Spasticity Management v.3.6
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Achieving Goals
Military: Target dictates weapon, weapon dictates movement (Navy SEAL doctrine)
Civilian Application: Goal dictates treatment, treatment dictates action

Richard J. Machowicz: Unleash the Warrior Within, 2002
Goals of the Lecture
- Usefully define spasticity
- Review patient goal-setting guidelines
- Identify potential treatments with their possible side effects
- Learn to apply potential treatments to specific clinical situations

Definition of Spasticity
- Lance, 1980: “Motor disorder characterized by 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 neuron syndrome”

- Velocity-dependent exaggeration of responses to phasic stretch = tendon jerks or passive movement
- Limitations of Definition
- Downplays importance of sensory ("afferent") contribution to spasticity
- Many of the most disabling manifestations of spasticity are not velocity dependent
- Many patterns of spasticity are not explained by hyperexcitability of reflexes alone

- Upper Motor Neuron Syndrome
- Collection of symptoms and signs caused by damage to the brain and spinal cord
- “Positive signs”: Spasticity, Hyper-Reflexia, Abnormal cutaneous responses, Tremor, Abnormal posturing/dystonias
- “Negative signs”: Weakness, Loss of motor control, Sensory loss, Ataxia, Autonomic dysfunction, bowel/bladder dysfunction

Etiologies
- Stroke
- Cerebral palsy
- Multiple sclerosis
- Traumatic brain injury
- Spinal cord injury
- Hypoxic brain or spinal cord injury
- Neurodegenerative disease
- Mass Lesions
- Idiopathic
- Etiologies
- The great majority of conditions causing spasticity are not curable (yet).
- Therefore, spasticity encompasses a large number of symptoms which we can only hope to manage, rather than eliminate.
- Prevention of secondary complications, like joint contractures, is critical in the context of renewed hope for a cure for many causes of spasticity.
- Terms of Confusion
- Spasticity
- Spasm
- Spastic co-contracture
- Spastic Dystonia
- Spasmodic Torticollis (aka cervical dystonia)
- Definitions: Clarified
- Spasticity: As previously stated. It is a symptom of upper motor neuron damage, not a diagnosis.

Spasmodic Torticollis, aka cervical dystonia: Idiopathic movement disorder causing muscle over-activity with abnormal stereotyped movement patterns affecting the neck and shoulders. The most common type of Focal Dystonia; a diagnosis.

- Muscle “Spasm”
Involuntary, regional, painful muscle tightening; usually a symptom of local injury.

- Most common example is the “back spasm” following lumbar muscle, tendon or ligament injury
- Most likely due to central mechanisms which function to limit further injury

- Spastic Co-contraction
- Sub-type of spasticity that results in simultaneous activation of both agonists and antagonists around a joint, resulting in loss of motor control, slow movement, abnormal positioning, and loss of range of motion –OR-
- **Spastic Co-Contraction**
  Excessive muscle contraction occurring in the antagonists during a voluntary command on agonists, even in the absence of phasic stretch

- **Phenomena Commonly Associated With Spasticity**
  - *Other types of muscle overactivity* (present with yawning, coughing, breathing, etc)
  - *Muscle shortening* = consequence of both immobilization and muscle overactivity
  - *Motor weakness*
  - Spastic Dystonia
- Sub-type of Spasticity with increased muscle tone present at rest.
- May lead to severe, permanent deformities and therefore is frequently described separate from other manifestations of spasticity.

