WELCOME!

We will start in a few minutes...
WSA WEBINAR
Advancing the Management of Spasticity

February 22nd, 2024 | 2:00 PM CET
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UP NEXT

Spasticity After stroke: Treatment intensification among patients with unmet needs

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Spasticity After Stroke: Treatment Intensification Among Patients With Unmet Needs

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www.world-stroke-academy.org
Learning Objectives

1. Explore *innovative approaches to intensify the treatment of spasticity* after stroke, focusing on patients with unmet needs and evolving clinical paradigms.

2. Gain insights into *adapting spasticity management for special populations*, including the elderly and individuals with limited access to healthcare resources, considering factors like frailty and unique challenges.

3. Recognize the importance of *multidisciplinary collaboration* in optimizing spasticity management and learn how different healthcare professionals can contribute to improved patient outcomes.
Post-Stroke Spasticity: Defined

• Involuntary muscle activity in central paresis
• Affected by slow or rapid passive joint movement or sensory stimulation\(^1\)
• Present in 25\% of stroke survivors\(^2\)
  • 39.5\% of stroke survivors with paresis
    • Almost 10\% of which developed severe or disabling spasticity

\(^1\) Source: www.world-stroke-academy.org
Why focus on spasticity?¹, ²

- Functional impact
  - Ambulation
  - ADLs
- Hygiene
- Pain
- Musculoskeletal issues (*more pain*)
  - Posture
  - Tendon/muscle/ligament length
- Nerve entrapment (*even more pain*)
How do we address spasticity?

As a multidisciplinary team
Rehabilitation Interventions

- Education
- Physical Intervention
- Positioning
## Rehabilitation Interventions

<table>
<thead>
<tr>
<th>Education for self-management$^3$</th>
<th>Physical Intervention$^3$-$^7$</th>
<th>Positioning$^3$, $^6$-$^7$</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Exercises</td>
<td>• Active/passive movement</td>
<td>• Daytime</td>
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<tr>
<td>• Stretches</td>
<td>• Weight bearing (altering</td>
<td>• Nighttime</td>
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<tr>
<td>• Positioning devices</td>
<td>sensory input)</td>
<td>• Serial casting</td>
</tr>
<tr>
<td>• Identifying triggers</td>
<td>• Strengthening (paired with</td>
<td></td>
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<td></td>
<td>chemodenervation and electrical</td>
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<td></td>
<td>stimulation)</td>
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<tr>
<td></td>
<td>• Prolonged stretch (paired</td>
<td></td>
</tr>
<tr>
<td></td>
<td>with positioning)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Aquatic Therapy</td>
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Patients with Unmet Needs

• Stroke survivors in chronic stages need intervention too
  • Refer for outpatient or home-health OT/PT

• Consider in-home or community-based interventions
  • Extension of therapists: personal trainers, community health workers

• Educate, educate, educate

• Think outside the box!
References


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POLLS
Management of spasticity in special populations: elderly, limited access, frailty

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Management of spasticity in special populations: elderly, limited access, frailty

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Introduction


• How does management differ in special populations?
• Special populations
  : elderly who are frail,
  : living in nursing homes, or
  : have limited access to treatments
Learning Objectives

At the end of the lecture, you would be able to

• outline issues with spasticity management in special populations
• recognise scenarios where spasticity management is warranted in special populations
• summarise spasticity management strategies in special populations
Issues with spasticity management in special populations

• There is some certainty about WHAT to do with spasticity, but less certainty about WHO and WHERE once stroke survivor leaves the hospital

• **WHAT:** Botulinum Toxin A, Rehabilitation therapy, Adjunct therapies

• **WHO:** Who will screen and refer in the community? Who to refer to?

• **WHERE:** Where will care be provided? Should we develop primary care services (in community), or improve access to specialist services (in hospitals)?

Scenarios where spasticity management is warranted in special populations

• Is spasticity causing difficulties in activities (e.g., standing, walking)?
• Is spasticity causing pain or discomfort (e.g., cosmesis reasons)?
• Is spasticity causing increase in caregiver burden (e.g., showering, dressing)?

