Assessment & Treatment of Spastic Muscle Overactivity

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Objectives

- Participants will be able to:
  - Identify types of muscle overactivity and their clinical presentations
  - Discuss the pathophysiology and origins of spastic muscle overactivity
  - Identify and differentiate various assessment tools used in assessing muscle overactivity
  - Discuss various treatment options for spastic muscle overactivity, including medical management and therapeutic management
What is “Spasticity?”

Most Common Definition:

Spasticity is “…a motor disorder characterized by velocity dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyper excitability of the stretch reflex…”

(Lance, 1980)
What is “Spasticity?”

Clinically:

- Just one type of muscle overactivity that occurs with CNS lesions.

- Most “talked about,” but other types of overactivity are usually grouped together as “spasticity.”

- Triggered by stretch and velocity

- By itself, may not cause a high level of disability
Spastic Dystonia

Muscle overactivity present at rest, which is spontaneous and has no primary triggering factor; causes deformation of joints and body postures. Frequently produces twisting or repetitive movements.
Other Types of Muscle Overactivity in CNS lesions

Hypertonic Rigidity
Similar resistance is felt with PROM regardless of the speed or direction of movement

Spastic Co-Contraction
Unwanted activity of antagonistic muscle group during voluntary agonistic movements (ex: activation of triceps during voluntary elbow flexion).
Other Types of Muscle Overactivity in CNS lesions

Contracture

- Fixed resistance to passive stretching of muscles due to shortening or wasting (atrophy) of muscle fibers or the development of scar tissue (fibrosis) over joints. Not technically muscle over-activity, but can be confused with over-activity. This is non-neural, but can be caused by neural components.
Pathophysiology

Immediate
- Paralysis
- Immobility
- Contracture

Damage to CNS
- Flaccidity

Immediate - Delayed
- Spasticity, Dystonia, Rigidity, Co-Contraction, etc

Delayed
- Plastic Changes
- Spinal
  - Supraspinal
- Overactivity
Immediate paralysis, leading to immobility

Active and passive tissue changes:
  - Type I fibers (slow/tonic) change to Type II (fast twitch/fatigable)
  - Muscle extensibility decreases due to a decrease in the # of sarcomeres and connective tissue accumulation in extrafusal fibers
  - These tissue changes lead to increases in stretch transmission to muscle spindle

Plastic changes within the CNS
  - Rerouting at spinal level
  - Recruitment of new motor pathways
How Does Spasticity Develop?

- Possible mechanisms of spasticity
  - Increased neuronal excitability
  - Enhanced excitatory synaptic input
  - Segmental afferents
  - Regional excitatory interneurons
  - Descending pathways, i.e., lateral vestibulospinal tract
  - Reduced inhibitory synaptic input
  - Renshaw cell recurrent inhibition
  - Ia inhibitory interneurons
  - Ib afferent fibers
  - Change in intrinsic electrical properties of the neuron
  - Change in passive membrane electrical properties
  - Change in voltage sensitive membrane conductance
  - Enhanced stretch-evoked synaptic excitation of neurons
  - γ efferent hyperactivity
  - Excitatory interneurons more sensitive to muscle afferent
Major Descending Pathways Controlling Spinal Reflexes
Origins of Muscle Overactivity

Cerebral -
BI, CVA, CP, MS
Lack of inhibition messages being sent

Spinal - SCI, MS
Inhibitory message is sent but cannot be received due to disruption of spinal cord
Cortical vs Spinal Spasticity

- Muscle tone is affected by:
  - the absence of an intact corticospinal system
  - An imbalance of inputs from reticulospinal and other descending pathways to the motor and interneuronal circuits of the spinal cord
Cortical vs Spinal Spasticity

- Indirect cortical pathways to spinal cord (e.g., cortico-reticulospinal) pathways usually has inhibitory influence on muscle tone, especially limb extensor tone
Cortical vs Spinal Spasticity

Other consequences of damage to direct and indirect corticospinal tracts

- Loss of descending tonic or phasic excitatory and inhibitory inputs to the spinal motor apparatus
- Alterations in the segmental balance of excitatory and inhibitory control
- Denervation supersensitivity
- Neuronal sprouting
Cortical Spasticity

- Selective damage to area 4 in the cerebral cortex of primates produces paresis that improves with time, but increases in muscle tone are not a prominent feature.
- Lesions involving area 6 cause impairment of postural control in the contralateral limbs.
- Combined lesions of areas 4 and 6 cause both paresis and spasticity to develop.
Spinal Changes

- During the development of spasticity, the spinal cord undergoes neurophysiologic changes in the excitability of motor neurons, interneuronal connections, and local reflex pathways.
- The excitability of alpha motor neurons is increased, as is suggested by enhanced H-M ratios and F-wave amplitudes.
- Judged by recordings from Ia spindle afferents, muscle spindle sensitivity is not increased in human spasticity.
Flexor vs Extensor Spasticity

- Reticulospinal tract → flexion, typically
- Vestibulospinal tract → extension, typically
Pathophysiology of Spasticity

