Optimizing the Management of Spasticity in Spinal Cord Disorders

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Objectives

At the end of the session, participants will be able to:

1. Define spasticity & disabling spasticity;
2. List outcome measures for spasticity;
3. Compare the relative advantages and disadvantages of different treatment options;
4. Optimize treatment strategies following clinical assessment.
The Impact of Spasticity following Spinal Cord Disorders

- Spasticity is common following SCI/D
  - Reported prevalence between 65 – 93% following spinal cord damage.¹

- Individuals with SCD perceive spasticity as a problem
  - In a community survey, spasticity reported as a significant problem by 17% and a moderate problem by 28% of participants.²

- Spasticity can limit physical abilities
  - Transfers, positioning and mobility, ADLs, social participation²
  - Can also have positive effects, e.g. for transfers

- Spasticity impacts health and quality of life (QoL)
  - Disturbed sleep, fatigue and pain, increased risk of injury, pressure ulcers, negative self-image, etc.¹

What is Spasticity?

I shall not today attempt further to define the kinds of material but I know it when I see it.

___ Potter Stewart ___

AZ Quotes
Signs & Symptoms of Upper Motor Neuron Syndrome

- Velocity-dependent increased resistance to passive stretch
- Exaggerated deep tendon reflexes
- Clonus (rhythmic alternating contractions)
- Involuntary spasms (random contractions)
- Rigidity (co-contractions of agonist/antagonists)
- Presence of UMN signs (Babinski, Hoffman)
Ankle Clonus
Involuntary Lower Extremity Spasms
### Definitions of Spasticity

<table>
<thead>
<tr>
<th>Definition</th>
<th>Author, Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>“[…] the presence of a soft yielding resistance that appears only towards the end of passive stretch, and is associated with increased amplitude of tendon reflex.”</td>
<td>Denny-Brown, 30(p129) 1966</td>
</tr>
<tr>
<td>“A velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neurone syndrome”</td>
<td>Lance, 31(p485) 1980</td>
</tr>
<tr>
<td>“A motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes that result from abnormal intra-spinal processing of primary afferent input”</td>
<td>Young, 32(p513) 1994</td>
</tr>
<tr>
<td>“Muscle hypertonia, hyperactive deep tendon reflexes, clonus, and velocity dependent resistance to passive stretch”</td>
<td>Engsberg, 33(p223) 2002</td>
</tr>
<tr>
<td>“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”</td>
<td>Sanger, 34(p91) 2003</td>
</tr>
<tr>
<td>“An unusual tightening of muscles that feels like leg stiffness, jumping of legs, a repetitive bouncing of the foot, muscle cramping in the legs or arms, legs going out tight and straight or drawing up”</td>
<td>Rizzo, 29(p590) 2004</td>
</tr>
<tr>
<td>“An involuntary muscle overactivity, which may have several harmful effects such as pain, deformity, and impaired function”</td>
<td>Ward, 35(p35) 2003</td>
</tr>
<tr>
<td>“Disordered sensi-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles”</td>
<td>Pandyan, 36(p5) 2005</td>
</tr>
<tr>
<td>“Spasticity is defined as a motor disorder characterized by an involuntary, velocity-dependent increase in muscle tone (hypertonicity) that is associated with neurologic conditions or injury to the brain or spinal cord.”</td>
<td>Mullarkey, 37(p514) 2009</td>
</tr>
<tr>
<td>“Velocity dependent (increasing with faster movement of the limb) and varies in terms of direction of the stretch (with arm flexors and leg extensors being more affected)”</td>
<td>Ostrem, 38(p44) 2010</td>
</tr>
<tr>
<td>“Velocity dependence: […] the faster the stretch, the greater the muscle resistance”</td>
<td>Kheder, 39(p290-1) 2012</td>
</tr>
<tr>
<td>“Clasp-knife” phenomenon: […] the limb initially resists movement and then suddenly gives way […]”</td>
<td></td>
</tr>
<tr>
<td>“Stroking effect: stroking the surface of the antagonist muscle may reduce the tone in spasticity…”</td>
<td></td>
</tr>
<tr>
<td>“Distribution: […] differential distribution with antigravity muscles being more affected”</td>
<td></td>
</tr>
</tbody>
</table>

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Limitations of Lance Definition

“A velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neurone syndrome”


- Observed features do not result exclusive from hyperexcitability of stretch reflex
- Not all features are velocity dependent
- Fails to incorporate many common associated signs & symptoms - e.g., clonus, paroxysmal involuntary activation of muscles (spasms), etc.
- Influenced & exacerbated by afferent input unrelated to stretch reflex (e.g., UTIs, stool impaction)
The ABILITY Network is an international panel of clinical experts with experience in rehabilitation, research and the management of persons with SCI and spasticity

From Europe...
Carlotte Kiekens (BE)
Annick Viaene (BE)
Jesus Benito (ES)
Djamel Bensmail (FR)
Anand Nene (NL)

Arminda Lopes (PT)
Alexandre Campos (PT)
Per Ertzgaard (SE)
Bengt Skoog (SE)
Klemen Grabljevec (SI)

... and beyond
Peter New (AUS)
Anthony Burns (CA)
Indira Lanig (USA)
Gerard Bilsky (USA)
Michael Yochelson (USA)
Recommended Definitions

The Ability Network endorsed the following definitions:

- **Spasticity** – Disordered sensori-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles. (Pandyan AD et al. Disabil Rehabil 2005;27:2-6)

- **Disabling spasticity** – Spasticity which is perceived by the individual or caregivers as hindering body function, activities, and/or participation.