Expert consensus: Spasticity is a specialized problem post-stroke and preference is referral to a specialist stroke service/spasticity clinic if the patient is affected by the symptoms.
SR of guidelines used AGRE-IIL to appraise and identify high quality clinical practice guidelines. They include

- [Stroke] WSO Guidelines
- [Stroke] SF Guidelines (Australia)
- [Frailty] ICFSR Guidelines

Guideline recommendations on Spasticity post-stroke

<table>
<thead>
<tr>
<th>[Guideline] Recommendations</th>
<th>Rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>[World Stroke Organization] Chemo-denervation using botulinum toxin can be used to increase range of motion and decrease pain for patients with focal symptomatically distressing spasticity (upper and lower limbs)</td>
<td>Advanced system</td>
</tr>
<tr>
<td>[Stroke Foundation, Australia] • For stroke survivors with <strong>upper</strong> limb spasticity, Botulinum Toxin A in addition to rehabilitation therapy may be used to reduce spasticity, but is unlikely to improve activity or motor function. • For stroke survivors with <strong>lower</strong> limb spasticity, Botulinum Toxin A in addition to rehabilitation therapy may be used to reduce spasticity but is unlikely to improve motor function or walking. • For stroke survivors with spasticity, adjunct therapies to Botulinum Toxin A, such as electrical stimulation, casting and taping, may be used.</td>
<td>Weak For</td>
</tr>
<tr>
<td>[Stroke Foundation, Australia] • For stroke survivors with spasticity, acupuncture should not be used for treatment of spasticity in routine practice other than as part of a research study. (Lim et al 2015 [54]) • For stroke survivors, the routine use of stretch to reduce spasticity is not recommended.</td>
<td>Weak Against</td>
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</table>

Screen with validated rapid frailty instrument
Assess with clinical assessment
Treat with comprehensive management plan (multi-component, including social support)

Spasticity management strategies in special populations

- Botulinum Toxin A
- Rehabilitation therapy
- Adjunct therapies (e.g., electrical stimulation, casting and taping) +
- Comprehensive frailty management plan (*targeting sarcopenia, exhaustion, polypharmacy and other conditions)

Spasticity management strategies in special populations

✓ Checklists (e.g., Post-Stroke Checklist, Clinical Frailty Scale)
✓ Connector (e.g., community care staff, family, peers)
✓ Connecting system (e.g., map of local services and how to access, communication and/or referral pathways)

? Develop primary care services


Spasticity management strategies in special populations

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? Develop primary care services

Take Away Messages

• The biggest issues surrounding spasticity management in special populations is with detection of spasticity (“who”) and location of management (“where”).

• Spasticity management is warranted if spasticity is causing difficulties in activities, pain or discomfort, and increase in caregiver burden.

• In addition to Botulinum Toxin A, rehabilitation and adjunct therapies, management in the community should also include screening with “checklists”, identifying person to screen/detect and refer (“connector”), and creating resources/pathways to aid referral back to stroke specialist services (“connecting system”).
References

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Beyond Chemodenervation: Effective co-adjuvant therapies in the management of spasticity after stroke

Dr. Deniz Dishman
Faculty appointment, Department of Research at Cizik School of Nursing
Lead in Post-Stroke Pain Management Program
UTHealth Houston - Institute for Stroke and Cerebrovascular Disease
POLLS
Beyond Chemodenervation

Deniz Dishman, PhD, CRNA, NSPM-C

UTHealth Science at Houston
Institute for Stroke and Cerebrovascular Disease

www.world-stroke-academy.org
Introduction

Deniz Dishman holds a faculty appointment at the University of Texas Health Science Center at Houston in the Department of Research at Cizik School of Nursing. She also is leading the Post-Stroke Pain Management Program at UTHealth Science at Houston Institute for Stroke and Cerebrovascular Disease. Dr. Dishman’s research aims to improve post-stroke pain assessment and management, including the discovery of novel pain treatments.
Learning Objectives

• Describe the tight coupling between spasticity and pain.
• Identify non-pharmacologic co-adjuvant therapies in spasticity management
• Describe the evidence supporting the use of non-pharmacologic treatment modalities
• Describe the benefits and role of ultrasound imaging for precise, focal spasticity management
• Identify interventional modalities that may be useful as a co-adjuvant therapy in spasticity treatment
Chemodenervation

• Botulinum toxin (BoNT) – high level of evidence for its effectiveness in spasticity.¹ Most widely used over the past 30+ years.

• Induces relaxation - inhibits the presynaptic release of acetylcholine which keeps the muscle from contracting.

