Spasticity in Traumatic Brain Injury: Overview, Evaluation, Medical management

Nicholas Ketchum, MD
Assistant Professor
Medical College of Wisconsin
Department of Physical Medicine and Rehabilitation

Objectives
At the conclusion of this program, the learner will be able to:

1) Define spasticity and its clinical features
2) Name methods to assess spasticity clinically
3) Discuss advantages and disadvantages of spasticity
4) Assess a patient’s goals of treatment of spasticity
5) Identify various treatment options for problematic spasticity

Spasticity: Overview, Evaluation, Medical management

- Components of Upper Motor Neuron Syndrome
- Measures of spasticity
- Medical treatments as part of a comprehensive management program for spasticity

Upper Motor Neuron Syndrome
- Lesion of the upper motor neuron in the central nervous system
  - Brain
  - Spinal cord
- Overactivity or “Positive Signs”
- Underactivity or “Negative Signs”
- “Aggregate of positive and negative signs after an upper motor neuron lesion comprises the Upper Motor Neuron Syndrome”

Upper Motor Neuron Syndrome

- Positive Symptoms
  - Spasticity
  - Spastic co-contraction
  - Spastic dystonia
  - Flex/Ext synergistic muscle patterns
  - Reflex release phenomena
- Negative Symptoms
  - Weakness, fatigue
  - Loss of dexterity, balance
  - Loss of selective muscle control
- Rheologic/Soft Tissue Changes
  - Contracture, fibrosis, atrophy
  - Contribute most to disability
  - Most resistant to treatment
  - Must take care to prevent as much and as early as possible

Acute Care Unit

Rehabilitation

Damage to Central Motor Pathways

Paralysis

Immediate

Delayed

Plastic rearrangements

• Spinal reactivity

• Supraspinal command

Flaccidity

Immobilization in shortened position

Spasticity

Spastic Dystonia

Spastic Co-contraction

Other

Mechanisms of Impairment in Patients With Spasticity

Spasticity

- Velocity-dependent increase in tonic stretch reflex (muscle tone)
- Hyperexcitability of the stretch reflex
- One component of the upper motor neuron syndrome

(Mayer 2002)

Mechanisms of Spasticity

- Increased motoneuronal excitability
- Enhanced excitatory input
- Reduced inhibitory input
- Intrinsic neuronal changes
- Enhanced stretch evoked excitation
- Gamma hyperexcitability
- Excitatory interneurons more sensitive

(Mayer 2002)

Etiologies of Spasticity

- Cerebral Origin
  - Traumatic brain injury
  - Anoxic brain injury
  - Cerebral palsy
  - Stroke

- Spinal Origin
  - Spinal cord injury
  - Spinal cord lesion
  - Multiple sclerosis

Pathophysiology of Cerebral Origin Spasticity

Normal
- Normal brain delivers inhibitory neural signals to the spinal cord
- Inhibitory signals modulate reflex signals—tone remains normal

Damaged
- Damaged brain fails to generate or sends inadequate inhibitory signals
- Lack of neural inhibition leads to spasticity

Spastic Co-contraction

Estimated Prevalence of Spasticity and Problematic Spasticity in the United States

<table>
<thead>
<tr>
<th>Condition</th>
<th>Est. Patients</th>
<th>Prevalence Spasticity</th>
<th>Prevalence Problematic Spasticity</th>
</tr>
</thead>
<tbody>
<tr>
<td>TBI</td>
<td>5,300,000</td>
<td>1,325,000 (25%)</td>
<td>437,000 (33%)</td>
</tr>
<tr>
<td>Stroke</td>
<td>5,500,000</td>
<td>1,495,000 (27%)</td>
<td>448,500 (30%)</td>
</tr>
<tr>
<td>CP</td>
<td>764,000</td>
<td>649,400 (85%)</td>
<td>382,000 (50%)</td>
</tr>
<tr>
<td>MS</td>
<td>400,000</td>
<td>268,000 (67%)</td>
<td>152,000 (38%)</td>
</tr>
<tr>
<td>SCI</td>
<td>259,000</td>
<td>172,040 (68%)</td>
<td>83,490 (33%)</td>
</tr>
</tbody>
</table>