Pathophysiology of Spasticity: Established Mechanisms

- Alterations within the reflex arc
  - Change in muscle active properties (increased ratio torque/EMG)
  - Change in muscle passive properties (decreased extensibility)
  - Decreased presynaptic inhibition (paraplegics++)
- Increased fusimotor activity and increased excitability of the alpha motor neuron have not been established

- **Pathophysiology of Spasticity:**
  Established Mechanisms (cont’d)

- **Mechanisms outside the reflex arc**
  - Decreased reciprocal Ia inhibition on extensors
  - Decreased nonreciprocal Ib inhibition
  - Decreased inhibition from flexor reflex afferents

- **Phenomena Commonly Associated With Spasticity**
  - Abnormal cutaneous reflexes (Babinski’s sign)
  - Spastic dystonia
    - Muscle contraction present at rest, dependent on tonic stretch
    - Significant contribution to deformity
  - Spastic co-contraction
Abnormal antagonist contraction present during voluntary agonist effort, dependent on tonic stretch on antagonist

- Extrasegmental co-contraction
  - Abnormal contraction distant from the muscles involved in voluntary effort; contribution to synkinesia, overflow, athetosis, etc

- Pathologic Cutaneous or Nociceptive Responses

Exaggeration or disinhibition of complex motor patterns or “Primitive Reflexes”

Produce stereotyped patterns of muscle over-activity

- Complex Motor Patterns or “Primitive Reflexes”
- Polysynaptic spinal cord and/or brain-stem mediated movement patterns which occur in response to cutaneous or nociceptive external stimuli
- Produce stereotyped patterns of movement, present in infancy, which are controlled (or “incorporated”) by higher cortical centers with maturity
- Main source of motor behaviors in infants
- “Primitive Reflexes”
- Precursors to volitional, mature motor skills
- Replaced by “postural reactions” with maturity, which persist throughout life
- Incorporated with maturity, but not lost
- May be unmasked, or disinhibited by central nervous system injury
- Often designated by eponyms

- Moro Reflex
- Sudden neck extension or startling an infant produces multi-joint extension
Recognition of Unmasked “Primitive Reflexes” can lead to better clinical outcomes

Disabling Phenomena Associated With Spasticity

*Muscle shortening, motor weakness, and stretch-dependent muscle overactivity* (spastic co-contraction and spastic dystonia) are probably the most disabling features in patients with spasticity.

→ Three logical solutions in therapy:
**muscle lengthening, motor training, and muscle relaxation**

- **Disabling Phenomena Associated With Spasticity (cont’d)**

- **Muscle overactivity** is most often asymmetrical around joints, one agonist being more overactive (and shortened) than its antagonist.

→ Rationale for **focal** intervention, including **focal muscle relaxation** as first-line therapy

- **Consequences of Phenomena Associated With Spasticity**
Evaluation of Tone: Clinical Rating Scales

- Resistance to passive movement, whatever its mechanism (muscle overactivity, soft tissue contracture)
  - Ashworth, Modified Ashworth, Oswestry scales:
    o 0 to 4 scale of tone intensity
  - Degree of adductor muscle tone (Snow)

- Spasticity per se: Tardieu scale, measuring the spasticity angle = angle of movement arrest at slow speed minus angle of catch-and-release at fast speed

- Modified Ashworth Scale
  - 0: No increase in muscle tone
  - 1: Slight increase in muscle tone, manifested by a catch and release or minimal resistance at the end of range of motion when the affected part is moved in flexion and extension
1+: Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the range of motion
2: More marked increase in muscle tone throughout most of the ROM, but the part is still easily moved
3: Considerable increase in muscle tone, passive movement difficult
4: Affected part rigid in flexion or extension

Other Measures

Objective Measures
- Spasm frequency, pain
- Active range of motion
- Rapid alternating movements
- Gait (2 minutes or 10 meters)
- Electrophysiologic/biomechanical
- Pediatric-specific measures

**Functional Measures**
- **Performance** at the clinic (Frenchay, nine-hole peg test, etc)
- **Subjective report** by patient or caregiver
  - Global disability
  - ADL/hygiene
  - Patient/caregiver
- QOL

**Management of Spasticity**

**Management Goals**
No meaningful plan can be formulated without first determining the goals of treatment, including the following:
- Patient/caregiver goals
- Functional goals
- Technical goals

