MANAGEMENT OF SPASTICITY

CLINICAL IMPLICATIONS AND INTERVENTION

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Quinnipiac University
3/20/20
DISCLOSURE STATEMENT

• No relevant financial or nonfinancial relationships exist
COURSE OBJECTIVES

• Apply evidence-based practice to inform clinical decision making for treatment and management of spasticity.
• Explore examination and interventions for spasticity and potential complications through a case example.
• Understand the importance of interprofessional collaboration to maximize outcomes.
WHAT IS SPASTICITY?

• Neuromuscular overactivity resulting in extreme involuntary muscle contraction
• Abnormal increase in tone accompanied by resistance to active and passive movement
• Positive sign of CNS pathology
  • UMN damage
  • Interruption or interference within the descending motor pathways
• Velocity dependent
• Extent and location of the damage/lesion can impact the type and level of impairment
VARIATION IN MUSCLE TONE

- Flaccidity
- Hypotonicity (Low tone)
- Normal
- Hypertonicity (Spasticity)
- Rigidity
WHY DOES THIS MATTER TO US AS CLINICIANS?
BIOMECHANICAL CHANGES

- Disuse atrophy
- Shortening of soft tissue (contractures)
- Maladaptive movements
- Abnormal posture

What can this lead to?
- Pain
- Loss of range of motion
- Skin breakdown
- Fractures
EFFECT OF SPASTICITY ON FUNCTION

- Spasticity is velocity dependent; with increased speed increased spasticity can occur
  - E.g. how does this impact reach or ambulation?
- Spasticity can lead to involuntary activation
  - E.g. abnormal posturing, synergies, clonus
FUNCTIONAL IMPACT

• Impaired performance in daily occupations
  • Pain, spasms
  • Loss of range of motion
  • Loss of independence
  • Poor hygiene

• Silver lining...?
  • Transfers, postural control, mobility
  • Help maintain muscle mass
  • Reduce edema
  • Prevent DVT
What do you include in your assessment of tone?

What’s in your toolbox?
BRUNNSTROMS STAGES OF RECOVERY

1. Flaccid paralysis. No reflexes.
3. Spasticity is marked. Synergistic movements may be elicited voluntarily.
5. Spasticity wanes. Can move out of synergies although synergies still present.
6. Coordination and movement patterns near normal. Trouble with more rapid complex movements.

www.ebrsr.com
• Sensorimotor assessment that addresses the following areas:
  • Motor functioning (both UE and LE)
  • Sensation (light touch and position sense)
  • Joint functioning (ROM and pain)
• Assists in determining severity of stroke, describe motor recovery, and assessment and intervention
• Classifications for impairment severity proposed based on FMA total motor score (100 points)
  • Each of the domains can be assessed separately e.g. UE (66 points) LE (34 points)
<table>
<thead>
<tr>
<th>FUGL-MEYER ASSESSMENT UPPPER EXTREMITY (FMA-UE)</th>
<th>Examiner:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assessment of sensorimotor function</td>
<td></td>
</tr>
</tbody>
</table>

**A. UPPER EXTREMITY**

**Sitting position**

1. **Reflex activity**: sitting-position
   - Flexion, extension, and active finger flexion
   - Passive flexion
   - Sensory

2. **Vital signs**: vital signs
   - Blood pressure
   - Heart rate
   - Respiration

3. **Vital signs**: if any

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<tr>
<th>ID:</th>
<th>Date:</th>
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</table>

**B. REFLEXIVE MOVEMENT WITH SYMMETRY**

- **Shoulder abduction**: normal
- **Elbow flexion**: normal
- **Forearm supination**: normal

**C. PASSIVE MOBILITY**

- **Shoulder abduction**: normal
- **Elbow flexion**: normal
- **Forearm pronation**: normal

**D. HAND**

- **Support provided**: normal

**E. GRASP**

- **Thumb abduction**: normal
- **Finger abduction**: normal

**F. COORDINATION/SPR.**

- **Finger abduction**: normal

**G. COORDINATION/SPR.**

- **Finger flexion**: normal

<table>
<thead>
<tr>
<th>TOTAL A (max 5)</th>
<th>TOTAL B (max 11)</th>
<th>TOTAL C (max 14)</th>
<th>TOTAL D (max 24)</th>
<th>TOTAL A-D (max 64)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Approved by Fugl-Meyer AR 2010</td>
<td>Updated 2019-03-03</td>
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</tr>
</tbody>
</table>
## FUGL-MEYER ASSESSMENT
### LOWER EXTREMITY (FMA-LE)