Other Types of Movement Disorders

- **Dystonia:** Abnormal twisting, or repetitive movements
- **Chorea:** Involuntary and irregular dance-like movements
- **Athetosis:** Writhing, distal movements
- **Choreoathetosis:** Combination of both chorea and athetosis
- **Ataxia:** Flailing movements, wide-based gait

**How do we measure it?**

**Quantification of Spasticity**

- **Clinical**
  - Spasm frequency scale
  - Ashworth Scale
  - Tardieu method of assessment
- **Biomechanical**
  - Electrophysiologic – H-reflexes
  - Pendulum test
Spasm frequency scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No spasms</td>
</tr>
<tr>
<td>1</td>
<td>Spasms induced only by stimulation</td>
</tr>
<tr>
<td>2</td>
<td>Spasms occurring less than once per hour</td>
</tr>
<tr>
<td>3</td>
<td>Spasms occurring more than once per hour</td>
</tr>
<tr>
<td>4</td>
<td>Spasms occurring more than 10 times per hour</td>
</tr>
</tbody>
</table>

Ashworth Scale

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No increase in muscle tone</td>
</tr>
<tr>
<td>1</td>
<td>Slight increase in muscle tone giving a catch and release</td>
</tr>
<tr>
<td>2</td>
<td>More marked increase in tone but limb easily flexed</td>
</tr>
<tr>
<td>3</td>
<td>Considerable increase in tone – passive movement difficult</td>
</tr>
<tr>
<td>4</td>
<td>Limb rigid in flexion or extension</td>
</tr>
</tbody>
</table>

Tardeiu Scale

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No resistance throughout range of motion</td>
</tr>
<tr>
<td>1</td>
<td>Slight resistance throughout range of motion with no clear catch at a precise angle</td>
</tr>
<tr>
<td>2</td>
<td>Clear catch at a precise angle, followed by release</td>
</tr>
<tr>
<td>3</td>
<td>Fatigable clonus (&lt;10s) appearing at a precise angle</td>
</tr>
<tr>
<td>4</td>
<td>Non-fatigable clonus (&gt;10s) appearing at a precise angle</td>
</tr>
</tbody>
</table>

Revised Ashworth-Tardieu Scale (RATS)

- Move the isolated limb with a quick stretch (V3, >60 degrees/second) to assess and document the first catch (R1)
- Grade the degree of resistance to passive stretch:
  - 0 – no resistance
  - 1 – catch followed by release
  - 2 – mild increase in resistance
  - 3 – moderate increase in resistance
  - 4 – severe increase in resistance
- Document clonus during the exam: None, * = fatigable clonus (<10s), or ** = non-fatigable clonus (>10s)
- Slowly stretch the limb (V1) to assess and document end range (R2)
Example of the RATS

- R1 (First catch) = 90° moving the limb into extension produces a catch, noted at 90°
  - 0 degrees is full elbow extension/anatomical position
- Grade of resistance (Tone)/Clonus: 3=Moderate increase in tone, Clonus: * = Fatigable clonus
- R2 (End Range of motion) = 20° in this case the elbow lacks 20° of extension
- Recorded as:

<table>
<thead>
<tr>
<th>Muscle Group</th>
<th>R1</th>
<th>Tone/Clonus</th>
<th>R2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elbow flexors</td>
<td>90</td>
<td>3*</td>
<td>20</td>
</tr>
</tbody>
</table>

Quantification of Motor Control

- Clinical
  - Fugl-Meyer
  - UE test (Rancho, Wolf)
  - Timed walk, Get up & Go
- Biomechanical
  - Dynamic EMG
  - Motion Analysis Systems

So what do we see clinically?
Common upper limb patterns seen in upper motor neuron syndrome

- Adducted/internal rotated shoulder
- Flexed elbow
- Pronated forearm
- Flexed wrist
- Clenched Fist

