



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

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. Psychiatrists 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.

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 psychiatry and rehabilitation practices.

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 psychiatry 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.	A
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	
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

^aThe 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.	<ul style="list-style-type: none"> 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		<ul style="list-style-type: none"> 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		<ul style="list-style-type: none"> 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 fatigability.³¹
Upper limb function	Modified Frenchay Scale, Wolf Motor Function Test; Action Research Arm Test	<ul style="list-style-type: none"> 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	<ul style="list-style-type: none"> 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 non-surgical 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	<ul style="list-style-type: none"> • 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. 	<ul style="list-style-type: none"> • 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.	<ul style="list-style-type: none"> • 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 	<ul style="list-style-type: none"> • 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 administered 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.	<ul style="list-style-type: none"> • 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. 	<ul style="list-style-type: none"> • Alpha-2 agonist; use results in both direct impairment of excitatory amino acid release from spinal interneurons and a concomitant inhibition of facilitatory cerebrosplinal 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.	<ul style="list-style-type: none"> • Hepatotoxicity • Weakness • Fatigue • Drowsiness • Dizziness 	<ul style="list-style-type: none"> • 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 age⁵³; 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 baclofen⁸²; 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 blocks¹³² 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 scale¹¹¹ 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- μ g dose of baclofen is injected into the cerebrospinal fluid via lumbar puncture.¹⁵⁹ A 25- μ g 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 μ g 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)

	<ul style="list-style-type: none"> • 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	<ul style="list-style-type: none"> • 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	<ul style="list-style-type: none"> • 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	<ul style="list-style-type: none"> • 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	<ul style="list-style-type: none"> • 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	<ul style="list-style-type: none"> • 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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