Poststroke Spasticity Recognition and Treatment

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Disclosures

-Participated in spasticity advisory board

-Conducted research on botulinum toxin

-Filmed educational stretching videos to perform with spasticity

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Objectives

1. Participants will be able to identify post stroke spasticity to allow early identification
2. Participants will be able to name primary treatment objectives for post stroke spasticity
3. Participants will be able to name 3 different interventions to treat post stroke spasticity

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Outline/Agenda

Impact of stroke
Rehabilitation settings and team members
Post stroke spasticity pathophysiology
Tools for measuring spasticity and common patterns
Treatment modalities for post stroke spasticity
Barriers to treatment

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How a Stroke Affects the Body

Effects vary widely depending on stroke type and location in the brain

Nearly 800,000 people suffer a stroke annually in the US. Early recognition and treatment are crucial — "time is brain." Symptoms may include:

- Facial droop
- Arm/leg weakness
- Numbness & tingling
- Spasticity
- Muscle contracture
- Neglect (one side)
- Speech & swallowing difficulty
- Impaired vision
- Depression & cognitive issues

Source: Brown University Health Rehabilitation Services

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The Economic Costs of Stroke

Direct Costs	Indirect Costs
~$36 billion in direct medical costs (2019–2020)	~$20 billion in indirect costs from lost productivity
Rehabilitation care is a major driver of direct spending	Includes lost wages, reduced work capacity, and caregiver burden
Medicare is the primary payer; stroke is one of the most expensive conditions covered	Total US stroke costs exceed $56 billion annually

Medicare Advantage penetration is reshaping PAC access: MA plans cover >50% of beneficiaries in many states, with lower IRF utilization vs. fee-for-service Medicare. This shift impacts rehabilitation access and long-term outcomes.

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Postacute Care Settings for Stroke Rehabilitation

IRF
Inpatient Rehabilitation Facility
≥ 3 hrs/day, 5 days/wk of PT/OT/SLP. Physician-led multidisciplinary team. Preferred setting for patients with appropriate activity tolerance.

SNF
Skilled Nursing Facility
For patients unable to tolerate 15 hrs/wk therapy. Interdisciplinary PT, OT, SLP with weekly medical oversight. 24-hr nursing available.

HH
Home Health & Outpatient
PT, OT, SLP, nursing, & social work at home. Outpatient services are single-service encounters ordered by a clinician.

TR
Telerehabilitation & Day Rehab
Web-based therapy expanded during COVID-19. Day rehab offers multidisciplinary goal-oriented care up to 5x/week in outpatient settings.

Source: AHA/ASA Policy Statement, Ifejika et al., Stroke 2025

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What Drives Where Patients Go After Stroke?

GEOGRAPHIC FACTORS	INSURANCE FACTORS	CLINICAL FACTORS
Regional Variability	**Coverage Barriers**	**Patient Needs**
Key Drivers: • IRF/SNF availability varies by region (urban vs. rural) • Proximity to an IRF increases IRF utilization • Academic medical center affiliation boosts IRF use • Northeast vs. southeast disparities in facility density	**Key Drivers:** • Medicare Advantage vs. fee-for-service coverage differences • Prior authorization delays PAC access • Uninsured/Medicaid: 63% less likely to receive IRF care • Commercial plans have inequitable IRF coverage	**Key Drivers:** • Age, premorbid disability, functional level post-stroke • Caregiving availability and community support • No consensus algorithm for appropriate PAC level • Facility location and need to expedite patient flow
Impact	**Impact**	**Evidence**
• Rural residents more likely to have stroke • Stroke Belt: 2–4x higher mortality rates	• MA penetration lowers PAC utilization • Peer-to-peer reviews increase acute LOS	• IRF care linked to better functional outcomes • IRF associated with better cardiovascular outcomes for ICH

Adapted from Ifejika et al., AHA/ASA Policy Statement, Stroke 2025

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The Rehabilitation Team

A multidisciplinary team ensures the best treatment plan for each patient

Physical Therapy
Walking & mobility
Strength & endurance
Assistive devices & exoskeletons
Spasticity management

