Pathophysiology and Management of Spastic Hypertonia: Current Concepts

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Definition of Spasticity

"Spasticity is a 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."

Lance JW. *Spasticity: disordered motor control* 1980

Upper Motor Neuron Syndrome

Positive Symptoms

- Spasticity
- Clonus
- Flexor/extensor spasm
- Hyper-reflexia
- Dystonia
- Rigidity

Negative Symptoms

- Decreased dexterity
- Weakness
- Paralysis
- Fatigability
- Slowness of movement

Etiologies

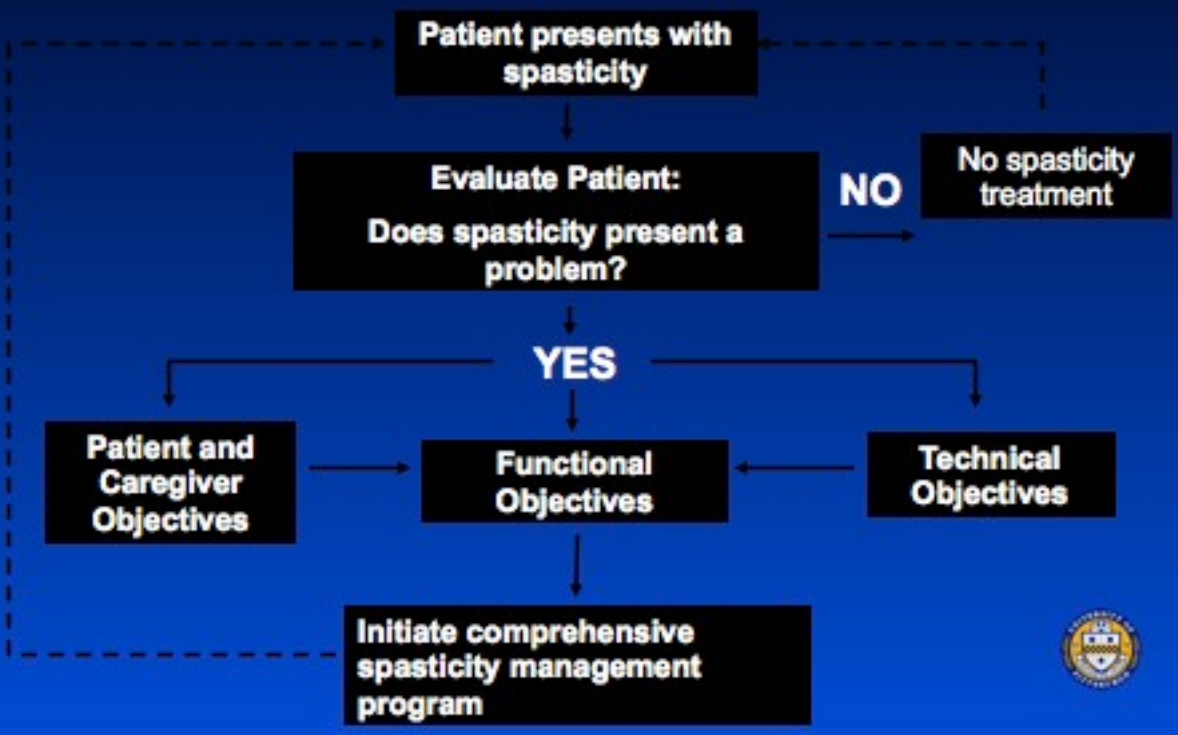
- Stroke
- Traumatic brain injury
- Multiple sclerosis
- Spinal cord injury
- Cerebral palsy
- Anoxia
- Neurodegenerative disease

