conditions or in pediatric conditions is less well-established.

PROCEDURAL MANAGEMENT

Intrathecal Baclofen

Intrathecal baclofen is useful in reducing spasticity while avoiding systemic adverse effects of oral medications such as sedation, or in patients who do not benefit from the maximum oral dose.[15] Intrathecal baclofen can reduce pain and improve general functional status, notably with gait and mobility.[16] Patients should have a trial injection of intrathecal baclofen to determine its efficacy. Following implantation of the pump, dosing can be titrated to achieve the best muscle tone for a patient's comfort and functional ability. Complications of pump placement include infection and catheter obstruction. The pump must be refilled, but the time between refills can be increased with gradual increases in the medication concentration.[17]

Selective Dorsal Rhizotomy

Selective dorsal rhizotomy (SDR) is a procedure that involves sectioning afferent nerve rootlets that innervate affected muscle groups, which reduces the exaggerated stretch reflexes of spasticity, leading to improvements in gross motor function and gait.[18] Additionally, patients who undergo SDR may be less prone to long-term complications of spasticity and thus require fewer surgeries.[19] The results of SDR also appear to be long-lasting; at 20-year follow-up, 91% of respondents reported that the procedure improved their quality of life and 88% said they would recommend the procedure to others.[20] Proper patient selection for this

procedure is crucial to maximizing outcomes. Patients who benefit most from the procedure are ambulatory with primarily lower extremity involvement, have good core and abdominal strength to support the trunk while walking, and the cognitive function and resources to participate in intensive therapy postoperatively.

ORTHOPEDIC SURGERIES FOR SPASTICITY

Orthopedic procedures for the treatment of spasticity can be grouped into the categories of tendon transfer, tendon lengthening or release, osteotomy, and arthrodesis.[3,5] Indications for surgery vary depending on the patient's age and goals, but they generally include fixed contractures, joint deformity, or joint dislocation that affects function, impairs hygiene, or causes significant pain.[2,21] Surgery aims to address deformities by stabilizing the contracted joint or correcting the muscle strength imbalance. Van Heest et al showed that children with spastic upper extremities who were suitable candidates for tendon transfers had greater improvement in function than repeated botulinum toxin injections or regular therapy sessions at 12 months of follow-up; this suggests that the surgical management of spasticity improves quality of life, provided that patients are suitable candidates for a procedure.[22]

Upper extremity

The shoulder's positioning in patients with spasticity is due to the unbalanced forces of the internal rotators: latissimus dorsi (LD), teres major (TM), pectoralis major (PM), and subscapularis.[3] For non-functional shoulders, complete release of the subscapularis, LD, and PM tendon insertions, while preserving the shoulder joint capsule, improved passive range of motion in flexion, extension, abduction, and external rotation. For patients with remaining shoulder function or some voluntary movement, fractional tendon lengthening of hypertonic muscle groups (PM, LD, and TM), is the preferred surgical procedure.[3,23] In shoulders with severe internal rotation and chronic posterior dislocation, proximal humerus derotational osteotomy helps to stabilize the joint and increase range of motion, especially in younger patients.[6,24]

Surgical treatment of elbow flexion deformities depends on their angles. Fixed contractures of less than 45 degrees are treated with fractional tendon lengthening of the elbow flexors, while those with worse contractures may require total tendon releases.[23] Fractional tendon lengthening involves the Z-lengthening approach, in which a tendon is split longitudinally and one of the resulting tendon limbs is reflected.[23,25] Both fractional tendon lengthening and complete tendon release of elbow flexors will increase range of motion and decrease pain in patients with spasticity.[23]

Contributions by the pronator teres and pronator quadratus make forearm pronation deformities far more common than supination ones.[26] Surgical correction is done via tendon lengthening of the pronator teres; full tendon release is done for patients without volitional activity of the muscle. Similarly, the pronator quadratus can also be partially or fully transected, depending on forearm function; in most cases, lengthening of the pronator teres is sufficient to improve supination.[5,26] In the rare case of spastic supination, the biceps tendon can be rerouted distally to wrap around the radius in order to restore the forearm to a neutral position.[26] Transfer of the flexor carpi ulnaris (FCU) dorsally to the extensor carpi radialis brevis (ECRB) or extensor digitorum communis muscles is often combined with pronator release or transfer to increase supination in patients with concomitant ulnar deviation deformities.

Flexed wrist, clenched fist, and thumb-in-palm deformity are classic deformities from spasticity, caused by hyperactivity of flexor muscles.[23,27] The most common procedure for surgical correction of wrist deformity is FCU to ECRB tendon transfer, which allows for increased extension and decreased ulnar deviation.[23] The tendon transfer can be passed ulnarly to aid in supination or radially to aid in pronation.[27] In patients with functional upper extremities, fractional lengthening of the flexor digitorum profundus (FDP), flexor digitorum superficialis (FDS), or flexor carpi radialis (FDR) via Z-lengthening can also improve hand function.[23,27] Patients who are skeletally mature can also have total wrist fusion for a severe flexion contracture [**Figure 1**].[6] Thumb-in-palm deformity can be addressed in a variety of surgical options, including tenotomy of the adductors, lengthening of the flexor pollicis longus (FPL), Z-plasty of the first web space, or tendon transfers of the extensors.[6] The surgical procedure depends on the specific tendinous structures that are abnormal and whether the first web space in implicated in the deformity.[27]

Figure 1. Twenty-year-old woman with left wrist contracture and a painful bunion deformity on left foot due to spasticity related to an intracranial bleed at age eight. [Radiographs are courtesy of Dr. Craig Eberson.]