Endorsement of Pandyan definition based on 4 factors:

1. The mention of motor control rather than motor disorder
2. Recognition that spasticity is not result exclusively due to hyperexcitability of the stretch reflex
3. Broad clinical applicability
4. Its incorporation of meaningful symptoms as experienced by persons living with spasticity
SPECIAL COMMUNICATION

Optimizing the Management of Disabling Spasticity Following Spinal Cord Damage: The Ability Network—An International Initiative

Anthony Scott Burns, MD, MSc,a,b Indira Lanig, MD,c Klemen Grabljevec, MD,d Peter Wayne New, PhD, MBBS,e,f Djamel Bensmail, MD,g Per Ertzgaard, MD,h,i Anand Vishwanath Nene, MB, MS(Orth), MChOrth, PhD,j,k

From the aDivision of Physiatry, Department of Medicine, University of Toronto, Toronto, Ontario, Canada; bBrain and Spinal Cord Rehabilitation Program, University Health Network—Toronto Rehabilitation Institute, Toronto, Ontario, Canada; cNorthern Colorado Rehabilitation Hospital, Johnstown, CO; dBrain Injury Rehabilitation Department, University Rehabilitation Institute, Ljubljana, Slovenia; eSpinal Rehabilitation Services, Department of Rehabilitation, Caulfield Hospital, Alfred Health, Melbourne, Victoria, Australia; fEpworth-Monash Rehabilitation Medicine Unit, Southern Medical School, Monash University, Melbourne, Victoria, Australia; gDepartment of Physical Medicine and Rehabilitation, R. Paimpont Hospital, Assistance publique - Hôpitaux de Paris, University of Versailles Saint Quentin, Garches, France; Departments of h,iRehabilitation Medicine, and Medicine and Health Sciences, Linköping University, Linköping, Sweden; jRoessingh Centre for Rehabilitation, Enschede, The Netherlands; and kRoessingh Research and Development, Enschede, The Netherlands.

Abstract

Optimizing the treatment of disabling spasticity in persons with spinal cord damage is hampered by a lack of consensus regarding the use of acceptable definitions of spasticity and disabling spasticity, and the relative absence of decision tools such as clinical guidelines and concise algorithms to support decision-making within the broader clinical community. Many people with spinal cord damage are managed outside specialist centers, and variability in practice result in unequal access to best practice despite equal need. In order to address these issues, the Ability Network—an international panel of clinical experts—was initiated to develop management algorithms to guide and standardize the assessment, treatment, and evaluation of outcomes of persons with spinal cord damage and disabling spasticity. To achieve this, consensus was sought on common definitions through facilitated, in-person meetings. To guide patient selection, an in-depth review of the available tools was performed and expert consensus sought to develop an appropriate instrument. Literature reviews are guiding the selection and development of tools to evaluate treatment outcomes (body functions, activity, participation, quality of life) as perceived by people with spinal cord damage and disabling spasticity, and their caregivers and clinicians. Using this approach, the Ability Network aims to facilitate treatment decisions that take into account the following: the impact of disabling spasticity on health status, patient preferences, treatment goals, tolerance for adverse events, and in cases of totally dependent persons, caregiver burden.

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Traumatic or nontraumatic spinal cord damage is defined as a pathologic injury or process affecting the function of the spinal cord. Spasticity is a common and debilitating secondary complication after spinal cord damage. Despite this, there is a scarcity of evidence-based guidelines to support the assessment and management of spasticity after spinal cord damage. This void contributes to fragmented care and unequal access to best clinical practice. In addition, there are no single, universally accepted definitions of spasticity or disabling spasticity. The development of evidence-based clinical guidelines and an algorithm for the assessment, treatment, and determination of outcomes after the treatment of spasticity arising from spinal cord damage would provide a valuable resource to specialist and nonspecialist clinicians who manage these people, while reducing variation in practice and optimizing treatment outcomes.
Pathophysiology of Spasticity
What Causes Spasticity?

Basic Theory
• Loss of descending inhibition to the motor neurons in spinal cord:
What Causes Spasticity?

Basic Theory
- Loss of descending inhibition to the motor neurons in spinal cord:
  - Spasticity of cerebral origin results from lack of descending inhibitory input due to injury to the brain
What Causes Spasticity?

**Basic Theory**

- Loss of descending inhibition to the motor neurons in spinal cord.