Treatment Goals

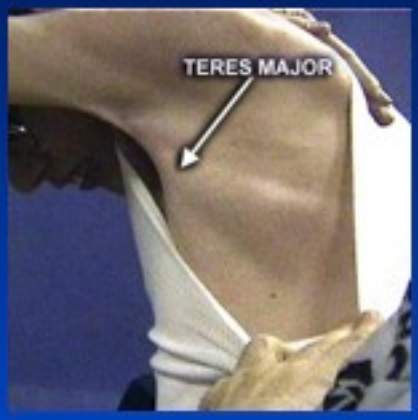
Improved	Decreased
ROM	Energy expenditure
Gait	Spasm frequency
Orthotic fit	Pain
Transfers	Caregiver burden
Seating	
Ease of hygiene	

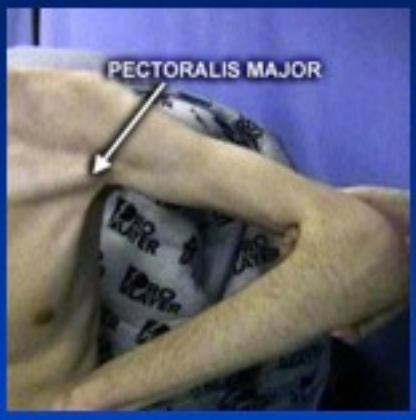
Algorithm for Spasticity

*Adapted from Brin, 1997



Upper Limb Spasticity Patterns





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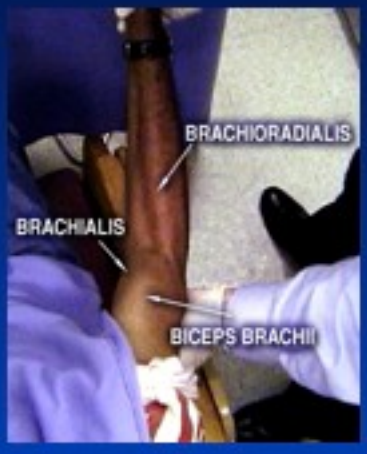

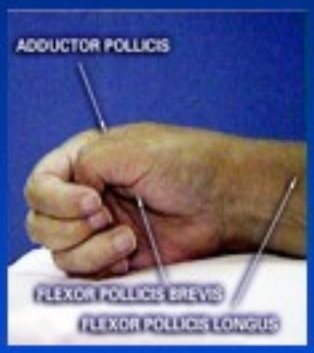

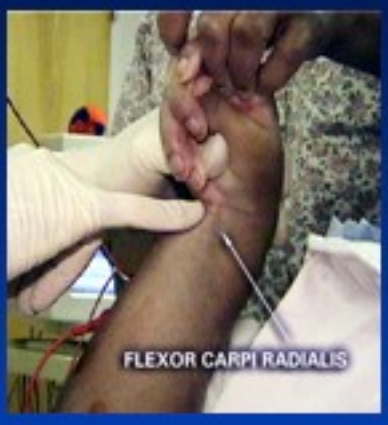

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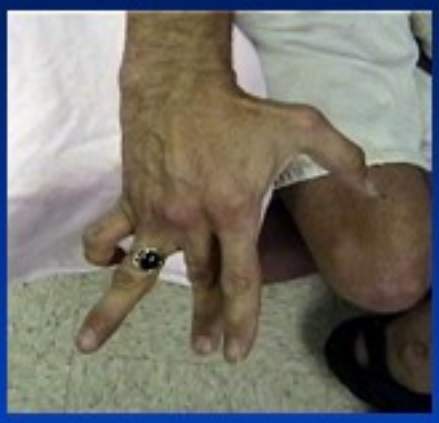
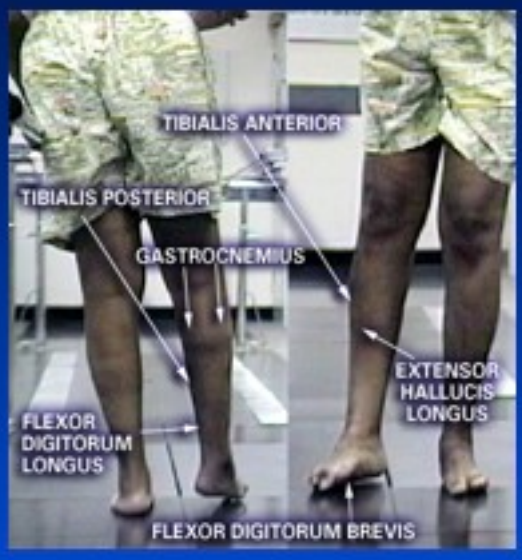
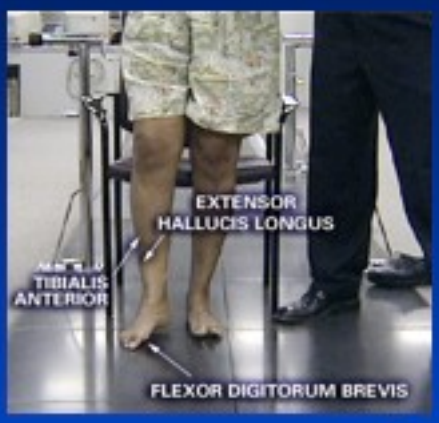