• Provides analgesia – proposed mechanisms include blockade of the cholinergic transmission in the nociceptive system, interaction with TRPV1 receptors, and inhibition of substance P, glutamate, and CRGP synaptic release, which are excitatory neurotransmitters that influence pain generation and transmission.²

• There is a large amount of data of its use in the chronic phases after stroke but further investigation is needed in the acute and subacute phases of stroke.²
• Guided ultrasound injection allows for more precise injection at target muscles – minimizes spread and subsequent weakness of nearby muscles. Better outcomes with US over electrical nerve stimulation.³
  • Lungo et al. 2022 found better reported outcomes in VAS, discomfort, and weakness in a 2-center RCT crossover study of BoNT-A injections using ultrasound vs. electrical nerve stimulation.³

• Asimakidou and Sidiropoulos (2023) - Bayesian Network Meta Analysis/SR showed that US is the best method to guide BoNT injections in limb spasticity, followed by ES and EMG.²
  • All three approaches were superior to manual needle placement based on surface anatomy with regard to the clinical outcome as assessed by MAS at 2 to 4 weeks after BoNT treatment of limb spasticity in adults.²

• 600u dosage limitation – if large muscle groups need to be treated then use in tandem with another therapy, i.e. stretching, casting, and more recently used techniques such as extracorporeal shock wave therapy
  • Intiso et al (2023) – Systematic Review investigated relationship between high dosing and improved function and analgesia – insufficient evidence⁴
Chemical Neurolysis – Phenol and Alcohol

• Phenol and alcohol neurolysis used for many years – pain and spasticity (spasticity treatment requires higher concentration than anesthetic doses). Works at alpha motorneurons – causes Wallerian degeneration of the axon.

• Faster onset (minutes vs 1 week), effects last longer than botulinum – months rather than weeks (3 to 9 months – depends on axonal regrowth)

• Ultrasound nerve identification decreases potential adverse effects i.e. loss of sensation, chemical neuritis, dyesthesia, neuropathic pain.\(^7\)

• Further studies needed to identify optimal dosing.\(^8\)
Adjuvant Therapies

Acupuncture – all types including using electrical stimulation, acupressure
• Yi et al (2024): Overview of systematic reviews - much variability (and weakness) in methods and reporting – weak evidence despite many clinical trials.  

Electroshock Waves – high-pressure air wave - targeted to specific location.
• Yang, Lew, Ozcakar, Wu (2021) – Systematic review showed ESWT has prominent/direct effects on spasticity parameters such as MAS and MTS scores; however, mixed results were shown regarding functional recovery. No standardized treatment.  

Repetitive Transcranial Magnetic Stimulation (rTMS)
• Xu et al (2020) SR/MA no significant improvement in MAS over sham treatment but subjects did demonstrate a change in MAS over the course of treatment.  

Transcranial Direct Stimulation (tDCS)
• Alsharam et al (2022) – Systematic review showed limited evidence and unclear treatment dosage among RCTs.  

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Cryoneurolysis – “freeze” therapy

Uses specialized probe capable of freezing ranging from -60 ° to -90 °C - Joule-Thomson effect (compression of gas through narrow aperture), depending on the type of gas used as the cryogen.

Tip of the cold probe causes body fluid to generate a ball of ice.

Rapid plunge in temperature causes Wallerian degeneration of targeted nerve, causing a secondary axonotmesis.

Epineurium and perineurium maintained - allows for axonal regeneration.

Blood vessels and surrounding tissues not affected - their freezing occurs at lower temperatures.

Axon will regrow after 3–6mos
• Winston et al (2023) prospective observational study followed subjects with spasticity over 1 year following therapy.\textsuperscript{11}

• 113 patients treated – ongoing study (only adverse event reporting)

• 9 (3.25\%) had dysesthesia attributed to application to mixed motor/sensory nerve – mostly transient and did not warrant treatment.

| TABLE 3. Summary of adverse effects reported from all participants in all clinical trials |
|-----------------------------------------------|--------|------------------|-----------------------------------------------|-----------------------------------------------|
| Adverse Effect                               | No. Patients Affected | Duration of Symptoms | Treated Nerve(s) Related to the Adverse Effect | Treatment for Adverse Effect |
| Skin infection                               | 1      | 1 mo             | MSCN                                          | Antibiotics                                  |
| Bruising or swelling                         | 2      | 2 wks            | Median trunk and MSCN                         | No treatment                                 |
|                                              |        | 1 mo             | Tibial trunk                                  | No treatment                                 |
| Nerve pain or dysesthesia                    | 9      | 1 mo             | Ulnar trunk                                   | Lidocaine and NSAID                          |
|                                              |        | 1 mo             | Median trunk                                  | No treatment                                 |
|                                              |        | 1 mo             | Median motor branch to flexor                 | Lidocaine and cortisone injection, topical lidocaine |
|                                              |        | 2 mos            | digitorum superficialis                       |                                              |
|                                              |        | 1 mo             | Tibial trunk                                  | No treatment                                 |
|                                              |        | Pain in 3 mos, numbness beyond 6 mos | Tibial motor branches to medial and lateral gastrocnemias | No treatment |
|                                              | 3 mos  | 3 mos            | Tibial trunk                                  | No treatment                                 |
|                                              | 3 mos  | 3 mos            | Tibial trunk                                  | Gabapentin 50\% and cortisone injection      |
|                                              | 1.5 mos| 3 mos            | Tibial trunk                                  | Gabapentin                                   |
| Cramping in antagonistic muscle              | 1      | 3 mos            | Tibial trunk                                  | Botulinum toxin injection                    |
|                                              |        | 3 mos            | Tibial trunk                                  | Topical treatment and 50 units of botulinum toxin |

