Spasticity Management

JOHN MACFARLANE, CONSULTANT IN REHABILITATION MEDICINE, MUH & CUH

LIZ O’SULLIVAN, SENIOR PHYSIOTHERAPIST IN NEUROSCIENCES, CUH
Disclosures

- John Macfarlane has received honoraria, educational support from Allergan, Ipsen & Merz (manufacturers of botulinum toxin)
- Liz O’ Sullivan has received educational support from Allergan & Merz (manufacturers of botulinum toxin)
Talk Outline

- Introduction
- Definitions of spasticity
- Measurement tools used to measure impairment
- Pharmacological & Non-pharmacological management
- Overview of evidence base for Botulinum Toxin
- Case-studies
- Summary
Spasticity?

- The Adducted/Internally Rotated Shoulder
- The Flexed Wrist
- The Pronated Forearm
- The Clinched Fist
- The Flexed Elbow
- The Thumb-in-Palm Deformity
Spasticity use?

- Systematic review of definitions used for spasticity and tone
- Limited to stroke and UL. Results: 250 papers identified
- Equated to ‘muscle tone’=88
- Lance Definition =78
- No Definition=78
- Own Definition=6
- Measures: Neurophys 47, Biomechanical 228, None 19
- Spasticity, an impairment that is poorly defined and poorly measured

Malhotra et al, Clin Rehab 2009
History of spasticity

- Spasm (from Greek) used by Hippocrates but for seizure
- English adjective ‘‘spastic’’, derived via Latin from the Greek spastikos (‘‘drawing in’’), may have been first used in the 16th century to describe contraction of muscles around wounds
- Little 1843 – modern use- cases of spastic diparesis
- ‘Spasticity’ first used by Erb 1875
- Then ‘spasticity’ was overused, not defined → confusion
- In 1954 Tardieu proposed a definition and a method to measure it
Spasticity – modern era

1980 Lance et al – consensus definition from a conference

"...a motor disorder, characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex as one component of the upper motor neuron (UMN) syndrome”

Criticisms & limitations

Many other definitions..............

2004 EU Spasm group: “disordered sensorimotor control resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles”

2005 Gracies: “an increase in the velocity-dependent reflexes to phasic stretch, detected and measured at rest”
The name “deforming spastic paresis” was then proposed to describe the clinical syndrome caused by lesions involving the corticospinal pathways.

This syndrome contains two disorders:

“Spastic Myopathy” - combining muscle shortening and loss of extensibility. Occurs within hours to days.

Neurological Disorder with 4 main components.................
Deforming Spastic Paresis- 4 neuro components

- **Spastic Dystonia** - an unwanted, involuntary muscle activity at rest, in the absence of any phasic stretch or voluntary effort, but sensitive to tonic stretch

- **Spastic Co-contraction** - unwanted, involuntary muscle activity in the antagonist, during voluntary effort directed to the agonist, aggravated by antagonist stretch

- **Spasticity** - an enhancement of the velocity-dependent responses to phasic stretch, detected and measured at rest

- **stretch-sensitive paresis** - corresponding to a decreased central command to the agonist, aggravated by antagonist stretch
Co-efficient of Impairment in deforming spastic paresis (Gracies 2015)

- Step 1: objective evaluation of active functional performance
- Step 2: assess passive soft tissue extensibility – slow stretch, Xv1
- Step 3: assess the response of the tested muscles to their own stretch (spasticity) – fast stretch Xv3
- Step 4: assess the capacity of agonist activation to overcome passive and active resistance of the tested muscle group
- Step 5: assess the repeatability of overcoming passive and active resistances of the tested muscle. One asks the patient to accomplish as many movements against the tested muscle in a given time
Clinical Measurement of ‘Spasticity’

- Tardieu Scale 1954
- Ashworth Scale 1964 - measures resistance to passive movement – 0, 1, 2, 3, 4
- Modified Ashworth Scale – 0, 1, 1+, 2, 3, 4
- King’s hypertonicity scale
- Coefficients of impairment

- MAS widely used in spasticity research – primary endpoint in most ‘spasticity’ trials
- MAS poor functional relevance and validity, doesn’t measure ‘spasticity’
Finally, ....assessment is introduced, leading to the staged calculation of four impairment coefficients (shortening, spasticity, weakness, fatigability), which should help guide the treatment towards predominantly muscular approaches (muscle modifications endeavours, e.g. by stretch programs), or neural approaches (e.g. training programs) or both.
Pathophysicsiology

Pathophysiological mechanisms in spasticity

- Presynaptic inhibition
- Post-activation depression
- $\gamma$-motoneuron
- $\alpha$-motoneuron intrinsic mechanisms
- Ib inhibitory interneuron
- Gr. II pathways
- Renshaw cell inhibition
- $\lambda$ afferent

Agonist muscle
How much of a problem is spasticity?

