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CLINICAL GUIDANCE

PM&R

AAPM&R consensus guidance on spasticity assessment and management

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Abstract

Background: The American Academy of Physical Medicine and Rehabilitation (AAPM&R) conducted a comprehensive review in 2021 to identify opportunities for enhancing the care of adult and pediatric patients with spasticity. A technical expert panel (TEP) was convened to develop consensus-based practice recommendations aimed at addressing gaps in spasticity care.

Objective: To develop consensus-based practice recommendations to identify and address gaps in spasticity care.

Methods: The Spasticity TEP engaged in a 16-month virtual meeting process, focusing on formulating search terms, refining research questions, and conducting a structured evidence review. Evidence quality was assessed by the AAPM&R Evidence, Quality and Performance Committee (EQPC), and a modified Delphi process was employed to achieve consensus on recommendation statements and evidence grading. The Strength of Recommendation Taxonomy (SORT) guided the rating of individual studies and the strength of recommendations.

Results: The TEP approved five recommendations for spasticity management and five best practices for assessment and management, with one recommendation unable to be graded due to evidence limitations. Best practices were defined as widely accepted components of care, while recommendations required structured evidence reviews and grading. The consensus guidance statement represents current best practices and evidence-based treatment options, intended for use by PM&R physicians caring for patients with spasticity.

Conclusion: This consensus guidance provides clinicians with practical recommendations for spasticity assessment and management based on the best available evidence and expert opinion. Clinical judgment should be exercised, and recommendations tailored to individual patient needs, preferences, and risk profiles. The accompanying table summarizes the best practice recommendations for spasticity assessment and management, reflecting principles with little controversy in care delivery.

INTRODUCTION

Spasticity is a common disorder encountered by physical medicine and rehabilitation (PM&R) physicians. As a component of the upper motor neuron syndrome, spasticity has numerous etiologies of brain (eg, stroke, cerebral palsy [CP], multiple sclerosis) and spinal (eg, traumatic, inflammatory) origin across the lifespan.[1] Spasticity can negatively affect function (eg, gait, transfers), interfere with positioning, cause pain and discomfort, and increase caregiver burden. However, spasticity can also facilitate improved function such as gait and positioning and assist with transfers.[2] In children, spasticity management may help achieve developmental milestones.

The definition of spasticity has evolved over the years and has been inclusive of neural mechanisms, and more recently, neuroanatomical aspects.[3] In 1980, Lance and colleagues defined spasticity at a consensus symposium as:

> a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex as one component of the upper motor neuron syndrome.[4]

Subsequently, Pandyan et al. attempted to validate Lance et al.'s definition by reviewing the literature and concluded that spasticity is not a pure motor disorder but rather a disorder of sensorimotor control and presents as muscle overactivity. Hence spasticity was redefined as:

> disordered sensorimotor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles.[5]

A more recent definition of spasticity is even broader and specifies a neuroanatomical substrate for the hyperexcitability. By this definition, spasticity is:

> manifested as velocity- and muscle length–dependent increase in resistance to externally imposed muscle stretch. It results from hyperexcitable descending excitatory brainstem pathways and from the resultant exaggerated stretch reflex responses. Other related motor impairments, including abnormal synergies, inappropriate muscle activation, and anomalous muscle coactivation, coexist with spasticity and share similar pathophysiological origins.[6]

Abnormal excitability of the stretch reflex is still the central mechanism as per this latest definition of spasticity. Spasticity may also be defined as a type of hypertonia as part of the upper motor neuron syndrome, which can also manifest as hyperreflexia, clonus, co-contractions, spreading of reflexes, spastic dystonia, associated reactions, motor overflow, and muscle spasms.[7] In this American Academy of Physical Medicine and Rehabilitation (AAPM&R) guidance statement, we provide a condensed review of evidence for the assessment and management of spasticity to offer practical, consensus-based recommendations to guide the management of patients presenting with spasticity. A full review of pathophysiology and pathologic effects is beyond the scope of this paper but is available elsewhere.[3,7,8]

Throughout this consensus guidance, we meticulously reference and integrate insights from previous consensus statements and guidelines. These established frameworks serve as foundational pillars, enriching our independent review and ensuring that our recommendations are rooted in a comprehensive understanding of the existing literature and expert opinions. Our aim in formulating this consensus guidance is to assist PM&R providers in the management of spasticity. In doing so, we sought to review treatment options, endorse the highest quality of evidence-based care, and encourage research where there are knowledge gaps. Multidisciplinary care is a cornerstone of PM&R, and every care team member should continuously measure and analyze outcomes to adjust care pathways to optimize outcomes aligned with patient goals. Beyond patient care, the hope is that a broadened understanding of current patient care practices will help identify areas of future research.

Methodology

In 2021, after the completion of a quality environment review to assess areas of opportunity to improve the care of adult and pediatric patients with spasticity, the AAPM&R convened a technical expert panel (TEP) to conduct an evidence review and develop consensus-based practice recommendations to address gaps in care for patients with spasticity. The Spasticity TEP held virtual meetings over a 16-month period. Early meetings focused on the formulation of search terms and research questions to guide a structured evidence review that was focused on key aspects of spasticity assessment and management that could be synthesized into practical guidance for clinicians with the intention of improving care. To assess the quality of evidence for the assessment and treatment recommendations, the evidence submitted in support of each recommendation was

reviewed and graded by the AAPM&R Evidence, Quality and Performance Committee (EQPC) prior to a consensus vote being conducted to finalize and grade the recommendations by the TEP. The TEP used a modified Delphi process to evaluate the recommendation statements and achieve consensus both on the recommendation language and quality of evidence. As recommendation statements were drafted, an initial consensus assessment survey was conducted online to determine if the majority of the TEP approved the wording of each recommendation statement. This initial assessment was followed by workgroup revisions and further development of evidence review narratives. Finally, through this iterative consensus process each guidance statement section's workgroup presented the evidence used to develop each recommendation for TEP discussion; this was followed by an online poll to assess agreement with recommendation language and classification of the evidence level into grades. To guide consensus achievement and ensure a patient-centric focus, the TEP and the EQPC used the Strength of Recommendation Taxonomy (SORT) to rate the quality of individual studies and the strength of recommendations offered based on a body of evidence.[9] As a result of this consensus process, the TEP approved five recommendations for spasticity management and five best practices covering assessment and management of spasticity and was unable to grade one recommendation. The AAPM&R defines best practices as components of care that are accepted by medical experts as a proper course of action for a certain type of disease, condition, or intervention; there is little to no controversy about the practices; and they can be widely used by health care professionals. Clinical recommendations are distinguished from best practices by requiring a structured review of evidence and an assessment or grading reflecting the quality of the evidence. In the event a recommendation cannot be graded due to limitations of the evidence, the TEP can assign a rating of "NG."

This consensus guidance statement is intended to reflect current practice in patient assessment and treatment options based on best available evidence and expert opinion from PM&R physicians who care for patients with spasticity on a regular basis. The information presented here by the AAPM&R Spasticity TEP should not preclude clinical judgment and must be applied in the context of and tailored to each specific patient, with adjustments for patient preferences, comorbidities, and other factors with consideration of risk profile and tolerance of each assessment and management activity. Table 1 summarizes best practice recommendations for spasticity assessment and management. These best practices reflect assessment principles that the Spasticity TEP noted as having little to no controversy in care delivery.

The following AAPM&R Clinical Recommendations for Spasticity Management (Table 2) are based on a structured evidence review and grading of the evidence to achieve consensus on management approaches. The AAPM&R Spasticity TEP recommends taking into account patient tolerance and risk profile when dealing with the treatment of spasticity. The existing literature lacks consistent and strong evidence concerning the advantages, disadvantages, and functional results of numerous interventions frequently employed in physiatry and rehabilitation practices.

TABLE 1 AAPM&R best practices for spasticity assessment.

AAPM&R Best Practices for Spasticity Assessment and Management
A-1: As a component of the initial patient evaluation, clinicians should assess the impact of spasticity on passive and active movement, ability to repeat movements, and function to guide its treatment/management.
A-2: Reassessment of spasticity should occur throughout the treatment course. Specifically, reassessment should occur before or at the time of each treatment to consider whether to continue the same treatment or to change the course of treatment.
A-3: Standardized measures to evaluate spasticity should be utilized at each evaluation to optimize consistency and to objectively measure response when an intervention is applied.
A-4: Treating spasticity should start with optimizing medical management. Physiatrists should make sure that patients are medically stable and address any medical problems that may exacerbate spasticity.
A-5: To assess the extent to which a patient's goals are being met, a goal attainment scale or other means of measuring treatment response may be considered in each reassessment.

Health equity and access to care

Medical literature discussing health disparities specifically in spasticity is limited; however, a greater body of research exists concerning the limited access to rehabilitation care for certain marginalized groups. Studies in the field of physiatry have emphasized that limited access to rehabilitation services poses a significant obstacle to achieving equitable outcomes. Narrative reviews have demonstrated health disparities across several domains for Black and Hispanic persons,[11,12] In these reviews, patients of color with stroke, traumatic brain injury, and spinal cord injury – all groups who develop spasticity as a sequela – demonstrated pervasive inequities. Furthermore, a study in 2017 found racial, gender, and geographical disparities in access to high-quality neurologic care.[13] Similar data have been found in children and youth with special health care needs, inclusive of children with CP.[14] A more recent retrospective cross-sectional study found disparities in access to spasticity chemodenervation proceduralists in the United States for Medicare patients, especially in nonurban and highly Hispanic

TABLE 2 AAPM&R clinical recommendations for spasticity management.

Management/treatment recommendation statement	SORT Grade[a]
Pharm-1: The AAPM&R Spasticity TEP suggests use of oral medications to manage generalized or systemic spasticity; oral medications can be used either exclusively or as a component of a multimodal treatment approach.	C
INJ-1: The AAPM&R Spasticity TEP recommends clinicians consider use of botulinum toxin A for management of focal upper and lower limb spasticity.	A
INJ-2: The AAPM&R Spasticity TEP suggests that clinicians consider use of phenol or alcohol blocks for management of focal spasticity.	C
SUR-1: The AAPM&R Spasticity TEP recommends use of intrathecal baclofen pump therapy (ITB) as an effective treatment of spinal or cerebral origin spasticity in appropriately identified patients.	A
SUR-2: The AAPM&R TEP recommends utilization of selective dorsal rhizotomy (SDR) to treat spasticity with proper patient selection focused on patients with primarily spasticity of the lower extremity (LE), adequate LE strength and selective motor control, and absence of significant contractures. Technical Note: Historically, the procedure has primarily been performed in children; more recently SDR in adults has been noted to be helpful in reducing spasticity, maintaining or improving level of ambulation, but with a higher propensity to develop new sensory deficits or neuropathic pain.[10]	A
NP-1: The AAPM&R Spasticity TEP recommends consideration of use of nonpharmacologic interventions from a range of treatment modalities, in conjunction with other therapeutic options to effect spasticity and facilitate the effects of pharmacologic and procedural interventions on spasticity and to improve function and decrease deleterious effects of contributing conditions.	NG

[a]The use of "TEP recommends" is based on a SORT evidence grade of A or B; the use of "TEP suggests" is based on a SORT evidence grade of C. "NG" recommendation cannot be graded due to limitations of the evidence.
Abbreviations: AAPM&R, American Academy of Physical Medicine and Rehabilitation; SORT, Strength of Recommendation Taxonomy; TEP, Technical Expert Panel.

communities.[15] Only 566 providers had ≥11 claims for chemodenervation for Medicare patients in 2017, a number that is shockingly low for the number of persons with problematic spasticity.

Not often discussed in this literature is financial disincentives for clinicians to provide this care to patients. Although procedures can offer benefits to patients, there are various factors including reimbursement issues, training requirements, equipment costs, liability concerns, and time constraints that all disincentivize physicians from performing these procedures in their office. The current reimbursement structure for chemodenervation procedures – including procurement of expensive botulinum toxins and not receiving full reimbursement from Medicare in return – along with specialized equipment and supplies needed, office staff for prior authorization/benefits coordination, does not adequately compensate physicians and makes it financially unattractive to do these procedures. It has now been shown that there are reductions in health care use and costs after spasticity management.[16] These data should be used to support the value of using botulinum toxin in treating poststroke spasticity, inclusive of having more incentives for physicians to perform these procedures.

Existing research also reveals that individuals from lower socioeconomic backgrounds and those with insufficient insurance coverage encountered delays in receiving timely stroke interventions.[17] Such treatment delays for acute stroke can have significant consequences on patient outcomes, inclusive of the emergence of spasticity resulting from more severe strokes.[18] This underscores the importance of implementing targeted interventions to overcome socioeconomic and insurance-related barriers that contribute to disparities.

The factors underlying disparities in spasticity care across the care continuum are multifaceted. Therefore, comprehensive and multidisciplinary strategies must be developed to counteract these effects. Interventions aimed at reducing the primary causes of spasticity and its associated conditions may potentially create opportunities for reducing both the occurrence and severity of spasticity. Educating individuals about spasticity symptom recognition and the importance of seeking treatment at the appropriate literacy level could also be a contributing solution.[19] Teaching more spasticity providers to provide treatments, including those in rural areas or resource-limited communities, could further contribute to reducing disparities in spasticity care. Additionally, a more diverse representation of physicians, including those from racial/ethnic minoritized groups or with disabilities, could help bridge health equity gaps of care for patients with spasticity.

One pivotal area regarding spasticity and access to care is the realm of telemedicine and remote monitoring. Recent literature explored telerehabilitation and spasticity evaluation via telemedicine, as well as remote monitoring technologies.[20,21] Such innovations hold tremendous potential, particularly in ensuring consistent follow-up care for individuals with spasticity, inclusive of those who have geographic or accessibility constraints. However, it is essential to recognize that while telemedicine offers promising avenues to expand care, it comes with inherent limitations, such as technological barriers, concerns for quality of remote assessment, and the necessity for robust infrastructure support.[22–25] Despite these challenges, telemedicine can significantly bolster equity and increase access to spasticity-related care.

INITIAL AND FOLLOW-UP ASSESSMENTS TO GUIDE SPASTICITY MANAGEMENT

Multiple assessment methods can be implemented to gather the information needed to guide the spasticity management plan. We recommend consistency in measurement tools beginning with the initial assessment and continuing through ongoing management. As a component of upper motor neuron syndrome, spasticity includes positive and negative signs/symptoms.[26] The positive signs/symptoms include excessive muscle tone, hyperactive stretch reflexes, clonus, and spasms. The negative signs/symptoms include incoordination, fatigue, weakness, and impaired motor control. The coexistence of the positive and negative symptoms exacerbates the functional disability and makes treatment particularly challenging. A comprehensive evaluation of a patient with spasticity should include a patient-centered history and physical examination that incorporates upper and lower limb functional ability, passive range of motion, and active range of motion with repetition of movement.[27] The purpose of the assessment is to set collaborative patient-centered goals for treatment and to evaluate the treatment response in a consistent and repeatable manner. Realistic treatment goal setting for patients (as well as family members and caregivers) is critical because it promotes motivation and cooperation as well as proper management of expectations and can favorably affect outcome.[28] The use of goal attainment scales has been shown to help organize, focus, and clarify the aims of treatment to optimize the clinical response.[29]

Table 3: Key Components for Spasticity Assessment provides an overview of commonly used tools to assess passive and active range of motion, repetitive motion, and upper and lower limb function, to guide treatment and for periodic reassessment and follow-up before and after interventions to treat spasticity. When selecting clinical measures for spasticity assessment, it is important to consider repeatability of the measures, ability to evaluate the potential effects of the treatment in consideration, and the level of impairment and function of the individual.