MSCN, musculocutaneous nerve; NSAID, nonsteroid anti-inflammatory drug.
Stellate Ganglion Block

Local anesthetic induced block of the sympathetic nervous system at the stellate ganglion, which is a synapse of sympathetic fibers in the sympathetic chain that lead to the head, face, neck, upper extremities, and heart.\textsuperscript{12-15}

Found anterior to the neck of the first rib and can extend up to the inferior aspect of the transverse process at C7 in 80\% of individuals.\textsuperscript{14}

Historically used to treat pain in the upper extremities, neck and face including complex regional pain syndrome, peripheral vascular disease, phantom limb pain, and postherpetic neuralgia.\textsuperscript{14} More recently, evidence demonstrates its effectiveness in the treatment of post-traumatic stress disorder and chronic, intractable, atypical chest pain.\textsuperscript{12,13,15}

Originally performed under fluoroscopy with increased rates of adverse events, such as intraarterial puncture. US guidance affords safety, relatively quick delivery, and increased patient comfort.
Can block peripheral nervous system, inhibit the function of preganglionic and postganglionic fibers, and suppress the muscle tension dominated by the sympathetic nerve fibers.

- Observational case of good efficacy and long standing (9 month) relief of generalized dystonia in head, jaw, and neck

Yung et al. (2023) examined SGB vs extracorporeal shock wave therapy in an RCT in 60 stroke survivors with limb spasticity

- SGB, ESWT, and SGB + ESWT groups + control group
- Upper limb score based on Fugl-Meyer Assessment in the SGB, ESWT, and SGB + ESWT groups were significantly increased (P < 0.05) over control.
- Compared with the SGB and ESWT groups, SGB + ESWT exhibited a higher upper limb function score (P < 0.05), while the MBI score was not significantly different (P > 0.05).
Shi et al (2023) Rat model (diabetic) of induced thalamic stroke to identify the effects of SGB on ischemic stroke.\textsuperscript{16}

- SGB could effectively improve the cerebral ischemia and neurological function of diabetic rats
- Main mechanism uncovered was that SGB reduced the phosphorylation of NF-κB p65 and inhibited inflammatory response
- SGB can improve brain blood circulation, aid damaged brain neurons, improve the blood supply of the limbs, relieve muscle spasticity, promote tissue metabolism and restore the limb functions by regulating the function of central and peripheral nerves
Growing body of evidence suggests that SGB significantly improves the prognosis of cerebrovascular events by alleviating cerebral vascular spasm, increasing brain oxygen supply, reducing the inflammatory response, and decreasing oxidative stress.

Recently, SGB has emerged as a novel treatment for various pathological pain conditions, such as complex regional pain syndrome, postoperative pain, and orofacial pain as well as conditions such as fibromyalgia and long covid.\textsuperscript{15}

For CPSP, case studies showed that a single SGB treatment considerably alleviated somatic pain and decreased the usage of analgesic medicines for at least one month.\textsuperscript{17}

Lynch et al (2023) reported in a case series of 285 patients GAD7 scores reduced by 50% in patients treated with SGB.\textsuperscript{18} Decreased anxiety and PTSD would also be of benefit to stroke survivors.
Take Away Messages

BoNT is an effective treatment for spasticity and spasticity related pain but better when used with imaging and other therapies.

Dosage ceilings limit the use of BoNT injections.

Effectiveness of BoNT, Phenol, cryoneurolysis depends on provider skill including the use of ultrasound.

Adjuvant therapies are available but further rigorous trials are needed. Therapies provide analgesia however additional pain assessment and treatment is critical.
Conclusions

• Spasticity is the most commonly reported sequela of stroke that hinders achieving better quality of life for stroke survivors and caregivers.

• The tight interlink between pain and spasticity warrants focused efforts to establish evidence-based guidelines for the early assessment and treatment of pain and spasticity after stroke.
References


References


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UPCOMING WEBINAR

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Stroke in cis/trans women: current data on clinical trials, hormonal therapies and stroke prevention

March 26th, 2024 | 3:00 - 4:00PM CET