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Alterations in Muscle Tone: Diagnosis and Management

Jill Seale, PT, PhD, NCS
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PhysicalTherapy.com

Objectives

Describe the continuum of muscle tone.
Identify the pathophysiology of altered muscle tone, specifically spasticity.
Identify at least thee of the functional impacts of altered muscle tone.
Compare and contrast at least two methods of management of alterations in muscle tone.
Lower Motor Neurons

Lundy-Ekman, 2012
Spinal Level
Stepping Pattern Generators
Descending Motor Pathways

Control Circuits

• Basal Ganglia

• Cerebellum
Motor Planning Areas

Lundy-Ekman, 1998
Upper Motor Neuron versus Lower Motor Neuron

• Upper motor neuron lesion
  – Increased tone (hypertonia)
  – Little to no muscle wasting
  – Muscle weakness
  – Hyperreflexia

• Lower motor neuron lesion
  – Flaccid (or low tone)
  – Significant, rapid muscle wasting
  – Decreased or absent reflexes

Upper Motor Neuron Syndrome

Positive Signs
• Spasticity
• Athetosis
• Primitive reflexes
• Rigidity
• Dystonia

Negative Signs
• Decreased strength
• Decreased motor control
• Decreased coordination
• Decreased endurance

Pathophysiology

Primary neuromuscular impairments

Secondary musculoskeletal

Shumway-Cook, Woollacott, 2007

What is tone?
Muscle Tone

- Tension in muscle, determined by mechanical factors and motor unit activity
- Some amount of muscle tone is normal
- Range of muscle tone, depending on demand
- How does/might stroke impact muscle tone, versus other neurological diagnoses?

Flaccidity and Hypotonia
Hypertonicity and Spasticity

Spasticity  (Lance 1980)

- Motor disorder
- *Velocity dependent increase in tonic stretch reflexes*
- Exaggerated DTRs
- Component of upper motor neuron syndrome
SPASM Consortium

• Disordered sensorimotor control, presenting as involuntary muscle activation following an upper motor lesion

http://research.ncl.ac.uk/spasm/

One more definition

• Spasticity is primarily a segmental sensorimotor reflex with afferent activity, elicited by muscle stretch, being abnormally processed in related cord segments, ultimately generating excessive drive on segmental alpha motor neurons innervating the very muscles being stretched. (Sheean, 2002)
Definitions

• Hypertonia: abnormally increased resistance to externally imposed movement about a joint
  • may be caused by spasticity, dystonia, rigidity, combination of features
• Spasticity: velocity-dependent resistance of muscle to stretch
  • hypertonia in which 1 or both of the following signs are present:
    1) resistance to externally imposed movement increases with increasing speed of stretch and varies with the direction of joint movement, and/or
    2) resistance to externally imposed movement rises rapidly above a threshold speed or joint angle


Pathophysiology of Spasticity
Pathophysiology of Spasticity

Cerebral Spasticity

- Enhanced excitability of monosynaptic pathways due to lack on inhibition
- Rapid build-up of reflex activity
- Bias toward overactivity in the antigravity muscles
Spinal Spasticity

- Removal of inhibition on segmental *polysynaptic* pathways
- Slow, progressive rise of excitatory state through cumulative excitation
- Afferent activity from one segment may lead to muscle response many segments away
- Flexors and extensors may be overexcited

Rigidity

![Image of rigidity](image-url)
A couple other terms

• Paralysis: loss of muscle function
• Paresis: weakness/decrease in muscle function
• Hemiplegia: paralysis of 1 side of body
• Hemiparesis: weakness of 1 side of body
• Paraplegia: Paralysis of all or part of trunk and BOTH LEs
• Tetraplegia: Paralysis of trunk, UEs, and LEs

Impact of Alterations in Muscle Tone

• Instability
• Lack of selective movements
• Inability to grade forces
• Loss of range of motion
• Gait alterations
• Positional challenges
• Pressure sores/skin breakdown
• Hygiene issues
• Pain
• Sleep
Adult Spasticity Management