Lower Limb Spasticity Patterns





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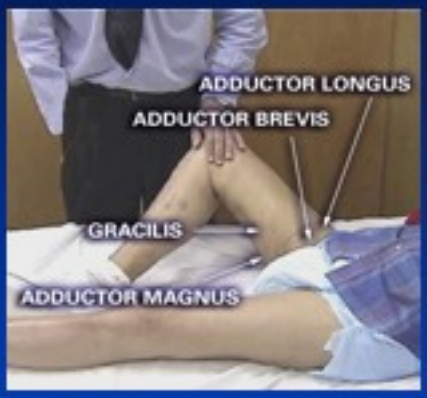
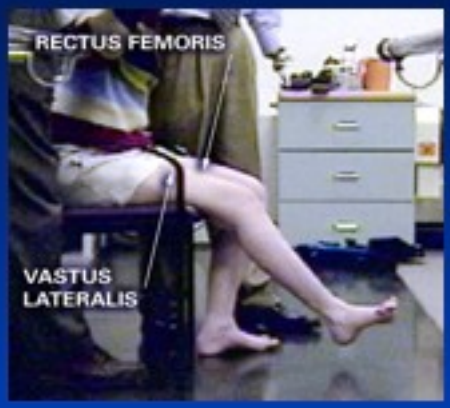
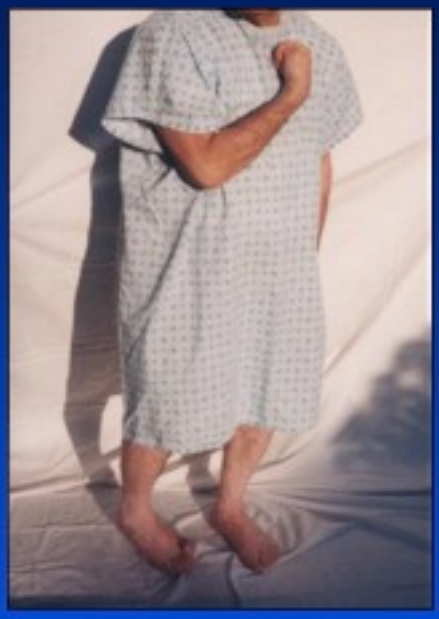
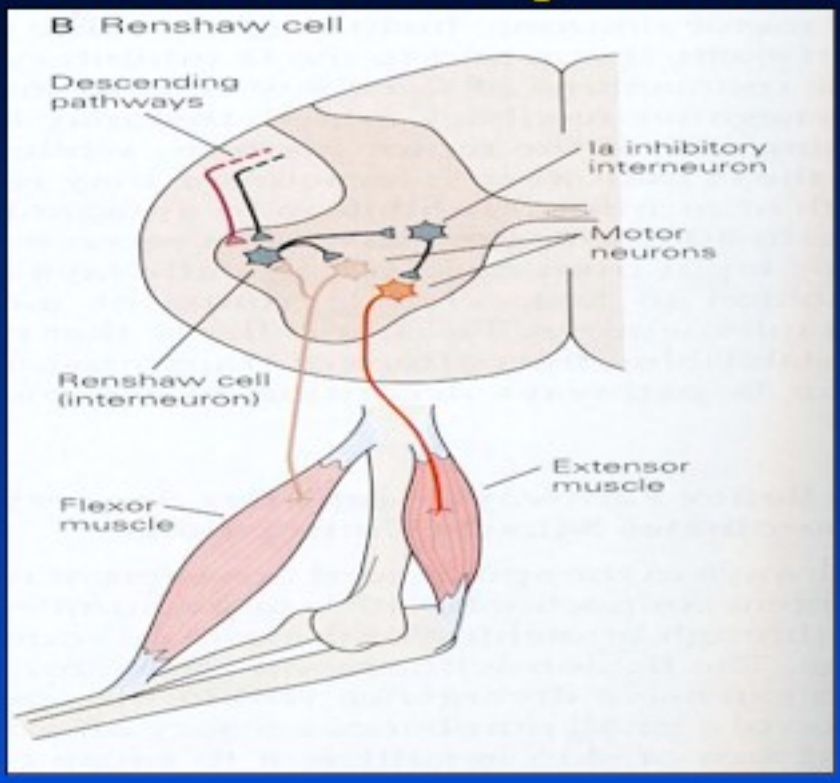