Common lower limb patterns seen in upper motor neuron syndrome

- Flexed Hip
- Adducted Hip
- Flexed Knee
- Excessive plantarflexion
- Pronated Ankle

Adducted Internally Rotated Shoulder

- Pectoralis major
- Latissimus dorsi
- Teres major
- Subscapularis

Flexed Elbow

- Brachioradialis
- Biceps
- Brachialis
Pronated Forearm

- Pronator teres
- Pronator quadratus

Flexed Wrist

- Flexor carpi radialis
- Flexor carpi ulnaris
- Flexor digitorum profundus
- Flexor digitorum sublimis

Clenched Fist

- Flexor digitorum profundus
- Flexor digitorum sublimis

Thumb-in Palm Deformity

- Adductor pollicis
- Flexor pollicis longus
- Flexor pollicis brevis
**Flexed Hip**
- Iliopsoas
- Rectus femoris
- Add long/brev
- Pectineus
- Gracilis
- Sartorius
- Tensor fasciae latae
- Gluteus med/min

**Adducted Hip**
- Add long/brev/mag
- Pectineus
- Gracilis
- Iliopsoas
- Med hamstrings
- Gluteus maximus

**Flexed Knee**
- Hamstrings
- Gracilis
- Sartorius
- Gastrocs

**Equinovarus Foot**
- Gatrocs/Soleus
- Tib post/ant
- Flexor dig long/brev
- Flexor hallucis long
Striatal Toe

- Ext hallucis longus

Toe Curling

- Flexor dig long/brev
- Flexor hall long/brev

Why do we care?

Symptoms/Signs of Spasticity

<table>
<thead>
<tr>
<th>Altered Mobility</th>
<th>PAIN</th>
<th>Bowel/Bladder Dysfunction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatigue &amp; Depression</td>
<td>Sleep Disturbance</td>
<td>Decreased Productivity</td>
</tr>
<tr>
<td>Social Isolation</td>
<td>Cognitive or Emotional Problems</td>
<td>Sexual Dysfunction</td>
</tr>
</tbody>
</table>
Disadvantages of Spasticity

- Interfere with ADL’s
- Painful spasms
- Impede ambulation
- Lead to contractures or dislocations
- Abnormal bone growth
- Skin breakdown
- Impair respiratory function
- Mask volitional movement

Factors That May Increase Spasticity

Uncontrollable
- Urinary tract infection
- Kidney stones
- Menses
- Bowel impaction or gas
- Deep vein thrombosis
- Pneumonia
- Wounds or infections
- Progression of disease

Controllable
- Stress
- Ingrown nails
- Restrictive clothing
- Fatigue
- Psychological factors
- Change in temperature or humidity

Spasticity Benefits

- Stability in sitting or standing
- Assists with transfers
- Prevention of edema
- Prevention of DVT
- Awareness of noxious stimuli
- Improves cough strength
- Improves venous return

How do we treat it?
Goal: To Relieve Symptoms or Reduce Impairments

- Pain reduction
- Reducing sleep disturbances
- Reducing disfigurement and improving body image
- Reducing flexor and extensor muscle spasms
- Delay or prevention of contracture/surgery
- Prevention of subluxation
- Pressure sore reduction
- Reduction of post-surgical muscle spasm
- Increased tolerance for orthotics/shoes/splints
- Reduce abnormal bone growth in children

Goal: To Improve Active Function

- Mobility (transfers, improved gait pattern)
- Improved balance
- Energy demand reduction
- Wheelchair management and mobility
- ADL’s: LE dressing, hygiene, bathing, toileting

Goal: To Improve Passive Function

- ADL’s: LE dressing, hygiene, bathing
- Toileting and perineal care
- Wheelchair and bed postioning
- Transfers
- Application of splints, orthoses, and shoewear
- Promotion of physical and occupational therapy programs