- Post stroke spasticity 4-42% (Wissel, Schelosky et al 2010)
- Contracture incidence post stroke 66% of stroke survivors (NIHSS>5) developed at least 1 contracture (Kwah et al 2012)
How Spasticity can present in the upper and lower limbs

- Flexed elbow
- Bent wrist
- Pronated forearm
- Clenched fist
- Thumb in palm
<table>
<thead>
<tr>
<th>Pattern</th>
<th>Muscle involved</th>
<th>Benefits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper limb</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Shoulder adduction, internal rotation and retraction\(^1\) | • Pectoralis major  
• Latissimus dorsi  
• Teres muscle group  
• Subscapularis  
• Rhomboids and interscapular muscles | • Sitting posture  
• Ease of dressing  
• Axillary hygiene  
• Improve balance and symmetry of gait and can sometimes help to reduce unwanted spasticity in the elbow and hand |
| Elbow flexion\(^2\)                          | • Biceps brachii  
• Brachialis  
• Brachioradialis | • Improve flexion deformity  
• Improve reach/retrieve |
| Pronation of the forearm\(^3\)               | • Pronator teres  
• Pronator quadratus | • Hand function |
| Flexed wrist and clenched hand\(^4\)         | • Flexor carpi ulnaris and radialis  
• Flexor digitorum superficialis and profundus  
• Flexor pollicis longus | • Maintain palmar skin hygiene  
• Improve grasp release |
How Spasticity can present in the lower limbs

- Adducted Thigh
- Flexed Knee
- Extended Knee
- Plantar Flexed Foot/Ankle
- Equinovarus Foot
- Striatal Toe
- Flexed Toe
Key steps to treatment of spasticity with Botulinum toxin: RCP 2018

Fig 2: Management strategy for adults with spasticity
Physical management of spasticity _Adapted from Buchanan & Hourihan 2016_

- Standing
- Functional electrical stimulation
- Wheelchair posture and seating
- Splinting and the use of Orthotics
- Stretches
- Active Exercise
- Passive Movement

Balance between movement and positioning
Physical management of spasticity

- COT & ACPIN 2015 Splinting Guideline
- Ankle: contracture correction
  "It is suggested that ankle casts are applied at end range to improve joint range of movement in conjunction with Botulinum toxin A when presenting with clinically significant spasticity_ 2B evidence"

Hand and wrist: contracture prevention

"It is suggested that splints in conjunction with Botulinum toxin A may reduce spasticity as a component in preventing loss of range of movement in selected cases_ 2C evidence"
<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Intervention</th>
<th>Key Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;4 years post stroke.</td>
<td>3 groups all received</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower limb spasticity _</td>
<td>BONT-A plus intervention</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ankle plantar flexors</td>
<td>3 month follow-up. No</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>control group.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>N=35</td>
<td>3 parallel interventions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;10 post Brain .Injury</td>
<td>for lower limb spasticity</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Over 12 weeks.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>N=70 Upper limb spasticity</td>
<td>trial. All subjects received BONT-A injections &amp; 10 day intervention &amp; 1 month follow-up</td>
<td></td>
<td></td>
</tr>
<tr>
<td>affecting wrist and fingers</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Copley et al 2013</td>
<td>Single case design</td>
<td>Splint vs no splint. Measures taken were ROM, MAS &amp; Modified Tardieu Scale over 5 time points over a 4 month follow-up</td>
<td>Individualized resting splints for adults with moderate Hypertonicity (&gt;1+ MAS) resulted in positive clinical effects to ROM, muscle stiffness &amp; spasticity.</td>
</tr>
<tr>
<td>N=10 Upper limb spasticity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>affecting wrist and fingers</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Cochrane Review (2017): Physical management of spasticity

- Stretch for the treatment and prevention of contractures.

- High-quality evidence that stretch did not have clinically important effects on joint mobility in people with neurological conditions if performed for less than 7 months.

- Short-term effects of stretch on QOL, pain, activity limitations & participation restrictions are uncertain.

(Harvey et al 2017)
Non-Pharmacological management of spasticity: NMES

- “Moderate” evidence for electro-neuromuscular stimulation as an adjunct therapy to conventional routine care (pharmacological and rehabilitation) in persons following stroke (Khan et al 2017).