Review of Normal Function and Pathophyisology of Spastic Hypertonia

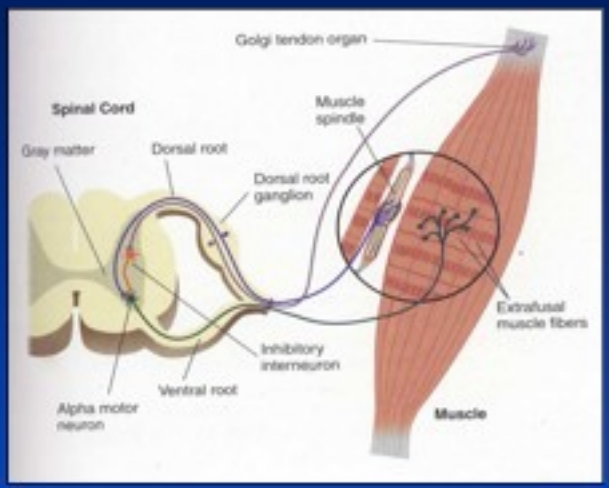


Loss of Descending Inhibition



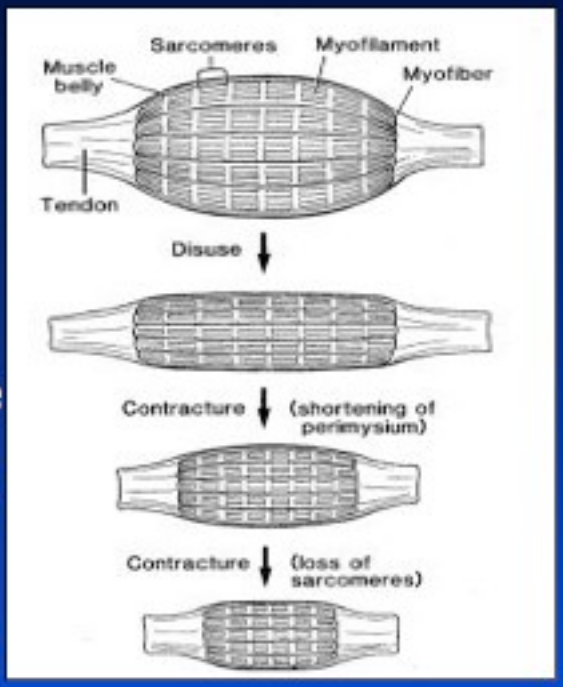
Alpha Motor Neuron Excitability

- Denervation Hypersensitivity
- Collateral Sprouting



Effects of Immobilization in Spasticity

Connective tissue deposition



Mechanisms of Impairment in Patients With Spasticity

Damage to Central Motor Pathways

Acute

Delayed

Paresis

CNS Plastic rearrangements
- *Supraspinal*
- *Spinal*

Immobilization in shortened position

Disuse

Muscle Overactivity

Soft tissue/Muscle rearrangements

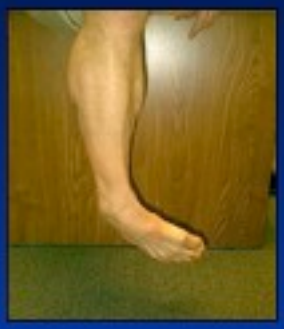
Spasticity
Spastic Dystonia
Spastic Co-Contraction

Contracture

Gracies. *Muscle & Nerve.* 2005;31:553.

Spastic Dystonia

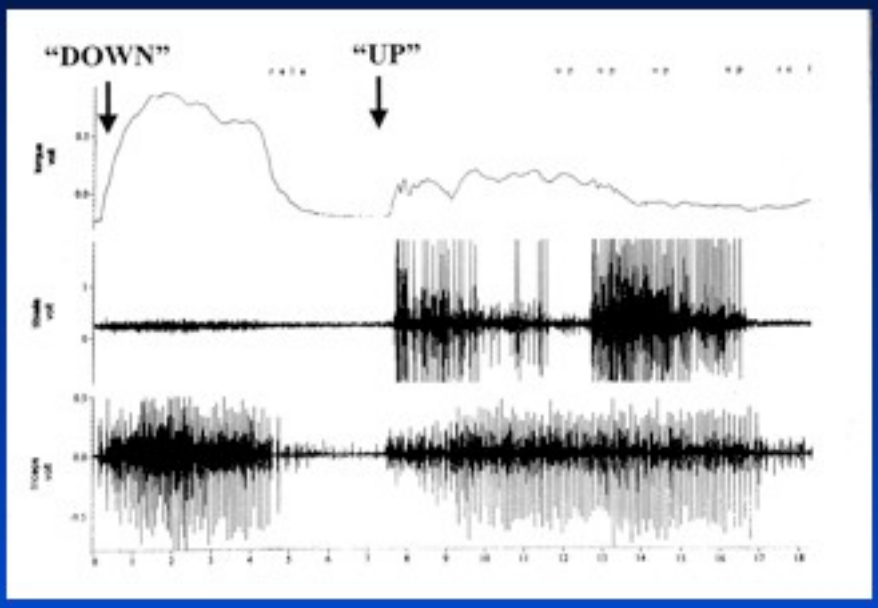




From Denny-Brown, 1966:

Tonic muscle contraction of both flexors and extensors in absence of phasic stretch or voluntary effort

Spastic Dystonia (Co-contraction)



Physical and Occupational

Modalities

- Cold (Ice), Biofeedback
- Acupuncture
- Vibration
- Electrical stimulation (NMES, FES, TES)
  - Efficacy not well documented
  - Used to:
    - Stimulate a weak agonist
    - Reduce spasticity in antagonist

Positioning Splints

- Upper and lower extremity
- Passive or dynamic
- Need to brace in appropriate positions
- ROM/stretching useful but hard to maintain with increased tone

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Modalities/Splinting in Spasticity

- Adjunct to pharmacological intervention, chemodenervation
- Not significantly helpful as monotherapy

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Spasticity: Pharmacological Treatment

Oral Medications

- Benzodiazepines
- Baclofen
- Dantrolene sodium
- Tizanidine
- Clonidine
- Cyproheptadine
- Gabapentin
- 4-AP