- Imbalance between excitatory and inhibitory impulses to the alpha motor neuron in the spinal cord
- Due to a loss of descending inhibitory input to the alpha motor neuron due to injury to the cortical spinal tracts
Treatment Options

- Botulinum Toxins
- Physical Modalities and Therapy
- Oral Drugs
- Surgery
- Phenol and Alcohol injections
- Intrathecal therapies
Considerations for Management Choices

- When considering management choices for Cerebral Origin muscle overactivity, we need to be aware of the effect of the medications on the brain’s recovery process.
- Many oral anti-spasticity medications have the side effect of making people sleepy, slowing cognitive processes, and may ultimately slow the recovery of function.
- The challenge is to manage abnormal tone without interfering with brain healing/recovery.
Best Practices
Spasticity Management

EFFECTIVE APPROACH

- An integrated and multidisciplinary program of physical and medical interventions
- A clear individualized patient management strategy

WHEN AND HOW TO TREAT

- Significance, i.e. disabling
- Distribution of spasticity
- Chronicity, severity, and cause
- Concomitant conditions
- Cost
- It’s never too late to treat spasticity
How can tx of spasticity facilitate motor recovery?

Brunstrom Stages of Recovery

**STROKE**

- **I Flaccidity**
- **II Synergies** Some spasticity
- **III Marked spasticity**
- **IV Out of synergy Less spasticity**
- **V Selective control of movement**
- **VI Isolated/coordinated movement**

Reversed “maladaptive plasticity”?
Goal-Setting

PRIMARY EFFECTS
- Focus
- Tone reduction
- Spasm reduction

Locus
- Central
- Peripheral

SECONDARY EFFECTS
- Symptom Relief
- Correct Deficit
  - Exercise
  - Compensation
  - Restitution
Spasticity is not the reason for treatment. Instead, it is the impact of spasticity on a person’s well-being.

Significance, not severity, of spasticity should dictate need for treatment.
Management Starts with Goal-Setting

TECHNICAL

- Decrease hypertonia (Ashworth)
- Decrease spasm frequency and severity
- Increase range of motion

FUNCTIONAL

- “PASSIVE”
  - Facilitating wearing of splints
  - Decreasing pain associated with abnormal posture and spasms
  - Improve nursing care

- “ACTIVE”
  - Increase performance

- OTHERS
  - Improve body image
Spasticity Management
Interdisciplinary Approach

Pharmacologic

Physical Intervention

Functional Re-training
Spasticity Management

Assessment  Goal-Setting  Choice of Treatment
Management Options - Therapy

Medical Management should always be supplemented by therapeutic management for optimal improvement!

Therapeutic Interventions:

- Weight bearing
- Serial Casting/Splinting
- Locomotor Training
- Positioning
- E-stim
- Strengthening
### Assessment of Muscle Overactivity

<table>
<thead>
<tr>
<th>Assessment</th>
<th>What are we assessing?</th>
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</thead>
<tbody>
<tr>
<td>Ashworth/Modified Ashworth</td>
<td>Resistance to passive range of motion (including, but not exclusive of, resistance due to spasticity)</td>
</tr>
<tr>
<td>Tardieu/Modified Tardieu Scale</td>
<td>Spasticity angle, Quality of movement at multiple velocities</td>
</tr>
<tr>
<td>Passive Range of Motion</td>
<td>Amount of passive movement allowed by the joint, soft tissue, muscle, etc. Goal is to assess without eliciting a stretch reflex</td>
</tr>
<tr>
<td>Active Range of Motion</td>
<td>How much active movement can be performed against the spastic antagonist (NOT strength!)</td>
</tr>
<tr>
<td>Functional activities</td>
<td>Quality of movement, speed of movement, postural alignment, energy expenditure, etc.</td>
</tr>
<tr>
<td>Patient/caregiver report</td>
<td>Pain, ease of care, transfers, quality of life, etc</td>
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</tbody>
</table>
### Ashworth/Modified Ashworth Scales

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No increase in tone</td>
</tr>
<tr>
<td>1</td>
<td>Slightly increased tone, with a catch &amp; release or minimal resistance at terminal ROM</td>
</tr>
<tr>
<td>1+</td>
<td>Slight increase, catch followed by minimal resistance throughout the remainder of the range (&lt;1/2 of the ROM) (only in MAS)</td>
</tr>
<tr>
<td>2</td>
<td>Marked increase through most of the ROM, but affected part is easily moved</td>
</tr>
<tr>
<td>3</td>
<td>Considerable increase, passive ROM difficult</td>
</tr>
<tr>
<td>4</td>
<td>Affected part is rigid</td>
</tr>
</tbody>
</table>

Passive movements of muscle groups should be performed over a **one-second** time frame.
### Variable reliability