**Rationale for Treatment**
- Muscle overactivity interferes with
  - Functioning
  - Positioning
  - Comfort
  - Care
- Overactivity in a muscle where it is not useful (e.g., knee extensors during transfers)
- Treatment is expected to provide meaningful improvement
- Technical Questions in the Diagnostic Exam
- Active function
  - How are overactivity and contracture impairing active movements?
  - Can performance of other muscles improve if free of opposing co-contraction?
- Passive function
- Are overactivity and/or contracture preventing full stretch and joint range of motion?
  - Which muscles are contributing to the pathologic posture?
- Technical Questions: Treatment
  - Generalized vs. focal spasticity
  - Response to previous treatments
  - Severity and progression of complications caused by spasticity
  - The needs and goals of other practitioners
- Muscle Identification
  - Clinical exam: Careful inspection and palpation are sufficient in most cases
    - Knowledge of functional anatomy is critical
  - Dynamic EMG (+/- needle)
- Overactivity, co-contraction
  - Motor point block
    - Lidocaine, etidocaine, bupivacaine
  - Gait laboratory analysis +/- needle EMG

- Considerations in Treatment Decisions

- Comorbidities
  - Degree of underlying selective control
  - Cognitive or psychiatric impairment, depression, degree of patient motivation
  - Degree of contracture

- Availability of care and support

- Reimbursement issues

- Potential for complications

- Spasticity Treatment General Guidelines

- Start with patient and/or caregiver goals
- Never forget: Not all Spasticity is Bad
- Set realistic goals
- Always try to think one move ahead
- Remember: Target (goal) dictates weapon (treatment), weapon dictates movement (action)

- The Interdisciplinary Team
  - Physiatrist
  - Neurologist
  - Orthopedist
  - Physical therapist
  - Occupational therapist
  - Speech therapist
  - Physician assistant
  - Nurses
  - Neurosurgeon
  - Social worker
- Pharmacist
- Billing specialist
- Possible Treatment Goals
  - Passive function and comfort
    - Increased ROM
    - Improved positioning
    - Increased ease of hygiene
    - Improved cosmesis
    - Decreased spasm frequency
    - Improved orthotic fit
    - Decreased pain
  - Active function
    - Improved upper limb use: reaching, grasping, releasing
    - Improved mobility
    - Improved gait
    - Decreased energy expenditure
- Management of Patients With Spasticity
↓ noxious stimuli

- Physical and occupational therapies, home exercise program (stretch and motor training+++)

- Other therapies
  - **LOCAL interventions**: botulinum toxin, alcohol, phenol, surgery
  - **SYSTEMIC interventions**: oral or intrathecal administration of drugs

- Occupational and Physical Therapy General Guidelines:

  - Individual assessment and professional judgment are the basis for all therapy
  - Interdisciplinary (transdisciplinary?) treatment models work best
  - Communication among team members is critical for best outcomes
- All guidelines courtesy of Kay Beegan LPT, Jon Beegan LPT, Kurt Jackson DPT, and many others I have shamelessly ripped off
- Slightly less general therapy guidelines

Treatment should always be meaningful to the patient, and require their active involvement.

The best therapists are imaginative, flexible and creative, especially in home health settings.

As much as possible, all treatments should be applicable for long-term use in the patient’s environment, inexpensive, and easily taught to the patient and/or caregivers.
- Sorta specific guidelines
- Splint overnight for range of motion, and through the day for function
- A simple orthotic the patient will wear is better than the ideal orthotic she won’t
- Closed chain stretching is much better than open chain stretching
- Range of motion exercises start with comfortable positioning, then joint mobs, then stretching and gross range of motion

- Who does this guy think he is?
- Range of motion once a day for low tone muscles, twice a day for high tone
- Scapular motion is key for the shoulder
- Unclenching spastic finger and thumb flexors is easier if the forearm is supinated
- RV wheel chucks make inexpensive and effective wedges for patients to stretch the gastrocnemius/soleus complex with standing
- Muscle Strengthening does NOT increase spasticity

- Local Treatments:
  - Chemodenervation
  - Injectable therapy
  - Local muscle weakening
    - Reversible
    - Temporary
    - Titratable to patient needs
Agents
- Botulinum toxin
- Alcohol
- Phenol

Botulinum Toxin
- Product of *Clostridium botulinum* bacteria
- Seven serotypes: A – G
  - A and B approved for clinical use

BTX-A
- Botox® (Allergan)
- Dysport® (Ipsen, Ltd.)