- Systematic review of 29 RCT’s concluded that NMES combined with other treatment modalities can be considered as a treatment option that provides improvements in spasticity and ROM in patients post stroke (Stein et al 2015).

- Usage of NMES with frequency 30-50Hz, pulse width 0.1-0.5ms for 30 minutes 5 times per week for 3 to 4 weeks were associated with successful results.
Electrical Stimulation

- Stein et al. 2015

### Experimental Stimulation

#### NMES on the Leg Spasticity

<table>
<thead>
<tr>
<th>Study or Subgroup</th>
<th>Mean</th>
<th>SD</th>
<th>Total</th>
<th>Mean</th>
<th>SD</th>
<th>Total</th>
<th>Weight</th>
<th>Mean Difference (95% CI)</th>
<th>Mean Difference (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baktiary and Fatemy 2008</td>
<td>-1.6</td>
<td>0.47</td>
<td>17</td>
<td>-1.1</td>
<td>0.66</td>
<td>18</td>
<td>7.6%</td>
<td>-0.50 [-0.88, -0.12]</td>
<td>-1.00 [-1.49, -0.51]</td>
</tr>
<tr>
<td>Johnson et al. 2004</td>
<td>-1</td>
<td>0.59</td>
<td>9</td>
<td>0</td>
<td>0.47</td>
<td>9</td>
<td>7.1%</td>
<td>-1.00 [-1.49, -0.51]</td>
<td>-1.00 [-1.49, -0.51]</td>
</tr>
<tr>
<td>Mesci et al. 2007</td>
<td>-1</td>
<td>0.99</td>
<td>17</td>
<td>0</td>
<td>0.94</td>
<td>18</td>
<td>6.2%</td>
<td>-1.00 [-1.64, -0.36]</td>
<td>-1.00 [-1.64, -0.36]</td>
</tr>
<tr>
<td>Mesci et al. 2009</td>
<td>-1.2</td>
<td>0.54</td>
<td>20</td>
<td>-0.05</td>
<td>0.8</td>
<td>20</td>
<td>7.9%</td>
<td>-1.05 [-1.40, -0.70]</td>
<td>-1.05 [-1.40, -0.70]</td>
</tr>
<tr>
<td>Sabut et al. 2011</td>
<td>-1.1</td>
<td>0.42</td>
<td>27</td>
<td>-0.5</td>
<td>0.39</td>
<td>24</td>
<td>8.5%</td>
<td>-0.60 [-0.82, -0.38]</td>
<td>-0.60 [-0.82, -0.38]</td>
</tr>
<tr>
<td><strong>Subtotal (95% CI)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td><strong>90</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Heterogeneity: Tau² = 0.03; Chi² = 7.74, df = 4 (P = 0.1); I² = 48%
Test for overall effect: Z = 6.46 (P < 0.00001)

#### NMES on the Wrist Spasticity

<table>
<thead>
<tr>
<th>Study or Subgroup</th>
<th>Mean</th>
<th>SD</th>
<th>Total</th>
<th>Mean</th>
<th>SD</th>
<th>Total</th>
<th>Weight</th>
<th>Mean Difference (95% CI)</th>
<th>Mean Difference (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boyaci et al. 2013</td>
<td>-0.09</td>
<td>0.7</td>
<td>11</td>
<td>0.6</td>
<td>0.57</td>
<td>10</td>
<td>6.8%</td>
<td>-0.69 [-1.23, -0.15]</td>
<td>-0.69 [-1.23, -0.15]</td>
</tr>
<tr>
<td>Chau et al. 2009</td>
<td>-0.4</td>
<td>0.72</td>
<td>10</td>
<td>-0.2</td>
<td>0.72</td>
<td>10</td>
<td>6.3%</td>
<td>-0.20 [-0.83, 0.43]</td>
<td>-0.20 [-0.83, 0.43]</td>
</tr>
<tr>
<td>de Kroon et al. 2004</td>
<td>1</td>
<td>1.4</td>
<td>13</td>
<td>0</td>
<td>0.47</td>
<td>15</td>
<td>5.4%</td>
<td>1.00 [0.20, 1.80]</td>
<td>1.00 [0.20, 1.80]</td>
</tr>
<tr>
<td>Hesse et al. 1998</td>
<td>-0.33</td>
<td>0.59</td>
<td>6</td>
<td>-0.16</td>
<td>0.5</td>
<td>6</td>
<td>6.4%</td>
<td>-0.17 [-0.79, 0.45]</td>
<td>-0.17 [-0.79, 0.45]</td>
</tr>
<tr>
<td>Kim and Lee 2014</td>
<td>-0.15</td>
<td>0.45</td>
<td>10</td>
<td>-0.06</td>
<td>0.44</td>
<td>9</td>
<td>7.6%</td>
<td>-0.09 [-0.49, 0.31]</td>
<td>-0.09 [-0.49, 0.31]</td>
</tr>
<tr>
<td>Mangold et al. 2009</td>
<td>-1.5</td>
<td>0.67</td>
<td>12</td>
<td>-2.5</td>
<td>0.67</td>
<td>11</td>
<td>6.8%</td>
<td>1.00 [0.45, 1.55]</td>
<td>1.00 [0.45, 1.55]</td>
</tr>
<tr>
<td><strong>Subtotal (95% CI)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td><strong>62</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Heterogeneity: Tau² = 0.24; Chi² = 25.66, df = 5 (P = 0.0001); I² = 91%
Test for overall effect: Z = 0.44 (P = 0.66)