Interdisciplinary Team

- Patient and family
- Physicians
  - physiatrist
  - neurologist
  - neurosurgeon
  - orthopedic surgeon
- Rehabilitation Nurse
- Nurse Practitioner
- Social Worker
- Physical therapist
- Occupational therapist
- Speech/Language Pathologist
- Dietician
- Psychologist
Comprehensive Spasticity Management

- Rehabilitation treatments
- Reduce nociceptive input
- Focal/Segmental Treatments:
  - Nerve/Motor point blocks
  - Tendon transfer/lengthening
- Generalized Treatments:
  - Oral/Intrathecal medications
  - Rhizotomy

Rehabilitation Treatments

- Physical/occupational/speech therapists are essential for successful spasticity management
- Assist with patient evaluation, education and setting goals
- Stretch shortened muscle/tendons
- Strengthen weak muscles
- Improve motor control

Rehabilitation Treatments

- Inhibitory/serial casting
- Dynamic splinting
- Weight bearing
- Neurofacilatory techniques
- Electrical stimulation (FES, NMES)
- Aquatic therapy
- EMG biofeedback
- Constraint induced movement therapy
- Robotic training
- Partial weight support treadmill training

Oral Antispasmodics
Oral Antispasmodics

Most common:
- Baclofen (Lioresal®)
- Diazepam (Valium®)
- Dantrolene sodium (Dantrium®)
- Tizanidine (Zanaflex®)

Pharmacology of Oral Antispasmodics - Baclofen

- **Baclofen**: Analog of GABA
- Binds to GABA\_B receptor
  - Presynaptically to Ia afferent sensory neurons
  - Postsynaptically to motor neurons
- Inhibits glutamate and aspartate release by blocking Ca influx
- Inhibits gamma motor neuron activity
- Reduces muscle spindle sensitivity
- As a result, inhibits mono- and poly-synaptic reflexes

Pharmacology of Oral antispasmodics - Baclofen
- Peak within 2hr
- Half-life 2.5–4hr
- Excreted unchanged in the kidneys
- Starting dose 5-10mg BID-TID
- Recommended max 80-100mg/day, see up to 300mg/day in practice
- First choice in spinal origin spasticity
- Less well-established in cerebral origin spasticity
- May be more effective in LE>UE (receptor density)


Pharmacology of Oral antispasmodics - Diazepam
- **Diazepam** (Valium®): Promotes release of GABA from GABA-A neurons
- does not bind directly to GABA receptor
  - Binds to benzodiazepine binding site
  - Results in increased presynaptic inhibition at spinal and supraspinal levels
  - Reduction of mono- and poly-synaptic reflexes at the spinal level

Pharmacology of Oral antispasmodics - diazepam

- Peak within 1–2hr
- Half-life 20–60hr
- Excreted unchanged in the kidneys
- Starting dose 2mg BID
- Recommended max 40–60mg/day
- First line in spinal origin spasticity
- Due to sedative effects, caution used in early stages of recovery if cerebral origin


Pharmacology of Oral antispasmodics - Diazepam

- Side effects
  - Sedation
  - Dizziness
  - Weakness
  - Ataxia
  - Decreased attention and memory
  - Avoid abrupt withdrawal


Pharmacology of Oral antispasmodics - dantrolene

- Dantrolene (Dantrium®): Acts peripherally on skeletal muscle
- Decreases calcium release from sarcoplasmic reticulum
- Uncouples electrical excitation from muscle contraction
- Diminishes force of muscle contraction
- Also affects intrafusal and extrafusal fibers
  - Muscle spindle sensitivity is reduced


Fig. 1: Mechanism of action of main pharmacological anti-spastic treatments.