  - Spasticity of cerebral origin results from lack of descending inhibitory input due to injury to the brain
  
  - Spasticity of spinal origin results from interruption of descending tracts that inhibit or modulate alpha and gamma motor neurons

- Plasticity in the spinal cord likely also contributes to and reinforces spasticity.
Loss of Inhibition in Spasticity

Normal Muscle Tone

Descending Inhibition
Sensory Excitation

Diminished Inhibition

Descending Inhibition
Sensory Excitation
## Physiologic Mechanisms of Spasticity

**Table 1.** The Likelihood of Involvement of the Various Mechanisms Thought to Contribute to Spasticity After Spinal Cord Injury and Their Extent of Significance

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Involvement in Spasticity</th>
<th>Significance for Spasticity</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enhancement in the excitability of motoneurons</td>
<td>Most likely</td>
<td>High</td>
<td>7, 10-12</td>
</tr>
<tr>
<td>Enhancement in the excitability of interneurons</td>
<td>Most likely</td>
<td>High</td>
<td>7, 13, 14</td>
</tr>
<tr>
<td>Axonal sprouting</td>
<td>Likely</td>
<td>High</td>
<td>15-17</td>
</tr>
<tr>
<td>Reduction in presynaptic inhibition</td>
<td>Likely</td>
<td>Moderate</td>
<td>18-20</td>
</tr>
<tr>
<td>Reduction in postactivation depression</td>
<td>Likely</td>
<td>Uncertain</td>
<td>21-24</td>
</tr>
<tr>
<td>Reduction in la-reciprocal inhibition</td>
<td>Likely</td>
<td>Unclear</td>
<td>25-27</td>
</tr>
<tr>
<td>Fusimotor hyperexcitability</td>
<td>Unlikely</td>
<td>None</td>
<td>28, 29</td>
</tr>
</tbody>
</table>

Assessment
“If you can’t measure it, you can’t manage it”

Peter Drucker
IMPORTANCE OF ASSESSMENT

- Facilitate a full appreciation of the impact of spasticity
- Identify the need for intervention and accompanying treatment goals
- Central to the determination of treatment efficacy
- Lack of consensus on clinical and functional measures suitable for routine assessment in clinical practice; end result is considerable variability in day-to-day clinical practice
SPECIAL COMMUNICATION

Clinical Assessment of Spasticity in People With Spinal Cord Damage: Recommendations From the Ability Network, an International Initiative

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Klemen Grabljevec, MD, Arminda Lopes, MD, Bengt Skoog, MD, PhD,
Anthony S. Burns, MD, MSc

From the *Roosevelt Center for Rehabilitation, Roessingh Research & Development, Enschede, the Netherlands; †Neurosurgery Department, Santa Maria Hospital, Lisbon, Portugal; ‡University Rehabilitation Institute, Ljubljana, Slovenia; §Centre of Physical and Rehabilitation Medicine of the South, São Brás de Alportel, Portugal; § Sahlgrenska University Hospital, Gothenburg, Sweden; and †University of Toronto, Toronto, ON, Canada.

Abstract

A thorough assessment of the extent and severity of spasticity, and its effect on functioning, is central to the effective management of spasticity in persons with spinal cord damage (SCD). These individuals however do not always receive adequate assessment of their spasticity. Inadequate assessment compromises management when the effect of spasticity and/or need for intervention are not fully recognized. Assessment is also central to determining treatment efficacy. A barrier to spasticity assessment has been the lack of consensus on clinical and functional measures suitable for routine clinical practice. To extend on existing work, a working group of the Ability Network identified and consolidated information on possible measures, and then synthesized and formulated findings into practical recommendations for assessing spasticity and its effect on function in persons with SCD. Sixteen clinical and functional measures that have been used for this purpose were identified using a targeted literature review. These were mapped to the relevant domains of the International Classification of Functioning, Disability and Health to assess the breadth of their coverage. Coverage of many domains was found to be lacking, suggesting a focus for future work. The advantages, disadvantages, and usefulness of the measures were assessed using a range of criteria, with a focus on usefulness and feasibility in routine clinical practice. Based on this evaluation, a selection of measures suitable for initial and follow-up assessments are recommended. The recommendations are intended to have broad applicability to a variety of health care settings where people with SCD are managed.

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Spasticity is a common and often debilitating complication of spinal cord damage (SCD). The Ability Network is an international collaboration of experts formed with the primary objective of addressing the assessment and management of spasticity in people with SCD. Although the prevalence of spasticity in people with SCD of at least 1-year duration has been estimated at 60% to 93%, there is considerable variability in how it has been defined in the medical literature. The Ability Network has previously recommended adoption of the definition by Pandyan et al., in which spasticity is defined as “disordered sensori-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles,”1,5 Spasticity not only affects persons with SCD but also the caregivers of those individuals. The Ability Network has therefore recommended a definition of disabling spasticity, which takes this into account, defining disabling spasticity as “spasticity which is perceived by the affected individual or caregivers as hindering body function, activities, and/or participation.” This definition conceptually incorporates the domains of the International Classification of Functioning, Disability and Health (ICF).1,5