Assessment

• Ashworth Scale
• Modified Ashworth Scale
• Tardieu Scale
• Oswestry Scale
• Spasm Frequency Scale
• Tone Assessment Scale
Table 3 Ashworth scale

- 0: No increased tone
- 1: Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion when the affected part is moved in flexion or extension
- 2: More marked increase in muscle tone through most of the range of motion, but affected part(s) easily moved
- 3: Considerable increase in muscle tone, passive movement difficult
- 4: Affected part(s) rigid in flexion or extension

Most Widely Used

Testing Spasticity with the Modified Ashworth Scale

- The MAS is done in supine (this will garner the most accurate and the lowest score; any tension anywhere in the body will increase spasticity)
- Because spasticity is "velocity dependent," the faster the limb is moved, the more spasticity is encountered, the MAS is done moving the limb at the "speed of gravity." This is defined as the same speed a non-spastic limb would naturally drop in other words fast.
- The test is done a maximum of three times for each joint. If it is done more than three times the short-term effect of a stretch impacts the score.
- The MAS is done prior to gastrocnemius testing. Gastrocnemius testing provides a stretch and the shorthand effect of a stretch impacts the score.

TARDIEU SCALE

This scale quantifies muscle spasticity by assessing the response of the muscle to stretch applied at specified velocities.

Grading is always performed at the same time of day, in a constant position of the body for a given limb. For each muscle group, reaction to stretch is rated at a specified stretch velocity with 2 parameters x and y.

Velocity to stretch (V)

- V1: As slow as possible
- V2: Speed of the limb segment falling
- V3: As fast as possible (to natural drop)

V1 is used to measure the passive range of Motion (PROM). Only V1 and V3 are used to rate spasticity.

Angle of muscle reaction (Y)

Measure relative to the position of minimal stretch of the muscle (corresponding angle).

Spasticity Angle

- R1: Angle of each seen at Velocity V2 or V3
- R2: Full range of motion achieved when muscle is at rest and tested at V1 velocity

Bond, Graham 1999
- A large difference between R1 & R2 values in the outer middle range of normal length indicates a large dynamic component.
- A small difference in the R1 & R2 measurement in the middle to inner range indicates predominantly fixed contracture.
Oswestry Spasticity Scale

- 0 – solely spasticity, no willed movement possible
- 1 – very severe spasticity, movement very poor
- 2 – severe spasticity, movement poor
- 3 – moderate spasticity, movement fair
- 4 – mild spasticity, movement good
- 5 – no spasticity, movement normal

- Goff B, *Physiotherapy*, 1976
Spasm Frequency Scale

<table>
<thead>
<tr>
<th>How often are muscle spasms occurring?</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>4</td>
</tr>
</tbody>
</table>

Additional Assessment

- Impairments
  - PROM, AROM, muscle length
  - Strength
  - Sensation
- Activity/function
  - Gait
  - Transfers
  - ADL
  - w/c mobility
- Quality of life
Rehabilitation Interventions for Spasticity Management

- Accurate assessment
- Maintaining/improving flexibility
- Strengthening
- Weightbearing
- Function
- Education
- Appropriate referral for medical interventions

Flexibility

- Target tissues: muscle, joint capsule, nerve
- PROM exercises not sufficient
- Need low load prolonged stretch
  - Positioning
  - Casting/splinting
- Special attention to 2 joint muscles
- AROM is best, E-stim may augment
- Remember your manual skills for joint and soft tissue mobilization
- Neural gliding is also necessary
The Effects of Stretching in Spasticity: A Systematic Review

Thomas J. Beswick, MD, Miroslava Nouman, MD, Karen Barker, PhD, Helen Owens, PhD,

Abstract: The effects of stretching in spasticity have been independently performed by numerous researchers in the field of neurorehabilitation. However, there is a lack of consensus on the optimal stretching techniques and their impact on spasticity. This systematic review aimed to summarize the current evidence on the effects of stretching in spasticity.