Quantitative methods such as motion capture using marker-based or markerless technology can now easily be integrated into clinical practice for reliable and repeated measurements.[32,33] In addition, electrophysiologic and biomechanical assessments can be used as adjuncts to clinical measures to quantify the associated abnormal muscle activity.[34–40] Concurrent measurement of biomechanical properties such as angle and torque with electromyography (EMG) can help distinguish

TABLE 3 Key components for spasticity assessment.

Assessment components	Examples of measurement tools	Clinical comments; description
Passive range of motion	Modified Ashworth scale (MAS), Tardieu Scale, goniometer, motion capture using marker-based and markerless technology.	• Assesses soft tissue length and extensibility. During passive range of motion, stiffness can be assessed using the MAS and spasticity may be assessed using the Tardieu Scale. Angle of catch or clonus upon fast passive stretch of the muscle group assessed provides insight on stretch reflex excitability. Restoring passive range of motion is an important treatment goal for many patients.[30]
Active range of motion		• Assesses underlying neurological ability and is a net result of agonist recruitment minus the combined resistance from passive soft tissue stiffness and spastic co-contraction in opposing muscle group. It is important to preserve any active movement ability the patient has.[31]
Rapid alternating movement		• Assesses coordination and fatigue by asking patient to perform a movement as fast as possible and repetitively. A decrease in the range of motion with repetition indicates spasticity-related fatiguability.[31]
Upper limb function	Modified Frenchay Scale, Wolf Motor Function Test; Action Research Arm Test	• Several validated functional task batteries may be used to provide information on functional ability. Patients can be rated based on the time taken to perform the task and the quality of the movement. Alternatively, a specific functional task relevant to the patient, for example, reaching for an object or grasping and releasing a cup, can be video recorded from a consistent angle to assess functional ability before and after the intervention.
Lower limb function	Gait assessment	• A walking test (10 m or 2 min) that challenges the patient as much as possible, should be used consistently to assess speed and compensatory strategies used in ambulatory patients.

between neural (spastic catch associated with increased EMG activity) and nonneural (spastic catch without increase in EMG activity) resistance to stretch on passive movement, which can assist with treatment planning and selection of the appropriate intervention. Gait analysis has been most frequently used to tailor interventions in children with CP. However, a randomized controlled trial that tested the superiority of instrumented gait analysis over usual care on outcomes showed low compliance with recommended interventions and no between-group differences.[41] Regardless of the measurement used, it is recommended that the same measures are used at initial and subsequent evaluations to assess the change in spasticity with the interventions.

Treating spasticity begins with optimizing medical management. The history and physical examination should include assessment of medical conditions that may exacerbate spasticity involving the genitourinary, gastrointestinal, musculoskeletal, vascular, and neurological systems. Successful management of medical exacerbation of spasticity requires an understanding of the underlying pathophysiology, early identification of the triggering factors, and implementation of treatment strategies that are generally known and widely used.[42] The assessments for spasticity can aid in distinguishing between (1) generalized hyperexcitability amenable to intervention with oral spasmolytics and/or intrathecal baclofen, (2) predominantly neural focal muscle overactivity that can be treated with focal neurolysis and/or chemodenervation using botulinum toxins, phenol or alcohol blocks, and (3) predominantly nonneural muscle shortening that can be treated with nonpharmacologic, surgical, and emerging methods.[43]

SPASTICITY MANAGEMENT

Early goal-directed spasticity management is instrumental in helping increase the likelihood of good outcomes and limiting complications[44,45] Unfortunately, a lack of universally standardized management and an abundance of therapeutic options make spasticity management a challenging task.[44] Management of spasticity is aimed at decreasing the intensity, frequency and functional impact of increased tone and prevention of musculoskeletal deformity that may occur as a result of chronic imbalance of muscle forces.[1] An assortment of physical and occupational therapies, oral and intrathecal medications, surgery, and focal denervation are used in the management of spasticity.[1] Although many treatment options are available, and have data to support their use, further research on efficacy is warranted to better understand optimal use strategies.[1]

Management of spasticity involves a range of therapeutic interventions, either used individually or in combination. Although not universally available, nonsurgical options with physiotherapy should be prioritized and used early in spasticity management. It is crucial to recognize the significance of nonpharmacological and nonsurgical treatments with physical and occupational therapeutic techniques as the initial steps in managing spasticity. Even if pharmacological or surgical interventions become necessary, a combination of therapeutic modalities within an interdisciplinary rehabilitation approach is advised, discouraging standalone approaches. Physiotherapy plays a central role, encompassing early intervention, maintenance of muscle length, joint alignment, prevention of complications, and task-specific training. Despite its integral role, there is a lack of robust clinical trial data supporting the role of physiotherapy in affecting spasticity, emphasizing the need for further research and evidence-based practices in spasticity management. An overview of systematic reviews of nonpharmacologic interventions for spasticity in adults was published in 2019 and in children in 2020.[46,47] Even with 18 systematic reviews for a range of nonpharmacologic interventions used in spasticity management, there was a lack of high-quality evidence for many modalities. Much of the evidence was moderate to low quality for rehabilitation programs, again, underscoring the importance of further research. Ultimately, a rehabilitation program should be patient centric and goal specific, employing multimodal treatments with both generalized and focal interventions.

The following sections review therapeutic options commonly used in patients with spasticity.

Oral medication options for spasticity management

Oral medications recommendation

Pharm-1: The AAPM&R Spasticity TEP suggests use of oral medications to manage generalized or systemic spasticity; oral medications can be used either exclusively or in combination, as a component of a multimodal treatment approach. (SORT C)

The pharmacologic management of spasticity is an integral component of the care of many neurologically impaired patients. Before treatment is initiated, the physician needs to assess the effects of spasticity and determine if the adverse effects outweigh any benefits of the spasticity.[48] The wide variety of oral medication options for treatment from which a practitioner may select are reviewed in the following narrative as well as in Table 4: Oral Medications for the Treatment of Spasticity. No medication is uniformly useful in the treatment of hypertonia. All drugs have potentially serious side effects and their negative effects should be weighed when prescribing the medication.[49–52]

Oral medications such as baclofen, diazepam, tizanidine, and dantrolene are common management options to consider in treating spasticity in children and

TABLE 4 Most commonly used oral medications in spasticity treatment.

Medication	Dosing information	Side effects and contraindication notes	Additional information
Baclofen References:[48–56]	Children: roughly based on body weight, and a typical starting dose is 2.5–5 mg/day, titrated up every few days to a maximum dose of 20 to 60 mg/day. Adults: Dosage begins approximately 5 mg orally two to three times a day, may be slowly titrated to recommended maximum dose of 80 mg/day; however, higher doses are well tolerated and can be therapeutic	• Drowsiness/sedation • Weakness • Constipation • Confusion • Hypotonia and ataxia • Sudden withdrawal of the drug may lead to seizures and hallucinations. • Adverse effects that are intolerable may preclude increasing the dose of baclofen to the desired level to achieve maximum reduction in spasticity.	• A GABA-B agonist is adept at reducing spasticity regardless of the source. It does not cross the blood–brain barrier easily as it is not very lipophilic and it may make it challenging to achieve a desired concentration at the site of action without titrating the dose up quite a bit. • The use of oral baclofen in neonates with spasticity is not well documented.
Diazepam References:[49, 50, 57–63]	Children: usual dose is 0.2 to 0.8 mg/kg of body weight per 24 h divided every 6 to 8 h. Adults: Recommended oral dose is starting with 2 mg twice a day and can be titrated up to 60 mg a day in divided doses. IV onset of action within 1 to 3 min, and oral dosing onset ranges between 15 and 60 min. Diazepam is long lasting with a duration of action of more than 12 h.	• Somnolence • Impaired memory/cognition • Respiratory depression with higher doses • Physical and psychological dependence • Hypotension • Blurred vision • Nausea and vomiting • Constipation • Contraindicated for patients with glaucoma, coma, and respiratory disorders. • Readily crosses the placental barrier and use during pregnancy may result in neonatal withdrawal soon after birth. • Elderly patients tend to have decreased renal function and clearing capability; therefore, this population is at an increased risk of diazepam accumulation and its major metabolites. • In long-term use and abrupt cessation, there is potential for hallucinations and epileptic seizures to occur.[60–62]	• A GABA-A agonist which facilitates postsynaptic effects of GABA resulting in increased presynaptic inhibition. • Careful dosing and monitoring is important due to potential for dependence and implications of withdrawal.
Tizanidine References:[50, 52, 64–71]	Children: initiate with 1 mg/day in 18 mo-7 y old children, 2 mg/day in 7–12 y old children as initial doses, and for those older than 12 y similar dosing to that in adults. Adults: Tizanidine is administrated orally as 2, 4, and 6 mg capsules or as 2 and 4 mg tablets. Dosage starts with 2 mg orally and may repeat every 6 to 8 h as needed. The dosage may gradually increase by 2 to 4 mg per dose of 1 to 4 days in between until there is a noticeably significant reduction of spasticity. Maximum dosing is three doses every 24 h, up to 36 mg daily.	• Sedation/drowsiness • Dizziness • Constipation • Hallucinations • Nervousness • Dyskinesia • Severe adverse reactions that are possible but extremely rare: anaphylaxis, severe hypotension, bradycardia, hepatotoxicity, and Stevens–Johnson syndrome.	• Alpha-2 agonist; use results in both direct impairment of excitatory amino acid release from spinal interneurons and a concomitant inhibition of facilitatory cerebrospinal pathways. • Monitoring of creatinine, liver functions, and blood pressure is necessary in patients with high-dose and prolonged therapy. • Extent of absorption is greater when taken with food. The tablet and capsule dosage forms are not bioequivalent when administered with food. • Recommendation is to taper the dose 2 to 4 mg per day to reduce the risk of tachycardia, rebound hypertension, and increased spasticity.

(Continues)

TABLE 4 (Continued)

Medication	Dosing information	Side effects and contraindication notes	Additional information
Dantrolene References:[49,50,72–75]	Children: starting dose of 0.5 mg/kg of body weight orally once a day and increasing to 12 mg/kg of body weight or 400 mg per day in divided doses. Adults: Dosage begins 25 mg 1–2 times a day and slowly titrated up to 400 mg/day with divided doses.	• Hepatotoxicity • Weakness • Fatigue • Drowsiness • Dizziness	• Dantrolene sodium is a postsynaptic muscle relaxant that lessens excitation-contraction coupling in muscle cells. It acts directly at the skeletal muscle to decrease contractility by inhibiting post synaptic release of calcium at the sarcoplasmic reticulum. • Dantrolene carries a black box warning for hepatotoxicity, therefore liver enzymes should be monitored. Although this has not been reported in children <16 years of age, routine liver function tests should be monitored.

Abbreviations: GABA, gamma amino butyric acid; IV, intravenous.

adults. Though they help decrease spasticity, this does not always translate into significant functional improvement, and sedation as a side effect may preclude use in patients. In addition, clonidine, cyproheptadine, ketamine and gabapentin have shown efficacy; however, more studies are required to confirm their place in therapy.[50,52] Only diazepam and oral dantrolene are approved by the U.S. Food and Drug Administration for children older than 5 years of age[53]; However, it may be used off label in younger children.

Baclofen

Baclofen is a gamma amino butyric acid (GABA)-B agonist and is the most commonly used drug for spasticity of spinal origin. The role of oral baclofen in the treatment of cerebral forms of spasticity remains questionable due to its interference with attention and memory in brain-injured patients. Otherwise, it has been shown to be safe and effective in long-term use.[49] Additionally, baclofen may improve bladder control by decreasing hyper reflexive contraction of the external urethral sphincter.

There are no data available about the outcomes on quality of life in patients treated with oral baclofen.[76] Case reports on baclofen use in two term infants showed improved tone and easier handling initially and better tracking and overall interactions at 4 months.[77] No adverse effects were noted in either study.

Diazepam

Diazepam acts on the central nervous system by stimulating GABA that inhibits the excitatory stimulation in the brain. It suppresses the activity of the brain and suppresses convulsions. It potentiates the inhibitory neurotransmitters in the limbic system and thereby decreases emotions and anxiety.[52,58,78] Diazepam is one of the oldest oral medications used to treat spasticity in children.[75] It has been used successfully to treat spinal hypertonia; however, it is not the drug of choice for patients with brain injuries due to cognitive side effects. Sudden stoppage of the drug is not recommended as it can cause withdrawal symptoms.[79,80]

In the United States, diazepam is a Schedule IV controlled substance with the potential for abuse. As a controlled substance, caution should be used in prescribing with special attention paid to preventing dependence and introducing tolerance with long-term treatment. Once an individual develops dependence, the risk of developing withdrawal symptoms increases. Signs of benzodiazepine withdrawal include tremor, rebound anxiety, perceptual disturbances, dysphoria, psychosis, agitation, irritability, restlessness, sweating, headache, confusion, myalgias, abdominal pain, and vomiting.

Tizanidine

Tizanidine is an alpha 2 agonist and has been shown to decrease spasticity.[58,64,66,75,78] Currently there is level C evidence for use in children, and an overall safety rating of good.[75] Despite its structural and biochemical similarity to clonidine, the cardiovascular properties of tizanidine are mild and transitory in relation to its activity as a muscle relaxant. These findings, together with a possible greater separation between myotonolytic and general central nervous system depressant activity than with other agents, make

tizanidine a valuable addition in the pharmacologic treatment of spasticity.

Tizanidine is generally well tolerated. However, reports exist of potential adverse effects on several organs such as cutaneous, gastrointestinal, neurologic, cardiovascular, endocrine, and respiratory systems.[68–70,81] Concomitant use of tizanidine with fluvoxamine or ciprofloxacin is contraindicated due to significant hypotension and increased psychomotor impairment.

Dantrolene

Dantrolene is the only medication that acts at the muscular level. Though its primary indication is for treatment of malignant hyperthermia, it is also used to treat spasticity. It can be less sedating than diazepam or baclofen[82]; however, adverse effects that should be considered are muscle weakness including diaphragmatic and accessory respiratory muscle weakness.[83]

The stability of dantrolene in a suspension form is short, creating significant administration problems in anyone who cannot swallow capsules. Dantrolene capsules can be emptied into food, but dosing is unreliable via this method if the dose is not a full capsule. This drug typically needs to be administered four times a day. Compliance can therefore be an issue.

Cannabinoids

Cannabinoids or cannabis are compounds found in the cannabis plant that have been used for their potential therapeutic effects on spasticity. They can be given orally, sublingually, or topically. Most studies to date have been done in persons with multiple sclerosis[84,85] Cannabinoids have muscle relaxant properties and may help reduce muscle stiffness and spasms associated with spasticity. There are also anti-inflammatory properties, as well as analgesic effects, which may contribute to an overall improvement in the symptoms associated with spasticity. A systematic review and meta-analysis published in the *Journal of the American Medical Association* (*JAMA*) found moderate-quality evidence for the treatment of chronic pain and spasticity[86] More research is needed to fully understand their mechanisms and effectiveness and risks.