<table>
<thead>
<tr>
<th>Author</th>
<th>Subjects</th>
<th>Results/Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sloan et al</td>
<td>34 hemiplegic</td>
<td>“MAS has acceptable interrater reliability for testing of upper limb spasticity, but not so for testing of the lower limb spasticity.”</td>
</tr>
<tr>
<td>Nuyens, et al</td>
<td>30 MS</td>
<td>“AS more reliable for muscles of the ankle than for muscles of the knee, and least reliable for muscles of the hip.”</td>
</tr>
<tr>
<td>Haas, et al</td>
<td>30 SCI</td>
<td>Interrater reliability varied between AS and MAS, between muscle groups (hip adductors &gt; hip extensors/flexors &gt; ankles plantarflexors), and between limbs. Recommended for both to be used with caution when assessing LE spasticity with SCI patients</td>
</tr>
<tr>
<td>Allison et al</td>
<td>30 TBI</td>
<td>Low interrater reliability for ankle plantarflexors, and argued that there was no support for continued use of MAS to assess PFs in pts with TBI.</td>
</tr>
<tr>
<td>Gregson, et al</td>
<td>32 acute CVA</td>
<td>Intra/inter-rater reliability found to be “good to very good for the elbow, wrist and knee, but less satisfactory over the ankle.”</td>
</tr>
<tr>
<td>Blackburn et al</td>
<td>36 CVA</td>
<td>Acceptable intra-rater reliability, but poor inter-rater reliability for MAS. Most agreement was with scores of “0,” so conclusion was that reliable measurements could be obtained to determine whether normal or low muscle tone is present or not.</td>
</tr>
</tbody>
</table>
“The results...are clear and tell us the Ashworth Scale has insufficient validity and reliability to be used as a measure of spasticity. However, we are left with the problem of how to measure spasticity in a valid and reliable way. The quest for this holy grail is ongoing.”

- Katharina S Sunnerhagen

So what else can we use??
Modified Tardieu Scale

Developed by Tardieu as a way to assess the velocity component of spasticity in 1950s, modified by Boyd and Graham

May be more useful to clinicians in assessing functional implications of spasticity, as well as effects of treatment

Measures spasticity using two parameters
- The spasticity angle
- The spasticity grade
Tardieu Scale – What does the evidence say?

More likely to identify presence of contractures than MAS/AS (Patrick, 2006)

More likely to correctly identify presence [but not severity] of spasticity than MAS when confirmed with EMG (Patrick, 2006)

Very good intra-rater reliability across 2 sessions in elbow flexors and ankle plantarflexors (Singh, 2011)

“In patients with severe brain injury and impaired consciousness the Modified Tardieu Scale provides higher test retest and inter-rater reliability compared with the Modified Ashworth Scale and may therefore be a more valid spasticity scale in adults.” Mehrholz, 2005

Variable reliability, which increases with training – – StrokEdge review
Tardieu Scale – Spasticity Angle

Range of motion measured at two different velocities
V1 – Slow as possible (R2)
V2 – Fast as possible (R1)

Large spasticity angles indicate a large dynamic component (spasticity), whereas small differences indicate predominantly muscle contracture.
<table>
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<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No resistance throughout passive movement</td>
</tr>
<tr>
<td>1</td>
<td>Slight resistance throughout passive movement</td>
</tr>
<tr>
<td>2</td>
<td>Clear catch at precise angle, interrupting passive movement, followed by release</td>
</tr>
<tr>
<td>3</td>
<td>Fatigable clonus (&lt;10 s when maintaining pressure) occurring at a precise angle, followed by release</td>
</tr>
<tr>
<td>4</td>
<td>Unfatigable clonus (&gt;10s when maintaining pressure) occurring at a precise angle</td>
</tr>
</tbody>
</table>

Notes:
- If spasticity angle = 0, grade must be a 0 or 1 by definition
- If spasticity angle > 0, grade must be at least a 2, even if no definite “release” felt
Active Range of Motion

Objective measure of how much the agonist can move against the overactive antagonist

- Example – To assess overactive biceps, we could measure the degrees of active *extension* that a patient can achieve.
- **NOT** looking at the *strength* of the extensors, but rather how much the overactivity of the flexors can be overcome
- Following treatment, would want to reassess to see if the patient can achieve more active movement,
- May be a more functional measurement than passive measurements
Ask Questions!

- How is this impacting their lives? *Severity ≠ Significance*
- What are their goals of treatment?

Observe!

- Watch them walk, transfer, propel w/c, eat, dress, etc
- How are they positioned?
- Video, video, video!!

Be Objective when you can!

- Use measures such as 10MWT, Gaitrite assessment, Modified Frenchay Arm Test, Disability Assessment Scale
Other Assessments

- Penn Spasm Frequency Scale
- Timed repeated contractions
- Barry Albright Dystonia Scale
Clinicians often focus on the direct effects of spasticity; however, it is the indirect/disabling effects of spasticity that most impact patients’ daily life.

Effective rehabilitation consists of appropriate assessment, and an individualized, interdisciplinary approach that may incorporate both pharmacologic and nonpharmacologic treatments.

Spasticity and other chronic stroke complications should be assessed and managed on a continual basis to sustain functional gains.
References

- www.wemove.org
References


References

- Sunnerhagen, K. Stop Using the Ashworth scale for the assessment of spastisity [letter]. *J Neurol Neurosurg Psychiatry* 2010. 81:2


Additional Resources


Additional Resources