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Spasticity

International classification of functioning, disability and health (ICF)
Guide to assessment of spasticity in persons with SCD

Clinical and functional measures for spasticity following SCD
Assess spasticity at routine visits and before and after interventions

Presence, extent and severity of spasticity

Impact of spasticity on function

Function

Pain present

Person can stand or ambulate

Optional (e.g., research)

Ashworth/Modified Ashworth
Penn Spasm Frequency Scale
Clonus Score
SCATS

ROM
Dynamometry

Numeric Pain Rating Scale

Berg Balance Scale
Timed Up & Go Test
10 m Walk Test
6 min Walk Test

WISCI II
PRESENCE, EXTENT, AND SEVERITY OF SPASTICITY (N = 7)

- Ashworth/Modified Ashworth Scale (MAS)
- Clonus score
- Numeric Pain Rating Scale
- Pendulum Test (Wartenburg)
- Penn Spasm Frequency Scale
- Range of motion/goniometry
- Spinal Cord Assessment Tool for Spastic Reflexes (SCATS)
FUNCTIONAL IMPACT OF SPASTICITY (N = 8)

- 6 minute walk test
- 10 meter walk test
- Berg Balance Scale
- Dynamometry
- Timed Up and Go
- Walking Index for Spinal Cord Injury (WISCI, WISCI II)
- Functional Independence Measure (FIM)
- Spinal Cord Independency Measure (SCIM)
International classification of functioning, disability and health (ICF)

Health condition (disorder or disease)

Body Functions & Structure

Activity

Participation

Contextual factors

Environmental Factors

Personal Factors

Spasticity
PRO, PROM, HRQoL

- PRO, Patient Reported Outcome
  - Denote the subjective patient experience, such as subjective symptoms, quality of life, subjective functional status, satisfaction with care and/or compliance with medication—essentially anything that patients know first-hand and is appropriate for them to report (Basch, E. 2014)

- PROM, Patient Reported Outcome Measures
  - Efforts to standardize and instrumentalize PRO. Mainly referring to HRQoL

- HRQoL, Health Related Quality of Life instruments
EXAMPLES OF PROMS & HRQOL MEASURES

- Generic measures of HRQoL
  - SF-36
  - WHOQOL-BREF

- Disease- or condition-specific measures (Spasticity)
  - SCI-SET (https://scireproject.com/)
  - PRISM

- Preference-based utility measures
  - EQ-5D
  - SF-6D
CAREGIVER BURDEN

- Spasticity can have significant consequences for caregivers
  - Generic HRQoL measures
    - Can be used with caregivers - do not provide insight into caregiver-specific problems
  - No specific caregiver burden scales developed for SCD
    - Caregiver Burden Scale (3 studies)
    - Zarit Burden Interview (1 study)
Review Article

A review and evaluation of patient-reported outcome measures for spasticity in persons with spinal cord damage: Recommendations from the Ability Network – an international initiative

Per Ertzgaard 1, Anand Nene 2, Carlotte Kiekens 3,4, Anthony S. Burns 5

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Context: Patient-reported outcome measures (PROMs) are valuable for capturing the impact of spasticity on health-related quality of life (HRQoL) in persons with spinal cord damage (SCD) and evaluating the efficacy of interventions.

Objective: To provide practical guidance for measuring HRQoL in persons with spasticity following SCD.

Methods: Literature reviews identified measures of HRQoL and caregiver burden, utilized in studies addressing spasticity in SCD. Identified measures were evaluated for clinical relevance and practicality for use in clinical practice and research. The PRISM, SCI-SET, EQ-5D and SF-36 instruments were mapped to the International Classification of Functioning, Disability and Health (ICF). The PRISM and SCI-SET were evaluated using the Consensus-based Standards for the Selection of Health Measurement Instruments (COSMIN) checklist.

Results: Two spasticity-specific, five generic, and four preference-based measures were identified. ICF mapping and the COSMIN checklist supported the use of the PRISM and SCI-SET in SCD. The SF-36 is considered the most useful generic measure; disability-adapted versions may be more acceptable but further studies on psychometric properties are required. The SF-36 can be converted to a preference-based measure (SF-6D), or alternatively the EQ-5D can be used. While no measures specific to caregivers of people with SCD were identified, the Caregiver Burden Scale and the Zarit Burden Interview are considered suitable.

Conclusion: Recommended measures include the PRISM and SCI-SET (condition-specific), SF-36 (generic), and Caregiver Burden Scale and Zarit Burden Interview (caregiver burden). Consideration should be given to using condition-specific and generic measures in combination; the PRISM or SCI-SET combined with SF-36 is recommended.