Injectables

Injectables recommendations

- INJ-1: The AAPM&R Spasticity TEP recommends clinicians consider use of botulinum toxin A for management of focal upper and lower limb spasticity. (SORT A)
- INJ-2: The AAPM&R Spasticity TEP suggests that clinicians consider use of phenol or alcohol blocks for management of focal spasticity. (SORT C)

Chemodenervation and neurolysis via localized injections can help provide focal spasticity relief.[44,80,87] Medications used in chemodenervation and neurolysis procedures include botulinum neurotoxin (BoNT), phenol, and alcohol.[80,87–90]

In the United States, health insurance limitations often affect the choice of therapeutic options. For example, BoNT injections are associated with significant costs, and repeated injections and dosage are often further restricted by finances for coverage of treatment. These limitations prevent the sole utility of chemodenervation for a multipattern treatment, for example, elbow flexion, clenched fist, stiff knee gait, and equinovarus of the foot. Consequently, phenol neurolysis (PN) and BoNT can be used in complement, with PN frequently reserved for proximal nerves and BoNT used for distal musculature.[91]

The following sections review considerations for use of BoNT and PN in spasticity management.

Botulinum toxin injections

A search of the published medical literature was conducted regarding spasticity treatment with botulinum toxins inclusive of upper and lower extremity spasticity treatment with abobotulinum toxin A, incobotulinumtoxinA, and onabotulinumtoxinA. This literature review also included search terms to understand current evidence for the use of botulinum toxins in the pediatric population. Intramuscular injections with botulinum toxin A are commonly used in the upper limbs of children with CP to manage pain, reduce caregiver burden, and improve function.[92] Intramuscular botulinum toxin A has been used in the upper limbs of children with CP to manage preoperative and postoperative pain, facilitate nursing, and achieve functional and/or cosmetic improvement of hand position.[92]

In 2008, with an update in 2016, the American Academy of Neurology published guidelines on the uses of botulinum neurotoxin for the treatment of conditions associated with movement disorders, inclusive of adult spasticity.[93] The guidelines found strong Level A evidence for treatment of upper extremity spasticity with abobotulinumtoxinA, incobotulinumtoxinA, and onabotulinumtoxinA. There was also strong Level A evidence for onabotulinumtoxinA and abobotulinumtoxinA for lower limb spasticity.

Four meta-analyses have been published regarding the use of botulinum toxin for the treatment of upper limb spasticity.[94–97] In the most recent study by Jia et al., a meta-analysis was conducted to evaluate the

efficacy of BoNT-A for the treatment of upper limb spasticity after stroke.[96] A total of 10 randomized, controlled studies evaluating multiple BoNT-A products from different manufacturers were included in the analysis. Participants receiving BoNT-A had a statistically significant reduction in finger Modified Ashworth Scale (MAS) score compared with those receiving placebo ($p = .008$). There was no statistically significant difference between groups in wrist, ($p = .42$), pain ($p = .61$), or Barthel index scores ($p = .87$). In the BoNT-A type subgroup analysis comparing the efficacy of Botox or Dysport to placebo, elbow MAS was more improved in the Dysport-treated participants ($p = .008$) compared with Botox-treated participants ($p = .84$).

Two systematic reviews[92,98] and two meta-analyses have been published regarding the use of botulinum toxins for the treatment of lower limb spasticity. In the most recent meta-analysis, the effectiveness of botulinum toxin on spasticity and gait of hemiplegic participants after stroke was evaluated.[97,99] A total of 12 of the 21 studies were included in the meta-analysis portion of the study. Following botulinum toxin injections, the MAS score was significantly improved (Hedges' g: −1.17; 95% confidence interval [CI]: −1.66 to −0.67; $p < .001$). The same applied for the 10 Meter Walk Test (−0.35; 95% CI: −0.68 to −0.02; $p = .016$). Adverse events consisted of mild injection-site pain, myalgia, weakness, or erythema. In children, evidence reviews have concluded there is some evidence to support the use of botulinum toxin A as an adjuvant treatment to other physical therapy (PT) regimens or placebo to reduce spasticity in children with CP in the short term.[99]

Given that use of botulinum toxin is individualized for patient treatment goals, there is real-world registry data that has been published. The Adult Spasticity International Registry (ASPIRE) study was a 2-year, multicenter, prospective, observational registry of patients receiving onabotulinumtoxinA treatment for lower limb spasticity that found doses ranged between 10 and 1100 units across all presentations. Of note was clinician and patient satisfaction/extreme satisfaction that treatment managed their spasticity.[100]

It is important to know there is lack of interchangeability between botulinum toxin products. Each preparation has a specific biological activity and dosing should be followed based on studies for that specific botulinum toxin.

Nerve blocks with phenol or alcohol

Phenol and alcohol nerve blocks can be used to treat spasticity, as an adjunct to oral medications and botulinum toxin injections, or as a standalone procedure. Phenol and ethyl alcohol denature protein resulting in a neuropraxic injury and may lead to subsequent Wallerian degeneration of the targeted nerve. The effects of these agents can also result in fibrosis, local vascular injury, or muscle necrosis due to nonspecific protein denaturation. Higher concentrations increase the risk of these side effects and there are reports of profound muscle necrosis with concentrations >75% of ethyl alcohol. Phenol is commonly injected at a concentration between 3% and 6% and ethyl alcohol at 40%–50%. Judicious application of the agent, skilled provider, and proper electrical stimulation guidance minimize these possible side effects. Phenol and ethyl alcohol have an average duration of effect of 3–9 months.

These agents are most commonly applied to isolated motor nerves or motor branches to minimize the risk of exposure to a sensory nerve that would result in painful dysesthesias. Common motor nerves that are candidates for neurolysis are the musculocutaneous nerve to the elbow flexors,[101–107] obturator nerve to the hip adductors,[108–119] and thoracodorsal nerve to the latissimus dorsi. Motor branches can also be targeted more distally where separated from sensory nerve fibers. Common sites are a motor branch from the sciatic nerve to the medial hamstrings, tibial nerve to the gastrocnemius,[120–130] radial nerve to the brachioradialis, or femoral nerve to the quadriceps.[131] The clinician should use electrical stimulation guidance to carefully localize the motor nerve. Precise localization reduces the volume needed for a successful block and minimizes potential side effects. Ideally, strong muscle contraction should be obtained at stimulation levels of <1 mA. Volumes vary but are commonly <1–2 mL per site. There is some evidence to suggest that use of ultrasound to aid in localization of the nerve provides an additional benefit for reducing overall volume of phenol.[103]

Numerous studies demonstrate the effectiveness of phenol and alcohol nerve blocks dating back to the 1960s. Specifically, phenol has been studied for use in treating spasticity that has occurred from stroke, spinal cord injury, brain injury, and CP. After musculocutaneous nerve blocks, decreased elbow flexor spasticity was noted immediately[101,103,104,106] and lasted as long as 6 months.[105] After obturator nerve blocks, decreased hip adductor spasticity was noted immediately[109,111,116] and lasted as long as 6 months after alcohol blocks[132] and 9 months after phenol blocks.[112] Obturator nerve blocks were associated with improved hygiene,[111,112] caregiver burden,[112] and gait on a four-point scale[111] and with a wider base of support.[113] Subscapular nerve blocks in a cohort with spastic hemiplegia were noted to improve range of motion and pain.[133] For the spastic ankle, tibial nerve blocks can improve ankle clonus, gait parameters, and range of motion.[127–129]

There are numerous references documenting the use of phenol or ethyl alcohol for motor nerve blocks in children with spasticity, primarily those with CP.[134–136] The most common injection sites for children are the obturator nerve, musculocutaneous nerve, and motor

branches of the sciatic nerve to the medial hamstring. Other motor nerves may be targeted as well. There has been little research comparing use of phenol to botulinum toxins or other spasticity management techniques in children. One study, by Wong et al., reported inferior effects on gait compared to botulinum toxins.[137] Two studies have demonstrated the effectiveness of combining phenol neurolysis with botulinum toxin injections. These authors note the benefit in children of being able to address more spastic muscles simultaneously, often a challenge due to the necessity of weight-based dosing.[88,138] A few additional studies noted similar effectiveness and side effect profile; earlier studies noted more adverse reactions that may be related to dosage and localization techniques.[139–141] The maximum safe dose is 30 mg/kg and <1 g for one treatment session.[136] General anesthesia or sedation is typically required for use in children due to the increased time needed for nerve localization and pain during the procedure.

Adverse effects of phenol nerve blocks primarily occur with higher doses of phenol, when phenol inadvertently spreads to adjacent nerves or when phenol was injected onto motor nerves with sensory fibers. Rates of dysesthesias following phenol or alcohol nerve blocks are estimated between 0% and 30%. A few studies of tibial nerve blocks report rates of dysesthesias around 10%.[124,126,129] There is also a report of unintentional foot drop from proximal tibial nerve blocks where phenol was thought to spread to the peroneal nerve. Morrison et al. monitored plasma phenol concentrations during phenol injections for spasticity and noted three episodes of mild dysrhythmias at higher plasma concentrations (>20 mcg/mL).[142]

Surgical options

Recommendation statements

- SUR-1: The AAPM&R Spasticity TEP recommends use of intrathecal baclofen pump therapy as an effective treatment of spinal or cerebral origin spasticity in appropriately identified patients. (SORT A)
- SUR-2: The AAPM&R TEP recommends use of selective dorsal rhizotomy (SDR) to treat spasticity with proper patient selection focused on patients with primarily spasticity of the lower extremity (LE), adequate LE strength and selective motor control, and absence of significant contractures. (SORT A)
  - Technical Note: Historically, the procedure has primarily been performed in children; more recently SDR in adults it has been noted to be helpful in reducing spasticity and maintaining or improving level of ambulation, but with a higher propensity to result in new sensory deficits or neuropathic pain.[10]

Intrathecal baclofen pump therapy

Enteral medications used to treat global or regional spasticity can have negative side effects on arousal and cognition due to systemic delivery. These treatment options and dosing can be further limited by potential interactions with medications used to treat common comorbidities, especially pain and/or seizures. Intrathecal baclofen therapy has been established to be effective for chronic spasticity of spinal (traumatic spinal cord injury and multiple sclerosis) or cerebral origin (acquired brain injury, CP, and stroke).[143–156] Bypassing the blood–brain barrier allows enhanced distribution to GABA receptors in the spinal cord and daily dosing in micrograms far less than the equivalent oral/parenteral baclofen dose with less potential for systemic effects and drug interactions. Although often relegated as an invasive intervention of "last resort," intrathecal drug delivery can be useful for reducing spasticity, optimizing patient functionality, and minimizing the use of systemic medications in appropriately selected patients relatively early during their treatment. Current intrathecal pump technology is also programmable with a handheld device to allow customized dosing throughout the day in accordance with timing of patient's symptoms and their daily routine.

Each patient should have previously trialed enteral baclofen and possibly other oral agents and either had an inadequate response or intolerable adverse effects with uptitration.[157] This also ensures they do not have a hypersensitivity to baclofen. Current guidelines recommend waiting for at least 1 year following brain or spinal cord injury before consideration of pump implant, though this does not always apply, and early placement has been shown to improve dysautonomia in select patients.[158]

An intrathecal baclofen trial is recommended to identify appropriate candidates and screen for adverse reactions prior to consenting for implantation although this may not be practical in some cases. Intrathecal baclofen trial can be performed with an intrathecal bolus or a catheter infusion. For intrathecal bolus trial, typically a 50-μg dose of baclofen is injected into the cerebrospinal fluid via lumbar puncture.[159] A 25-μg dose can be used in pediatric patients, patients who are prone to developing hypotonia with medications, or those who rely on hypertonicity for standing and ambulation. A trial can be repeated with higher doses up to 100 μg if the initial trial was not deemed successful. Peak effect is observed approximately 4 hours after injection with a typical total duration of effect of 8–10 hours. A catheter infusion trial over several days can also be considered and offers the advantage of having the ability to control catheter tip placement and more closely mimics the effects of continuous infusion with implantation.[160] However, the technical skill and risk of complications is increased with this trialing approach. Ideally, both types of trials should be performed under fluoroscopic guidance to ensure

placement of needle and/or catheter into intrathecal space on first attempt and prevent multiple dural punctures.[159]

Candidates for intrathecal therapy are typically referred to a surgeon for implantation following successful trial. Contraindications to implantation of an intrathecal infusion system include presence of infection (eg, meningitis, ventriculitis, cellulitis, or bacteremia), if the pump cannot be implanted 2.5 cm (1 inch) or less from the surface of the skin, insufficient body mass to accept pump bulk and weight, or spinal anomalies that complicate placement of the catheter. It is good practice to communicate to the implanting surgeon the recommended pump size as well as the intrathecal baclofen concentration and starting dose. The catheter is typically inserted into the intrathecal space at the L3–4 level and is advanced cephalad to approximately mid to lower thoracic levels. In some instances, it may be advanced higher in an attempt to have more effect on spasticity in the upper extremities but there is limited evidence regarding benefits and risks to this approach.[161,162] Following the implantation and titration phase of intrathecal therapy, maintenance consists of periodic refilling of the pump reservoir with new medication, troubleshooting any system malfunctions, and regularly replacing the pump prior to end of battery life. Physicians, patients, and/or caregivers should monitor for potential complications including infection, pocket refills due to refilling error, over/underdosing due to programming errors, motor stalls, and catheter malfunction. There are currently two programmable intrathecal pumps available on the market (Medtronic Synchromed II and Flowonix Prometra II). Guidelines for undergoing magnetic resonance imaging are different for each and should be reviewed with the patient and/or their caregiver.[163,164]

Intrathecal baclofen therapy has been shown to be effective for global or regional spasticity management in adult and pediatric patients with spinal cord injury, multiple sclerosis, acquired brain injury, CP, and stroke. It has been shown to be more effective than oral baclofen in reducing spasm frequency.[165] Patients with hereditary spastic paraparesis, stiff person syndrome, and other less common causes of spasticity and muscle overactivity may also benefit from intrathecal therapy.[165–169] This therapy can be used in conjunction with other enteral antispasticity medications as well as more local interventions such as botulinum toxin injections, neurolysis, and bracing to address focal issues to help optimize patient's function and quality of life. The evidence is limited regarding combination intrathecal therapy but reduction in noxious stimuli such as pain with intrathecal pain medications may further enhance spasticity mitigation in select patients.