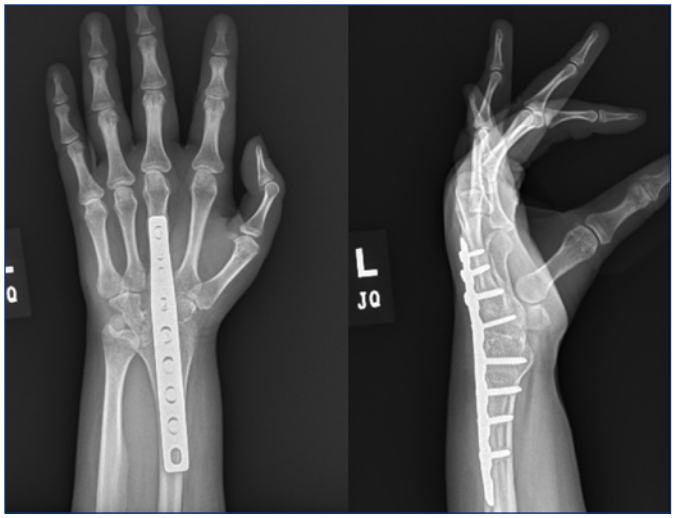
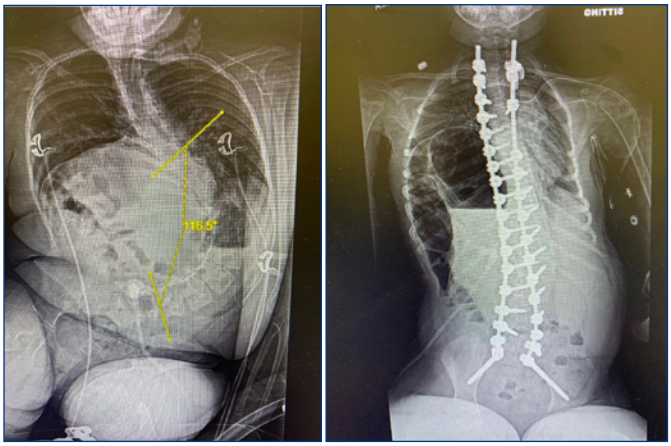


Figure 2. [A] Preoperative radiograph of a patient with severe neuromuscular spinal deformity secondary to cerebral palsy. **[B]** Postoperative radiograph after posterior spinal fusion from T2 to the pelvis, improving pulmonary function and allowing this patient to sit more comfortably.



Spine

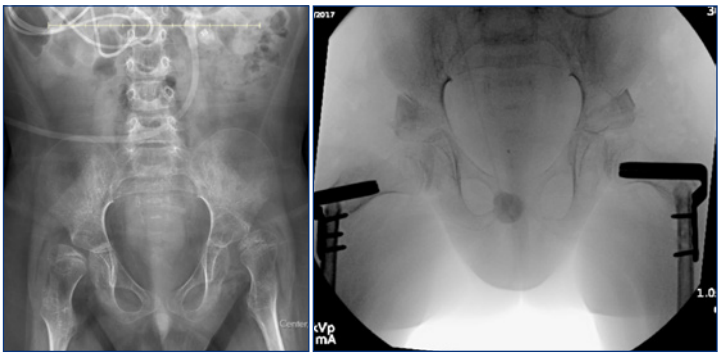
Cerebral palsy (CP) is the leading cause of neuromuscular scoliosis (NMS).[28] The progression of scoliosis curvature in CP patients can be rapid, particularly in those that are non-ambulatory, due to persistent muscle weakness, imbalance of forces, and decreasing bone density.[29] The resulting severe spinal deformity [**Figure 2A**] can cause adverse sequelae such as respiratory or cardiac compromise, pelvic obliquity, skin infections, poor nutritional status, and pain.[28,30] Nonoperative management with spinal bracing can delay surgical intervention, but has less efficacy in halting curve progression than in patients with adolescent idiopathic scoliosis.[29] Indications for surgery, traditionally a posterior spinal fusion, vary depending on the etiology of the deformity and patient circumstances, but is usually recommended in patients with progressing curves that are impacting balance or positioning with sitting or standing.[29,30] The extent of the spinal deformity determines the levels of spinal fusion; long fusions extending from T2 to the pelvis may be necessary in non-ambulatory children with CP [**Figure 2B**].[29,30] Posterior spinal fusion was previously combined with anterior procedures in patients with rigid spinal deformities or severe pelvic obliquity, but advances in implants and increased usage of traction devices have decreased the necessity of the combined anterior and posterior approach.[30] While surgical correction of neuromuscular scoliosis significantly improves quality of life, it also entails the highest surgical complication rate among all types of scoliosis. Seaver et al demonstrated a 10-year reoperation rate of 21.7%, most commonly due to implant failure and surgical site infections, in children with non-ambulatory CP who underwent posterior spinal fusion.[29,31] This is particularly notable as children who undergo posterior spinal fusion carry the implants within them as they transition to adulthood.[28] Thus, the final decision to pursue surgery requires detailed discussion of risks and benefits between providers and families in order to arrive at the optimal treatment plan.