Pharmacology of Oral antispasmodics - dantrolene

- Peak within 3-6hr
- Half-life 4-8hr
- Metabolized by the liver
  - Eliminated by the kidney
- Starting dose 25mg daily
- Recommended max 100mg 4x/day
- Preferred for cerebral origin spasticity
  - Controversial
  - May be less effective in MS


Pharmacology of Oral antispasmodics - tizanidine

- Tizanidine (Zanaflex®): Imidazoline derivative
- Alpha-2 adrenergic agonist
- Centrally acting
- Acts presynaptically at spinal level to inhibit excitatory amino acids (glutamate, aspartate)
- Inhibits polysynaptic reflexes by inhibiting effectiveness of excitatory amino acids
- Supraspinally inhibits locus cereleus, which in turn inhibits descending cerebrospinal pathway
- No effect on monosynaptic reflexes

Pharmacology of Oral antispasmodics - Tizanidine

- Peak within 0.75-2hr
- Half-life 2-4hr
- Metabolized by the liver
  - Need to check LFTs
  - Eliminated by the kidney and in stool
- Starting dose 2mg BID
- Slow taper upwards
- Recommended max 32mg/day
- Can see paradoxical worsening of spasticity in higher doses


Side effects
- Dry mouth
- Dizziness
- Weakness
- Hepatotoxicity
  - Must check LFTs prior to starting and periodically during treatment
  - More selective than clonidine, fewer CV side effects


Pharmacology of Oral antispasmodics - Clonidine

- Clonidine: Imidazoline derivative
- Alpha-2 adrenergic agonist
- Decreases tonic facilitation
- Enhances alpha-2 mediated presynaptic inhibition
- Mixed clearance (50% liver)
- Half-life 5-19hr
- Starting Dose 0.05mg/BID
- Recommended Max Dose 0.4mg/day
- CV side effects
  - Lower BP
  - Rebound HTN


Additional oral agents

- Gabapentin
- Sinemet
- Cyproheptadine
- Valproic Acid
- Cannabinoids
- Vigabatrin
- Tiagabine

Focal Treatments

- Local Anesthetics:
  - Etidocaine (0.5-1.0%)
  - Bupivicane (0.25-0.75%)
- Chemodenervation:
  - Botulinum Toxin A and B
- Chemical Neurolysis:
  - Phenol (3-7%)
  - Ethyl alcohol (5-99%)
- Orthopedic Procedures

Botulinum Neurotoxin (BoNT)

- Intra-muscular injections can be used to reduce focal muscle overactivity
- Affects both intrafusal and extrafusal muscle
- May affect nociceptor pathways via C and A delta fibers and substance P
- Can be used to block salivary and sweat gland
- Used to treat numerous disorders: dystonia, spasticity, ophthalmologic, GI/GU, dermatologic and pain.

FDA Approved BoNT and Recommended Names

- Botox® - OnabotulinumtoxinA
- Dysport® - AbobotulinumtoxinA
- Xeomin® - IncobotulinumtoxinA
- Myobloc® - RimabotulinumtoxinB

BoNT Mechanism of Action

Local, Temporary
Cholinergic Chemodenervation
BoNT Injection Techniques

- Considerable variation among clinicians
- Optimal dose, volume, and dilution
- Injection technique
- Injection sites
- Timing

Endplate Zone Location

Deshpande, Gormley, Carey, Neurotox Res. 2006
Ultrasound Guided BoNT Injection

Optimal BoNT Dosage Factors:

- Treatment Goal
- Number of targeted muscles
- Size of targeted muscles
- Degree of muscle over-activity
- Magnitude of desired response
- Duration of desired response

Therapeutic Dose Window

- Too low
  - No clinical effect
- Too high
  - Target muscle too weak
  - Adjacent muscle weakness
  - No additional therapeutic weakness

“Treatment Failure”

- Inappropriate patient selection
- Inappropriate goal selection
- Wrong muscle targeted
- Poor muscle localization
- Insufficient dose
- Mishandling of toxin
- Poor follow-up
- Neutralizing antibody formation
BoNT Adverse Effects

- Excess weakness, nausea, fatigue, pain, bronchitis, dysphagia, dry mouth
- Vary with dose, injection site, product
- Antibody-mediated resistance < 1%
- Minimize risk of resistance by using lowest effective dose with at least 3 month between injections
- Not recommended in patients who are pregnant, lactating or have a NMJ disorder