Selective dorsal rhizotomy

SDR is an irreversible spasticity reducing procedure, hence careful patient selection is of paramount importance. General criteria used for patient selection include prematurity, diplegia CP, Gross Motor Function Classification System (GMFCS) I to III, age between 4 and 10 years, pure spasticity, antigravity hip flexor strength, adequate cognition, absence of contractures, and periventricular leukomalacia with no basal ganglia or thalamic lesions on imaging.[170]

The surgical technique involves the dorsal roots at the L1–S1/S2 level being accessed either through a 1–2 level laminectomy at the conus medullaris level (park) or a multilevel laminectomy at the cauda equina level.[171] These roots are divided into rootlets and then electrically stimulated. The resultant motor or reflex response of the LE muscles is monitored by EMG and palpation. An electrostimulation response is considered abnormal if there is a sustained motor response or if a muscle not typically innervated at that root level responds. A rootlet with a single-twitch motor response is considered normal. If an abnormal response is seen, then that rootlet is cut. The premise with this selective technique is that only the sensory fibers feeding into the patient's spasticity will be cut, thus reducing spasticity but preserving sensation.[171]

The percentage of dorsal rootlets cut varies between surgeons and typically ranges between 25% and 40%,[170] although some centers are known to transect up to 75% rootlets.[172] Following a rhizotomy, the change in tone creates an opportunity to change motor patterning. This requires postoperative PT for several weeks slowly tapering the intensity of therapy over several months.[173] The initial intensity of PT varies among centers from early aggressive outpatient PT 2×/wk to 6 weeks of intensive inpatient PT.[174,175]

SDR is effective in reducing spasticity, with reduction in spasticity noted as far out as 17 years after surgery.[176] This reduction in spasticity could be assigned to the natural history wherein there is a reduction in spasticity from ages 4 to 12,[177] but children 10 years post SDR had normalized their MAS scores to a 1 in all muscle groups compared to those without an SDR who had only partial reduction in spasticity. These findings imply that reduction in spasticity could be due to SDR rather than natural history alone.[178] Children with diplegic CP, GMFCS I to III seem to have reduction in spasticity maintained through early adulthood.[174,176,179]

Following an SDR, lower extremity deep tendon reflexes and clonus are usually permanently ablated.[180] Absence of the patellar reflex can lead to patients having an increase in falls from inadvertent knee flexion in stance because of the absence of the normal knee extension reflex when the knee is suddenly flexed.[181] Patients usually adapt to these issues over time.

Hamstring contractures can be particularly troublesome after a rhizotomy and could adversely affect gait.[172] Following a rhizotomy, recommendations are for patients with knee flexion contractures to wear knee immobilizers at night through their adolescent growth spurt.[170,176]

Established functional outcomes of SDR are limited by small sample sizes, heterogeneous outcome measures, and lack of control groups. SDR may help increase Gross Motor Function Measure,[182–184] prevent loss of function,[183,184] and increase independence,[182,183]. Although an improvement in quality-of-life measures may be present at 24 months post surgery,[185] these improvements may not persist 10 years post surgery.[178]

SDR is a safe procedure and long-term complications are generally rare. The most common transient issues seen include dysesthesias, urinary retention, numbness, infections,[186,187] and cerebral spinal fluid leak.[188] Dysesthesia risk increases with an increased number of rootlets dissected but usually lasts only several weeks and responds to gabapentin.[189] In the early stages of rehabilitation, nocturnal leg spasms can occur but usually respond to diazepam.[190]

SDR in adult patients with CP helps reduce spasticity and maintain or improve level of ambulation, but adults have a higher propensity to develop new sensory deficits or neuropathic pain.[10]

Additional surgical options

A nonselective ventral dorsal rhizotomy is another surgical option for managing spasticity and dystonia in children GMFCS IV or V with significant hypertonia affecting their comfort, care, and positioning.[191,192] The roots are accessed similar to an SDR, either through a 1–2 level laminectomy at the conus medullaris level or a multilevel laminectomy at the cauda equina level. Both the sensory or dorsal and motor or ventral rootlets typically are cut from L1 to S2 in a nonselective fashion.[192] The percentage of rootlets cut averages 50% to 80%.[191,192] Because the ventral rootlets are also cut, there usually is a reduction in spasticity and dystonia, but some dystonia can return after 1 to 2 years. Ventral dorsal rhizotomies can overly weaken the legs, which can decrease standing ability.[191,192]

Additional surgical options exist and may be used in treating and managing spasticity.[136,193] We mention examples here; however, a full review of evidence is beyond the scope of this guidance statement.

- Correction of bony deformity or soft tissue contracture: Can result in decreased pain and improved biomechanics.[194]
- Tendon transfer: Redirection of the muscle vectors to promote a more balanced limb.[195,196]
- Selective peripheral neurotomy/neurectomy: surgically sectioning of nerve branches to spastic muscles decreasing ability of the peripheral nerve to stimulate muscle contraction.[197,198]

Nonpharmacologic options to manage spasticity

Recommendation statement

- NP-1: The AAPM&R Spasticity TEP recommends consideration of use of nonpharmacologic interventions from a range of treatment modalities, in conjunction with other therapeutic options to effect spasticity and facilitate the effects of pharmacologic and procedural interventions on spasticity and to improve function and decrease deleterious effects of contributing conditions. (NG)

Nonpharmacologic interventions are considered a mainstay in the care of patients with spasticity due to their role in improving function, avoiding secondary impact of spasticity and augmenting the response to other interventions. There are numerous nonpharmacological interventions that have been suggested for spasticity management; however, the evidence supporting or refuting each intervention has not been comprehensively delineated and these interventions are not considered definitive treatment. Studies on efficacy of nonpharmacologic therapeutic options for spasticity have primarily been focused on improvement in function and not on impact on tone nor on optimal dosing (eg, frequency and direction) nor on required intensity. Although a change in spasticity may have occurred concurrent with the improvement in function, it is not a commonly measured outcome. However, there is evidence that including direct therapy care without investigating specific interventions for patients with spasticity can have a positive impact on spasticity as well as function.[187,199–203]

The AAPM&R Spasticity TEP advises that management of spasticity be tailored to each patient based on the consideration of risk profile and tolerance of each intervention. When considering an intervention approach, it is important to understand potential barriers introduced by the intervention as well as those that are unique to patient demographics and condition severity. This is especially true for the nonpharmacologic interventions, for example:

- Many flexibility and strength programs require skilled education to administer and may require equipment which could impose access to care issues.
- Patients with impaired mobility and motor control may require assistance to perform flexibility and strength and conditioning programs.

The evidence review for this guidance statement was focused on the effect of interventions on spasticity and change in spasticity as an outcome. A systematic review of systematic reviews focused on nonpharmacologic intervention options for spasticity noted that

TABLE 5 Nonpharmacologic interventions used in spasticity management.

Improving flexibility Intended effect on spasticity: Improve the viscoelastic properties of musculotendinous unit to allow greater range prior to triggering spasticity. The level of efficacy for any specific intervention to have an effect on spasticity is dependent on whether or not it improves flexibility.	
Active and passive nonsurgical technique examples: passive, active, functional, and positional stretching including static and dynamic bracing (orthotics) and serial casting.	• Three systematic reviews found inconclusive results with no evidence for short- or long-term effects on spasticity in different neurological cohorts.[46,203–205] • In patients with cerebral palsy, evidence that stretching alone improves spasticity is limited, and sustained stretching using a brace or serial casting is probably more effective.[206] • A meta-analysis on effectiveness of static stretching positioning on poststroke upper-limb spasticity and mobility revealed very low-quality evidence that static stretching with positioning orthoses reduces wrist flexion spasticity after stroke as compared with no therapy.[207] • Furthermore, we found low-quality evidence that static stretching by simple positioning is not better than conventional physiotherapy for preventing loss of mobility in the shoulder and wrist.[207] • There is some evidence if pairing with focal tone management injections that delaying cast application can result in decreased treatment time to effect on flexibility.[208,209] • Overstretching can cause pain and in extreme cases can negatively affect orthopedic integrity.[209–211]
Improving strength and endurance Intended effect on spasticity: Spastic muscles are weaker and stiffer and result in functional limitations including mobility. Improving strength and endurance is intended to counter decreased activity, which can lead to impairment of the viscoelastic properties of musculotendinous unit triggering a stretch reflex in a shorter range.	
Strength and endurance therapy examples: Exercise programs to improve movement, to improve strength and endurance, and targeted strengthening activities in therapy (eg, in direct therapies, home program activities, community activities, and adaptive sports)	• A Cochrane review notes low-level evidence for physical activity programs used in isolation or in combination with other interventions (pharmacological or nonpharmacological) in improving spasticity in adults with multiple sclerosis.[212] • Prior concerns that ‘‘overstrengthening’’ spastic muscles would worsen spasticity but this has not been found to be true.[213,214]
Improving motor learning, proficiency, and control Intended effect on spasticity: These techniques are thought to change access to movement patterns by effecting the central nervous system and impacting neuroplasticity by reorganization of the motor cortex.	
Motor learning, proficiency, and control examples: Direct therapy for task specific training, gait training, constraint-induced movement therapy, bimanual therapy, mirror therapy, biofeedback, robotic therapy, and virtual reality training	• Key messages from Cochrane Review on constraint-induced movement therapy (CIMT) in the treatment of the upper limb in children with unilateral cerebral palsy: CIMT may work better than another upper-limb therapy carried out at low intensity (low dose) for improving children’s ability to use both hands together. CIMT appears no more effective than another upper-limb therapy carried out at a high dose or equal dose. CIMT appears to be safe. More well-designed research is needed for strong conclusions to be made.[215] • Studies have demonstrated the clinical effect of Kinesio taping on hand spasticity reduction in patients with stroke, providing that Kinesio taping allows the stretching of the muscles and normalizes muscle tone, thereby allowing sensory feedback between the central and peripheral nervous systems.[216,217] • Studies suggest that biofeedback combined with standard physiotherapy could produce improvements in muscle strength, functional recovery, and gait quality compared to standard physiotherapy alone.[218]
Decrease discomfort Intended effect on spasticity: modalities that may be used for direct effects on peripheral nerves, muscle spindles, and/or to facilitate motor control or strength or improve comfort. These interventions are often used as an adjunct to direct therapy or as part of a home program.	
Electric stimulation therapies: Examples: Neuromuscular electrical stimulation (NMES), transcutaneous electrical nerve stimulation (TENS), and cyclic functional electrical stimulation (FES Bike)	• Physical modalities such as NMES applied to spastic muscles may be reasonable to improve spasticity temporarily as an adjunct to rehabilitation therapy.[219] • When combined with other interventions exhibited significant reduction in spasticity and increase in range of motion in persons with stroke.[220]

TABLE 5 (Continued)

	• In a study investigating effectiveness of electrical stimulation as an adjunct to BoNT-A in reducing spasticity in adults, electrical stimulation reduced spasticity and may boost action of BoNT-A therapy.[221] • Another review summarized the effect of TENS for management of limb spasticity. Although "some" evidence for TENS in improving spasticity was reported, evidence was insufficient to support TENS as an adjunct therapy to active therapies (such as BoNT-A, physical therapy, etc.)[221–223] • Electrical stimulation (e stim) can have direct effect on peripheral nerve function by restoration of postsynaptic depression -presynaptic inhibition to spastic muscles, Renshaw cell inhibition by depolarizing alpha motor neurons. E stim to facilitate antagonist muscle activity can inhibit spastic muscle activity.[224]
Cryotherapy	• Temporarily decreases spasticity by reducing muscle spindle activity that may increase pain threshold. • Inexpensive option that is often combined with active therapy and used to hinder muscle hypertonia and clonus during casting procedures. • Randomized controlled trials are based on small sample size with a focus on stroke patients.[225,226]
Cryoneurolysis	• Recent studies suggest that cryoneurolysis has potential to be a safe spasticity treatment, adverse effects are limited and manageable, but more research is needed.[227]
Thermotherapy: Heat including ultrasound	• Reported to decrease muscle tone, reduce muscle spasms, and increase pain threshold.[228] • No studies on the long-term impact on spasticity.[229]
Dry needling	• There is no consistent data to suggest a sustained benefit from dry needling. Some studies note benefits of dry needling lasting 1 month or less.[230] • Thin monofilament needles without medication are inserted into the muscles, targeting trigger points, in an effort to release muscle tension.[231] • Proposed to restore muscle architecture and contractile properties, decrease endplate noise, and have beneficial effects on brain activity.[232]
Acupuncture, including electro-acupuncture	• Moderate level evidence for electro-acupuncture combined with conventional routine care (pharmacological and rehabilitation) in reduction in upper-limb and lower-limb spasticity, improved overall motor function, activities of daily living.[233] • It is proposed that acupuncture works by decreasing the pain-spasm cycle, spinal motor neuron regulation, and neurochemical regulation, though exact mechanisms are not clear.[234]

evidence remains unclear for many treatment options.[46] Additionally, although gaps in the literature exist, findings suggest that an integrated multidisciplinary, goal-centered management approach is essential to providing a long-term, comprehensive care for spasticity. Specific areas where more research is needed include therapy components, modalities, duration, and setting.[46]

Some of the nonpharmacologic interventions used in spasticity management are noted to provide beneficial effects, although clarity on optimal timing, duration, and intensity of many therapies is limited. Table 5 summarizes the most common nonpharmacologic interventions used in spasticity and notes the quality of evidence for each modality in specific conditions and populations. Table 5 was developed by compiling findings from published systematic reviews for the effectiveness of interventions in the management of spasticity.

The following nonpharmacologic treatment options are known to be used in practice but more often are used in research settings. These treatment options are included to provide information should patients or caregivers inquire about use. The AAPM&R Spasticity TEP is not providing a recommendation on their use or non-use due to lack of studies specifically related to outcomes in spasticity including long-term efficacy and safety.

- Hyperbaric oxygen therapy
- Transcranial direct current stimulation
- Intermittent theta burst stimulation

- Repetitive transcranial magnetic stimulation
- Electromagnetic therapy (pulsed electromagnetic therapy; magnetic pulsing device)
- Whole-body vibration or focal vibration
- Shock wave therapy

Nonpharmacologic interventions are integral to PM&R practice; however, these interventions are not studied for effectiveness in rigorous clinical trials with specific end points that can be interpreted for clinical guidance. Our literature review confirms findings from recent systematic reviews and meta-analysis that translation of studies to practice has significant limitations for most interventions. Studies are commonly not randomized controlled trials or are based on very small sample sizes and heterogenous populations. Although some interventions offer promise for spasticity, additional studies are needed to further refine knowledge on duration of effect and implementation protocols.

SUMMARY

The AAPM&R embarked on this initiative to produce a condensed review of evidence for the assessment and management of spasticity to offer practical, consensus-based recommendations for PM&R clinicians. The TEP identified a wealth of evidence to support the recommendations outlined in Table 1; however, there is considerable heterogeneity in patient populations, practice, and research, which limits the ability to make focused, strong recommendations to guide all management scenarios. Key findings that influenced the determination of the TEP's assessment and management recommendations include:

- Holistic and patient-centered evaluations, treatment, and reassessments.
  - Assessment of the impact of spasticity management is not limited to the reduction of objective measures of tone but should also include the impact on function and quality of life
- Importance of multimodal therapies
  - Spasticity interventions should often be used in combination for maximal benefit. Coordination of care between physiatrists, physical and/or occupational therapists, caregivers, and surgeons, when appropriate, is key to achieving optimal outcomes.
- Lack of studies that explore effectiveness across conditions (traumatic brain injury, CP, multiple sclerosis, etc.)
  - Although there have been marked gains and improvements in management of spasticity, more work needs to be done to differentiate pathologic mechanisms and more targeted treatment for conditions causing spasticity.
- Access to care as an obstacle
  - Numerous socioeconomic factors affect access to care, and these factors need to be taken into consideration and addressed at both an individual patient as well as societal level.