Occupational Therapy
Daily living activities
Personal hygiene & dressing
Return to work & hobbies
Cognitive rehabilitation
Spasticity management
Return to driving

Speech-Language Pathology
Swallowing assessment
Modified barium swallow studies
Speech & language therapy
Alternative communication tools
Cognition

Physiatry
Recovery guidance (acute → outpatient)
Nerve, muscle & brain disorders
Care navigation
Focus on independence & safety

Source: Brown University Health Rehabilitation Services

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The Scope of Poststroke Spasticity

A common, disabling, and undertreated complication

25–80%	1.8–5.6M	>100M	4×
of stroke survivors develop spasticity	affected in the United States alone	affected globally after stroke	higher cost of care when spasticity present

Consequences of Undertreated Spasticity

- Impaired functional mobility & independence
- Pain, disturbed sleep, reduced rehab participation
- Joint contractures & fixed deformity
- Skin breakdown & pressure injury risk
- Increased caregiver burden
- Preventable long-term disability

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Understanding Spasticity: A Multidomain Syndrome

Three interacting domains degrade functional mobility

1 Involuntary Muscle Overactivity

- Velocity-dependent stretch reflex hyperreflexia
- Clonus (rhythmic contractions)
- Postural hypertonia / spastic dystonia
- Involuntary muscle spasms

2 Impaired Voluntary Motor Control

- Weakness in proximal-to-distal gradient
- Loss of selective motor control
- Flexion & extension synergy patterns
- Effort-dependent movement limitations

3 Passive Tissue Remodeling

- Muscle stiffness & shortening
- Fibrosis of muscle & joint capsules
- Hyaluronan accumulation
- Fixed joint contractures

Together these three domains form a self-reinforcing triad driving chronic disability.

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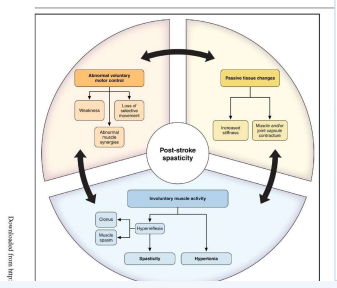
Figure 1. Spectrum of Motor Dysfunction in Poststroke Spasticity



Key Concept

Spasticity is narrowly defined as velocity-dependent hyperactivity after rapid muscle movement. The full clinical syndrome is broader and encompasses three interacting domains:

Abnormal Voluntary Motor Control

Weakness, loss of selective movement, abnormal muscle synergies

Involuntary Muscle Activity

Hyperreflexia, clonus, muscle spasm, hypertonia, and spasticity

Passive Tissue Changes

Increased stiffness and muscle/joint capsule contracture

Source: Bandela et al. Stroke. 2026;57:e146–e159

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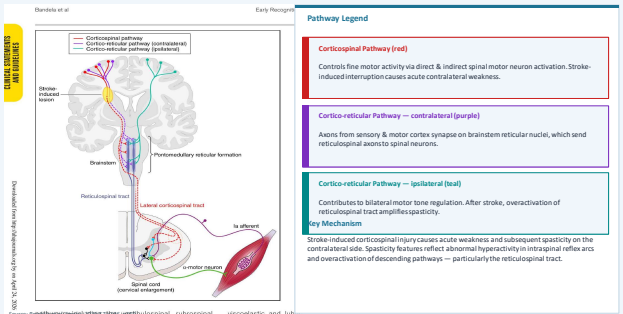
Figure 2. Proposed Mechanisms of Supraspinal Involvement in Poststroke Spasticity



Pathway Legend

Corticospinal Pathway (red)

Controls fine motor activity via direct & indirect spinal motor neuron activation. Stroke-induced interruption causes acute contralateral weakness.

Cortico-reticular Pathway — contralateral (purple)

Axons from sensory & motor cortex synapse on brainstem reticular nuclei, which send reticulospinal axons to spinal neurons.

Cortico-reticular Pathway — ipsilateral (teal)

Contributes to bilateral motor tone regulation. After stroke, overactivation of reticulospinal tract amplifies spasticity.

Key Mechanism

Stroke-induced corticospinal injury causes acute weakness and subsequent spasticity on the contralateral side. Spasticity features reflect abnormal hyperactivity in intraspinal reflex arcs and overactivation of descending pathways — particularly the reticulospinal tract.

