"Open Arms"

Medical Management of the Stroke Rehab Patient.

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Disclosures

- I have no financial/non-financial relationships to disclose.
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Objectives

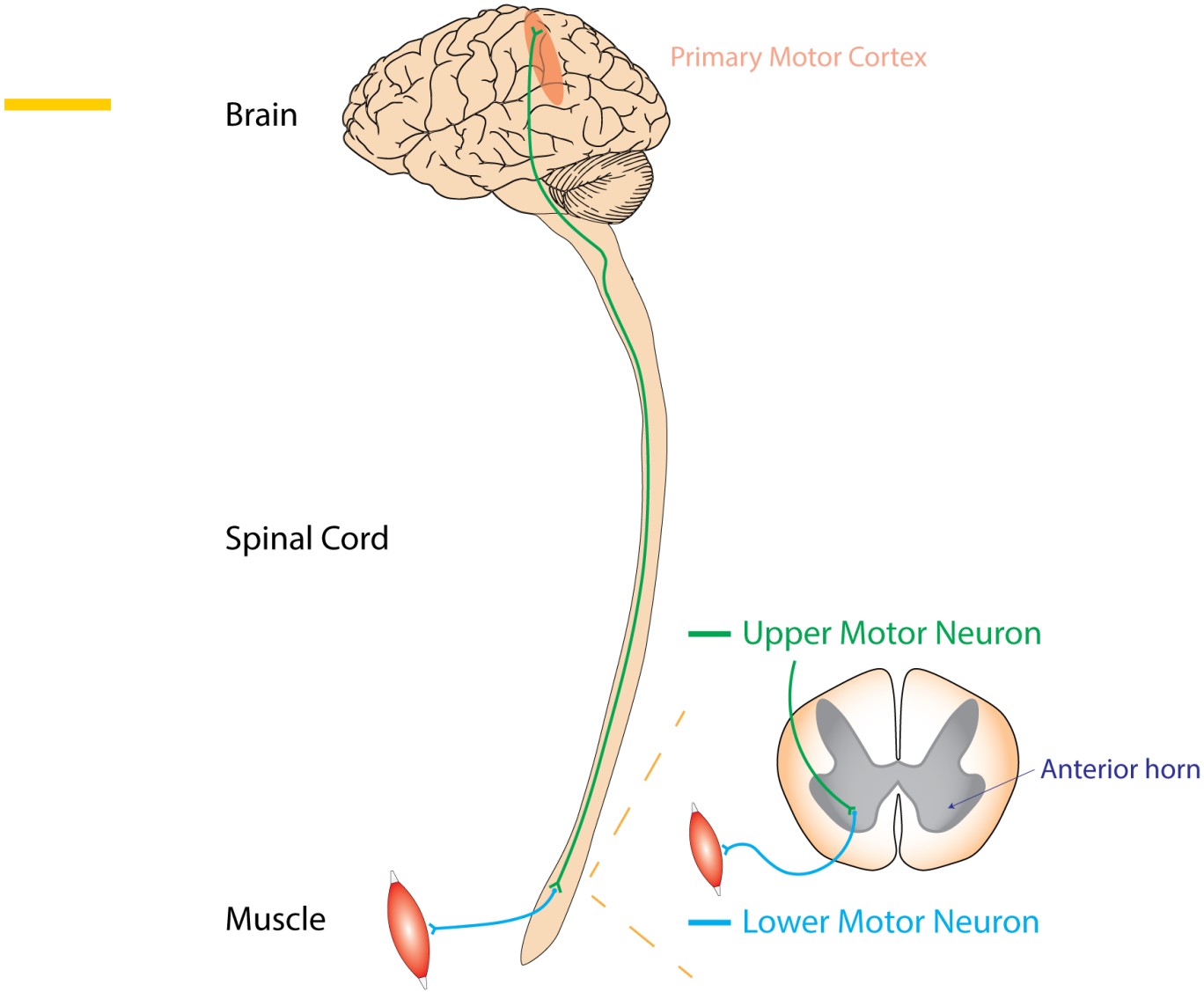
- Describe the methods used to increase mobility and independence of the post-acute stroke patient
- Define spasticity
- Describe how spasticity is assessed
- Describe medical management to help w/ spasticity during acute inpatient rehabilitation hospitalization

What is spasticity?

- A motor disorder characterized by an abnormal, **velocity-dependent increase in tonic stretch reflexes (muscle tone)** with exaggerated phasic stretch reflexes (tendon jerks, clonus) resulting from hyperexcitability of the stretch reflex.

What is spasticity?

- Due to an upper motor neuron dysfunction
- Lesions proximal to the alpha motor neuron (spinal cord, brain) resulting in **loss of descending inhibition** and hypersensitivity of the reflex arc in the spinal cord.