Consensus guidance statements are valuable but come with inherent limitations, which the TEP recognizes. These statements may introduce biases based on experts' perspectives and experiences, potentially leading to recommendations that are not fully evidence based. The process is also resource intensive, and the updates can be challenging, inclusive of when sections were written and by the time the publication comes out into the literature. Additionally, this guidance statement did not incorporate patient perspective, although we did prioritize patient-centered outcomes. Finally, this guidance statement was funded exclusively by AAPM&R, and although disclosures of financial conflicts of interest were reviewed throughout the development process, panel members have ties to industry and were accordingly managed to minimize bias.

Developments in the field of neurorehabilitation progress rapidly, and there are several ongoing and potential future directions in spasticity assessment and management. Tools exist to both quantitatively and qualitatively assess the severity of spasticity inclusive of electrodiagnostic, mechanical, and ultrasound measures.[235] Advanced imaging techniques could lead to more precise interventions and deeper insights into the neuromuscular mechanisms underlying spasticity.

As we learn more about spasticity, we realize that spasticity is an evolving condition with more than one underlying pathophysiologic process. As we better understand the underlying neurochemical processes involved in spasticity, different treatments may specifically target those processes. Advanced neuromodulation techniques, such as transcranial magnetic stimulation and transcranial direct current stimulation hold promise for modulating neural circuits involved in spasticity. There is also continued research into new medications and drug delivery methods that may lead to more effective therapeutic management in spasticity. Neurosurgical techniques, such as cerebellar stimulation for spasticity in patients with CP or deep brain stimulation of the internal capsule, are also emerging as treatments.[236] Furthermore, innovative neurorehabilitation techniques can enhance our traditional rehabilitation programs. This includes advancements in robotics and exoskeleton technology, which can be designed to provide support and target therapy to affected muscles and joints. More research into stem cell therapy and regenerative therapies will need to be done to learn about approaches that may repair damaged neural pathways and promote tissue healing, which can alleviate spasticity and possibly improve motor function. As we learn about the effects of neuroinflammation after

acquired neurologic injuries and its impact on spasticity, this could lead to the development of targeted immunomodulatory treatments in the future.

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REFERENCES

1. Chang E, Ghosh N, Yanni D, Lee S, Alexandru D, Mozaffar T. A review of spasticity treatments: pharmacological and interventional approaches. *Crit Rev Phys Rehabil Med*. 2013;25(1–2):11-22. doi:10.1615/CritRevPhysRehabilMed.2013007945
2. Gormley ME Jr, Krach LE, Piccini L. Spasticity management in the child with spastic quadriplegia. *Eur J Neurol*. 2001;8(Suppl 5):127-135. doi:10.1046/j.1468-1331.2001.00045.x
3. Raghavan P. Neural basis of spasticity. In: Raghavan P, ed. *Spasticity and Muscle Stiffness: Restoring Form and Function*. 1st ed. Springer Nature; 2022.
4. Lance JW. Symposium synopsis. In: Feldman RG, Young RR, Koella WP, eds. *Spasticity: Disordered Motor Control*. Year Book Medical Publishers; 1980.
5. Pandyan AD, Gregoric M, Barnes MP, et al. Spasticity: clinical perceptions, neurological realities and meaningful measurement. *Disabil Rehabil*. 2005;27(1–2):2-6. doi:10.1080/09638280400014576
6. Li S, Francisco GE, Rymer WZ. A new definition of poststroke spasticity and the interference of spasticity with motor recovery from acute to chronic stages. *Neurorehabil Neural Repair*. 2021;35(7):601-610. doi:10.1177/15459683211011214
7. Mayer NH, Herman RM. Phenomenology of muscle overactivity in the upper motor neuron syndrome. *Eura Medicophys*. 2004;40(2):85-110.
8. Raghavan P, Stecco A, Menon R, Cowman MK, Regatte R. Mechanisms of development of passive mechanical muscle stiffness. In: Raghavan P, ed. *Spasticity and Muscle Stiffness: Restoring Form and Function*. 1st ed. Springer Nature; 2022.
9. Ebell MH, Siwek J, Weiss BD, et al. Strength of recommendation taxonomy (SORT): a patient-centered approach to grading evidence in the medical literature. *J Am Board Fam Pract*. 2004;17(1):59-67. doi:10.3122/jabfm.17.1.59
10. Park TS, Uhm SY, Walter DM, Meyer NL, Dobbs MB. Functional outcome of adulthood selective dorsal rhizotomy for spastic diplegia. *Cureus*. 2019;11(7):e5184. doi:10.7759/cureus.5184

11. Odonkor CA, Esparza R, Flores LE, et al. Disparities in health care for black patients in physical medicine and rehabilitation in the United States: a narrative review. *PM R*. 2021;13(2):180-203. doi:10.1002/pmrj.12509
12. Flores LE, Verduzco-Gutierrez M, Molinares D, Silver JK. Disparities in health care for hispanic patients in physical medicine and rehabilitation in the United States: a narrative review. *Am J Phys Med Rehabil*. 2020;99(4):338-347. doi:10.1097/PHM.0000000000001342
13. Saadi A, Himmelstein DU, Woolhandler S, Mejia NI. Racial disparities in neurologic health care access and utilization in the United States. *Neurology*. 2017;88(24):2268-2275. doi:10.1212/WNL.0000000000004025
14. Houtrow A, Martin AJ, Harris D, et al. Health equity for children and youth with special health care needs: a vision for the future. *Pediatrics*. 2022;149(Suppl 7):e2021056150F. doi:10.1542/peds.2021-056150F
15. Kazerooni R, Healy S, Verduzco-Gutierrez M. Disparities in access to spasticity chemodenervation specialists in the US: a retrospective cross-sectional study. *Am J Phys Med Rehabil*. 2023;22:203-207. doi:10.1097/PHM.0000000000002375
16. Esquenazi A, Bloudek L, Migliaccio-Walle K, et al. Healthcare resource utilization and costs among patients with post-stroke spasticity before and after spasticity management including onabotulinumtoxina. *J Rehabil Med*. 2023;55:jrm11626. doi:10.2340/jrm.v55.11626
17. de Havenon A, Sheth K, Johnston KC, et al. Acute ischemic stroke interventions in the United States and racial, socioeconomic, and geographic disparities. *Neurology*. 2021;97(23):e2292-e2303. doi:10.1212/WNL.0000000000012943
18. Bai YL, Hu YS, Wu Y, et al. Long-term three-stage rehabilitation intervention alleviates spasticity of the elbows, fingers, and plantar flexors and improves activities of daily living in ischemic stroke patients: a randomized, controlled trial. *Neuroreport*. 2014;25(13):998-1005. doi:10.1097/WNR.0000000000000194
19. Larkin T, Martinez V, Scully T, Martinez D, Hayes C, Verduzco-Gutierrez M. Upper extremity spasticity: the quality of online patient resources. *Am J Phys med Rehabil*. 2023;24:18-23. doi:10.1097/PHM.0000000000002297
20. Verduzco-Gutierrez M, Romanoski NL, Capizzi AN, et al. Spasticity outpatient evaluation via telemedicine: a practical framework. *Am J Phys Med Rehabil*. 2020;99(12):1086-1091. doi:10.1097/PHM.0000000000001594
21. Kim J, Sin M, Kim WS, et al. Remote assessment of post-stroke elbow function using internet-based telerobotics: a proof-of-concept study. *Front Neurol*. 2020;11:583101. doi:10.3389/fneur.2020.583101
22. Tenforde AS, Alexander JJ, Alexander M, et al. Telehealth in PM&R: past, present, and future in clinical practice and opportunities for translational research. *PM R*. 2023;15(9):1156-1174. doi:10.1002/pmrj.13029
23. Norman J, Stowers J, Verduzco-Gutierrez M. Parking meters to touch screens: the unforeseen barriers that expansion of telemedicine presents to the disability community. *Am J Phys Med Rehabil*. 2021;100(11):1105-1108. doi:10.1097/PHM.0000000000001771
24. Verduzco-Gutierrez M, Lara AM, Annaswamy TM. When disparities and disabilities collide: inequities during the COVID-19 pandemic. *PM R*. 2021;13(4):412-414. doi:10.1002/pmrj.12551
25. Annaswamy TM, Verduzco-Gutierrez M, Frieden L. Telemedicine barriers and challenges for persons with disabilities: COVID-19 and beyond. *Disabil Health J*. 2020;13(4):100973. doi:10.1016/j.dhjo.2020.100973
26. Walshe FM. Contributions of John Hughlings Jackson to neurology. A brief introduction to his teachings. *Arch Neurol*. 1961;5:119-131. doi:10.1001/archneur.1961.00450140001001
27. Gracies JM, Bayle N, Vinti M, et al. Five-step clinical assessment in spastic paresis. *Eur J Phys Rehabil Med*. 2010;46(3):411-421.
28. Turner-Stokes L, Ashford S, Esquenazi A, et al. A comprehensive person-centered approach to adult spastic paresis: a consensus-based framework. *Eur J Phys Rehabil Med*. 2018;54(4):605-617. doi:10.23736/S1973-9087.17.04808-0
29. Ashford S, Turner-Stokes L. Goal attainment for spasticity management using botulinum toxin. *Physiother Res Int*. 2006;11(1):24-34. doi:10.1002/pri.36
30. Baude M, Ghedira M, Pradines M, Gracies J-M. Clinical assessment of the syndrome of spastic paresis. In: Raghavan P, ed. *Spasticity and Muscle Stiffness: Restoring Form and Function*. 1st ed. Springer Nature; 2022.
31. Gracies JM. Coefficients of impairment in deforming spastic paresis. *Ann Phys Rehabil med*. 2015;58(3):173-178. doi:10.1016/j.rehab.2015.04.004
32. Lam WWT, Tang YM, Fong KNK. A systematic review of the applications of markerless motion capture (MMC) technology for clinical measurement in rehabilitation. *J Neuroeng Rehabil*. 2023;20(1):57. doi:10.1186/s12984-023-01186-9
33. Scott B, Seyres M, Philp F, Chadwick EK, Blana D. Healthcare applications of single camera markerless motion capture: a scoping review. *PeerJ*. 2022;10:e13517. doi:10.7717/peerj.13517
34. Bui HT, Gagnon C, Audet O, Mathieu J, Leone M. Measurement properties of a new wireless electrogoniometer for quantifying spasticity during the pendulum test in ARSACS patients. *J Neurol Sci*. 2017;375:181-185. doi:10.1016/j.jns.2017.01.065
35. Germanotta M, Taborri J, Rossi S, et al. Spasticity measurement based on tonic stretch reflex threshold in children with cerebral palsy using the PediAnklebot. *Front Hum Neurosci*. 2017;11:277. doi:10.3389/fnhum.2017.00277
36. Li X, Shin H, Li S, Zhou P. Assessing muscle spasticity with myotonometric and passive stretch measurements: validity of the Myotonometer. *Sci Rep*. 2017;7:44022. doi:10.1038/srep44022
37. Plantin J, Pennati GV, Roca P, et al. Quantitative assessment of hand spasticity after stroke: imaging correlates and impact on motor recovery. *Front Neurol*. 2019;10:836. doi:10.3389/fneur.2019.00836
38. Pennati GV, Plantin J, Borg J, Lindberg PG. Normative NeuroFlexor data for detection of spasticity after stroke: a cross-sectional study. *J Neuroeng Rehabil*. 2016;13:30. doi:10.1186/s12984-016-0133-x
39. Wang R, Herman P, Ekeberg Ö, Gäverth J, Fagergren A, Forssberg H. Neural and non-neural related properties in the spastic wrist flexors: an optimization study. *Med Eng Phys*. 2017;47:198-209. doi:10.1016/j.medengphy.2017.06.023
40. Wu YN, Park HS, Chen JJ, Ren Y, Roth EJ, Zhang LQ. Position as well as velocity dependence of spasticity-four-dimensional characterizations of catch angle. *Front Neurol*. 2018;9:863. doi:10.3389/fneur.2018.00863
41. Rasmussen HM, Pedersen NW, Overgaard S, et al. Gait analysis for individually tailored interdisciplinary interventions in children with cerebral palsy: a randomized controlled trial. *Dev Med Child Neurol*. 2019;61(10):1189-1195. doi:10.1111/dmcn.14178
42. Flanagan SR, Cynthia H, Petrucelli R, Ragucci M. Medical exacerbation of spasticity. In: Raghavan P, ed. *Spasticity and Muscle Stiffness: Restoring Form and Function*. 1st ed. Springer Nature; 2022.
43. Raghavan P. Framework for the treatment of spasticity and muscle stiffness. In: Raghavan P, ed. *Spasticity and Muscle Stiffness: Restoring Form and Function*. 1st ed. Springer Nature; 2022.
44. Francisco GE, Li S. Spasticity. In: Cifu DX, ed. *Physical Medicine and Rehabilitation*. 5th ed. Elsevier; 2016:487-489.
45. Pierson SH. Outcome measures in spasticity management. *Muscle Nerve Suppl*. 1997;6:S36-S60.
46. Khan F, Amatya B, Bensmail D, Yelnik A. Non-pharmacological interventions for spasticity in adults: an overview of systematic reviews. *Ann Phys Rehabil Med*. 2019;62(4):265-273. doi:10.1016/j.rehab.2017.10.001