**Assessment of sensorimotor function**

<table>
<thead>
<tr>
<th>Examiner:</th>
<th>FMA-LE PROTOCOL</th>
<th>Rehabilitation Medicine, University of Gothenburg</th>
</tr>
</thead>
</table>

**ID:**

**Date:**

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### E. LOWER EXTREMITY

<table>
<thead>
<tr>
<th>Reflex activity, supine position</th>
<th>Flexors: knee flexors</th>
<th>Extensors: patellar, Achilles (at least one)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None can be elicited</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

**Subtotal I (max 4):**

---

### II. Volitional movement within synergies

<table>
<thead>
<tr>
<th>Flexor synergy: Maximal hip flexion (adduction/external rotation), maximal flexion in knee and ankle joint (paraspinal tendons to ensure active knee flexion)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee flexion</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Extensor synergy: From flexor synergy to the hip extension/adduction, knee extension and ankle Plantar flexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip flexion</td>
</tr>
</tbody>
</table>

**Subtotal II (max 16):**

---

### III. Volitional movement mixing synergies

<table>
<thead>
<tr>
<th>Sitting position, knee 10 cm from the edge of the chair</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee flexion from actively or passively extended knee</td>
</tr>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ankle dorsiflexion with unstable side</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
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</tbody>
</table>

**Subtotal III (max 6):**

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### IV. Volitional movement with little or no synergy

<table>
<thead>
<tr>
<th>Standing position, hip at 90°</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee flexion to 90° or hip at 0°</td>
</tr>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ankle dorsiflexion with unstable side</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

**Subtotal IV (max 4):**

---

### V. Normal reflex activity

<table>
<thead>
<tr>
<th>Reflex activity, supine position, assessed only if full score of 4 points is achieved in part IV, compare with the unaffected side</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 of 3 reactions markedly hyperactive or at least 1 reflexes lively maximum of 1 reflex lively, none hyperactive</td>
</tr>
</tbody>
</table>

**Subtotal V (max 2):**

**Total E (max 28):**

---

### F. COORDINATION / SPEED

- **Supine, after one trial with both legs, eyes closed, need to knee cap of the opposite leg, 5 times as fast as possible:**
  - Tremor: at least 1 completed movement
    - marked: 0
    - slight: 1
    - none: 2
  - Dystonia: pronounced or unsystematic slight and systematic no dystonia
    - 0
    - 2-5s
    - >2s
  - Time: 8 or more seconds slower than unaffected side, 2-5 seconds slower than unaffected side less than 2 seconds difference
    - 0
    - 2

**Total F (max 9):**

---

### H. SENSATION, lower extremity

<table>
<thead>
<tr>
<th>Light touch: leg, foot sole</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hypoesthesia or dyssynergia</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Position: hip, knee, ankle, plantar flexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

**Total H (max 12):**

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### I. PASSIVE JOINT MOTION

<table>
<thead>
<tr>
<th>Lower extremity supine position, compare with the unaffected side</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</tbody>
</table>

**Total J (max 20):**

---

### J. JOINT PAIN during passive motion, lower extremity

<table>
<thead>
<tr>
<th>Pronounced pain during movement or very painful at the end of the movement</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Only few degrees (&lt;10° hip)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
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</tbody>
</table>

**Total E-F (motor function):**

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**TOTAL (max 34):**

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Approved by Fugl-Meyer AR 2010

Updated 2019-03-03

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Approved by Fugl-Meyer AR 2010

Updated 2019-03-03
MODIFIED ASHWORTH SCALE

- Used to assess resistance to PROM due to spasticity
  - 0 No increase in muscle 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(s) is moved in flexion or extension
  - 1+ Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM
  - 2 More marked increase in muscle tone through most of the ROM, but affected part(s) easily moved
  - 3 Considerable increase in muscle tone, passive movement difficult
  - 4 Affected part(s) rigid in flexion or extension
TARDIEU SCALE AND MODIFIED TARDIEU SCALE (MTS)

- Clinical measure of muscle spasticity
- Spasticity is quantified by assessing muscle response to stretch applied at given velocities using a 0-5 scale
  - As slow as possible (V1)
  - Speed of the limb segment falling under gravity (V2)
  - As quickly as possible (V3)
- Joint angle measurement
  - R1 (angle of catch following a fast velocity stretch – during V3)
  - R2 (PROM – during V1)
  - The difference between the two measures R2-R1 represents the velocity-dependent tone or where the movement is influenced by spasticity