#### NMES on the Elbow Spasticity

<table>
<thead>
<tr>
<th>Study or Subgroup</th>
<th>Mean</th>
<th>SD</th>
<th>Total</th>
<th>Mean</th>
<th>SD</th>
<th>Total</th>
<th>Weight</th>
<th>Mean Difference (95% CI)</th>
<th>Mean Difference (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barker et al. 2008</td>
<td>-1</td>
<td>1.49</td>
<td>10</td>
<td>0</td>
<td>0.99</td>
<td>10</td>
<td>3.9%</td>
<td>-1.00 [-2.10, 0.10]</td>
<td>-1.00 [-2.10, 0.10]</td>
</tr>
<tr>
<td>Chau et al. 2009</td>
<td>0.1</td>
<td>0.55</td>
<td>10</td>
<td>0.2</td>
<td>0.73</td>
<td>10</td>
<td>6.7%</td>
<td>-0.10 [-0.67, 0.47]</td>
<td>-0.10 [-0.67, 0.47]</td>
</tr>
<tr>
<td>Hesse et al. 1998</td>
<td>-0.83</td>
<td>0.86</td>
<td>6</td>
<td>0.16</td>
<td>0.5</td>
<td>6</td>
<td>5.4%</td>
<td>-0.99 [-1.79, -0.19]</td>
<td>-0.99 [-1.79, -0.19]</td>
</tr>
<tr>
<td>Kim and Lee 2014</td>
<td>-0.1</td>
<td>0.36</td>
<td>10</td>
<td>-0.11</td>
<td>0.57</td>
<td>9</td>
<td>7.2%</td>
<td>0.01 [0.07, 0.48]</td>
<td>0.01 [0.07, 0.48]</td>
</tr>
<tr>
<td><strong>Subtotal (95% CI)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td><strong>62</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Heterogeneity: Tau² = 0.14; Chi² = 6.47, df = 3 (P = 0.09); I² = 54%
Test for overall effect: Z = 1.51 (P = 0.13)

### FES

<table>
<thead>
<tr>
<th>Study or Subgroup</th>
<th>Mean</th>
<th>SD</th>
<th>Total</th>
<th>Mean</th>
<th>SD</th>
<th>Total</th>
<th>Weight</th>
<th>Mean Difference (95% CI)</th>
<th>Mean Difference (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duarte et al. 2011</td>
<td>-0.6</td>
<td>0.48</td>
<td>13</td>
<td>-0.6</td>
<td>0.66</td>
<td>11</td>
<td>48.8%</td>
<td>0.00 [-0.47, 0.47]</td>
<td>0.00 [-0.47, 0.47]</td>
</tr>
<tr>
<td>Hesse et al. 1998</td>
<td>-1.17</td>
<td>0.4</td>
<td>6</td>
<td>-0.5</td>
<td>0.73</td>
<td>6</td>
<td>36.6%</td>
<td>-0.67 [-1.34, -0.00]</td>
<td>-0.67 [-1.34, -0.00]</td>
</tr>
<tr>
<td>Johnson et al. 2004</td>
<td>-1</td>
<td>1.01</td>
<td>10</td>
<td>0</td>
<td>1.75</td>
<td>8</td>
<td>14.4%</td>
<td>-1.00 [-2.36, 0.36]</td>
<td>-1.00 [-2.36, 0.36]</td>
</tr>
<tr>
<td><strong>Total (95% CI)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td><strong>29</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Heterogeneity: Tau² = 0.12; Chi² = 3.75, df = 2 (P = 0.15); I² = 47%
Test for overall effect: Z = 1.32 (P = 0.19)
Key steps to Goal setting