47. Novak I, Morgan C, Fahey M, et al. State of the evidence traffic lights 2019: systematic review of interventions for preventing and treating children with cerebral palsy. *Curr Neurol Neurosci Rep*. 2020;20(2):3. doi:10.1007/s11910-020-1022-z
48. Gormley ME Jr. Treatment of neuromuscular and musculoskeletal problems in cerebral palsy. *Pediatr Rehabil*. 2001;4(1):5-16. doi:10.1080/13638490151068393
49. Katz RT. Management of spasticity. *Am J Phys Med Rehabil*. 1988;67(3):108-116. doi:10.1097/00002060-198806000-00004
50. Kita M, Goodkin DE. Drugs used to treat spasticity. *Drugs*. 2000; 59(3):487-495. doi:10.2165/00003495-200059030-00006
51. Basmajian JV, Shankardass K, Russell D, Yucel V. Ketazolam treatment for spasticity: double-blind study of a new drug. *Arch Phys Med Rehabil*. 1984;65(11):698-701.
52. Rode G, Maupas E, Luaute J, Courtois-Jacquin S, Boisson D. Traitements médicamenteux de la spasticité [medical treatment of spasticity]. *Neurochirurgie*. 2003;49(2–3 Pt 2):247-255.
53. Reilly M, Liuzzo K, Blackmer AB. Pharmacological management of spasticity in children with cerebral palsy. *J Pediatr Health Care*. 2020;34(5):495-509. doi:10.1016/j.pedhc.2020.04.010
54. Romito JW, Turner ER, Rosener JA, et al. Baclofen therapeutics, toxicity, and withdrawal: a narrative review. *SAGE Open Med*. 2021;9:20503121211022197. doi:10.1177/20503121211022197
55. Meythaler JM, Kowalski S. Pharmacologic management of spasticity: oral medications. In: Brashear A, Elovic EP, eds. *Spasticity: Diagnosis and Management*. 1st ed. Demos Medical Publishing; 2011:199-227.
56. Schulz E, Mathew OP. Is oral baclofen effective in neonatal hypertonia? *J Child Neurol*. 2012;27(2):197-199. doi:10.1177/0883073811416238
57. 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. doi:10.1016/j.ocl.2010.06.001
58. Verrier M, Ashby P, MacLeod S. Effect of diazepam on muscle contraction in spasticity. *Am J Phys Med*. 1976;55(4):184-191.
59. Wilson LA, McKechnie AA. Oral diazepam in the treatment of spasticity in paraplegia a double-blind trial and subsequent impressions. *Scott Med J*. 1966;11(2):46-51. doi:10.1177/003693306601100202
60. Abbruzzese G. The medical management of spasticity. *Eur J Neurol*. 2002;9(Suppl 1):30-61. doi:10.1046/j.1468-1331.2002.0090s1030.x
61. Sellers EM, Busto U. Diazepam withdrawal syndrome. *Can Med Assoc J*. 1983;129(2):97-100.
62. Leung FW, Guze PA. Diazepam withdrawal. *West J Med*. 1983; 138(1):98-101.
63. Robinson GM, Sellers EM. Diazepam withdrawal seizures. *Can Med Assoc J*. 1982;126(8):944-945.
64. Palazón García R, Benavente Valdepeñas A, Arroyo RO. Protocolo de uso de la tizanidina en la parálisis cerebral infantil [Protocol for tizanidine use in infantile cerebral palsy]. *An Pediatr (Barc)*. 2008;68(5):511-515. doi:10.1157/13120053
65. Kamen L, Henney HR 3rd, Runyan JD. A practical overview of tizanidine use for spasticity secondary to multiple sclerosis, stroke, and spinal cord injury. *Curr Med Res Opin*. 2008;24(2): 425-439. doi:10.1185/030079908x261113
66. Członkowski A, Mirowska D. Pharmacotherapy for spasticity. *Ortop Traumatol Rehabil*. 2002;4(1):54-56.
67. Ono H, Matsumoto K, Kato K, et al. Effects of tizanidine, a centrally acting muscle relaxant, on motor systems. *Gen Pharmacol*. 1986;17(2):137-142. doi:10.1016/0306-3623(86)90130-8
68. Ghanavatian S, Derian A. Tizanidine. In: StatPearls, ed. *Treasure Island*. StatPearls Publishing; 2023.
69. Malanga G, Reiter RD, Garay E. Update on tizanidine for muscle spasticity and emerging indications. *Expert Opin Pharmacother*. 2008;9(12):2209-2215. doi:10.1517/14656566.9.12.2209
70. Landau WM. Tizanidine and spasticity. *Neurology*. 1995;45(12): 2295-2296. doi:10.1212/wnl.45.12.2295
71. Vásquez-Briceño A, Arellano-Saldaña ME, León-Hernández SR, Morales-Osorio MG. Utilidad de la tizanidina. Seguimiento de un ano en el tratamiento de la espasticidad en la paralisis cerebral infantil [The usefulness of tizanidine. A one-year follow-up of the treatment of spasticity in infantile cerebral palsy]. *Rev Neurol*. 2006;43(3):132-136.
72. Tilton A, Vargus-Adams J, Delgado MR. Pharmacologic treatment of spasticity in children. *Semin Pediatr Neurol*. 2010;17(4): 261-267. doi:10.1016/j.spen.2010.10.009
73. Pinder RM, Brogden RN, Speight TM, Avery GS. Dantrolene sodium: a review of its pharmacological properties and therapeutic efficacy in spasticity. *Drugs*. 1977;13(1):3-23. doi:10.2165/00003495-197713010-00002
74. Saulino M, Jacobs BW. The pharmacological management of spasticity. *J Neurosci Nurs*. 2006;38(6):456-459.
75. Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society, Delgado MR, Hirtz D, et al. Practice parameter: pharmacologic treatment of spasticity in children and adolescents with cerebral palsy (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. *Neurology*. 2010;74(4):336-343. doi:10.1212/WNL.0b013e3181cbcd2f
76. Ertzgaard P, Campo C, Calabrese A. Efficacy and safety of oral baclofen in the management of spasticity: a rationale for intrathecal baclofen. *J Rehabil med*. 2017;49(3):193-203. doi:10.2340/16501977-2211
77. Moran LR, Cincotta T, Krishnamoorthy K, Insoft RM. The use of baclofen in full-term neonates with hypertonia. *J Perinatol*. 2005; 25(1):66-68. doi:10.1038/sj.jp.7211194
78. Campistol J. Farmacos empleados por via oral para el tratamiento de la espasticidad [orally administered drugs in the treatment of spasticity]. *Rev Neurol*. 2003;37(1):70-74.
79. Fehlings D, Brown L, Harvey A, et al. Pharmacological and neurosurgical interventions for managing dystonia in cerebral palsy: a systematic review. *Dev Med Child Neurol*. 2018;60(4):356-366. doi:10.1111/dmcn.13652
80. 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.
81. Lapeyre E, Kuks JB, Meijler WJ. Spasticity: revisiting the role and the individual value of several pharmacological treatments. *NeuroRehabilitation*. 2010;27(2):193-200. doi:10.3233/NRE-2010-0596
82. Peck J, Urits I, Crane J, et al. Oral muscle relaxants for the treatment of chronic pain associated with cerebral palsy. *Psychopharmacol Bull*. 2020;50(4 Suppl 1):142-162.
83. Harvison PJ. Dalteparin. In: Enna SJ, Bylund DB, eds. *xPharm: The Comprehensive Pharmacology Reference*. Elsevier; 2007.
84. Martinez-Paz C, García-Cabrera E, Vilches-Arenas Á. Effectiveness and safety of cannabinoids as an add-on therapy in the treatment of resistant spasticity in multiple sclerosis: a systematic review. *Cannabis Cannabinoid Res*. 2023;8(4):580-588. doi:10.1089/can.2022.0254
85. Yadav V, Bever C Jr, Bowen J, et al. Summary of evidence-based guideline: complementary and alternative medicine in multiple sclerosis: report of the guideline development subcommittee of the American Academy of Neurology. *Neurology*. 2014;82(12):1083-1092. doi:10.1212/WNL.0000000000000250
86. Whiting PF, Wolff RF, Deshpande S, et al. Cannabinoids for medical use: a systematic review and meta-analysis [published correction appears in *JAMA*. 2015;314(5):520] [published correction appears in *JAMA*. 2015;314(8):837] [published correction appears in *JAMA*. 2015;314(21):2308] [published correction appears in *JAMA*. 2016;315(14):1522]. *JAMA*. 2015;313(24):2456-2473. doi:10.1001/jama.2015.6358

87. Francisco GE, McGuire JR. Poststroke spasticity management. *Stroke*. 2012;43(11):3132-3136. doi:10.1161/STROKEAHA.111.639831
88. Ploypetch T, Kwon JY, Armstrong HF, Kim H. A retrospective review of unintended effects after single-event multi-level Chemoneurolysis with botulinum toxin-a and phenol in children with cerebral palsy. *PM R*. 2015;7(10):1073-1080. doi:10.1016/j.pmrj.2015.05.020
89. Furr-Stimming E, Boyle AM, Schiess MC. Spasticity and intrathecal baclofen. *Semin Neurol*. 2014;34(5):591-596. doi:10.1055/s-0034-1396012
90. Awaad Y, Rizk T, Siddiqui I, Roosen N, McIntosh K, Waines GM. Complications of intrathecal baclofen pump: prevention and cure. *ISRN Neurol*. 2012;2012:575168. doi:10.5402/2012/575168
91. Karri J, Mas MF, Francisco GE, Li S. Practice patterns for spasticity management with phenol neurolysis. *J Rehabil Med*. 2017;49(6):482-488. doi:10.2340/16501977-2239
92. Farag SM, Mohammed MO, El-Sobky TA, ElKadery NA, ElZohiery AK. Botulinum toxin a injection in treatment of upper limb spasticity in children with cerebral palsy: a systematic review of randomized controlled trials. *JBJS Rev*. 2020;8(3):e0119. doi:10.2106/JBJS.RVW.19.00119
93. Simpson DM, Hallett M, Ashman EJ, et al. Practice guideline update summary: botulinum neurotoxin for the treatment of blepharospasm, cervical dystonia, adult spasticity, and headache: report of the Guideline Development Subcommittee of the American Academy of Neurology. *Neurology*. 2016;86(19):1818-1826. doi:10.1212/WNL.0000000000002560
94. Andringa A, van de Port I, van Wegen E, Ket J, Meskers C, Kwakkel G. Effectiveness of botulinum toxin treatment for upper limb spasticity poststroke over different ICF domains: a systematic review and meta-analysis. *Arch Phys Med Rehabil*. 2019;100(9):1703-1725. doi:10.1016/j.apmr.2019.01.016
95. Dong Y, Wu T, Hu X, Wang T. Efficacy and safety of botulinum toxin type A for upper limb spasticity after stroke or traumatic brain injury: a systematic review with meta-analysis and trial sequential analysis. *Eur J Phys Rehabil Med*. 2017;53(2):256-267. doi:10.23736/S1973-9087.16.04329-X
96. Jia S, Liu Y, Shen L, Liang X, Xu X, Wei Y. Botulinum toxin type a for upper limb spasticity in poststroke patients: a meta-analysis of randomized controlled trials. *J Stroke Cerebrovasc Dis*. 2020;29(6):104682. doi:10.1016/j.jstrokecerebrovasdis.2020.104682
97. Sun LC, Chen R, Fu C, et al. Efficacy and safety of botulinum toxin type A for limb spasticity after stroke: a meta-analysis of randomized controlled trials. *Biomed Res Int*. 2019;2019:8329306. doi:10.1155/2019/8329306
98. Cofré Lizama LE, Khan F, Galea MP. Beyond speed: gait changes after botulinum toxin injections in chronic stroke survivors (a systematic review). *Gait Posture*. 2019;70:389-396. doi:10.1016/j.gaitpost.2019.03.035
99. Varvarousis DN, Martzivanou C, Dimopoulos D, Dimakopoulos G, Vasileiadis GI, Ploumis A. The effectiveness of botulinum toxin on spasticity and gait of hemiplegic patients after stroke: a systematic review and meta-analysis. *Toxicon*. 2021;203:74-84. doi:10.1016/j.toxicon.2021.09.020
100. Esquenazi A, Bavikatte G, Bandari DS, et al. Long-term observational results from the ASPIRE study: OnabotulinumtoxinA treatment for adult lower limb spasticity. *PM R*. 2021;13(10):1079-1093. doi:10.1002/pmrj.12517
101. Alfaro A, Tsai T. Phenol injections to musculocutaneous nerves: decrease in spasticity for elbow flexion, and complications. *Muscle Nerve*. 2016;54(3):569.
102. Keenan MA, Tomas ES, Stone L, Gerstén LM. Percutaneous phenol block of the musculocutaneous nerve to control elbow flexor spasticity. *J Hand Surg Am*. 1990;15(2):340-346. doi:10.1016/0363-5023(90)90120-g
103. Matsumoto ME, Berry J, Yung H, Matsumoto M, Munin MC. Comparing electrical stimulation with and without ultrasound guidance for phenol neurolysis to the musculocutaneous nerve. *PM R*. 2018;10(4):357-364. doi:10.1016/j.pmrj.2017.09.006
104. Zhang B, Darji N, Francisco GE, Li S. The time course of onset and peak effects of phenol neurolysis. *Am J Phys Med Rehabil*. 2021;100(3):266-270. doi:10.1097/PHM.0000000000001563
105. Kong KH, Chua KS. Neurolysis of the musculocutaneous nerve with alcohol to treat poststroke elbow flexor spasticity. *Arch Phys Med Rehabil*. 1999;80(10):1234-1236. doi:10.1016/s0003-9993(99)90021-7
106. Lee DG, Jang SH. Ultrasound guided alcohol neurolysis of musculocutaneous nerve to relieve elbow spasticity in hemiparetic stroke patients. *NeuroRehabilitation*. 2012;31(4):373-377. doi:10.3233/NRE-2012-00806
107. Lee J, Lee YS. Percutaneous chemical nerve block with ultrasound-guided intraneural injection. *Eur Radiol*. 2008;18(7):1506-1512. doi:10.1007/s00330-008-0909-x
108. Akkaya T, Unlu E, Alptekin A, Gumus HI, Umay E, Cakci A. Neurolytic phenol blockade of the obturator nerve for severe adductor spasticity. *Acta Anaesthesiol Scand*. 2010;54(1):79-85. doi:10.1111/j.1399-6576.2009.02130.x
109. Alfaro A, Flancia F. Phenol injections to obturator nerves: decrease in spasticity, and complications. *Poster 90*. 2015;9S(7):S120-S121. doi:10.1016/j.pmrj.2015.06.129
110. Alsuhabani A, Ethans K, Casey A, Skrabek R, Chateau D, Sutherland E. Ultrasound guided phenol block of the obturator nerve for severe adductor spasticity: a pilot study. *Int J Neurorehabilitation*. 2016;3(2):1-5.
111. Ghai A, Sangwan SS, Hooda S, Garg N, Kundu ZS, Gupta T. Evaluation of interadductor approach in neurolytic blockade of obturator nerve in spastic patients. *Saudi J Anaesth*. 2013;7(4):420-426. doi:10.4103/1658-354X.121074
112. Lam K, Wong D, Tam CK, et al. Ultrasound and electrical stimulator-guided obturator nerve block with phenol in the treatment of hip adductor spasticity in long-term care patients: a randomized, triple blind, placebo controlled study. *J Am Med Dir Assoc*. 2015;16(3):238-246. doi:10.1016/j.jamda.2014.10.005
113. Ofluoglu D, Esquenazi A, Hirai B. Temporospatial parameters of gait after obturator neurolysis in patients with spasticity. *Am J Phys Med Rehabil*. 2003;82(11):832-836. doi:10.1097/01.PHM.0000091986.32078.CD
114. Park ES, Rha DW, Lee WC, Sim EG. The effect of obturator nerve block on hip lateralization in low functioning children with spastic cerebral palsy. *Yonsei Med J*. 2014;55(1):191-196. doi:10.3349/ymj.2014.55.1.191
115. Razaq S, Fahim A, Arshad AM. Obturator nerve block with aqueous phenol reduces hip adductor spasticity – a single centre experience. *Pakistan J Neurol Surg*. 2020;15(4):30-35.
116. Selmi NH, Şahin S, Gurbet A, et al. Obturator nerve block in adductor spasticity: comparison of peripheral nerve stimulator and ultrasonography. *Turk J Anaesth Reanim*. 2013;41:121-125. doi:10.5152/TJAR.2013.49
117. Viel EJ, Perennou D, Ripart J, Pélissier J, Eledjam JJ. Neurolytic blockade of the obturator nerve for intractable spasticity of adductor thigh muscles. *Eur J Pain*. 2002;6(2):97-104. doi:10.1053/eujp.2001.0269
118. Wassef MR. Interadductor approach to obturator nerve blockade for spastic conditions of adductor thigh muscles. *Reg Anesth*. 1993;18(1):13-17.
119. Yaşar E, Tok F, Taşkaynatan MA, Yilmaz B, Balaban B, Alaca R. The effects of phenol neurolysis of the obturator nerve on the distribution of buttock-seat interface pressure in spinal cord injury patients with hip adductor spasticity. *Spinal Cord*. 2010;48(11):828-831. doi:10.1038/sc.2010.34
120. Alfaro A. Tibial nerve injections with phenol decreased spasticity and improved ankle dorsiflexion. Poster at the American Academy of Physical Medicine and Rehabilitation annual meeting. Baltimore, MD. 2022,S1(14).
121. Kocabas H, Salli A, Demir AH, Ozerbil OM. Comparison of phenol and alcohol neurolysis of tibial nerve motor branches to the