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Oral Medications

Benefits

1. Easy to administer
2. Global treatment
3. Decrease in hyperreflexia
4. Reduction in painful spasms

Side Effects

1. Sedation
2. Weakness
3. Confusion
4. Nausea
5. Dizziness/orthostasis
6. Lower seizure threshold

Chemodenervation in Spasticity

Chemodenervation

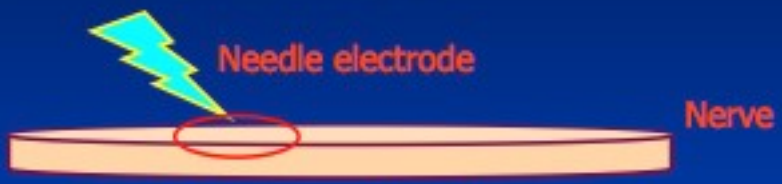
- Local muscle relaxation
- Injectable therapy
- Temporary, potentially reversible, titratable
  - Botulinum toxin
  - Phenol
  - Ethyl alcohol

Alcohol/Phenol: Histologic Effects

- Denatures protein; tissue necrosis; destroys all size axons especially on nerve outer aspect
- Low cost
- Lack of antigenicity

Alcohol/Phenol: Histologic Effects

- Duration related to area of denervated segment injected and dose



- Regeneration (re-growth of axons) occurs with fibrosis after variable period of time

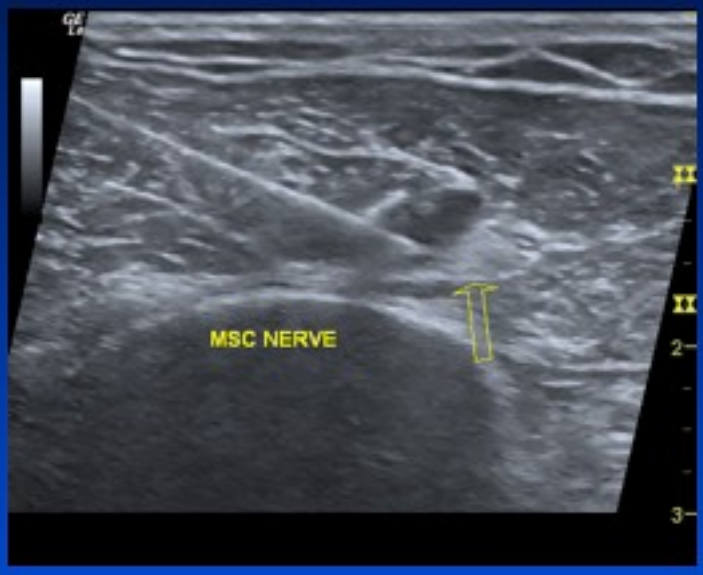
Alcohol/Phenol: Duration of

- 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%)

Common Neurolysis

- Obturator nerve
- Musculocutaneous nerve
- Motor point to Rectus Femoris
- Motor point to Hamstrings
- Motor point to Tibial nerve
- Femoral nerve

Phenol Nerve Block to Musculocutaneous Nerve



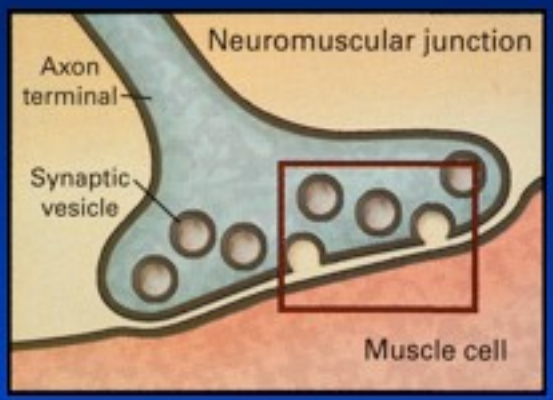
Botulinum Toxin

- Purified toxin from clostridia bacteria
- Many serotypes based on target receptor of toxin
- Approved for treatment of cervical dystonia (Dysport-Abo, Myobloc-Rima, Botox-Ona) and upper limb post stroke spasticity (Botox-Ona)