- Identify presenting problems
- Are they amenable to treatment?
- With what intervention?
- Identify broad goal areas
- Are they worthwhile?
- Define SMART goals
- Evaluate goal achievement post intervention
### Verbal rating

<table>
<thead>
<tr>
<th>At Baseline</th>
<th>With respect to this goal do they have?</th>
<th>Some function</th>
<th>No function (as bad as they could be)</th>
</tr>
</thead>
<tbody>
<tr>
<td>At Outcome:</td>
<td>Yes</td>
<td>A lot more</td>
<td>A little more</td>
</tr>
<tr>
<td>Was the goal achieved?</td>
<td>Yes</td>
<td>As expected</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>Partially achieved</td>
<td>No change</td>
</tr>
<tr>
<td></td>
<td>Got worse</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Numerical conversion

<table>
<thead>
<tr>
<th></th>
<th>-1</th>
<th>-2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+2</td>
<td>+2</td>
</tr>
<tr>
<td></td>
<td>+1</td>
<td>+1</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
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<tr>
<td></td>
<td>-1</td>
<td>-1</td>
</tr>
<tr>
<td></td>
<td>-1</td>
<td>-2</td>
</tr>
<tr>
<td></td>
<td>-2</td>
<td></td>
</tr>
</tbody>
</table>
GAS category tool – the 6 essentials

1) Pain/discomfort
2) Involuntary movements
3) ROM/contracture prevention
4) Passive function
5) Action function/Mobility
6) Cosmesis
<table>
<thead>
<tr>
<th>ICF Level</th>
<th>Problem</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Impairment</td>
<td>Muscle spasms</td>
<td>Pain</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Difficulty with seating and posture</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fatigue</td>
</tr>
<tr>
<td></td>
<td>Abnormal trunk and limb posture</td>
<td>Contractures</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Limb deformity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pressure ulcers/other tissue viability problems</td>
</tr>
<tr>
<td></td>
<td>Pain</td>
<td>Distress and low mood</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Poor sleep patterns</td>
</tr>
<tr>
<td>Activity</td>
<td>Loss of active function</td>
<td>Reduced mobility and dexterity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Difficulty with sexual intercourse</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Difficulty with continence</td>
</tr>
<tr>
<td></td>
<td>Loss of passive function</td>
<td>Difficulty with care and hygiene</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Increased carer burden</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Difficulty with wheelchair seating or bed positioning</td>
</tr>
<tr>
<td>Participation</td>
<td>Impact of any/all of the above</td>
<td>Poor self-esteem / self-image</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reduced social interaction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Impact on family relationships</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Impact on work</td>
</tr>
</tbody>
</table>
Measuring Spasticity & Related factors

- **Impairment level**
  - Modified Ashworth Scale
  - Tardieu scale
  - ROM
  - Pain scale

- **Activity Level**
  - Passive function: ARMA / LEG A
  - Active: Timed10M walk, ARAT

(RCP 2018)
Pharmacological Management

- Oral
- Localised Injections
- Intrathecal
- Inhaled!
Oral Agents

- Baclofen
- Tizanidine
- Dantrolene
- Diazepam
- Clonazepam
- Cannabinoids-Sativex
- Gabapentin*
- Pregabalin*

Overall evidence weak- MAS rather than functional/care goals

- Clonidine*
- Levetriacetam*
- Vigabatrin*
- Fampridine*
- Piracetam*

*Unlicensed use

- **N=60, TBI, stroke**
- **1° outcome - MAS at wrist**
- **2° MAS at elbow and fingers**
  
  DAS, MFS, 10m, A/Es

A/E=50% TZD, 20%BTX, 10% PLC

Adapted from Simpson et al
Case Study 1: Mr X left sided weakness post stroke

<table>
<thead>
<tr>
<th>Impairment</th>
<th>Clonus left ankle</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Activity limitation</strong></td>
<td>Active: Reduced transfer &amp; mobility ability</td>
</tr>
<tr>
<td></td>
<td>Passive: Difficulty applying splint &amp; increased care-giver burden</td>
</tr>
<tr>
<td><strong>Restriction in participation</strong></td>
<td>Reduced ability to visit home, family &amp; friends. Impact on quality of life</td>
</tr>
</tbody>
</table>
Goals of management

- Decrease frequency of left ankle clonus when sitting, standing and carrying out transfers by one month post injection.