gastrocnemius muscle for treatment of spastic foot after stroke: a randomized controlled pilot study. *Eur J Phys Rehabil Med*. 2010;46(1):5-10.
122. Khalili AA, Betts HB. Peripheral nerve block with phenol in the management of spasticity. Indications and complications. *JAMA*. 1967;200(13):1155-1157.
123. Khalili AA, Harmel MH, Forster S, Benton JG. Management of spasticity by selective peripheral nerve block with dilute phenol solutions in clinical rehabilitation. *Arch Phys Med Rehabil*. 1964; 45:513-519.
124. Kirazli Y, On AY, Kismali B, Aksit R. Comparison of phenol block and botulinus toxin type A in the treatment of spastic foot after stroke: a randomized, double-blind trial. *Am J Phys Med Rehabil*. 1998;77(6):510-515. doi:10.1097/00002060-199811000-00012
125. Manca M, Merlo A, Ferraresi G, Cavazza S, Marchi P. Botulinum toxin type A versus phenol. A clinical and neurophysiological study in the treatment of ankle clonus. *Eur J Phys Rehabil Med*. 2010;46(1):11-18.
126. On AY, Kirazli Y, Kismali B, Aksit R. Mechanisms of action of phenol block and botulinus toxin type A in relieving spasticity: electrophysiologic investigation and follow-up. *Am J Phys med Rehabil*. 1999;78(4):344-349. doi:10.1097/00002060-199907000-00010
127. Petrillo CR, Chu DS, Davis SW. Phenol block of the tibial nerve in the hemiplegic patient. *Orthopedics*. 1980;3(9):871-874. doi: 10.3928/0147-7447-19800901-10
128. Petrillo CR, Knoploch S. Phenol block of the tibial nerve for spasticity: a long-term follow-up study. *Int Disabil Stud*. 1988; 10(3):97-100. doi:10.3109/09638288809164120
129. Chua KS, Kong KH. Clinical and functional outcome after alcohol neurolysis of the tibial nerve for ankle-foot spasticity. *Brain Inj*. 2001;15(8):733-739. doi:10.1080/02699050010009775
130. Jang SH, Ahn SH, Park SM, Kim SH, Lee KH, Lee ZI. Alcohol neurolysis of tibial nerve motor branches to the gastrocnemius muscle to treat ankle spasticity in patients with hemiplegic stroke. *Arch Phys Med Rehabil*. 2004;85(3):506-508. doi:10.1016/s0003-9993(03)00468-4
131. Demir Y, Şan AU, Kesikburun S, Yaşar E, Yılmaz B. The short-term effect of ultrasound and peripheral nerve stimulator-guided femoral nerve block with phenol on the outcomes of patients with traumatic spinal cord injury. *Spinal Cord*. 2018;56(9):907-912. doi:10.1038/s41393-018-0142-7
132. Kong KH, Chua KS. Outcome of obturator nerve block with alcohol for the treatment of hip adductor spasticity. *Int J Rehabil Res*. 1999;22(4):327-329. doi:10.1097/00004356-199912000-00011
133. Hecht JS. Subscapular nerve block in the painful hemiplegic shoulder. *Arch Phys Med Rehabil*. 1992;73(11):1036-1039.
134. Vadivelu S, Stratton A, Pierce W. Pediatric tone management. *Phys Med Rehabil Clin N Am*. 2015;26(1):69-78. doi:10.1016/j.pmr.2014.09.008
135. Evans SH, Cameron MW, Burton JM. Hypertonia. *Curr Probl Pediatr Adolesc Health Care*. 2017;47(7):161-166. doi:10.1016/j.cppeds.2017.06.005
136. Nahm NJ, Graham HK, Gormley ME Jr, Georgiadis AG. Management of hypertonia in cerebral palsy. *Curr Opin Pediatr*. 2018;30(1):57-64. doi:10.1097/MOP.0000000000000567
137. Wong AM, Chen CL, Chen CP, Chou SW, Chung CY, Chen MJ. Clinical effects of botulinum toxin A and phenol block on gait in children with cerebral palsy. *Am J Phys Med Rehabil*. 2004; 83(4):284-291. doi:10.1097/01.phm.0000118038.02326.ca
138. Gooch JL, Patton CP. Combining botulinum toxin and phenol to manage spasticity in children. *Arch Phys Med Rehabil*. 2004; 85(7):1121-1124. doi:10.1016/j.apmr.2003.09.032
139. Easton JK, Ozel T, Halpern D. Intramuscular neurolysis for spasticity in children. *Arch Phys Med Rehabil*. 1979;60(4):155-158.
140. Spira R. Management of spasticity in cerebral palsied children by peripheral nerve block with phenol. *Dev Med Child Neurol*. 1971;13(2):164-173. doi:10.1111/j.1469-8749.1971.tb03241.x
141. Yadav SL, Singh U, Dureja GP, Singh KK, Chaturvedi S. Phenol block in the management of spastic cerebral palsy. *Indian J Pediatr*. 1994;61(3):249-255. doi:10.1007/BF02752218
142. Morrison JE Jr, Matthews D, Washington R, Fennessey PV, Harrison LM. Phenol motor point blocks in children: plasma concentrations and cardiac dysrhythmias. *Anesthesiology*. 1991; 75(2):359-362. doi:10.1097/00000542-199108000-00027
143. Meythaler JM. Use of intrathecal baclofen in brain injury patients. *Arch Phys Med Rehabil*. 1994;75(9):1036. doi:10.1016/0003-9993(94)90755-2
144. Meythaler JM, McCary A, Hadley MN. Prospective assessment of continuous intrathecal infusion of baclofen for spasticity caused by acquired brain injury: a preliminary report. *J Neurosurg*. 1997; 87(3):415-419. doi:10.3171/jns.1997.87.3.0415
145. Meythaler JM, Guin-Renfroe S, Hadley MN. 11. Continuously infused intrathecal baclofen (Itb) for spastic-hypertonia in adult cerebral palsy. *Am J Phys Med Rehabil*. 1998;77(2):173. doi:10.1097/00002060-199803000-00037
146. Meythaler JM, Guin-Renfroe S, Hadley MN. Continuously infused intrathecal baclofen for spastic/dystonic hemiplegia: a preliminary report. *Am J Phys Med Rehabil*. 1999;78(3):247-254. doi:10.1097/00002060-199905000-00012
147. Meythaler JM, Guin-Renfroe S, Brunner RC, Hadley MN. Intrathecal baclofen for spastic hypertonia from stroke. *Stroke*. 2001; 32(9):2099-2109. doi:10.1161/hs0901.095682
148. Meythaler JM, Guin-Renfroe S, Law C, Grabb P, Hadley MN. Continuously infused intrathecal baclofen over 12 months for spastic hypertonia in adolescents and adults with cerebral palsy. *Arch Phys Med Rehabil*. 2001;82(2):155-161. doi:10.1053/apmr.2001.19246
149. Meythaler JM, Steers WD, Tuel SM, Cross LL, Haworth CS. Continuous intrathecal baclofen in spinal cord spasticity. A prospective study. *Am J Phys Med Rehabil*. 1992;71(6):321-327. doi:10.1097/00002060-199212000-00003
150. Coffey JR, Cahill D, Steers W, et al. Intrathecal baclofen for intractable spasticity of spinal origin: results of a long-term multicenter study. *J Neurosurg*. 1993;78(2):226-232. doi:10.3171/jns.1993.78.2.0226
151. Albright AL, Gilmartin R, Swift D, Krach LE, Ivanhoe CB, McLaughlin JF. Long-term intrathecal baclofen therapy for severe spasticity of cerebral origin. *J Neurosurg*. 2003;98(2): 291-295. doi:10.3171/jns.2003.98.2.0291
152. Albright AL, Barron WB, Fasick MP, Polinko P, Janosky J. Continuous intrathecal baclofen infusion for spasticity of cerebral origin. *JAMA*. 1993;270(20):2475-2477. doi:10.1001/jama.1993.03510200081036
153. Penn RD, Kroin JS. Long-term intrathecal baclofen infusion for treatment of spasticity. *J Neurosurg*. 1987;66(2):181-185. doi: 10.3171/jns.1987.66.2.0181
154. Ordia JI, Fischer E, Adamski E, Spatz EL. Chronic intrathecal delivery of baclofen by a programmable pump for the treatment of severe spasticity. *J Neurosurg*. 1996;85(3):452-457. doi:10.3171/jns.1996.85.3.0452
155. 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. *Neuromodulation*. 2002;5(1):16-24. doi:10.1046/j.1525-1403.2002._2004.x
156. Becker R, Alberti O, Bauer BL. Continuous intrathecal baclofen infusion in severe spasticity after traumatic or hypoxic brain injury. *J Neurol*. 1997;244(3):160-166. doi:10.1007/s004150050067
157. Saulino M, Ivanhoe CB, McGuire JR, Ridley B, Shilt JS, Boster AL. Best practices for intrathecal baclofen therapy: patient selection. *Neuromodulation*. 2016;19(6):607-615. doi:10.1111/ner.12447
158. Hoarau X, Richer E, Dehail P, Cuny E. Comparison of long-term outcomes of patients with severe traumatic or hypoxic brain injuries

treated with intrathecal baclofen therapy for dysautonomia. *Brain Inj*. 2012;26(12):1451-1463. doi:10.3109/02699052.2012.694564
159. Boster AL, Bennett SE, Bilsky GS, et al. Best practices for intrathecal baclofen therapy: screening test. *Neuromodulation*. 2016; 19(6):616-622. doi:10.1111/ner.12437
160. Francisco GE, Saulino MF, Yablon SA, Turner M. Intrathecal baclofen therapy: an update. *PM R*. 2009;1(9):852-858. doi:10.1016/j.pmrj.2009.07.015
161. Burns AS, Meythaler JM. Intrathecal baclofen in tetraplegia of spinal origin: efficacy for upper extremity hypertonia. *Spinal Cord*. 2001;39(8):413-419. doi:10.1038/sj.sc.3101178
162. Grabb PA, Guin-Renfroe S, Meythaler JM. Midthoracic catheter tip placement for intrathecal baclofen administration in children with quadriparetic spasticity. *Neurosurgery*. 1999;45(4):833-837. doi:10.1097/00006123-199910000-00020
163. Medtronic. *MRI Guidelines for Medtronic Implantable Infusion Systems*. Medtronic Inc.; 2020:1-18 https://mriquestions.com/uploads/3/4/5/7/34572113/synchromed_ii_mri_m005186c_a_001_view.pdf (Accessed on 9/25/23)
164. Flowonix. *Prometra® and Prometra® II Programmable Pumps Magnetic Resonance Imaging (MRI) Safety Information*. Flowonix: 2017;1-10. https://flowonix.com/sites/default/files/pl-15200-02_-_prometra_and_prometra_ii_programmable_pumps_mri_scan_instructions.pdf (Accessed on 9/25/23)
165. McCormick ZL, Chu SK, Binler D, et al. Intrathecal versus oral baclofen: a matched cohort study of spasticity, pain, sleep, fatigue, and quality of life. *PM R*. 2016;8(6):553-562. doi:10.1016/j.pmrj.2015.10.005
166. Pucks-Faes E, Dobesberger J, Hitzenberger G, et al. Intrathecal baclofen in hereditary spastic paraparesis. *Front Neurol*. 2019; 10:901. doi:10.3389/fneur.2019.00901
167. Abbatemarco JR, Willis MA, Wilson RG, Nagel SJ, Machado AG, Bethoux FA. Case series: intrathecal baclofen therapy in stiff-person syndrome. *Neuromodulation*. 2018;21(7): 655-659. doi:10.1111/ner.12765
168. Penn RD, Mangieri EA. Stiff-man syndrome treated with intrathecal baclofen. *Neurology*. 1993;43(11):2412. doi:10.1212/wnl.43.11.2412
169. Patatoukas D, Rovlias A, Moumtzi H, et al. Hereditary spastic paraparesis and intrathecal baclofen. *Ann Phys Rehabil Med*. 2014;57:e49. doi:10.1016/j.rehab.2014.03.177
170. Wang KK, Munger ME, Chen BP, Novacheck TF. Selective dorsal rhizotomy in ambulant children with cerebral palsy. *J Child Orthop*. 2018;12(5):413-427. doi:10.1302/1863-2548.12.180123
171. Peacock WJ, Arens LJ. Selective posterior rhizotomy for the relief of spasticity in cerebral palsy. *S Afr Med J*. 1982;62(4): 119-124.
172. Park TS, Johnston JM. Surgical techniques of selective dorsal rhizotomy for spastic cerebral palsy. Technical note. *Neurosurg Focus*. 2006;21(2):e7.
173. Graham D, Aquilina K, Cawker S, Paget S, Wimalasundera N. Single-level selective dorsal rhizotomy for spastic cerebral palsy. *J Spine Surg*. 2016;2(3):195-201. doi:10.21037/jss.2016.08.08
174. Josenby AL, Wagner P, Jarnlo GB, Westbom L, Nordmark E. Motor function after selective dorsal rhizotomy: a 10-year practice-based follow-up study. *Dev Med Child Neurol*. 2012; 54(5):429-435. doi:10.1111/j.1469-8749.2012.04258.x
175. Farmer JP, Sabbagh AJ. Selective dorsal rhizotomies in the treatment of spasticity related to cerebral palsy. *Childs Nerv Syst*. 2007;23(9):991-1002. doi:10.1007/s00381-007-0398-2
176. Tedroff K, Löwing K, Åström E. A prospective cohort study investigating gross motor function, pain, and health-related quality of life 17 years after selective dorsal rhizotomy in cerebral palsy. *Dev Med Child Neurol*. 2015;57(5):484-490. doi:10.1111/dmcn.12665
177. Hägglund G, Wagner P. Development of spasticity with age in a total population of children with cerebral palsy. *BMC Musculoskelet Disord*. 2008;9:150. doi:10.1186/1471-2474-9-150
178. Munger ME, Aldahondo N, Krach LE, Novacheck TF, Schwartz MH. Long-term outcomes after selective dorsal rhizotomy: a retrospective matched cohort study. *Dev Med Child Neurol*. 2017;59(11):1196-1203. doi:10.1111/dmcn.13500
179. Dudley RW, Parolin M, Gagnon B, et al. Long-term functional benefits of selective dorsal rhizotomy for spastic cerebral palsy. *J Neurosurg Pediatr*. 2013;12(2):142-150. doi:10.3171/2013.4.PEDS12539
180. Tedroff K, Hägglund G, Miller F. Long-term effects of selective dorsal rhizotomy in children with cerebral palsy: a systematic review. *Dev Med Child Neurol*. 2020;62(5):554-562. doi:10.1111/dmcn.14320
181. Grootveld LR, van Schie PE, Buizer AI, et al. Sudden falls as a persistent complication of selective dorsal rhizotomy surgery in children with bilateral spasticity: report of 3 cases. *J Neurosurg Pediatr*. 2016;18(2):192-195. doi:10.3171/2016.2.PEDS15527
182. McLaughlin J, Bjornson K, Temkin N, et al. Selective dorsal rhizotomy: meta-analysis of three randomized controlled trials. *Dev Med Child Neurol*. 2002;44(1):17-25. doi:10.1017/s0012162201001608
183. Daunter AK, Kratz AL, Hurvitz EA. Long-term impact of childhood selective dorsal rhizotomy on pain, fatigue, and function: a case-control study. *Dev Med Child Neurol*. 2017;59(10):1089-1095. doi:10.1111/dmcn.13481
184. Bolster EA, van Schie PE, Becher JG, van Ouwerkerk WJ, Strijers RL, Vermeulen RJ. Long-term effect of selective dorsal rhizotomy on gross motor function in ambulant children with spastic bilateral cerebral palsy, compared with reference centiles. *Dev Med Child Neurol*. 2013;55(7):610-616. doi:10.1111/dmcn.12148
185. Summers J, Coker B, Eddy S, et al. Selective dorsal rhizotomy in ambulant children with cerebral palsy: an observational cohort study. *Lancet Child Adolesc Health*. 2019;3(7):455-462. doi:10.1016/S2352-4642(19)30119-1
186. Trost JP, Schwartz MH, Krach LE, Dunn ME, Novacheck TF. Comprehensive short-term outcome assessment of selective dorsal rhizotomy. *Dev Med Child Neurol*. 2008;50(10):765-771. doi:10.1111/j.1469-8749.2008.03031.x
187. Novak I, McIntyre S, Morgan C, et al. A systematic review of interventions for children with cerebral palsy: state of the evidence. *Dev Med Child Neurol*. 2013;55(10):885-910. doi:10.1111/dmcn.12246
188. Wach J, Yildiz ÖC, Sarikaya-Seiwert S, Vatter H, Haberl H. Predictors of postoperative complications after selective dorsal rhizotomy. *Acta Neurochir*. 2021;163(2):463-474. doi:10.1007/s00701-020-04487-3
189. Aldahondo N, Krach LE. Poster 447 dysesthesias after selective dorsal rhizotomy: risk factors, presentation, and treatment. *PM&R*. 2012;4(S10):S343-S344. doi:10.1016/j.pmrj.2012.09.1055
190. Geiduschek JM, Haberkern CM, McLaughlin JF, Jacobson LE, Hays RM, Roberts TS. Pain management for children following selective dorsal rhizotomy. *Can J Anaesth*. 1994;41(6):492-496. doi:10.1007/BF03011543
191. Abdel Ghany WA, Nada M, Mahran MA, et al. Combined anterior and posterior lumbar rhizotomy for treatment of mixed dystonia and spasticity in children with cerebral palsy. *Neurosurgery*. 2016;79(3):336-344. doi:10.1227/NEU.0000000000001271
192. Albright AL, Tyler-Kabara EC. Combined ventral and dorsal rhizotomies for dystonic and spastic extremities. Report of six cases. *J Neurosurg*. 2007;107(4 Suppl):324-327. doi:10.3171/PED-07/10/324
193. Merlo A, Galletti M, Zerbinati P, et al. Surgical quadriceps lengthening can reduce quadriceps spasticity in chronic stroke patients. A case-control study. *Front Neurol*. 2022;13:980692. doi:10.3389/fneur.2022.980692
194. Chang CH, Chen YY, Yeh KK, Chen CL. Gross motor function change after multilevel soft tissue release in children with cerebral palsy. *Biom J*. 2017;40(3):163-168. doi:10.1016/j.bj.2016.12.003