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Pathophysiology of Poststroke Spasticity

Understanding the neural and biomechanical mechanisms

01 Corticospinal Tract Injury

Stroke interrupts descending corticospinal fibers → immediate motor weakness (paresis). This is the primary driver of early motor deficits.

02 Spinal Reflex Hyperexcitability

Days to weeks post-stroke: abnormal facilitation of intraspinal monosynaptic circuits (Ia/Ib afferents → motor neurons) produces hyperreflexia — the hallmark of spasticity.

03 Brainstem Pathway Dysregulation

Pathologic overactivation of reticulospinal and other extrapyramidal tracts amplifies motor tone dysregulation. The reticulospinal tract receives particular attention.

04 Secondary Tissue Remodeling

Elevated hyaluronan & collagen accumulation → muscle stiffness, fibrosis, joint contractures. These biomechanical changes persist even after neuromodulatory treatment.

Animal models remain limited — rodent neural pathways differ from humans, hindering translation of mechanistic insights to clinical practice.

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Upper Extremity Post-Stroke Spasticity

Characteristic posture patterns and clinical presentation

Image: Upper extremity spasticity pattern post-stroke

Typical Posture Pattern

- **Shoulder**
  Adduction and internal rotation
- **Elbow**
  Flexion and pronation
- **Wrist**
  Flexion and ulnar deviation
- **Fingers**
  Flexion; clenched fist
- **Thumb**
  Adduction; thumb-in-palm

~43% of stroke patients develop spasticity; 15.6% severe

Source: StatPearls (NCBI); Brown University Health Rehabilitation Services

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Lower Extremity Post-Stroke Spasticity

Characteristic posture patterns and functional impact on gait and mobility

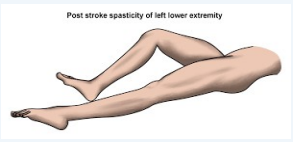

Image: Lower extremity spasticity pattern post-stroke

Typical Posture Pattern

- **Hip**
  Adduction and internal rotation
- **Knee**
  Extension (stiff-knee gait)
- **Ankle**
  Plantarflexion and inversion (equinovarus)
- **Toes**
  Flexion or clawing; striatal toe
- **Gait**
  Circumduction pattern; reduced clearance

LE spasticity can aid standing transfers but impairs gait safety

Source: StatPearls (NCBI); Brown University Health Rehabilitation Services

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Assessment & Diagnosis of Spasticity

Clinical scales, electrophysiology, and emerging technologies

Clinical Rating Tools

Modified Ashworth Scale (MAS)

Most widely used; grades resistance to slow passive movement. Score ≥1 indicates spasticity. Rapid but subjective; does not account for velocity dependence.

Modified Tardieu Scale

More precise: tests at multiple velocities & measures catch angle. Better assesses reflex hyperexcitability. Less used in routine settings due to time required.

5-Step Functional Framework

Evaluates passive movement, active movement, fatigability, and upper & lower limb function. Integrates real-world functional impact of spasticity.

Emerging Objective Technologies

- Biomechanical sensors — quantify movement resistance and joint stiffness
- Surface electromyography — detect myoelectric activity during passive stretch
- Automated motion analysis — provide detailed kinematic data
- H-reflex amplitude & reciprocal inhibition studies — neurophysiologic assessment
- Imaging-based predictors — identify lesion patterns associated with spasticity risk
- Wearable IMU + sEMG — continuous at-home monitoring during subacute recovery

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Modified Ashworth Scale (MAS)

The most widely used clinical tool for measuring increased muscle tone

Grade	Description	Clinical Meaning
0	No increase in muscle tone	Normal tone
1	Slight increase; catch and release or minimal resistance at end of ROM	Mild spasticity
1+	Slight increase; catch, then minimal resistance through less than half of ROM	Mild-moderate
2	More marked increase through most of ROM, but part easily moved	Moderate spasticity
3	Considerable increase in tone; passive movement difficult	Severe spasticity
4	Affected part rigid in flexion or extension	Rigidity

Developed by Bohannon & Smith (1987) as a revision of the original Ashworth Scale (1964). Assessed by moving a joint through passive ROM. Used by physiatrists, neurologists, PTs, and OTs. ~43% of stroke patients develop spasticity.