Keywords: Muscle spasticity, Spinal cord diseases, Spinal cord injuries, Patient reported outcome measures, Health-related quality of life

Introduction

Spasticity is a common feature of many neurological conditions characterized by upper motor neuron pathology. Examples include stroke, multiple sclerosis, cerebral palsy, traumatic brain injury, and spinal cord damage (SCD). This report focuses on SCD. It summarizes the deliberations and findings of the Outcomes and Access working group of the Ability Network (AN), an international panel of clinical experts with the overarching goal of addressing challenges and barriers to optimizing the management of disabling spasticity in people with SCD.
Time and Motivation
<table>
<thead>
<tr>
<th>Body function &amp; structure</th>
<th>Activity</th>
<th>Participation</th>
<th>HRQoL</th>
<th>cost-effectiveness</th>
</tr>
</thead>
</table>

- Assessment - individual
- Outcome - individual
- Outcome – clinic/department
- Research
Treatment
Spasticity Treatment Options

- **Reversible**
  - Oral Medications
  - ITB Therapy
  - Rehabilitation Therapy

- **Focal**
  - Injection Therapy

- **General**
  - Neurosurgery

- **Permanent**
  - Orthopedic Surgery
Spasticity Treatment Options

- Oral Medications
- ITB Therapy
- Rehabilitation Therapy
- Injection Therapy

- General
- Reversible
- Focal

- Neurosurgery
- Orthopedic Surgery
- Permanent
Spasticity Treatment Options

- Oral Medications
- ITB Therapy
- Rehabilitation Therapy
- Neurosurgery
- Orthopedic Surgery
- Injection Therapy
- Reversible
- Permanent
Chemical Neurolytics

- Injected directly into targeted nerves or muscles
- Targeted and less prone to systemic side effects
- Efficacy impacted by skill and experience of clinician
  • Improved with EMG or U/S localization
- Not practical for generalized, multi-segmental spasticity

- **Phenol Injections** – neurolysis of nerve.
  • Cheap
  • Dose dependent & injection localization critical
  • Waning clinician familiarity (largely supplanted by Botox)
  • Pain at administered site, causalgia w/ sensory nerve injury

- **Botulinum Toxin** - binds to presynaptic NMJ and prevents acetycholine release.
  • Expensive
  • Efficacy begins within 3-7 days and last 2-6 months
  • Dosing limits number of muscles which can be injected
Spasticity Treatment Options
Orthopedic Surgical Intervention

- Tendon Lengthening & Transfer - preferred method
  - Restores full passive range with some residual muscle tension.
  - Muscle must be immobilized under tension.

- Osteotomy - for skeletal deformity
  - Restore boney architecture, muscle-length can be improved.
  - Used along with tendon lengthening.

- Arthrodesis - joint fusion
  - When the above are prohibited.
  - Stabilize unstable joints (subtalar, thumb, wrist).
Spasticity Treatment Options

Diagram showing treatment options:
- Oral Medications
- ITB Therapy
- Rehabilitation Therapy
- Injection Therapy
- Orthopedic Surgery
- Neurosurgery

Options are categorized by reversibility and permanence:
- Reversible
- Focal
- General
- Permanent
Neurosurgery

- **Dorsal rhizotomies** - Regional spasticity.
  - Cut dorsal roots
  - Historically utilized in cerebral palsy patients

- **Peripheral neurotomies** - Focal spasticity.
Spasticity Treatment Options

- Oral Medications
- ITB Therapy
- Rehabilitation Therapy
- Neurosurgery
- Orthopedic Surgery
- Injection Therapy

Reversible vs. Permanent
Focal vs. General
Passive Range of Motion/Stretching
ORIGINAL ARTICLE

Effects of 6 months of regular passive movements on ankle joint mobility in people with spinal cord injury: a randomized controlled trial

LA Harvey¹, RD Herbert², J Glinsky¹, AM Moseley² and J Bowden¹

¹Rehabilitation Studies Unit, Northern Clinical School, Faculty of Medicine, The University of Sydney, Sydney, New South Wales, Australia and ²Musculoskeletal Division, The George Institute for International Health, Sydney, New South Wales, Australia

Study design: Assessor-blinded within-subject randomized controlled trial.
Objective: To determine the effects of 6 months of regular passive movements on ankle joint mobility in people with spinal cord injury.
Setting: Community, Australia.
Methods: A total of 20 people with tetraplegia living in the community had one ankle randomized to a control group and the other to an experimental group. Carers administered passive movements to participants’ experimental ankles for 10 min, 10 times a week for 6 months. The control ankles were left untreated. The primary outcome was passive ankle dorsiflexion range of motion.
Results: Adherence was high (mean adherence rate of 96%). Ankle dorsiflexion range of motion decreased by a mean (s.d.) of 2° (4) in control ankles and increased by 2° (4) in experimental ankles. The mean (95% confidence interval, CI) effect on ankle dorsiflexion range of motion was 4° (95% CI, 2–6°).
Conclusion: Regular passive movements have small effects on ankle joint mobility. It is unclear if these effects are clinically worthwhile.