Hip

Patients with spasticity are prone to hip abnormalities such as acetabular dysplasia, coxa valga, and femoral anteversion. Hypertonic hip flexors and adductors overpower the weaker hip extensors and abductors, shifting the center of rotation of the hip from the femoral head to the lesser trochanter and leading to pathologic remodeling of the femoral head and acetabulum.[32] Untreated deformities can cause subluxation and eventual dislocation of the hip (**Figure 3A,B**), along with early osteoarthritis due to the loss of joint integrity.[33]

Hip dislocation often progresses silently, especially in non-ambulatory children with CP. As a significant percentage of these patients are intellectually disabled or nonverbal, it is challenging for parents to identify hip pathology from observation of patients alone. Periodic surveillance with both clinical and radiographic examination is recommended, accompanied by hip joint measurements such as the Reimer migration index (RMI) and the acetabular index (AI) to allow for prompt recognition and surgical intervention.[33] In children with cerebral palsy, orthopedic surgeries of the spastic hip can be categorized into preventive, reconstructive, and salvage procedures, with differing goals, indications, techniques, and success rates.[34] (**Table 2**)

Figure 3. Eight-year-old male patient with spasticity and hip subluxation after spinal cord injury and traumatic brain injury at age one. [**A**] Preoperative radiograph demonstrating bilateral hip dislocation and deformity. [**B**] Postoperative radiograph after soft tissue release and femoral and pelvic lengthening for hip relocation. The operation reduced the difficulties the patient had with posture and sitting.



FOOT AND ANKLE

Equinovarus is the most common foot and ankle deformity seen in patients with strokes and cerebral palsy.[35] Other deformities in patients with spasticity include plano-valgus, toe flexion, and equinovalgus. Surgical management is recommended for patients who are older or unresponsive to

Table 2. Goals, indications, and descriptions of preventive, reconstructive, and salvage procedures in the correction of hip deformities.[2,34]

	Goals	Indications	Techniques	Success
Preventive	Delay hip subluxation or dislocation	30% ≤ RMI < 60% Limited hip abduction (<30°)	**Soft tissue procedures:** adductor tenotomy, iliopsoas release, semitendinosus lengthening	Dependent on pre-operative severity of hip abnormality. Increased RMI associated with decreased success.
Reconstructive	Maintain or rebuild a located hip with preserved mobility	RMI > 40%	**Bony procedures:** **Femur:** proximal femoral varus derotation osteotomy **Acetabulum:** Osteotomies (Dega, Periacetabular, Pemberton, Pericapsular, Chiari pelvic, Salter innominate)	Positive outcomes after long follow-up. Complications include dislocation recurrence, osteonecrosis of femoral head, heterotopic ossification.
Salvage	Reduce pain or increase abduction to maintain hygiene when hip cannot be reconstructed to be mobile or located anymore	Severe dysplasia and degenerative changes that are beyond the scope of reconstructive surgeries	**Resection:** Castle procedure (remove proximal femur, reattach rectus and vastus lateralis to femoral shaft, redirect gluteal muscles to between femur and acetabulum) **Redirection:** subtrochanteric valgus osteotomy **Arthroplasty:** total hip replacement	Inferior outcomes to reconstructive procedures due to high rates of complications.

nonoperative approaches, and the procedure depends on the severity and degree of involvement of specific muscle groups. Hoke lengthening (percutaneous triple hemi-section tenotomy) can be performed for patients with an Achilles tendon contracture.[2] For equinus deformities that are dynamic and combined with other abnormalities, gastrocnemius-soleus lengthening can be considered as well. Spastic equinovarus can also be approached with the split anterior tibialis tendon transfer (SPLATT) procedure, a rerouting of half of the tibialis anterior, which has demonstrated marked success in correcting the varus part of the deformity. Alternatively, fractional lengthening of the posterior tibialis tendon and transfers of the flexor hallucis longus and flexor digitorum longus may be done for varus correction.[35] In children, intramuscular lengthening of the posterior tibial tendon, Achilles lengthening, and SPLATT or split posterior tibial tendon transfer (SPTTT) anterior to the Achilles tendon into the peroneus brevis tendon are all effective procedures to reduce equinovarus deformities that are flexible. In more rigid deformities, osteotomy of the midfoot/calcaneus or triple arthrodesis (in skeletally mature patients) may be needed.

CONCLUSION

Spasticity results from many neurologic conditions and poses a significant challenge to the independence and quality of life of patients. Depending on the goals of care and patient function, a variety of combinations of nonoperative and operative approaches can be utilized for managing this condition. Orthopedic surgeries that treat spasticity aim to restore mobility, decrease long-term complications, and reduce pain, thereby improving the quality of life for these patients.

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