Phenol and Alcohol Injections

- Phenol (benzyl alcohol) 3%–6%
- Ethyl alcohol 35%–60%
- Clinical use
  - Motor nerve block
  - Motor point block
- Neurolytic mechanism
  - Denatures protein; tissue necrosis
- Regrowth of axons after variable period of time

Phenol/Alcohol: Clinical Effects

- Almost immediate effects
- Duration of effect (variable)
  - 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
    - 6–12 months (Tardieu et al, 45%)
    - 2–3 years in limited patients

Phenol/Alcohol: Clinical Conditions

- Adducted shoulder
  - Pect major
- Flexed elbow
  - Musculocutaneous n.
- Adducted hip
  - Obturator n.
- Flexed knee
  - Hamstrings
Phenol/Alcohol: Adverse Effects

- Injection-site pain
- Localized swelling
- Paresthesia
- Excessive motor weakness
- Deep venous thrombosis
- Dysesthesia
- Systemic effects
  - Tremor, convulsions
  - CNS depression
  - CV collapse

Temporary Block with Local Anesthetics

- Often done on inpatient services
  - Quick (minutes) onset of action
  - Can help differentiate spasticity from contracture
- Useful as an adjunct to therapy
  - Spasticity often a barrier to patient progress
  - Can help with ability to stand, fit of orthoses

Temporary Block with Local Anesthetics

- Useful to assess functional impact of decreasing spasticity of specific muscles
  - Also helps plan future treatment
- Can see longer lasting effects outside medication MOA

Temporary Block with Local Anesthetics

- Also done pre/postop amputation
  - Need <25° from full knee extension to accommodate BKA prosthesis
  - Need <20° flexion to accommodate AKA prosthesis
Intrathecal Delivery of Baclofen

Intrathecal Baclofen (ITB) Therapy

Used to treat individuals with severe spasticity due to:
- Spinal cord injury
- Multiple sclerosis
- Brain injury
- Stroke
- Cerebral Palsy

Benefits of ITB

- Minimizes effects of oral therapy
  - lethargy
  - drowsiness
  - confusion
- May eliminate need for other spasticity meds.
- Can be used in combination with oral or chemodenervation therapy.
- Can be programmed to patients own schedule.

Oral Baclofen vs. ITB

<table>
<thead>
<tr>
<th>Oral 60 mg/d</th>
<th>IT - Chronic 200 ug/d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baclofen ng/ml</td>
<td>Baclofen ng/ml</td>
</tr>
<tr>
<td>0</td>
<td>400</td>
</tr>
<tr>
<td>Plasma</td>
<td>Plasma (est)</td>
</tr>
<tr>
<td>100</td>
<td>300</td>
</tr>
<tr>
<td>CSF</td>
<td>CSF</td>
</tr>
<tr>
<td>200</td>
<td>200</td>
</tr>
<tr>
<td>300</td>
<td>300</td>
</tr>
<tr>
<td>400</td>
<td>400</td>
</tr>
</tbody>
</table>
Potential Downfalls

- Surgery is involved
- Battery life
- Potential pump failure
- Overmedication
- Withdrawal/undermedication
- Cost
- Frequency of follow-ups. Adherence to regime

SynchroMed® Infusion System Components

- **Pump**
  - infuses drug at programmed rate

- **Catheter**
  - delivers drug to the intrathecal (subarachnoid) space of the spinal cord

- **Programmer**
  - allows for precise dosing
  - easily adjustable dosing

ITB Pump is the size of a hockey puck

Pump placement

- ITB pumps are placed SQ into the abdomen.
- A catheter is tunneled into the intrathecal space
- Placement is from L3/4 to T 6
- May be as high as cervical region
- Off label Intraventricular
What can go wrong?