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No resistance throughout the course of the passive movement.</td>
</tr>
<tr>
<td>1</td>
<td>Slight resistance throughout the course of the passive movement, with no clear catch at a precise angle.</td>
</tr>
<tr>
<td>2</td>
<td>Clear catch at a precise angle, interrupting the passive movement, followed by a release.</td>
</tr>
<tr>
<td>3</td>
<td>Fatigable clonus (&lt;10 seconds when maintaining pressure) occurring at a precise angle.</td>
</tr>
<tr>
<td>4</td>
<td>Infatigable clonus (&gt;10 seconds when maintaining pressure) occurring at a precise angle.</td>
</tr>
<tr>
<td>5</td>
<td>Joint is immobile.</td>
</tr>
<tr>
<td>MODIFIED ASHWORTH</td>
<td>MODIFIED TARDIEU</td>
</tr>
<tr>
<td>---------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>• Commonly used clinical measure of spasticity</td>
<td>• Has the ability to differentiate between contracture and spasticity</td>
</tr>
<tr>
<td>• Widely used in research</td>
<td>• Uses velocity as part of the equation to identify and differentiate neural</td>
</tr>
<tr>
<td>• Does not differentiate spasticity from contracture</td>
<td>limitations</td>
</tr>
<tr>
<td>• May not be able to detect small changes</td>
<td>• Velocity dependent (spasticity)</td>
</tr>
<tr>
<td>• Measures muscle tone at one, unspecified velocity</td>
<td>• Passive stiffness (soft tissue limitations)</td>
</tr>
<tr>
<td>• Does not gauge velocity-dependent increase</td>
<td></td>
</tr>
<tr>
<td>• Can cause variation in overall reliability</td>
<td></td>
</tr>
</tbody>
</table>
How do we make decisions regarding treatment?

Decreased spasticity does not always imply improved function.
28 y/o male presents to acute care hospital s/p motorcycle vs. truck collision. At admission patient presents with GCS 11. Patient was diagnosed with R SAH. He has no significant PMHx. Previously working full time as a foreman for construction company. Patient lives with wife in a ranch style home. Patient is L hand dominant.

Patient's goal is to return to work and get back on his motorcycle.

Examination findings

Functional status: Min-Mod A for ADLs and mobility

PROM UE/LE is WNL

Tone assessment:
L LE: MAS 1 knee extensors, two beat clonus at the L ankle with quick stretch into DF
L UE: MAS 1+ biceps, pecs and finger flexors

Strength of UE/LE on L is emerging - grossly 2- throughout
BREAK INTO SMALL GROUPS AND DISCUSS

- Will you intervene to address this patient’s tone?

- If yes, how so…
  - What treatment strategies might you use? Why?
  - What are your goals?

- Consider the setting and the importance of an interdisciplinary team
Plan ahead... Think prevention!

Consider joints most likely to be affected by changes in tone

Maintain neutral joint positions
INTERVENTION: ACUTE CARE

- Patient & family Education
- Caregiver Education and Instruction

**Patient experience**

- Importance of re-assessment, communication & collaboration across disciplines
- Team approach – Physiatrist, Neurologist, OT, PT, Nursing staff, Orthotist
Patient is now 5 weeks post injury and has been participating in inpatient rehabilitation.

- Examination findings
- Functional status: Supervision-Min A for ADLs and mobility including ambulation using SBQC

- PROM
  - L UE: elbow extension = -40' from 0'
  - L LE WNL except ankle DF = -10' from 0

- Tone assessment:
  - L LE: MAS 2 knee extensors, multi-beat clonus at the L ankle with quick stretch into DF
  - L UE: MAS 3 biceps, MAS 2 pecs and finger flexors

- Strength of UE/LE on L: synergistic movement, unable to isolate to perform MMT
• Will you intervene to address this patient’s spasticity?

• If yes, how so?
  • What treatment strategies might you use? Why?
  • What are your goals?

• Consider the setting and the importance of an interdisciplinary team
M3 – Assessment of Spasticity

M 3.1 Individuals with traumatic brain injury with spasticity should be assessed and provided with a coordinated plan for interdisciplinary management including:

- Identification and management of aggravating factors such as pain, bladder or bowel distention, skin irritation and infection
- Use of specific treatment modalities such as serial casting or removable splints
- Use of anti-spasticity medications (See section M4 for more details)
- Rehabilitation interventions that consider a range of motion, flexibility and positioning routine.