195. Ellington MD, Scott AC, Linton J, Sullivan E, Barnes D. Rectus emoris transfer versus rectus intramuscular lengthening for the treatment of stiff knee gait in children with cerebral palsy. *J Pediatr Orthop*. 2018;38(4):e213-e218. doi:10.1097/BPO.0000000000001138
196. Kläusler M, Speth BM, Brunner R, Tirosh O, Camathias C, Rutz E. Long-term follow-up after tibialis anterior tendon shortening in combination with Achilles tendon lengthening in spastic equinus in cerebral palsy. *Gait Posture*. 2017;58:457-462. doi:10.1016/j.gaitpost.2017.08.028
197. Leclercq C, Perruisseau-Carrier A, Gras M, Panciera P, Fulchignoni C, Fulchignoni M. Hyperselective neurectomy for the treatment of upper limb spasticity in adults and children: a prospective study. *J Hand Surg Eur*. 2021;46(7):708-716. doi:10.1177/17531934211027499
198. Bollens B, Deltombe T, Detrembleur C, Gustin T, Stoquart G, Lejeune TM. Effects of selective tibial nerve neurotomy as a treatment for adults presenting with spastic equinovarus foot: a systematic review. *J Rehabil Med*. 2011;43(4):277-282. doi:10.2340/16501977-0786
199. Thibaut A, Wannez S, Deltombe T, Martens G, Laureys S, Chatelle C. Physical therapy in patients with disorders of consciousness: impact on spasticity and muscle contracture. *NeuroRehabilitation*. 2018;42(2):199-205. doi:10.3233/NRE-172229
200. Thibaut A, Chatelle C, Ziegler E, Bruno MA, Laureys S, Gosseries O. Spasticity after stroke: physiology, assessment and treatment. *Brain Inj*. 2013;27(10):1093-1105. doi:10.3109/02699052.2013.804202
201. Morgan C, Fetters L, Adde L, et al. Early intervention for children aged 0 to 2 years with or at high risk of cerebral palsy: international clinical practice guideline based on systematic reviews. *JAMA Pediatr*. 2021;175(8):846-858. doi:10.1001/jamapediatrics.2021.0878
202. Morgan C, Darrah J, Gordon AM, et al. Effectiveness of motor interventions in infants with cerebral palsy: a systematic review. *Dev Med Child Neurol*. 2016;58(9):900-909. doi:10.1111/dmcn.13105
203. Bovend'Eerdt TJ, Newman M, Barker K, Dawes H, Minelli C, Wade DT. The effects of stretching in spasticity: a systematic review. *Arch Phys Med Rehabil*. 2008;89(7):1395-1406. doi:10.1016/j.apmr.2008.02.015
204. Katalinic OM, Harvey LA, Herbert RD, Moseley AM, Lannin NA, Schurr K. Stretch for the treatment and prevention of contractures. *Cochrane Database Syst Rev*. 2010;1(9):CD007455. doi:10.1002/14651858.CD007455.pub2
205. Katalinic OM, Harvey LA, Herbert RD. Effectiveness of stretch for the treatment and prevention of contractures in people with neurological conditions: a systematic review. *Phys Ther*. 2011;91(1):11-24. doi:10.2522/ptj.20100265
206. Pin T, Dyke P, Chan M. The effectiveness of passive stretching in children with cerebral palsy. *Dev Med Child Neurol*. 2006;48(10):855-862. doi:10.1017/S0012162206001836
207. Salazar AP, Pinto C, Ruschel Mossi JV, Figueiro B, Lukrafka JL, Pagnussat AS. Effectiveness of static stretching positioning on post-stroke upper-limb spasticity and mobility: systematic review with meta-analysis. *Ann Phys Rehabil Med*. 2019;62(4):274-282. doi:10.1016/j.rehab.2018.11.004
208. Booth MY, Yates CC, Edgar TS, Bandy WD. Serial casting vs combined intervention with botulinum toxin A and serial casting in the treatment of spastic equinus in children. *Pediatr Phys Ther*. 2003;15(4):216-220. doi:10.1097/01.PEP.0000096382.65499.E2
209. Dai AI, Demiryürek AT. Serial casting as an adjunct to botulinum toxin type A treatment in children with cerebral palsy and spastic paraparesis with scissoring of the lower extremities. *J Child Neurol*. 2017;32(7):671-675. doi:10.1177/0883073817701526
210. Lee SJ, Sung IY, Jang DH, Yi JH, Lee JH, Ryu JS. The effect and complication of botulinum toxin type a injection with serial casting for the treatment of spastic equinus foot. *Ann Rehabil Med*. 2011;35(3):344-353. doi:10.5535/arm.2011.35.3.344
211. Glanzman AM, Kim H, Swaminathan K, Beck T. Efficacy of botulinum toxin A, serial casting, and combined treatment for spastic equinus: a retrospective analysis. *Dev Med Child Neurol*. 2004;46(12):807-811. doi:10.1017/s0012162204001410
212. Amatya B, Khan F, La Mantia L, Demetrios M, Wade DT. Non pharmacological interventions for spasticity in multiple sclerosis. *Cochrane Database Syst Rev*. 2013;2(2):CD009974. doi:10.1002/14651858.CD009974.pub2
213. Merino-Andrés J, García de Mateos-López A, Damiano DL, Sánchez-Sierra A. Effect of muscle strength training in children and adolescents with spastic cerebral palsy: a systematic review and meta-analysis. *Clin Rehabil*. 2022;36(1):4-14. doi:10.1177/02692155211040199
214. Rogers A, Furler BL, Brinks S, Darrah J. A systematic review of the effectiveness of aerobic exercise interventions for children with cerebral palsy: an AACPDM evidence report. *Dev Med Child Neurol*. 2008;50(11):808-814. doi:10.1111/j.1469-8749.2008.03134.x
215. Hoare BJ, Wallen MA, Thorley MN, Jackman ML, Carey LM, Imms C. Constraint-induced movement therapy in children with unilateral cerebral palsy. *Cochrane Database Syst Rev*. 2019;4(4):CD004149. doi:10.1002/14651858.CD004149.pub3
216. Huang YC, Chen PC, Tso HH, Yang YC, Ho TL, Leong CP. Effects of kinesio taping on hemiplegic hand in patients with upper limb post-stroke spasticity: a randomized controlled pilot study. *Eur J Phys Rehabil Med*. 2019;55(5):551-557. doi:10.23736/S1973-9087.19.05684-3
217. Santamato A, Micello MF, Panza F, et al. Adhesive taping vs. daily manual muscle stretching and splinting after botulinum toxin type A injection for wrist and fingers spastic overactivity in stroke patients: a randomized controlled trial. *Clin Rehabil*. 2015;29(1):50-58. doi:10.1177/0269215514537915
218. Woodford H, Price C. EMG biofeedback for the recovery of motor function after stroke. *Cochrane Database Syst Rev*. 2007;2007(2):CD004585. doi:10.1002/14651858.CD004585.pub2
219. Winstein CJ, Stein J, Arena R, et al. Guidelines for adult stroke rehabilitation and recovery: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2016;47(6):e98-e169. doi:10.1161/STR.0000000000000098 Erratum in: *Stroke*. 2017;48(2):e78. Erratum in: *Stroke*. 2017;48(12):e369.
220. Stein C, Fritsch CG, Robinson C, Sbruzzi G, Plentz RD. Effects of electrical stimulation in spastic muscles after stroke: systematic review and meta-analysis of randomized controlled trials. *Stroke*. 2015;46(8):2197-2205. doi:10.1161/STROKEAHA.115.009633
221. Intiso D, Santamato A, Di Rienzo F. Effect of electrical stimulation as an adjunct to botulinum toxin type A in the treatment of adult spasticity: a systematic review. *Disabil Rehabil*. 2017;39(21):2123-2133. doi:10.1080/09638288.2016.1219398
222. Korzhova J, Sinitsyn D, Chervyakov A, et al. Transcranial and spinal cord magnetic stimulation in treatment of spasticity: a literature review and meta-analysis. *Eur J Phys Rehabil Med*. 2018;54(1):75-84. doi:10.23736/S1973-9087.16.04433-6
223. Mills PB, Dossa F. Transcutaneous electrical nerve stimulation for management of limb spasticity: a systematic review. *Am J Phys Med Rehabil*. 2016;95(4):309-318. doi:10.1097/PHM.0000000000000437
224. Fang CY, Lien AS, Tsai JL, et al. The effect and dose-response of functional electrical stimulation cycling training on spasticity in individuals with spinal cord injury: a systematic review with meta-analysis. *Front Physiol*. 2021;12:756200. doi:10.3389/fphys.2021.756200
225. Alcantara CC, Blanco J, De Oliveira LM, et al. Cryotherapy reduces muscle hypertonia, but does not affect lower limb strength or gait kinematics post-stroke: a randomized controlled crossover study. *Top Stroke Rehabil*. 2019;26(4):267-280. doi:10.1080/10749357.2019.1593613

226. Garcia LC, Alcântara CC, Santos GL, Monção JVA, Russo TL. Cryotherapy reduces muscle spasticity but does not affect proprioception in ischemic stroke: a randomized sham-controlled crossover study. *Am J Phys Med Rehabil*. 2019;98(1):51-57. doi:10.1097/PHM.0000000000001024
227. Winston P, MacRae F, Rajapakshe S, et al. Analysis of adverse effects of cryoneurolysis for the treatment of spasticity. *Am J Phys Med Rehabil*. 2023;102(11):1008-1013. doi:10.1097/PHM.0000000000002267
228. Gracies JM. Physical modalities other than stretch in spastic hypertonia. *Phys Med Rehabil Clin N Am*. 2001;12(4):769-vi.
229. Smania N, Picelli A, Munari D, et al. Rehabilitation procedures in the management of spasticity. *Eur J Phys Rehabil Med*. 2010;46(3):423-438.
230. Fernández-de-Las-Peñas C, Pérez-Bellmunt A, Llurda-Almuzara L, Plaza-Manzano G, De-la-Llave-Rincón AI, Navarro-Santana MJ. Is dry needling effective for the management of spasticity, pain, and motor function in post-stroke patients? A systematic review and meta-analysis. *Pain Med*. 2021;22(1):131-141. doi:10.1093/pm/pnaa392
231. Tang L, Li Y, Huang QM, Yang Y. Dry needling at myofascial trigger points mitigates chronic post-stroke shoulder spasticity. *Neural Regen Res*. 2018;13(4):673-676. doi:10.4103/1673-5374.230293
232. Hadi S, Khadijeh O, Hadian M, et al. The effect of dry needling on spasticity, gait and muscle architecture in patients with chronic stroke: a case series study. *Top Stroke Rehabil*. 2018;25(5):326-332. doi:10.1080/10749357.2018.1460946
233. Cai Y, Zhang CS, Liu S, et al. Electroacupuncture for poststroke spasticity: a systematic review and meta-analysis. *Arch Phys Med Rehabil*. 2017;98(12):2578-2589. doi:10.1016/j.apmr.2017.03.023
234. Zhu Y, Yang Y, Li J. Does acupuncture help patients with spasticity? A narrative review. *Ann Phys Rehabil Med*. 2019;62(4):297-301. doi:10.1016/j.rehab.2018.09.010
235. Howard IM, Patel AT. Spasticity evaluation and management tools. *Muscle Nerve*. 2023;67(4):272-283. doi:10.1002/mus.27792
236. Franzini A, Cordella R, Nazzi V, Broggi G. Long-term chronic stimulation of internal capsule in poststroke pain and spasticity. Case report, long-term results and review of the literature. *Stereotact Funct Neurosurg*. 2008;86(3):179-183. doi:10.1159/000120431

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