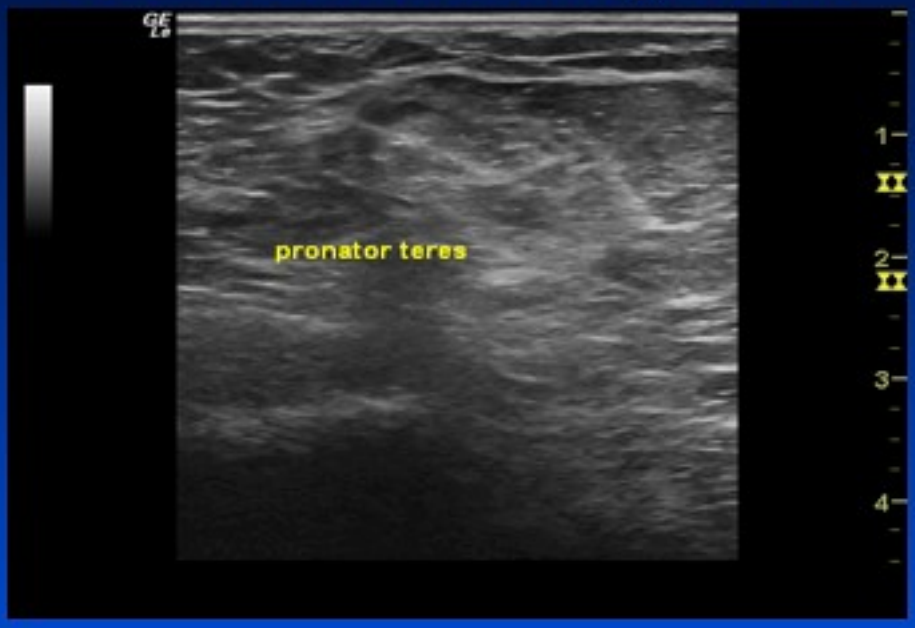
Botulinum Toxin Serotypes

Toxin	Target
Tetanus	Synaptobrevin
Botulinum Toxin A	SNAP-25
B	Synaptobrevin
C	Synaptobrevin
D	Synaptobrevin
E	SNAP-25
F	Synaptobrevin

Motor End Plate



Botulinum toxin injection



BTX-A: Mechanism of Action

- RE-ESTABLISHING
- Single nerve sprout establishes new neuromuscular junction. Muscle tone is restored and spasms return, making it necessary to repeat injections approximately every 3 to 6 months, depending on individual patient response.

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Clinical Effects of Botulinum Toxins

- Injected into overactive muscles
- Focal, temporary chemodenervation
- Onset usually within 24-72 hours
- Maximum effect at approximately 4 to 6weeks
- Clinical benefit usually >12 weeks
- Can be used with other therapies

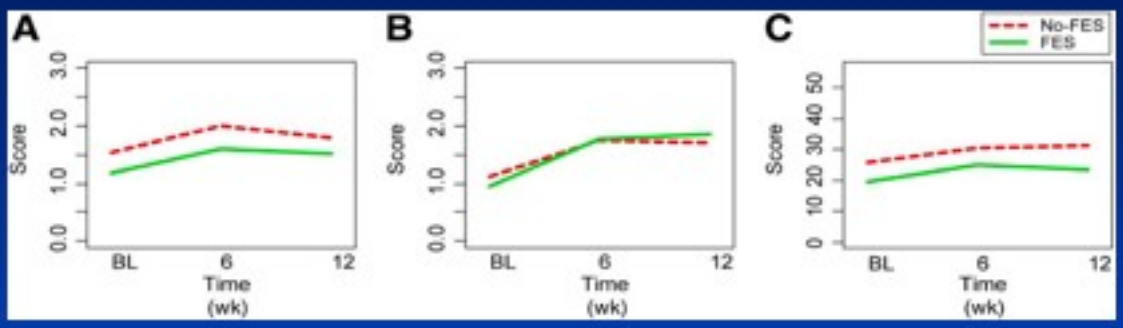
Phenol/Botulinum Toxin Combination Therapy