Source: Bohannon & Smith, 1987; StatPearls (NCBI)

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Post-Stroke Spasticity Risk Classification: Traffic Light System

LOW RISK Green	MEDIUM RISK Amber	HIGH RISK Red
Periodic Monitoring	**Routine Referral**	**Urgent Referral**
Risk Indicators: • Persistent dexterity problems • Absence of increased tone • Re-evaluate at 3–6 months	**Risk Indicators:** • MAS score ≥1 at 1–14 days • Left-sided weakness or paresis • Involuntary muscle contractions • Reduced sensitivity (1–5 days) • Left-sided visual inattention	**Risk Indicators:** • MAS ≥2 in 1 joint or ≥1 in 2+ joints • Upper limb weakness / finger dexterity problems • Reduced sensorimotor function • Functional impairment in daily living
Next Steps • Refer to community physical therapist • Provide patient/carer information • Enable self-referral if needed	**Next Steps** • Refer to multidisciplinary team • Follow-up at 6 weeks then regularly • Initiate physical therapy	**Next Steps** • Immediate spasticity specialist referral • Urgently initiate physical therapy • Access to full MDT & first-line treatments

Note: A patient's risk level is determined by their HIGHEST ranked risk factor.

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The Case for Early Intervention

Defined as treatment initiated within the first 3 months after stroke onset

Critical Window for Neuroplasticity

The first 3–6 months is when intensive task-specific training is most effective. Spasticity can emerge within weeks, interfering with voluntary movement and rehab participation. Early treatment during peak plasticity may optimize long-term gains.

Prevent Secondary Musculoskeletal Changes

Severe weakness and postural hypertonia → prolonged immobility → loss of sarcomeres, decreased muscle fascicle length, and increased stiffness. Early mobility interventions can reduce contracture risk and preserve range of motion.

Reduce Long-Term Disability & Costs

Delayed treatment leads to pain, progressive motor dysfunction, and caregiver burden. Early botulinum toxin was found to significantly reduce contracture treatment costs without increasing overall costs of care.

Key caveat: Targeting spasticity alone may not improve function if underlying weakness is not simultaneously addressed. Integrated, adaptive interventions are essential.

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Can Spasticity Be Prevented?

Identifying high-risk individuals and intervening proactively

High-Risk Indicators

- Severe initial motor paresis
- Early hyperreflexia in the acute phase
- Hemorrhagic stroke or anterior circulation ischemic stroke
- Lesions involving internal capsule, basal ganglia, or brainstem
- Stroke lesion diameter > 3 cm
- NIHSS score ≥ 10
- Cortical or subcortical lesion affecting corticospinal tract

Preventive Strategies

- Mobilization within 24–72 hours of stroke onset
- Passive & active range-of-motion exercises
- Robotic-assisted therapy for proximal joint stabilization
- Stretching devices and positioning orthoses
- Early botulinum toxin combined with motor training

(shown superior to delayed or separate use)

- Pharmacologic treatment within first 3 months may

reduce risk of persistent spasticity

- Early interdisciplinary risk-stratified management

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Evidence-Based Management of Poststroke Spasticity

Summary across three primary treatment domains

Involuntary Muscle Activation		Abnormal Voluntary Control		Passive Tissue Changes	
Botulinum Toxin A (BoNT-A)	A*	**Task-Specific Training**	A*	**Early Mobilization & Weight-Bearing**	A*
First-line; reduces MAS consistently; early use with ultrasound guidance enhances outcomes		Improves motor control and function; strong early evidence		Prevents contractures; reduce hyperreflexia when started early	
Intrathecal Baclofen	A	**Constraint-Induced Movement Therapy**	A	**Extracorporeal Shockwave Therapy**	A
Effective for generalized spasticity; requires monitoring		Best for higher-functioning patients		Short-term MAS reduction; radial may be superior	
Transcutaneous Electrical Nerve Stim.	A	**Functional Electrical Stimulation**	A*/	**Positioning Orthoses / Serial Casting**	A/B
Effective in lower limbs; upper limb efficacy unclear		Enhances voluntary activation; timing matters		Modest MAS improvements; prevents contracture progression	
Oral Antispasticity Agents	B/C	**Noninvasive Brain Stimulation**	A	**Hyaluronidase**	Emerging
Modest tone reduction; limited by side effects		tDCS/rTMS; strongest support in upper limbs		Improves MAS & ROM in subacute/chronic spasticity	