Keywords: stretching, spasticity, neurorehabilitation

The Evidence for Prolonged Muscle Stretching in Ankle Joint Management in Upper Motor Neuron Lesions: Considerations for Rehabilitation

Ali A Bani-Ahmad

Department of Physical Therapy, University of Tabuk (U.T.), Tabuk, Kingdom of Saudi Arabia (KSA)

Abstract

A normal functional ankle joint is a key factor for a successful gait. Many studies reported significant changes in ankle properties in upper motor neuron lesions (UMNs). As clinicians, muscles stretching approaches are of the most commonly used interventions in rehabilitation. However, there is a need for a more systematic approach to stretching in terms of the features of the stretching approaches such as duration and frequency as well as the compatibility and viewpoint of a stretching approach. This review is an attempt to synthesize findings from studies on "prolonged" stretching approaches in patients with UMNs, including stroke, spinal cord injuries and traumatic brain injuries. The review also investigated the important features of successful stretching in terms of reducing spasticity, improving the Active Range of Motion (AROM), Passive Range of Motion (PROM) and gait training in upper motor neuron lesions. Therefore, studies evaluating the effectiveness of "prolonged" stretching on spastic ankle plantar flexors muscles and its complications were critically reviewed and the level of evidence was analyzed. This review will add stronger understanding with regard to stretching considerations in rehabilitation following UMNs for clinicians as well as researchers to propose exciting possibilities for future research.
Strengthening

• A spastic muscle is a weak muscles
• The muscles that oppose spastic and/or contracted muscles are ALSO weak muscles
• Close chain progressing to open chain
• Isometric, eccentric, concentric
• Specificity of strengthening is important
• Strengthening DOES NOT promote/increase spasticity
  • Pak and Patten, *Top Stroke Rehabil*, 2008

Weightbearing

• Can help *normalize* muscle tone
• HOWEVER, good biomechanical alignment is critical for weightbearing to be effective
• Weightbearing + biomechanical alignment = best chance for more normal muscle tone
• Good biomechanical alignment allows antagonist to activate
• Get activation and good control in weightbearing prior to progressing to open chain
## Oral Medications

<table>
<thead>
<tr>
<th>Drug</th>
<th>Daily Max Dose</th>
<th>Mechanism of Action</th>
<th>Common Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baclofen</td>
<td>80 mg divided in 4 doses</td>
<td>GABA analogue</td>
<td>Drowsiness, dizziness, weakness</td>
</tr>
<tr>
<td>Clonidine</td>
<td>.1 mgqd</td>
<td>Alpha-adrenergic receptor agonist</td>
<td>Bradycardia, hypotension, depression</td>
</tr>
<tr>
<td>Dantrolene Sodium</td>
<td>100 mgqd</td>
<td>Blocks release of Ca from SR</td>
<td>Muscle weakness, hepatotoxicity</td>
</tr>
<tr>
<td>Tizanidine</td>
<td>36 mg</td>
<td>Alpha adrenergic receptor agonist</td>
<td>Drowsiness, dry mouth</td>
</tr>
<tr>
<td>Gabapentin</td>
<td>600-800 mgqd</td>
<td>GABA analogue</td>
<td>Drowsiness, dizziness, ataxia</td>
</tr>
</tbody>
</table>

### Pros
- Non-invasive
- Non permanent
- Effective management of + signs

### Cons
- Effects ebb and flow
- Must take on schedule
- Sedating side-effects
Oral Medications

• Clinical usefulness limited by side effects
• Lack of high-quality evidence
• Choose medication based on “side effect profile”
• Minimal dose, minimal side effect
• Should not be the “first-line” of treatment
  • Watanabe TK. PM R, 2009

Chemical Denervation

• Chemical Neurolysis: phenol or alcohol applied to nerve via injection with EMG guidance
  • Cause demyelination of axon
  • Effects last up to 6 months
• Neuromuscular Blockade: Botulinum neurotoxin injected into muscle, binds to presynaptic cholinergic nerve terminal; blocks release of ACH
  • This is not just Botox
Advantages/Disadvantages

• Chemical Neurolysis
  • Advantages: better effect on larger muscles, cost is minimal
  • Disadvantages: Difficult procedure, risk of sensory complications, muscle become fibrotic after repeated injections