- Improve transfers and mobility, decrease level of assistance required and recommence mobilising by 1/12 post injection.

- Improve patient’s quality of life through improved participation as measured by Numeric rating scale (NRS) by 1/12 post injection.
Dosage of Xeomin

- 170U Gastrocnemius (85U to each head, 2 sites)
- 80U Soleus
- Injections carried out under Ultra-sound guidance
### Results

<table>
<thead>
<tr>
<th></th>
<th>Pre-injection</th>
<th>1 month post-injection</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ankle ROM</strong> (knee extended)</td>
<td>40° PF → 5° PF</td>
<td>40° PF → 0° Plantigrade</td>
</tr>
<tr>
<td><strong>ROM</strong> (knee flexed)</td>
<td>40° PF → Plantigrade 0°</td>
<td>40° PF → 5° Dorsi-flexion</td>
</tr>
<tr>
<td><strong>Clonus Score ankle</strong></td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>(knee extended)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Clonus Score ankle</strong></td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>(knee flexed)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>MAS ankle</strong> (knee flexed/</td>
<td>2</td>
<td>1+</td>
</tr>
<tr>
<td>extended)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Results post injection

- Reduced clonus joint angle changed from 20° PF pre to 5° DF post-injection.
- MAS reduced from 2 to 1+
- Ankle ROM improved by 5° with knee flexed and extended. Manca et al (2010) reported a median of 10° improvement in passive ROM but this was after a longer follow-up period of six months.
- Improved symmetry of transfers with increased weight-bearing through left leg. Able to step transfer with minimal assistance of 1.
- Walking short distances wearing a dynamic AFO with assistance of 1 to a maximum of 6 m, 20 steps in 36 secs.
### Case Study 2: Mr Y, left sided weakness post stroke

| Impairment | Spasticity involving finger and thumb flexors  
|            | Pain on stretch |
| Activity limitation | Passive: Difficulty with personal hygiene, dressing and applying splint  
|            | Increased care-giver burden |
| Restriction in participation | Impact on quality of life  
|            | Appearance of hand |
Goals of management:

- Decrease spasticity
- Improve ROM - To reduce risk of contracture
- Decrease pain
- Easier application of resting splint
- PADL’S
- Improved quality of life
Dosage of Xeomin

- 75U Flexor Digitorum Superficialis
- 75 U Flexor Digitorum Profundus
- 50 U Flexor Pollicis Longus
- Under ultra-sound guidance
- Maximum total dose per treatment session is 500 U in the upper limb
Results
Results

- Improved ROM of wrist and fingers: ability to weight-bear through left upper limb as part of rehabilitation.
- Decrease in MAS scores from 2 to 1
- Easier application of splint able to tolerate it over-night
- Improved quality of life

<table>
<thead>
<tr>
<th>OCM</th>
<th>Pre-Botox</th>
<th>Post-Botox</th>
</tr>
</thead>
<tbody>
<tr>
<td>PROM fingers flex @MCP/PIP/DIP</td>
<td>30/30/30</td>
<td>0/0/0</td>
</tr>
<tr>
<td>ARMa</td>
<td>12</td>
<td>3</td>
</tr>
<tr>
<td>MAS</td>
<td>2/4</td>
<td>1/4</td>
</tr>
</tbody>
</table>
Baseline data for 975 patients across 57 sites in 14 countries

- 82% spasticity post stroke
- 2/3 had received previous Botulinum toxin injections
- Median time to first injection from diagnosis was 1 yr
- Upper limb muscles most commonly injected
- Instrumental injection guidance used 73% of time
- Common primary goal areas were passive function (31%) and pain (25%)
Intrathecal Baclofen

- MS and SCI
- Test dose procedure
- CSF leak
- A/E- Respiratory & coma
- Catheter kink/ blockage, infection

**Effect of Intrathecal Baclofen on Pain and Quality of Life in Poststroke Spasticity. RCT n=60, Creamer et al, 2018**
In Summary

- Bont-A is useful in the management of **focal spasticity/multi-focal** but it should be used as part of an *integrated multidisciplinary approach* and accompanied by a rehabilitation programme.

- Most evidence is for **passive** goals rather than active function

- Overall need for more and better research
Guidelines

- Simpson DM, et al


References


References


- College of Occupational Therapists and Association of Chartered Physiotherapists in Neurology (2015). ‘Splinting for the prevention and correction of contractures in adults with neurological dysfunction: Practice guideline for occupational therapists and physiotherapist’
References


References