** Evidence of effectiveness when given early (<3 months from stroke onset) — Tilborg et al. 2025*

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Intrathecal Baclofen (ITB) for Spasticity

What Is ITB?

- Programmable pump delivers baclofen directly into the intrathecal space
- Bypasses blood-brain barrier for targeted spinal cord action
- GABA-B receptor agonist reducing excitatory neurotransmission

Indications

- Severe, generalized spasticity unresponsive to oral medications
- Both upper and lower limb spasticity of spinal and cerebral origin
- Positive response to trial bolus injection required before implant

Procedure & Dosing

- Surgically implanted pump with intrathecal catheter
- Dose titrated over weeks to months for optimal response
- Pump reservoir refilled every 1–6 months depending on dosage

Benefits & Risks

- Reduces tone with fewer systemic side effects vs. oral baclofen
- Improves comfort, hygiene, positioning, and ease of care
- Risks: infection, catheter malfunction, withdrawal syndrome

Key Considerations

- Requires long-term follow-up and pump maintenance; abrupt withdrawal can be life-threatening
- Best outcomes when combined with ongoing rehabilitation and therapy
- May be used in combination with botulinum toxin for focal spasticity

Source: Bandela et al., Stroke 2026; Bavikatte et al., J Cent Nerv Syst Dis 2021

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Botulinum Toxin for Spasticity Treatment

What Is Botulinum Toxin?

•Neurotoxin that blocks acetylcholine release at the neuromuscular junction
•Reduces involuntary muscle overactivity without systemic sedation
•FDA-approved formulations: onabotulinumtoxinA, abobotulinumtoxinA, incobotulinumtoxinA

How It Works

•Injected directly into spastic muscles under guidance (EMG, ultrasound, or e-stim)
•Onset: 3–7 days; peak effect: 2–6 weeks
•Duration: ~12 weeks; repeat injections every 3–6 months

Clinical Evidence

•Strongest evidence among pharmacological interventions (Cochrane review)
•Reduces muscle tone (MAS), improves ROM and functional goals
•Best outcomes when combined with rehabilitation (stretching, splinting, task-specific training)

Treatment Goals

•Reduce pain and discomfort from spastic muscles
•Improve functional mobility, ADLs, and quality of life
•Prevent contractures and secondary musculoskeletal complications

Key Considerations

•Early intervention (within 3 months post-stroke) may prevent development of fixed contractures
•Goal-oriented treatment planning using GAS (Goal Attainment Scaling) improves outcomes
•Multidisciplinary approach essential: physiatry, PT, OT, and nursing collaboration

Sources: Bandela et al., Stroke 2026; Francisco et al., J Rehabil Med 2021; Bavikatte et al., J CNS Dis 2021

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Treatment Evidence: Botulinum Toxin

ASPIRE Study (Adult Spasticity International Registry) — 1-year follow-up, n=731

85%	of patients reported satisfaction with botulinum toxin for spasticity relief	**93%**	of clinicians expressed satisfaction with treatment outcomes
91%	of patients would definitely/probably continue treatment	**98%**	of physicians would recommend continuing botulinum toxin

Adjuvant Therapies That Enhance Botulinum Toxin Outcomes:

- Extracorporeal shock wave therapy — improved spasticity and pain outcomes vs electrical stimulation alone
- Electrical stimulation of injected muscles — may boost botulinum toxin efficacy (optimal protocol TBD)
- Casting and adhesive taping — casting provides better spasticity, range of motion, and gait outcomes

Evidence note: Cochrane review found botulinum toxin has the strongest evidence among pharmacological interventions for post-stroke spasticity