Keywords: ankle; stiffness; spinal cord injury; rehabilitation
Randomised trial of the effects of four weeks of daily stretch on extensibility of hamstring muscles in people with spinal cord injuries

Lisa A Harvey¹, Adrian J Byak¹, Marsha Ostrovskaya¹, Joanne Glinsky¹, Lyndall Katte² and Robert Herbert³

¹Royal Rehabilitation Centre, Sydney  ²The Prince Henry Hospital, Sydney  ³The University of Sydney

The aim of this assessor-blind randomised controlled trial was to determine the effect of four weeks of 30 minute stretches each weekday on extensibility of the hamstring muscles in people with recent spinal cord injuries. A consecutive sample of 16 spinal cord-injured patients with no or minimal voluntary motor power in the lower limbs and insufficient hamstring muscle extensibility to enable optimal long sitting were recruited. Subjects' legs were randomly allocated to experimental and control conditions. The hamstring muscles of the experimental leg of each subject were stretched with a 30 Nm torque at the hip for 30 minutes each weekday for four weeks. The hamstring muscles of the contralateral leg were not stretched during this period. Extensibility of the hamstring muscles (hip flexion range of motion with knee extended, measured with a 48 Nm torque at the hip) of both legs was measured by a blinded assessor at the commencement of the study and one day after the completion of the four-week stretch period. Changes in hamstring muscle extensibility from initial to final measurements were calculated. The effect of stretching was expressed as the mean difference in these changes between stretched and non-stretched legs. The mean effect of stretching was 1 degree (95% CI -2 to 5 degrees). Four weeks of 30 minute stretches each weekday does not affect the extensibility of the hamstring muscle in people with spinal cord injuries. [Harvey LA, Byak AJ, Ostrovskaya M, Glinsky J, Katte L and Herbert R (2003): Randomised trial of the effects of four weeks of daily stretch on extensibility of hamstring muscles in people with spinal cord injuries. Australian Journal of Physiotherapy 49: 176-181]

Key words: Contracture; Muscles; Quadriplegia; Rehabilitation
# Pharmacological Intervention

<table>
<thead>
<tr>
<th>Drug</th>
<th>Site of Action</th>
<th>Adverse Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Oral Drugs</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Diazepam</em></td>
<td>Brainstem reticular formation and spinal polysynaptic pathways</td>
<td>Fatigue; reduced motor coordination, intellect, attention, memory</td>
</tr>
<tr>
<td><em>Dantrolene Sodium</em></td>
<td>Skeletal muscle calcium stores</td>
<td>Hepatotoxicity, generalized muscle weakness</td>
</tr>
<tr>
<td><em>Oral Baclofen</em></td>
<td>GABA-b receptors</td>
<td>Drowsiness, confusion, headache, lethargy</td>
</tr>
<tr>
<td><em>Tizanidine Hydrochloride</em></td>
<td>a2-adrenergic receptors</td>
<td>Dizziness, sedation, dry mouth</td>
</tr>
<tr>
<td><strong>Intrathecal Drugs</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intrathecal Baclofen</td>
<td>Gaba-b receptors</td>
<td>Pump malfunction/ dislocation</td>
</tr>
</tbody>
</table>
Baclofen (oral)

- Most commonly prescribed medication for spasticity of CNS etiology.
- GABA agonist that binds to GABA-b (inhibitory) receptors in the CNS.
- Oral baclofen has supraspinal activity that contributes to side effects.
  - sedation, excessive weakness, dizziness, mental confusion, somnolence.
- Reported incidence of adverse effects has ranged from 10% to 75%.
  (Dario A, Tomei G. Drug Safety 2004; 27: 799-818)
- ~25-30% SCI & MS patients fail to respond adequately to oral baclofen.
Intrathecal Baclofen (ITB)

1. Pump is implanted in a pocket under the skin of the abdomen.
2. The catheter is tunneled under the skin to the back.
3. The catheter tip is inserted into the intrathecal space surrounding the spinal cord.
4. Baclofen is delivered directly to the cerebrospinal fluid surrounding the spinal cord.
Intrathecal baclofen is most effective treatment for severe generalized, refractory spasticity
## Basic criteria for ITB therapy

### Indications for ITB therapy:

- **Spasticity is severe** – the patient presents with increased tone that causes significant pain or interferes with function and/or care, which may be accompanied by spasms.
- **Spasticity is unresponsive** to oral Baclofen or the patient experiences intolerable central nervous system (CNS) side effects at effective doses.
- **Alternate treatment modalities** have not been effective/sufficient to manage spasticity.
- **Patient has sufficient psychosocial support and resources** to consistently meet refill follow-up care requirements (approximately 3-6 visits annually).
Goals of ITB Therapy?

Examples:

- Facilitate completion of activities of daily living (ADLs)
- Decrease caregiver burden
- Improve sleep
- Decrease pain (related to spasticity/spasms)
- Prevent contractures
- Improve mobility/transfers
- Improve wheelchair sitting
- Improve gait (ambulatory patients)
Advantages of ITB Therapy?