BTX-B (Elan)
- Myobloc™ (USA)
- NeuroBloc® (Europe)

Botulinum Toxin: Clinical Effects in Spasticity
- Direct injection in affected muscle(s)
- Therapeutic effect usually within 24–72 hours (BTX-A)
- Peak effect at 1–2 weeks (BTX-A)
- Duration of clinical benefits: 12–20 weeks (BTX-A and BTX-B)
- May be used in combination with
  - Alcohol/phenol
  - Systemic treatments (oral or intrathecal)

**Botulinum Toxin: Safety**

- Dose-dependent, localized weakness
  - Potentially a problem in distal upper-limb injections: unintended weakness in nontargeted muscles when diffusion/injection beyond injected area

- Contraindicated during pregnancy, lactation, or neuromuscular disease
- No reported anaphylactic reactions
Current Treatment Options: Nerve Blocks
Nerve blocks may reduce focal or regional muscle overactivity by decreasing afferent and efferent nerve impulses.
- Peripheral nerves may be blocked at a distance from or very close to (even in) the muscle they innervate.
- Blocks may be transient for diagnostic or other short-term purposes.
- Blocks can produce long-term effects through nerve destruction.

Alcohol/Phenol: Histologic Effects
- Local anesthetic effect (used in some throat lozenges)
- Denatures protein; tissue necrosis; destroys all size axons, especially on nerve’s outer aspect
- 98% alcohol/5% phenol in water solution produces axonal destruction
- Duration of effect related to length of denervated segment
- Regeneration (regrowth of axons) occurs with fibrosis after variable period of time

- Alcohol/Phenol: Clinical Effects
- Almost immediate effects
  - Monophasic, constant denervation
  - ↓ of stretch reflex greater than ↓ strength
- Duration of effect: variable
  - Alcohol
    - 6–12 months (Tardieu et al, 45%)
    - 2–3 years in limited patients
  - Phenol
    - Range 10–850 days, average 10–11 months
    - 10–11 months (Khalili et al, 2%–3%)
    - 9–22 months (Petrillo et al, 5%)
    - 1–36 months (Easton et al, 5%)
- Alcohol/Phenol: Adverse Effects
  - Excessive Weakness
  - Neuropathic pain
  - Skin and muscle induration/scarring
- Phenol/BTX Combination Therapy
  - Phenol
    - Large, proximal muscles
  - BTX
    - Small, distal muscles
    - Selective targeting
- Local Treatments: Surgical Modalities
  - Selective dorsal rhizotomy (SDR)
  - DREZotomy (dorsal root entry zone)
  - Neurotomy
- Orthopedic procedures
  - Musculotendinous lengthening
  - Joint fusion surgery for stability
  - Bone surgery for rotational abnormalities
  - Tendon transfers
- Selective Dorsal Rhizotomy
  - Sectioning of afferent nerve rootlets
    - L2 to S2
  - EMG guidance
    - Selected roots only
- Indications
  - Pure spasticity with minimal/no athetosis
  - Good strength, motor and trunk control
- Contraindications
  - Trunk, lower-extremity, unilateral, or antigravity muscle weakness
  - Hypotonia
- DREZotomy
- Microsurgical Dorsal Root Entry Zone lesion
- Ventrolateral aspect of DREZ
- Rootlets in dorsolateral sulcus

- **Ablative surgery**
  - Destroys nociceptive (lateral) and myotatic (central) fibers
  - Spares part of lemniscal (medial) fibers

- Indicated following failure of conservative options
  - Conditions: spasticity, neurogenic and cancer pain