(Adapted from ABIKUS 2007, G63, p. 26)
MEDICAL MANAGEMENT

- Oral medications
  - Baclofen – Level 4 evidence may improve LE spasticity, unclear UE
  - Dantrolene sodium
  - Tizanidine (Zanaflex)
- ITB
  - Continuous administration of medication directly into CSF
  - Considered when other options exhausted (medications, botox, casting etc.)
  - Complications – infection, over-dose/withdrawal

What are the pharmacological implications for the therapist?
WHAT’S IN YOUR TOOLBOX?

- NDT/Feldenkrais
- Weight bearing/deep pressure
- Traction
- Rhythmic rotation
- Thermal application
- Taping
- Vibration
- Electrical Stimulation
- Aquatics
- Neurodevelopmental positions
- Sustained positioning
WHAT DO WE KNOW ABOUT STRETCH?

Is stretching an effective treatment for spasticity or muscle contracture due to spasticity?

For how long? How often?

Long term effect?
Examples: Serial casting, splinting, standing frame

In general a longer duration stretch results in longer duration of tone reduction

Serial casting

- Shown to be both safe and effective for adults and children with spasticity
- Large variation in protocols
- Indications for casting

*When do YOU use serial casting?*

*How do you maintain gains achieved during serial casting process?*
ADDITIONAL INTERVENTION

Neuromuscular Electrical Stimulation:

- Commonly used as a rehabilitation modality and adjunct to intervention e.g. Botox, NDT
- May acutely decrease spasticity
- *Research needed to determine if spasticity reduction increased motor control!*

Taping:

- Control joint position
- Reduce excitability of spastic muscle fibers
- *Research needed to determine effectiveness!*
Patient is now 10 weeks post injury and has been participating in outpatient services.

- Baclofen 20mg qid

- Examination findings
  - Functional status: (S) for ADL and Mod I for mobility

- Tone assessment:
  - L LE: MAS 1+ knee extensors, multi-beat clonus at the L ankle with quick stretch into DF
  - L UE: MAS 2 biceps/pecs, MAS 3 finger flexors

- PROM:
  - L elbow –10’ from 0’
  - L hand and wrist to neutral
  - L ankle DF to neutral

- UE/LE strength on L:
  - LE grossly 4/5 except ankle DF 3/5
  - Proximal UE grossly 3/5 within available ROM
  - Distal UE utilizing gross grasp with limited release
• Will you continue to treat this patient’s spasticity?

• If yes, how so?

• What additional treatment strategies might you use? Why?
  • What are your goals?

• Consider the setting and the importance of the interdisciplinary team
CANNABIS

• American Academy of Neurology – reviewed 34 studies on effects of medical marijuana in neurologic disorders
  • Conclusion – there is evidence (strong?) for use of medical marijuana to reduce spasticity and pain
    BUT...
    • Large mixed population (Epilepsy, MS, Huntington's)
    • Multiple routes of administration included (oral, inhaled)
• National Academy of Sciences published "The Health Effects of Cannabis and Cannabinoids: The current state of evidence and recommendations for research" in 2017
  • No information specific to ABI/TBI for treatment of spasticity
**BOTULINUM TOXIN (BOTOX) INJECTIONS**

<table>
<thead>
<tr>
<th>Icon</th>
<th>Description</th>
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<tr>
<td>🌶️</td>
<td>Treats local spasticity</td>
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<tr>
<td>🍈</td>
<td>Intramuscular injection directly into target muscle (Type A = Botox)</td>
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<tr>
<td>⏰</td>
<td>Effect should be noted within 10 days Peak effect occurs 4-6 months</td>
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</tbody>
</table>

What is the role of OT/PT?
WHAT IS BEST PRACTICE?

- Casting
- Taping
- E-stim
- Stretching
EQUIPMENT

- Seating and positioning
  - Accomodation vs. Corrective
  - Posterior pelvic tilt to reduce extensor hypertonicity
  - Foot plate in PF
- Orthotics
  - Custom fabrication is usually best
  - Examples:
    - AFO with tone reducing modifications – extended foot/toe plate / inhibitor bar under first met head
    - UE splint incorporating reflex-inhibiting pattern to inhibit spasticity
THE TEAM APPROACH

• Shared goals/aims
• Each professional member must understand their specific contribution and share expertise
• Communication
RESOURCES

• Clinical Practice Guidelines for moderate to severe TBI
  • https://braininjuryguidelines.org/modtosevere/

• Evidence Based Review of moderate to severe Acquired Brain Injury (ERABI)
  • https://erabi.ca/

• Evidence Based Review of Stroke Rehabilitation (EBRSR)
  • http://www.ebrsr.com/

• Stroke engine
  • https://www.strokengine.ca/en/
REFERENCES