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Botulinum Toxin

- BoNT-A has been used for post-stroke spasticity for ~30 years — accepted standard of care for focal spasticity
- Safe and effective for upper and lower limb spasticity with active and passive functional gains
- First-line pharmacological treatment for focal and multi-focal spasticity within a multidisciplinary team (MDT) approach
- Based on extensive literature searches and clinical experience consensus

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Individualized Approach: Goal-Setting

- Patient-centred collaborative approach — agree goals with patients and caregivers
- Goal Attainment Scaling (GAS): tailored individual goals, sensitive to changes in symptoms, impairment, and function
- ~50% of expert group use GAS always or often
- Start with a modest list of goals; reassess and extend over time as results become apparent
- Goals must be well-defined (e.g. reduction in pain intensity vs. duration)
- Re-evaluate and re-prioritize goals throughout the continuum of care

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Treatment Timing & Frequency

- Earlier BoNT-A treatment post-stroke achieves better outcomes than delayed treatment
- Duration of effect varies by patient — saturates at approximately 3 months at higher doses
- 79% of patients preferred shorter injection intervals (35.5% at 12 weeks)
- Patient satisfaction decreases as effects wear off — the "roller-coaster effect"
- Flexible re-injection: treat before emergence of problems

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Adjunctive Therapies Overview

- BoNT-A injections should always be followed with a physical therapy program
- High-level evidence suggests adjunct therapies may improve outcomes post-injection

Current Use by Expert Group (n = 19):

- **Skilled physical therapy** — used "most" or "almost always" by 89.5%
- **Self-rehabilitation** — used "most" or "almost always" by 89.5%
- **Orthosis** — used "sometimes" or "most" by 89.5%
- **FES** — used rarely (63.2%) or sometimes (36.8%)
- **ESWT** — rarely available (94.7% never)

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Casting, Splinting & Taping

- Lower limb: BoNT-A + casting gives better and longer-lasting results than stretching alone
- Prolonged stretching via night cast (4 months) enhances long-lasting therapeutic benefit of BoNT-A
- Serial casting may prevent equinovarus deformity and improve walking quality in chronic stroke
- Upper limb: adhesive taping more effective than manual stretching + palmar splint; Recommended if available, especially with high risk of soft-tissue shortening
- Critical need for more robust RCTs with detailed injection and casting/taping protocols

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Orthotic Management of Post-Stroke Spasticity

AFO, PRAFO, and WHO devices for lower and upper extremity spasticity control

AFO

Ankle-Foot Orthosis

Ankle / Foot

- Manages foot drop and equinovarus posture during gait
- Provides mediolateral ankle stability during stance phase
- Improves gait speed, cadence, stride length, and dynamic balance
- Available as rigid, hinged, or posterior leaf-spring designs
- Prolonged stretching effect on plantarflexor muscles

PRAFO

Pressure Relief AFO

Ankle / Heel

- Offloads heel pressure to prevent ulceration in immobile patients
- Resists development of plantarflexion contractures at night
- Metal upright prevents displacement during patient movement
- Can serve as evaluation orthosis immediately post-stroke
- Dual-purpose: pressure relief and early mobilization support

WHO

Wrist-Hand Orthosis

Wrist / Hand

- Stabilizes wrist and fingers in functional position
- Provides passive stretching to reduce wrist flexor spasticity
- Prevents clenched fist deformity and flexion contractures
- Available as static (resting) or dynamic (functional) designs
- Supports hygiene maintenance and pain reduction

Source: Doucet et al., Phys Ther 2021; Anatomical Concepts Inc.; Lannin & Herbert, Cochrane Database; StatPearls (NCBI)

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Other Adjunctive Therapies

- **CIMT** Significant improvements at 6 months in spasticity (MAS), arm utilization (MAL), and mobility (ARAT) when combined with BoNT-A. Recommended post-injection.
- **ESWT** Reduces spasticity alone and with BoNT-A. Three sessions provide longer effect (16 vs. 8–12 weeks). BoNT-A + ESWT more effective than BoNT-A + ES in forearm.
- **FES/ES** Level I evidence for MAS improvement by at least one grade as adjunct. Optimal protocols (Hz, timing, duration) remain unclear. Recommended if available.
- **Robotics** Too early for formal recommendations. Intensive early rehab may prevent later spasticity. Likely to gain importance as repetition is key.
- **Self-Rehab** Positive benefits with task-oriented exercise, home-based and tele-rehabilitation. Recommended to supplement clinician-administered physiotherapy.