- **Orthopedic Procedures**
  - ↑ function by
    - Adjusting length of muscles by **lengthening** the appropriate musculotendinous unit
    - Distributing mechanical power by **tendon transfers**
    - ↑ gait by performing **rotational osteotomies**
    - ↑ stability with the use of selective **joint fusions**
Orthopedic Procedures: Tendon Transfers

Goals
- Weaken the overactive muscle
- Modify muscle’s primary function

Common techniques
- Split anterior tibialis transfer (SPLATT)
- Pronator to supinator transfer
- Tenodesis grasp
- Flexor carpi ulnaris transfer to extensor carpi radialis brevis or longus

Bone Surgery for Rotational Abnormalities

Lever-arm dysfunction

Surgeries designed to improve these problems include the following:
- Varus derotational osteotomies of the femur
- Tibial and fibular osteotomies
- Calcaneal osteotomies for severe pes valgus (flat foot)

- **Postoperative Management After Musculoskeletal Surgery**
  - Quick mobilization
  - Immediate postoperative motion/exercise
  - PT/OT 2\textsuperscript{nd} or 3\textsuperscript{rd} day postprocedure
  - Early gait training
  - Short braces
    - Fixed
    - Articulated
    - Dynamic

- **Medication Adverse Effects**
- All medications used for spasticity management may produce increased
weakness and sedation, especially at high doses.

- **Current Treatment Options:**
  - **Systemic Drugs—Baclofen**
  - GABA agonist
  - Mechanism: altered release of excitatory neurotransmitters and substance P in the segmental spinal cord
  - Depresses monosynaptic and polysynaptic transmission

- **Baclofen**
  - **Adverse Effects**
  - Sedation
  - Weakness
  - Hypotonia
  - Confusion, ataxia
  - Fatigue, dizziness
  - ↓ seizure threshold
  - Withdrawal seizures
- Memory impairment
- Depression, tolerance

**Clinical Effects**

- ↓ resistance of PROM*
- ↓ hyperreflexia
- ↓ painful spasms
- ↓ anxiety

Warning: administer with caution in patients with renal failure

- **Dantrolene Sodium**

  **Clinical Effects**

  - ↓ resistance to PROM
  - ↓ hyperreflexia
  - ↓ muscle tone
  - ↓ spasms
  - ↓ clonus

  **Adverse Effects**

  - Weakness (including ventilatory muscles)
  - Drowsiness
- Lethargy
- Nausea, diarrhea
- Paresthesia

**Current Treatment Options:**
**Oral/Systemic Agents—**
- Tizanidine

- An $\alpha_2$-adrenergic agonist
- Mechanism: reduces excitatory amino acid release and substance P

**Tizanidine**

**Clinical Effects**

- ↓ tone
- ↓ spasm frequency
- ↓ hyperreflexia
- Antinociceptive effect

**Adverse Effects**
- Drowsiness
- Sedation, depression, lethargy
- Weakness
- Dizziness
- Dry mouth
- Orthostatic hypotension

**Systemic Treatments:**
**Intrathecal Drug Delivery**
(baclofen, clonidine)
- Implantable, programmable pump
- Has been used in management of severely affected patients, whatever the etiology

**Intrathecal Treatments:**
**Requirements**
- No skin or systemic infection present
- Abdominal wall must be intact
• Previous sites of abdominal surgical procedures must be far from site of pump implantation
• Restriction of cerebrospinal fluid flow must be ruled out
• Responsible patient or caregiver
• Potential Benefits of Intrathecal Treatment
  • Provides relief when oral regimen or neurolysis fails
  • Electronic pumps may allow precise titration adjusted to patient’s needs
  • Reduces ↑↑ tone
  • Reduces sleep disturbances caused by uncontrolled spasms
- Spasticity Management: Case Studies

- The Interdisciplinary Team
- Physiatrist
- Neurologist
- Orthopedist
- Physical therapist
- Occupational therapist
- Speech therapist
- Physician assistant
- Nurses
- Neurosurgeon
- Social worker
- Pharmacist
- Billing specialist