- Drug delivered directly to the site of action (spinal cord)
- Central side effects (brain) minimized such as drowsiness or confusion
- Higher baclofen concentrations (CSF) than those attainable via the oral route.
- Pump can be non-invasively programmed to deliver a range of infusion rates in customized dosing patterns
- Reversible
Medtronic Targeted Drug Delivery
(4 Generations)

- 1\textsuperscript{st} Generation – SynchroMed
- 2\textsuperscript{nd} Generation – SynchroMed EL
- 3\textsuperscript{rd} Generation – SynchroMed II
- 4\textsuperscript{th} Generation – SynchroMed II
Test Dose Trial
Baclofen injection – bolus via lumbar puncture

Recommended concentration for screening test 50 microgram (µg)/ml

Screening test may be repeated at increased doses if patient does not have positive response to first dose

<table>
<thead>
<tr>
<th>Screening dose</th>
<th>Drug volume</th>
</tr>
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<tbody>
<tr>
<td>25 µg</td>
<td>0.5 ml</td>
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<tr>
<td>50 µg</td>
<td>1.0 ml</td>
</tr>
<tr>
<td>75 µg</td>
<td>1.5 ml</td>
</tr>
<tr>
<td>100 µg</td>
<td>2.0 ml</td>
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</tbody>
</table>

Pharmacokinetics

<table>
<thead>
<tr>
<th>Pharmacokinetics</th>
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<tbody>
<tr>
<td>Onset of action</td>
</tr>
<tr>
<td>Peak effect</td>
</tr>
<tr>
<td>Duration of action</td>
</tr>
</tbody>
</table>
Intrathecal Baclofen Trial

Before ITB
Spasticity of Spinal Origin

- 97% of patients with spasticity of spinal origin demonstrate a positive response to the screening test (Penn RD. J Neurosurg. 1992;77:236-240)
Test Dose Trial
Understanding patient’s response

• **Intrathecal bolus injection:**
  - ‘Light switch’ that turns spasticity off

• **Long-term ITB Therapy® with Implanted Pump:**
  - ‘Dimmer switch’ that allows dose to be adjusted precisely
  - Some patients can retain some functional spasticity while muscle strength and control are developed
Spasticity affects function/ADL, impairs care
Risk of potential clinical complications

Evaluate spasticity,
Consider previous treatment history

Define goals and outcome measures for
SCD spasticity

Manage secondary aggravating factors, e.g. noxious stimuli

Effective/successful
Goals met

Spasticity still problematic
Goals still to be achieved

Disabling elements are FOCAL

Disabling elements are MULTI-SEGMENTAL or GENERALIZED

Intrathecal Baclofen Pump Implantation

Intrathecal Baclofen Screening Test

Screening Test Positive

Screening Test Negative

Pump Titration & Management

Effective/successful
Goals met

Oral Pharma Treatments

Physiotherapy

Injectable Pharma Treatments

Surgical Treatments
SPECIAL COMMUNICATION

Optimizing the Management of Spasticity in People With Spinal Cord Damage: A Clinical Care Pathway for Assessment and Treatment Decision Making From the Ability Network, an International Initiative

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Abstract

The recognition, evaluation, and management of disabling spasticity in persons with spinal cord damage (SCD) is a challenge for health care professionals, institutions, health systems, and patients. To guide the assessment and management of disabling spasticity in individuals with SCD, the Ability Network, an international panel of clinical experts, developed a clinical care pathway. The aim of this pathway is to facilitate treatment decisions that take into account the effect of disabling spasticity on health status, individual preferences and treatment goals, tolerance for adverse events, and burden on caregivers. The pathway emphasizes a patient-centered, individualized approach and the need for interdisciplinary coordination of care, patient involvement in goal setting, and the use of assessment and outcome measures that lend themselves to practical application in the clinic. The clinical care pathway is intended for use by health care professionals who provide care for persons with SCD and disabling spasticity in various settings. Barriers to optimal spasticity management in these people are also discussed. There is an urgent need for the clinical community to clarify and overcome barriers (knowledge-based, organizational, health system) to optimizing the management of spasticity in people with SCD.

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Persons with spinal cord damage (SCD), either traumatic or nontraumatic, are often affected by significant spasticity. The prevalence of spasticity in people with SCD lasting at least 1 year has been estimated at 65% to 93%.3 Spasticity is a dynamic condition that can, in some cases, cause profound disability—either alone or in interplay with other conditions associated with SCD (e.g., pain, weakness, pressure ulcers or other wounds, infection). In surveys addressing the perceived importance of problems, individuals with SCD consistently rank spasticity among their top-4 life concerns.2 In a community sample of people living with SCD, 17% reported

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https://doi.org/10.1016/j.apmr.2018.01.017
CASE #1 – SPINAL CORD DAMAGE

J.W. is a 62 year old male who tripped over a curb while walking his dog.

Before he could extend his arms he fell forward striking his head on the ground.

J.W. was unable to move his arms and legs and was transported to University Hospital.

CT imaging of the head and neck was unremarkable with the exception of degenerative cervical spondylosis with prominent osteophytes adjacent to the C4-5 endplates.

Magnetic resonance imaging (MRI) revealed the presence of a congenitally narrow cervical canal, multi-level degenerative disk disease, and bulging of the posterior longitudinal ligament. Increased intramedullary T2 signal was visible posterior to the C4 vertebral body.

