Spasticity

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Spasticity

- 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

- J.W. Lance, 1980
Upper Motor Neuron Syndrome

• Characterized by:
  – Spasticity
  – spontaneous spasms
  – Weakness
  – loss of discrete distal movements
  – abolished abdominal reflexes
  – extensor plantar responses
Upper Motor Neuron Syndrome

- Develops after lesions to the cortex, corona radiata and internal capsule
- Other descending pathways are involved
- Does not develop following isolated lesions of the motor cortex or pyramidal tract
Spasticity

• Manifestation of UMN paralysis

• Additional Features
  • Positive: spasms
  • Negative: weakness loss of dexterity
Spastic Conditions

- Cerebral Palsy
- Stroke
- Multiple sclerosis
- Brain injury
- Spinal cord injury
- Anoxia
- Neurodegeneration
- **SPASTICITY**
  - Resistance to passive movement Bidirectional
  - Velocity dependent
  - Increased DTR's
  - Clasp-knife
  - Clonus

- **RIGIDITY**
  - Resistance to passive movement unidirectional (flexor or ext)
  - Velocity independent
  - Normal DTRs
  - Lead pipe
  - No clonus
Reflexes
Muscle Spindles

built around 3-13 small intrafusal muscle fibers
- attached to glycocalyx of the large extrafusal muscle fibers
- central portion of each fiber serves as sensory receptor
- motor innervation by gamma motoneurons
- sensory innervation of central portion via:
  - Ia fibers (annulospiral ending around nuclear bag and nuclear chain fibers)
  - II fibers (from nuclear chain fibers)
- detects changes (and rate of change) in muscle LENGTH

- two types of sensory responses:
  i. static response (nuclear chain fibers via II fibers)
     - active for as long the receptor remains stretched

  ii. dynamic response (nuclear bag fibers via Ia fibers)
     - response to a sudden increase in spindle length
     - only active while length is actually increasing
     - more powerful than static response
Golgi Tendon Organs

- encapsulated sensory receptor through which a few muscle tendon fibers pass
- sensory innervaton by Ib nerve fibers
- detects changes in muscle TENSION
**Alpha Motoneurons**
- give rise to large A-alpha nerve fibers
- excites several skeletal muscle fibers - a motor unit

**Gamma Motoneurons**
- give rise to smaller A-gamma fibers
- excite intrafusal muscle fibers

**Renshaw Cells**
- a type of interneuron located in ventral horn of spinal cord
- axons of motor neurons send collaterals to Renshaw cells
- these then inhibit other nearby motor neurons: *recurrent inhibition*
- helps to “sharpen” the signal
The Muscle Stretch Reflex

- stretching of the muscle causes stretching of the muscle spindle
- Ia afferents synpase directly on alpha motoneuron
- stimulates muscle contraction
- a monosynaptic reflex

- coincident with this is *reciprocal innervation*
- Ia afferents stimulates an inhibitory interneuron
- interneuron inhibits alpha motoneuron of the antagonist muscle
The Flexor Reflex

- afferents via II fibers and IV fibers (touch, pressure, joint receptors, nociceptors)
- activate flexor motoneurons, inhibit extensor motoneurons
- facilitated by corticospinal and rubrospinal pathways

- flexor responses can be released by loss of corticospinal/rubrospinal input
- clinical correlates:
  - positive Babinski response
  - exaggerated leg flexor and adductor response
Mechanisms

- exact mechanism of Spasticity in humans is not clear, possible factors include:

1. **Alpha Motoneuron Hyperexcitability**
   - imbalance in supraspinal excitatory vs. inhibitory inputs to the alpha MN

2. **Fusimotor Hyperactivity**
   - hyperactivity of gamma MN can lead to increase sensitivity of muscle spindle to stretch
   - likely not enough to solely explain the clinical picture

3. **Decrease in Presynaptic Inhibition of Ia terminals**
   - interneurons mediate presynaptic inhibition of the Ia terminals on the alpha MN
   - these interneurons are controlled by descending tracts
   - loss of this inhibition can produce a greater afferent stimulus to the alpha MN as a result of stretch
Mechanisms cont...

4. *Decreased Inhibition from Type II Afferents*
   - afferents for the static response also normally have an inhibitory effect via interneurons
   - interneurons are controlled by descending tracts

5. *Decreased Recurrent Inhibition from Renshaw Cells*
   - the inhibitory effect of the Renshaw cells are also subject to supraspinal control

6. *Decreased Reciprocal Ia Inhibition*
   - normal inhibition of antagonist muscle during muscle stretch may be lost
   - interneurons controlling reciprocal inhibition have supraspinal input
Cerebral Palsy

**Definition**
-a diagnosis
-represents a collection of clinical syndromes
-disorders are:
  - non-progressive
  - neurological injury is not an ongoing process
  - growth and development can cause tone and range of motion changes
  - these alter gait patterns and the ability to accomplish ADLs
  - results in functional losses or failure to progress
  - children can improve in areas of motor control and language skills
characterized by alterations in movements and posture, refers to a persistent abnormality of control of movement and posture
occurs as a result of injury to the immature brain
  - immaturity defined as 2-3 years of age and younger
  - some sources include children up to age 5-7
Epidemiology

- Incidence of CP: 1.5-3/1000 live births
- Low birth weight infants
  - 1500-2500g: 13-14/1000 live births
  - <1500g: 90-91/1000 live births
- Survival after 1 year: severe 60%, mild >90%
- Predictive factors:
  - Maternal preeclampsia
  - Infection (TORCH, GBS)
  - Late gestational bleeding
  - Multiple gestation
  - IUGR
  - Non-vertex presentation
  - Abruptio placentae
  - Fetal distress
Etiology

- cannot be attributed to a single causative factor or event
- up to 20% no identifiable cause
- caused by anything that results in injury to the developing brain:
  - migrational defects
  - infarction
  - intracranial hemorrhage
  - infection
  - chemical injury
  - hypoxia
- categorized by time frame and pathology
**Time of Insult**

1. during Gestation  
   stroke, TORCH infections (Toxo, other, rubella, CMV, herpes)
2. Perinatal Period  
   Hypoxia
3. Neonatally  
   GBS meningitis, IVH
4. Early Childhood  
   TBI secondary to abuse or accident  
   Infections: encephalitis, meningitis  
   Hypoxic-ischemic injury - near drowning, house fires  
   Status epilepticus

-90% Congenital CP - result from insults occurring antenatally, at birth, or in first post-natal month

-~16% Acquired CP - caused by damage after first month of life

*delivery related hypoxia is an infrequent cause
Classification

-six primary clinical syndromes constitute the diagnosis of CP:
  -pyramidal CP - 4 forms
  -extrapyramidal CP
  -Cerebellar or ataxic CP

-mixed clinical types
  spastic-athetoid
  -spastic-dystonic
  -spastic-rigid