Spasticity

Neurosurgery Residents' Seminar

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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

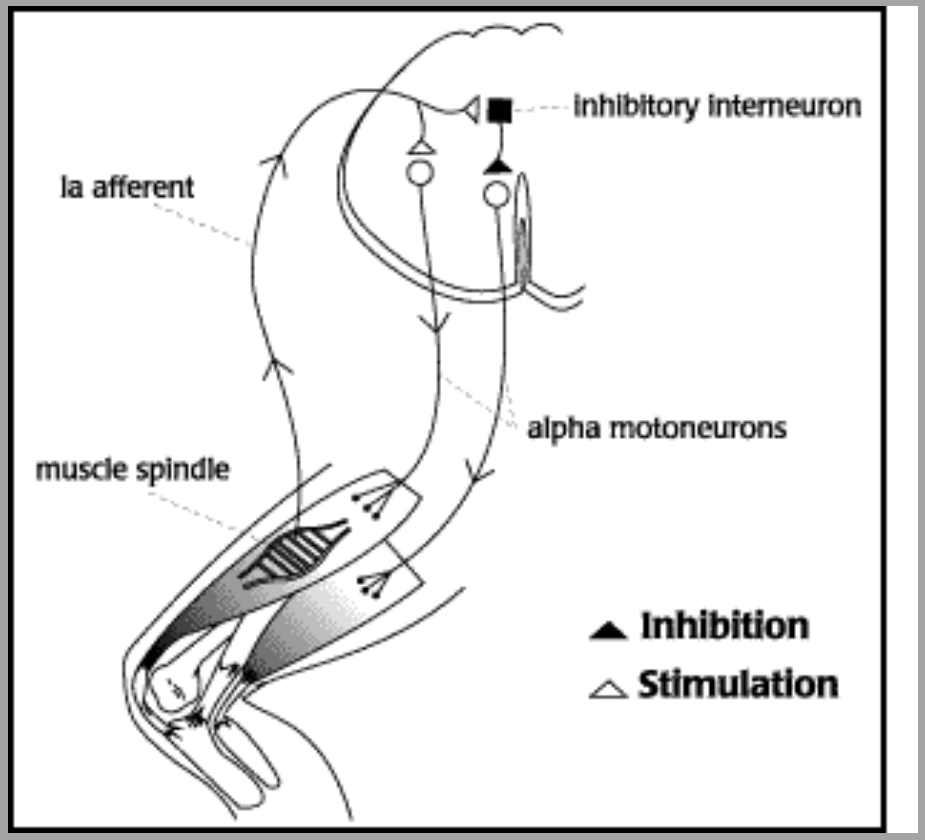
Clasp-knife

Clonus

- **RIGIDITY**

- Resistance to passive movement unidirectional (flexor or ext)
- Velocity independent
- Normal DTRs
- Lead pipe
- No clonus

Reflexes


inhibitory interneuron
Ia afferent
alpha motoneurons
muscle spindle
Inhibition
Stimulation

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

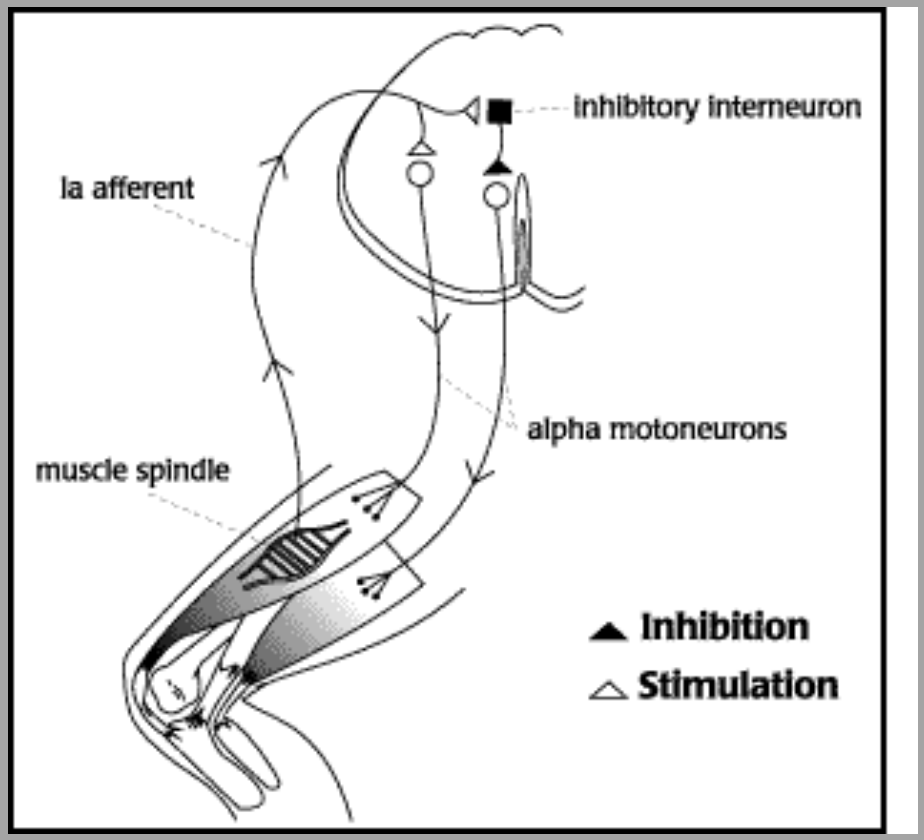
Renshaw Cells

- 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 anatagonist 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

-result is 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

occur 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)
- -lategestational 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