Francisco et al. • Journal of Rehabilitation Medicine • 2021

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Key Consensus Statements

- ✓ Patient-centred collaborative approach with individualized goals using GAS
- ✓ Higher doses of botulinum toxin are safe and enable more treatment goals
- ✓ Flexible re-injection intervals based on patient needs, not rigid 12-week schedules
- ✓ Use instrumental injection guidance (EMG / ES / US) to optimize muscle localization
- ✓ Always combine BoNT-A with physical therapy and adjunctive therapies
- ✓ Self-rehabilitation programmes should supplement clinician-administered physical therapy

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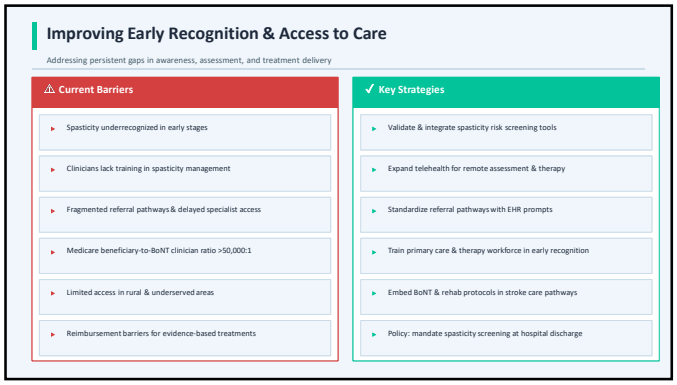
Improving Early Recognition & Access to Care
Addressing persistent gaps in awareness, assessment, and treatment delivery
⚠ Current Barriers
Spasticity underrecognized in early stages
Clinicians lack training in spasticity management
Fragmented referral pathways & delayed specialist access
Medicare beneficiary-to-BoNT clinician ratio >50,000:1
Limited access in rural & underserved areas
Reimbursement barriers for evidence-based treatments
✓ Key Strategies
Validate & integrate spasticity risk screening tools
Expand telehealth for remote assessment & therapy
Standardize referral pathways with EHR prompts
Train primary care & therapy workforce in early recognition
Embed BoNT & rehab protocols in stroke care pathways
Policy: mandate spasticity screening at hospital discharge

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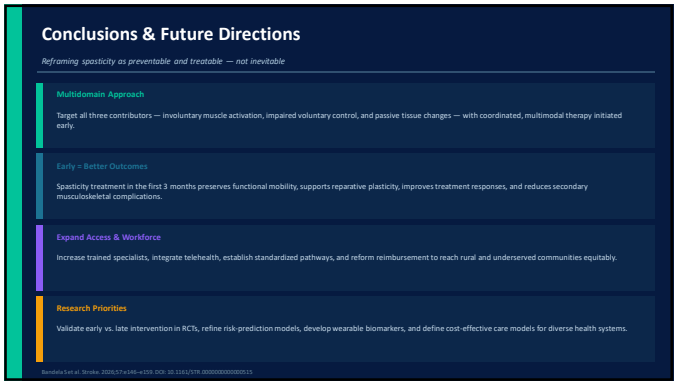
Conclusions & Future Directions
Reframing spasticity as preventable and treatable — not inevitable
Multidomain Approach
Target all three contributors — involuntary muscle activation, impaired voluntary control, and passive tissue changes — with coordinated, multimodal therapy initiated early.
Early = Better Outcomes
Spasticity treatment in the first 3 months preserves functional mobility, supports reparative plasticity, improves treatment responses, and reduces secondary musculoskeletal complications.
Expand Access & Workforce
Increase trained specialists, integrate telehealth, establish standardized pathways, and reform reimbursement to reach rural and underserved communities equitably.
Research Priorities
Validate early vs. late intervention in RCTs, refine risk-prediction models, develop wearable biomarkers, and define cost-effective care models for diverse health systems.