• Neuromuscular Blockade
  • Advantages: less painful, easier to perform, no sensory side effects, not permanent
  • Disadvantages: only reinject every 3 months, not permanent, cost, can develop antibodies

Intrathecal Baclofen (ITB)

How ITB Therapy Works

• Uses an implantable, programmable SynchroMed® II pump to deliver precise amounts of Lioresal® Intrathecal (baclofen injection) directly to site of action at spinal cord via the cerebrospinal fluid
• Since ITB Therapy delivers baclofen directly to spinal cord, a fraction of the oral medication dose may be needed
ITB

- Baclofen is diffused into cerebrospinal fluid (CSF) in intrathecal space
  - Catheter attached to mechanical pump implanted in pt
- Mechanism of action: presynaptic inhibition – GABA b receptor agonist
- Inhibits both mono and polysynaptic reflexes
- Advantages
  - Reversible, easy to titrate does, fewer side effects, improved function/ease of care
- Disadvantages
  - Mechanical complications, refills required (at least every 3 months), cost

Evidence

- ITB for post-stroke hypertonia
  - Improved FIM, SIP, and AS
  - No adverse effect on strength in unaffected limbs
  - Ivanhoe et al, APMR, 2006
- ITB Consensus Panel Guidelines
  - Collaboration between therapists and physicians
  - Positive effects of ITB
  - For those who did not respond or tolerate other treatment interventions
  - As early as 3-6 months post stroke
  - Optimal does is goal dependent
  - No evidence regarding superior dosing mode
    - Francisco GE et al, Top Stroke Rehabil, 2006
Intrathecal v Oral

Intrathecal
- Lower dose needed
- Potentially fewer side effects

Oral
- Low blood/brain barrier penetration
- High systemic absorption/low CNS absorption
- Lack of preferential distribution to spinal cord
- Adverse effects of sedation

Orthopedic Surgery

- Soft tissue
  - Selective percutaneous myofascial lengthening
  - Lengthening
  - Tendon transfer
  - Releases

- Skeletal procedures
  - Osteotomies
  - Fusions
Neurosurgical Interventions

- Selective Dorsal Rhizotomy - selective destruction of problematic nerve roots
  - Nerve roots where spasticity are located are identified using EMG
  - Selectively lesioned

Medical and Surgical Interventions

Graham HK et al, Gait Posture, 2000
Combining Rehab and Pharmacology

• Focal spasticity for MS
  • BoNT-A alone compared with BoNT-A + physiotherapy
  • BoNT-A + physiotherapy provided best response
  • Giovanelli et al, Clin Rehabil, 2007
• Review paper of rehab procedures in spasticity management
  • Integration of rehab measures with pharmacologic interventions
  • Smania et al, Euro J Phys Rehabil Med, 2010
• Rehab therapies after BoNT A for limb spasticity
  • Ergometer cycling, e-stim, stretch, CIMT, task specific motor training, exercise programs
  • Combo therapies “slightly” more effective than BoNT-A alone
  • Kinnear et al, Phys Ther, 2014.

The Controversy

• “Assumption that spasticity is direct cause of disordered movement”
• We assess spasticity in resting limbs, yet we associate it with movement disorders
• No direct causation between spasticity and function
• Hypertonia associated with contracture — abnormal movement may be more about stiffness of passive tissues
• So what is that excess muscle activity:
  • Compensatory behavior
  • Over recruitment when demand exceeds capacity
Case Examples

So what do we do?

• Improve our movement analysis to discern causes of abnormal movement
• Manipulate the person, environment, and/or task to get a more normal movement
  • Fix biomechanical constraints or compensate for them
  • Manipulate task and/or environment difficulty to better match demand to capacity
• Increase patient’s capacity...how?
• Decrease degrees of freedom
• More ideas?
Spasticity versus Musculoskeletal Contracture

• Diagnostics:
  • Tardieu Scale (R1 vs R2)
  • End feels
  • Lidocaine block
  • Evaluation under anesthesia
• Question must be answered prior to surgical intervention
• Spasticity must be treated prior to surgical interventions for contracture!

Questions?

• jseale27@sbcglobal.net