- <1-2% Infection
- 2% skin erosion
- 6-7% Mechanical: catheter migration, breakage, kinking
- 5-10% CSF Leak
- 15% Seroma
- Battery replacement ~6-7 years
- Overdose/Withdrawl or Side Effects of Baclofen

Potential catheter disruptions

- Break
- Kink or Hole
- Pump Disconnect
- Large to Small Catheter Disconnect

Signs and Symptoms to Watch For

- **OVERDOSE**
  - Drowsiness
  - Dizziness
  - Seizures
  - Respiratory depression
  - Decreased LOC
  - Flaccid tone

- **WITHDRAWL**
  - Increased spasticity
  - Irritation
  - Tachycardia
  - Fever of unknown origin
  - Severe puritis

Remember factors that may increase spasticity

- Pain
- Infection
- Constipation
- Immobility
- Incisions
- Quick titration of oral antispasmotic agents
- Anxiety
Potential Complications
- Human error overdose
- Catheter Migration
- Catheter Crack
- Disconnect
- Missed refill withdrawal
- Device Failure
- Seroma
- Infection
- Dehiscence

Baclofen Overdose
- Progressive decrease in tone to flaccid
- Confusion
- Hypotension
- Bradycardia
- Hypoventilation to apnea
- Coma

Baclofen Withdrawal Physi ology
- Loss of GABA mediated inhibition
  - Hypermetabolic
  - Spasticity/rigidity
  - Central effect,
    - Seizures
    - Hallucinations

Baclofen Withdrawal Early Symptoms
- Itching
- Irritation/Dysphoria
- Rebound spasticity
  - Rigidity
  - Tachycardia
  - Fever
  - BP changes
Medicating patient in withdrawal

- PM&R Pump team available 24/7
- Restart oral Baclofen at pre-surgery dose
- Benzo’s: activate central receptors and restore neuronal inhibition to endpoint of muscle relaxation, normothermia and stabilization of BP
- Cyproheptadine: Serotonin antagonist and antihistamine
- Dantrium: reduce depolarization-induced calcium release from sarcoplasmic reticulum. NOT GABA-ERGIC (decreases risk hyperthermia and Rhabdo)

Other considerations

- Pump patient may be admitted for revision of catheter or end of battery life replacement of device.
- Not everything that is wrong with the patient is a result of the Baclofen pump,
- HOWEVER:
  - Pump adjustments may improve other issues
  - Improve cough strength
  - Decrease pulling against braces/casts
  - Improve positioning

In the news...

Medtronic in FDA Consent Decree Over Its SynchroMed Infusion Pump

Medtronic agrees to stop widespread distribution of the product.

Federal authorities reached a proposed consent agreement with medical device maker Medtronic Inc. over flaws in its SynchroMed drug infusion pump for cancer and pain medicine, a device that has been linked to serious injuries and deaths in recent years.

The Justice Department, working with the Food and Drug Administration, filed the consent decree along with a legal complaint alleging that Medtronic and two executives—Chairman and CEO

In the news...

Under the proposed decree, Medtronic agreed to stop manufacturing and distributing new versions of the SynchroMed II implantable pump except in extraordinary cases, such as when a treating physician certifies that the pump is medically necessary. The device also dispenses medicine to alleviate severe chronic spasticity.
Medtronic may provide SynchroMed drug infusion pumps as follows:

- **Replacement Patients:**
  - The agreement permits Medtronic to provide a replacement SynchroMed pump because existing pump patients may be adversely affected by suddenly discontinuing their use of the pump.

- **New Patients:**
  - The agreement permits Medtronic to provide a SynchroMed pump for a new patient when, in the professional judgment of a physician, the pump is medically necessary to treat one or more of the following medical conditions, and the benefits of such treatment outweigh the risks:
    - severe spasticity
    - chronic intractable pain
    - severe chronic pain
    - primary or metastatic cancer

**Summary**

- Spasticity is only one component of the UMN syndrome
- Spasticity is more than hyperexcitability of stretch reflex
- Spasticity is prevalent in society
- Impacts patients daily functioning
- Not all spasticity is problematic
- Assessment should be done of both active and passive condition
- Multiple treatment strategies exist
- Providers have the tools to help patients with this disabling symptom of injury/disease
- Comprehensive spasticity management and realistic goals are essential to optimizing functional outcomes

**Selected references**

Thank you!