Brain
Primary Motor Cortex
Spinal Cord
Upper Motor Neuron
Anterior horn
Muscle
Lower Motor Neuron

Upper Motor Neuron Signs

Positive signs:

- Spasticity
- Hyperreflexia
- Clonus
- Rigidity
- Primitive reflexes reappear (+ babinski response)

Negative signs:

- Weakness
- Paralysis/paresis
- Atrophy
- Loss of voluntary movement/coordination

Modified Ashworth Scale (MAS)

0 - No increase in tone (i.e. normal)

1 - Slight increase in muscle tone, w/ catch and release or minimal resistance at the end ROM

1+ - Slight increase in muscle tone w/ catch followed by minimal resistance through the reminder (but less than half) ROM

2 - Marked increase in muscle tone through most of the ROM, but **extremity still easily moved**

3 - Considerable increase in muscle tone, **passive movement difficult**

4 - Affected part rigid in flexion or extension

Deep Tendon Reflex Grading

- 0 - Absent
- 1 - Diminished but present; minimal
- 2 - Normal
- 3 - Brisk and excessive
- 4 - Very brisk, often with rhythmic reflex contractions (clonus)

Brunnstrom's post-stroke recovery scale

1. Flaccidity
2. Spasticity appears
   - Basic synergy patterns appear (commonly flexion in the upper extremities and extension in lower extremities)
3. Patient gains voluntary control over synergies
   - Increase in spasticity
4. Some movement patterns out of synergy are mastered
5. If progress continues, more complex movement combinations are learned as the basic synergies lose their dominance
6. Disappearance of spasticity
7. Normal function is restored

Pharmacotherapy for Spasticity

- 4 oral drugs FDA approved
  - Baclofen
  - Dantrolene
  - Diazepam
  - Tizanidine

Baclofen

- Mechanism: GABA agonist at **$GABA_B$** receptors (B for Baclofen)
  - Inhibits Gamma Motor Neuron activity and decreases muscle spindle sensitivity to spinal reflexes
- Side effects: **Sedation/drowsiness**, patient can develop tolerance, **lowers seizure threshold**, weakness, GI upset
- Precautions: Sudden withdrawal can lead to seizures, hallucinations, and rebound spasticity w/ fever
- **Renally cleared** (only one of the FDA approved meds)
- Dosing: start w/ 5mg BID or TID and increase by 5mg/day up to 80mg/day (FDA's recommended max dose)
  - Caveat...some patients can still get benefit from >80mg/day dosing (i.e. 40mg TID, etc.), beware of side effects...

Dantrolene

- Mechanism: Acts peripherally in the striated muscle by blocking Ca^{2+} release from the sarcoplasmic reticulum
- Side effects: Liver toxicity (~1%), drowsiness/sedation (usually more mild), weakness, fatigue, GI upset
- **Hepatic clearance** - monitor LFTs
- Classically the preferred option for spasticity w/ cerebral origin (CVA, TBI)
- Used to treat malignant hyperthermia, neuroleptic malignant syndrome and fever from Baclofen withdrawal
- Dosing: Start at 25mg BID, max dose of 400mg/day (between 2-3 doses)

Diazepam

- Mechanism: facilitates GABA's effects at the **$GABA_A$ receptor**
- Side effects: **Sedation (very high)**, memory impairment, decreased REM sleep
- Precautions: not your 1st choice w/ TBI given memory impairment, CNS depression w/ EtOH use
- OD on diazepam?? Give them some flumazenil
- **Hepatically cleared**
- Dosing: 2mg BID or 4mg QHS, Max dose 60mg/day

Tizanidine

- Mechanism: **Central acting α-2 adrenergic agonist** that is thought to enhance presynaptic inhibitory modulation of spinal reflexes
- Side effects: Sedation/drowsiness (up to 50%), liver damage, hypotension (less than clonidine), dry mouth, bradycardia, dizziness
- **Hepatically cleared** - monitor LFTs
- Dosing: Start at 2-4mg/day (usually QHS to start), Max dose: 36mg/day

Things to consider upon discharge

- PM&R follow-up
  - Is it available?
  - Is it necessary?
  - Are there certain items that need to be communicated to the outpatient provider so that they don't get missed?
    - Current functional status
    - Current medications for spasticity and possible changes to consider
    - Chemodenervation?

Chemodenervation

- Unfortunately, it is unlikely to be covered as an inpatient
- Good reason to ensure patient has PM&R outpatient follow-up if felt to be beneficial

Chemodenervation

- 7 serotypes (A-G), *Clostridium botulinum* bacteria
- 4 w/ FDA approval
- Mechanism: blocks presynaptic release of acetylcholine at the neuromuscular junction
- Rule of 3's
  - Onset ~3 days
  - Peak effect ~3 weeks
  - Duration ~ 3 months

References

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Questions?

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Thank you

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