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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

- Image showing the brain with pathways labeled:
  - Motor Cortex
  - Thalamus
  - Basal Ganglia
  - Cerebellum
  - Effector Muscle
  - Motor neuron final common pathway
  - Efferent to muscle spindle
Pathophysiology of Spasticity

Cerebral Spasticity

- Enhanced excitability of monosynaptic pathways due to lack of inhibition
- Rapid build-up of reflex activity
- Bias towards 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
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

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No increased tone</td>
</tr>
<tr>
<td>1</td>
<td>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</td>
</tr>
<tr>
<td>2</td>
<td>More marked increase in muscle tone through most of the range of motion, but affected part(s) easily moved</td>
</tr>
<tr>
<td>3</td>
<td>Considerable increase in muscle tone, passive movement difficult</td>
</tr>
<tr>
<td>4</td>
<td>Affected part(s) rigid in flexion or extension</td>
</tr>
</tbody>
</table>

Testing Spasticity with the Modified Ashworth Scale (Sunnerhagen KS, Olver J, Francisco GE, Neurology, 2013)

- 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 as 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 posteroanterior testing. Somatosensory testing provides a stretch and the short-term 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 segments falling |
| V3 | As fast as possible (> natural drop) |

V3 is used to measure the passive range of Motion (PROM). Only V2 and V3 are used to rate spasticity.

Quality of muscle reaction (X)

<table>
<thead>
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<tbody>
<tr>
<td>0</td>
<td>No resistance throughout passive movement</td>
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<tr>
<td>1</td>
<td>Slight resistance throughout, with no clear catch at a precise angle</td>
</tr>
<tr>
<td>2</td>
<td>Clear catch at a precise angle followed by release</td>
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<tr>
<td>3</td>
<td>Fatiguable clonus (&lt;10s/sec) occurring at a precise angle</td>
</tr>
<tr>
<td>4</td>
<td>Unfatiguable clonus (&gt;10s/sec) occurring at a precise angle</td>
</tr>
<tr>
<td>5</td>
<td>Joint Immobile</td>
</tr>
</tbody>
</table>

Angle of muscle reaction (Y)

Measure relative to the position of minimal stretch of the muscle (corresponding at 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

* A large difference between R1 & R2 values in the outer to 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 contraction.
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

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Table 1: Tone Assessment Scale

<table>
<thead>
<tr>
<th></th>
<th>0</th>
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<th>2</th>
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</tr>
</tbody>
</table>

*Gregson et al., Arch Phys Med Rehabil, 1999*
Spasm Frequency Scale

How often are muscle spasms occurring?

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No spasms</td>
</tr>
<tr>
<td>1</td>
<td>Spasms induced only by stimulation</td>
</tr>
<tr>
<td>2</td>
<td>Spasms occurring less than once per hour</td>
</tr>
<tr>
<td>3</td>
<td>Spasms occurring between 1 and 10 times per hour</td>
</tr>
<tr>
<td>4</td>
<td>Spasms occurring more than 10 times per hour</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 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 (UiT), 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 within the affected ankle 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 an in-depth evaluation of research on prolonged stretching in terms of the functional stretching approaches such as duration and frequency as well as the compatible measures of a successful stretching approach. This review is an effort to synthesize findings from studies on “prolonged” stretching approaches in patients with UMN including stroke, spinal cord injuries and traumatic brain injuries. The review also investigated the compatible features of successful stretching regimens in terms of reducing spasticity, improving the Active Range of Motion (AROM), Passive Range of Motion (PROM) and gait training of spastic patient with upper motor neuron lesions. Therefore, studies evaluating the effectiveness of “prolonged” stretching on spastic ankle planter flexor muscles and its complications were critically reviewed and the level of evidence were analyzed. This review will add stronger understanding with regard to stretching considerations in rehabilitation following UMN for clinicians as well researchers to propose exciting possibilities for future research.

J Nov Physiotherapies, 2016
Strengthening

- A spastic muscle is a weak muscle
- 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 mg qd</td>
<td>Alpha-adrenergic receptor agonist</td>
<td>Bradycardia, hypotension, depression</td>
</tr>
<tr>
<td>Dantrolene Sodium</td>
<td>100 mg qd</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 mg qd</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 sites of action at spinal cord via the cerebrospinal fluid
- Since ITB Therapy delivers baclofen directly to spinal cord, a fraction of the total medication dose may be needed

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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

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**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

Combining Rehab and Pharmacology

- **Focal spasticity for MS**
  - BoNT-A alone compared with BoNT-A + physiotherapy
  - BoNT-A + physiotherapy provided best response
- **Review paper of rehab procedures in spasticity management**
  - Integration of rehab measures with pharmacologic interventions
- **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

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?

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