- **Phenol**
  - **Large, proximal muscles**
  - **Spares toxin dosing**
- **BTX**
  - **Small, distal muscles**
  - **Selective targeting**

Nonresponse to Botulinum Toxin

- Reasons for primary and secondary non-response
  - Dose may be too low
  - Muscles injected/technique may require modification
  - May be change in pattern of muscle involvement
  - Inappropriate reconstitution or storage of toxin
  - Neutralizing antibodies may be present

Cyclic Functional Electrical Stimulation Does Not Enhance Gains in Hand Grasp Function When Used as an Adjunct to OnabotulinumtoxinA and Task



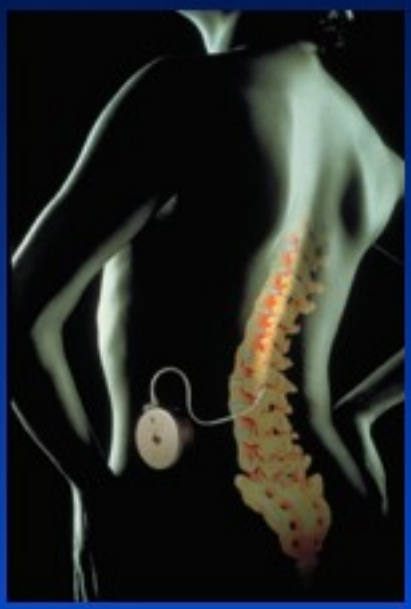
A) Motor Activity Log–Observation, (B) Motor Activity Log–Self-Report, (C) ARAT

Weber 2010 Arch Phys Med Rehabil

Orthopedic Procedures: Tendon Transfers

- **Goals**
  - Weaken the overactive muscle
  - Modify primary function
- **Common techniques**
  - Split anterior tibialis transfer (SPLATT)
  - Achilles tendon lengthening
  - FDL lengthening
  - Pronator to supinator transfer
  - Tenodesis grasp
  - Flexor carpi ulnaris transfer to extensor carpi radialis brevis or longus

Intrathecal Delivery of



Advantages of ITB Therapy

- Reversible
- Global spasticity management
- Programmable
  - allows dose titration to give optimal benefit
- Effective in reducing spasticity
  - trunk and lower > upper extremities
  - cerebral and spinal origin

Potential Risks of ITB Therapy

- Drug side effects
  - Most common
    - hypotonia, somnolence, nausea/vomiting, headaches, dizziness
- System and procedural complications
  - Infection risk 1%
- Overdose or withdrawal from lack of refill

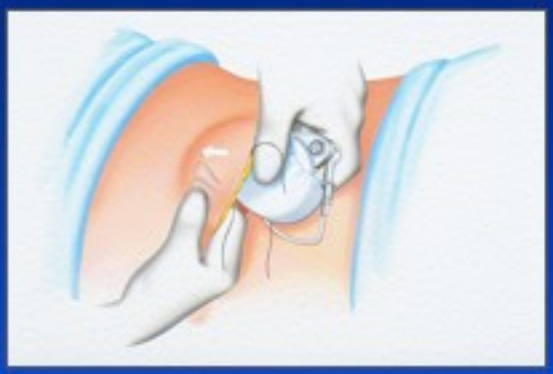
Catheter Implant

- Insert the introducer needle into the intraspinal space at the L3-L4 level
- Remove the needle stylet and confirm CSF backflow

Inserting catheter with guide wire through the introducer needle

Pump Implant

- Abdominal incision
  - make a pocket for the pump no deeper than 2.5 cm or 1 inch



Spasticity: Summary of Treatment Options

- Rehabilitation
- Oral medication
- Intrathecal baclofen
- Chemodenervation
- Orthopedic surgery
- Other neurosurgery

Multi-modal approach based on functional goals. Not all spasticity requires treatment