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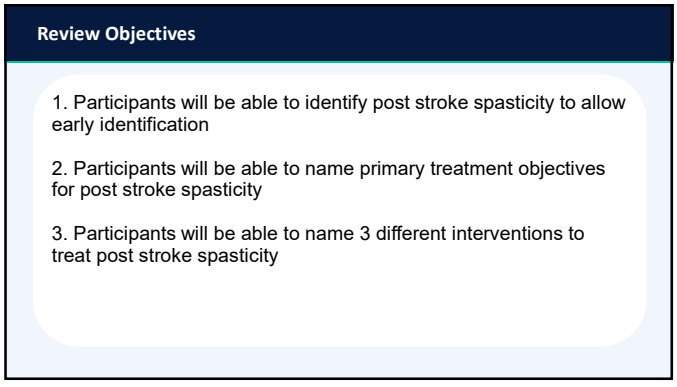
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Questions ???

Thank You

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References

Bandela S, McPherson L, Harvey RL, Awosika O, Aggarwal D, Liu CY, Raghavan P, Goldberg MP; on behalf of the American Heart Association Stroke Council; Council on Cardiovascular and Stroke Nursing; Council on Basic Cardiovascular Sciences; and Council on Lifestyle and CardiometabolicHealth. Early recognition and intervention for poststroke spasticity: a scientific statement from the American Heart Association. *Stroke*. 2026;57:e146–e159.

Bavikatte G, Subramanian G, Ashford S, Allison R, Hicklin D. Early identification, intervention and management of post-stroke spasticity: expert consensus recommendations. *J Cent Nerv Syst Dis*. 2021;13:1–8.

Brown University Health. Stroke rehabilitation and recovery. Brown University Health website. https://www.brownhealth.org/centers-services/rehabilitation-services. Accessed 2026.

Campanini I, Bò MC, Salsi F, Bassi MC, Damiano B, Scaltriti S, Lusuardi M, Merlo A. Physical therapy interventions for the correction of equinus foot deformity in post-stroke patients with triceps spasticity: a scoping review. *Front Neurol*. 2022;13:1026850.

Francisco GE, Balbert A, Bavikatte G, Bensmail D, Carda S, Deltombe T, Draulans N, Escaldi S, Gross R, Jacinto J, Ketchum N, Molteni F, Moraleda S, O'Dell MW, Reebye R, Sätterö P, Verduzco-Gutierrez M, Walker H, Wissel J. A practical guide to optimizing the benefits of post-stroke spasticity interventions with botulinum toxin A: an international group consensus. *J Rehabil Med*. 2021;53:jrm00134.

Ifejika NL, Awosika OO, Black T, Duncan PW, Harvey RL, Katz DI, Kimberley TJ, Lutz B, O'Neil F, Stein J, Yallapragada AV, Reeves MJ. Improving access to stroke rehabilitation and recovery: a policy statement from the American Heart Association/American Stroke Association. *Stroke*. 2025;56:e218–e233.

Kim W-S, Abo M, Soekadar SR, Pistarini C. Editorial: Transitional and long-term continuous care & rehabilitation after stroke. *Front Neurol*. 2022;13:965762.

Lee KB, Hong BY, Kim JS, Sul B, Yoon SC, Ji E-K, Son DB, Hwang BY, Lim SH. Which brain lesions produce spasticity? An observational study on 45 stroke patients. *PLoS One*. 2019;14(1):e0210038.

Marshall I, McKevitt C, Wang Y, Wafa H, Skolarus L, Bhalla A, Muruet-Gutierrez W, Emmett E, Sommerville P, Birns J, Sackley C, Clarke S, Hamidi F, Stevens E, Rudd AG, Rodgers H, Wolfe CDA. Stroke pathway — an evidence base for commissioning — an evidence review for NHS England and NHS Improvement. *NIHR Open Res*. 2022;2:43.

Morone G, Baricich A, Paolucci S, Bentivoglio AR, De Blasis P, Carlucci M, Violi F, Levato G, Pani M, Carpagnano LF, Spandonaro F, Picelli A, Smania N. Long-term spasticity management in post-stroke patients: issues and possible actions—a systematic review with an Italian expert opinion. *Healthcare*. 2023;11(6):783.

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