Taken to the O.R. next day for C4-5 laminectomies, C3-6 partial laminectomies, and C3-6 posterior instrumented fusion.
CASE #1 – HOSPITAL PRESENTATION

- Alert & oriented to person, place, and time.
- UEx motor function - trace elbow flexors.
- LEx motor function - 2/5 hip adductors, knee extensors, ankle plantarflexors.
- C3 sensory level bilaterally.
- No volitional contraction of external sphincter; bulbocavernosus reflex absent.
- UEx & LEx flaccid with pROM; deep tendon reflexes absent.
- Neurological classification – C3 AIS grade C
CASE #1 – REHABILITATION & FUNCTIONAL OUTCOMES

- Inpatient rehabilitation x 3 months; outpatient rehabilitation x 4 months
- Progressed C3 AIS D
- Independent household ambulator with forearm crutches & bilateral AFOs
- Motorized scooter used in community
- Modified independent for ADLs; utilizes bath bench and raised toilet seat
CASE #1 – INITIAL ASSESSMENT

- When assessed 9 months post-injury, J.W. voiced several concerns:

  - Reported feeling “stiff” and his upper body “was like a block of wood”
  - Dressing is difficult; wife assists with donning his shirt and styling his hair.
  - Sleep is poor and his legs “jump all night”. Tired in the morning and his wife is sleeping in a separate bed.
  - Frustrated with “slow” walking; difficult to get started after standing.
  - Legs jerk unexpectedly and he has fallen 3x in the past 2 months. Feels his symptoms are worse in the winter.
PRESENCE, EXTENT, AND SEVERITY OF SPASTICITY (N = 7)

- Ashworth/Modified Ashworth Scale (MAS)
- Clonus score
- Numeric Pain Rating Scale
- Pendulum Test (Wartenburg)
- Penn Spasm Frequency Scale
- Range of motion/goniometry
- Spinal Cord Assessment Tool for Spastic Reflexes (SCATS)
CASE #1 – PRESENCE, EXTENT, & SEVERITY

- pROM was performed of the upper and lower extremities:
  - Shoulder abduction limited to 90° on the left & 100° on the right
  - Shoulder external rotation ~60° bilaterally
  - Lacking 10-20° terminal elbow extension bilaterally

- Tone was assessed in the upper and lower extremities using the MAS:
  - Shoulder abductors MAS 3 bilaterally
  - Elbow flexors MAS 2 bilaterally
  - Hip/knee extensors MAS 2 bilaterally
  - Ankle plantarflexors MAS 2 bilaterally

- Other significant findings included:
  - Presence of bilateral Hoffman, Chaddock, and Babinski signs
  - Sustained bilateral clonus at the ankles

- SCATS completed to document extent of spasticity:
  - Clonus subscale – 3 (severe) bilateral
  - Flexor spasm subscale – 1 (mild) bilateral
  - Extensor subscale – 2 (moderate) bilateral
FUNCTIONAL IMPACT OF SPASTICITY (N = 8)

- 6 minute walk test
- 10 meter walk test
- Berg Balance Scale
- Dynamometry
- Timed Up and Go
- Walking Index for Spinal Cord Injury (WISCI, WISCI II)
- Functional Independence Measure (FIM)
- Spinal Cord Independence Measure (SCIM)
CASE #1 – INITIAL ASSESSMENT (FUNCTIONAL IMPACT)

- Gait was assessed:
  - Speed was diminished
  - Base of support widened with forearm crutches
  - ‘Stiff’ with decreased knee flexion during swing phase
  - Periodic patellar clonus/spasms during early stance
  - Foot clearance diminished but adequate with ankle foot orthoses (AFOs)

- 10 meter walk was performed:
  - 10 MWT = 0.6m/s

- Berg Balance Scale (BBS) completed:
  - BBS score = 38/56 (medium fall risk)
CASE #1 PATIENT REPORTED OUTCOMES

- University Hospital (research?)
- Patient relatively independent – minimal aid in ADL
- Walking with very low speed
- Poor sleep

Choice of instruments:
- No caregiver burden instrument
- SF-36 (generic)
- SF-6D (preference-based utility instrument)
- SCI-SET (symptom check-list and condition-specific instrument)
- Clear treatment goals according to SMART
CASE #1 TREATMENTS—GENERALIZED SPASTICITY

- Physiotherapy
- Identify/treat secondary contributors to spasticity

Trial of oral medications
- baclofen*
- tizanidine*/clonidine
- dantrolene*
- benzodiazepines

Upper limbs: pect major
- botulinum toxin *: consider depending on response

Order of oral trial
- Best evidence *
Treatment Response:

- Cognitive side effects from baclofen at 10mg tid with no benefit
- Started on tizanidine: partial response at 24mg/d → sedation
- Dantrolene initiated and titrated to 25mg TID; discontinued after transaminases (AST/ALT) increased

?
CASE #1 FURTHER OPTIONS

- Discussion of ITB as option
- Proceed to test dose
Questions?

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