

MOTOR PHYSIOLOGY

I. PERIPHERAL MOTOR MECHANISMS

a. Organization

i. Basic circuit

1. Afferent receptors from muscle, tendon, skin
2. Efferents from alpha motor neuron in spinal cord
3. Can function segmentally, or due to higher level control centers

ii. **Motor unit** = motor neuron and all fibers it innervates (small unit = finer control)

iii. Types of muscle fiber

1. **Red muscle** (posture):

- a. small body and axon, small motor unit
- b. high metab rate (high blood supply), resistant to fatigue
- c. low tension, slow contraction rate
- d. tonic nerve activity (always firing APs)
- e. easily excitable

2. **White muscle** (fight or flight):

- a. large body and axon, large motor unit
- b. low metab rate (low blood supply), easily fatigue
- c. high tension, fast contraction rate
- d. phasic nerve activity (only when necessary)
- e. require greater excitatory input

3. Relationship of muscle fiber size to properties = **size principle**

4. In a heterogeneous muscle, small fibers active most of the time, larger fibers recruited only when maximal effort is demanded

b. Biochemical mechanisms

i. Mechanical event (“**twitch**”)

1. At transverse tubule, AP dives into sarcoplasmic reticulum
2. releases Ca^{++} ; interaction of myosin and actin → contraction

ii. **Temporal summation**- elicit twitch events in succession

iii. **Spatial summation** – simultaneous twitch in multiple fibers

1. More active motor units → steadier total tension of muscle

c. Receptors

i. Muscle spindles

1. Parallel to muscle fibers – attached to same tendons → can meas length
2. Consists of **intrafusar fibers**, arranged as a **nuclear bag** or **nuclear chain**
3. **IA afferent** – ends on nuclear bag/chain, cell body in DRG
 - a. Enters dorsal root of spinal cord → synapse on spinal interneurons → other parts of nervous system
 - b. OR → alpha motor neuron (monosynaptic contact) → back to same muscle

- c. **Stretch/myotatic reflex:** lengthen muscle → incr firing in IA fibers → synapse with alpha motor neurons → contraction
 - 4. **Gamma motor neuron** (efferent)– cell body in ventral horn of SC
 - a. Innervates polar contractile ends of muscle spindle
 - b. Can maintain tension in spindle, even as muscle shortens: contraction at polar ends → tension in middle → incr IA firing → stretch reflex
 - c. **Compensatory loading:** even as muscle shortens, spindle maintains sensitivity – keeps receptors in tune, allows IA fibers to support alpha MN discharge
 - 5. Pathways
 - a. **Direct pathway:** activate alpha motor neuron
 - b. **Indirect pathway:** activate gamma → IA → alpha MN
 - c. **Alpha-gamma coactivation:** dir and indir pathways at once
 - ii. Golgi tendon organs
 - 1. Embedded in tendons – in series with muscle fibers → measure tension
 - 2. Excite golgi tendon → **IB afferent** incr firing → inhibits alpha MN (relaxation) = **Golgi tendon/inverse myotatic reflex**

II. SPINAL ORGANIZATION AND REFLEXES

- a. Spinal processing
 - i. Divergence
 - 1. **Amplification:** one cortical neuron to many alpha MNs
 - 2. **Distribution:** sensory afferents → interneurons/cerebellum/motor cortex
 - ii. Convergence
 - 1. **Assured output** (ie many nociceptors on one alpha MN)
 - 2. **Redundant input** (ie vestibular neuron, visual and sensory cx all on alpha MN) – if one impaired, can still function – for very imp't functions
 - 3. By temporal or spatial summation
 - iii. Signal Modification
 - 1. Signal inversion: activate one muscle, inhibit another (ie flexor/extensor)
 - 2. Positive feedback: muscle can extend beyond stimulus
 - 3. Negative feedback: sensory afferent truncated relative to input
 - *Dale's Law – given neuron can only secrete one kind of NT (+ or -)
 - use interneurons to modify signal
- b. Modulation of output
 - i. Neurons in the same pool are fractionated = excited to different degrees:
 - 1. Neurons in **firing zone** – sufficient excitation to reach threshold
 - 2. Neurons in **subliminal fringe** – excited but don't reach threshold
 - ii. Facilitation
 - 1. Stimulation of two inputs with overlapping subliminal cells
 - 2. Response **larger** than algebraic sum of two

- iii. Occlusion
 1. Stimulation of two inputs with overlapping firing zones
 2. Response smaller than algebraic sum
- c. Spinal Reflexes
 - i. **Stretch/Myotatic reflex**
 1. Stretch → IA fibers → excite alpha MN of same muscle
 2. IA collaterals → inhibit antagonistic muscles
 3. Maintain position (in extensors – maintain posture)
 - ii. **Clasp Knife/Inverse Stretch reflex**
 1. Initial resistance to stretch; increase stretch → rigidity “melts away”
 2. Golgi tendon organ – autogenic inhibition of muscle, contraction of antagonists and contralateral muscle
 - iii. **Flexion reflex/Nociceptive reflex** (withdrawal in response to pain)
 1. Noxious stimulus → A-delta afferents → contract ipsilateral flexors, relax ipsilateral extensors
 2. **Crossed extension reflex:** contralateral: contract extensors, relax flexors (support body weight) = double reciprocal innervations
 - iv. Spinal stepping
 1. Proprioceptors sense pressure on foot → coordinate extension of limb, flexion of contralateral
 2. Single stimulation can initiate rhythmic alternate stepping

III. SPINAL CORD AND BRAINSTEM MECHANISMS

- a. Peripheral mechanisms
 - i. Afferents
 1. **Primary (IA):** dynamic – responds to steady level of stretch and rate of change of length
 2. **Secondary (group II):** respond only to amount of stretch
 - ii. Efferents
 1. **Fusimotor plate (Gamma I)** – makes afferents more responsive to rate of change (more dynamic)
 2. **Fusimotor tail (gamma II)** - makes more responsive to level of stretch (more static)
 - iii. Which gamma fiber is active determines whether muscle spindle responds to extent of stretch or rate of change → adaptable CNS
 - iv. If muscle spindle can tell rate, can predict stretching → eliminates tremor (oscillation caused by loop delay and muscle overcompensating for stretch)
- b. Brainstem mechanisms
 - i. Reticular formation
 1. **Facilitatory reticular formation:** large, rostral; activates alpha MN of extensors, inhibit flexors

2. **Inhibitory reticular formation:** small, caudal; activates flexion, inhibits extension
 3. Higher motor centers (via basal ganglia and cerebellum) - inhibit facilitatory RF, excite inhibitory RF
 4. Decerebrate rigidity –due to loss of these descending inputs
 5. Ascending sensory input and vestibular nuclei – excite facilitatory RF
- ii. Body orienting reflexes
1. Receptors: visual, somatic proprioception, labyrinth in inner ear
 2. (Review of pathways)
 3. Information processed in upper brainstem, lots of redundancy
 4. Allows us to keep gaze constant
- iii. [Brainstem transection – not in lecture]
1. Between brainst and spinal cord → **spinal animal:** loss of higher facilitatory influences, but can elicit spinal reflexes
 2. In brainstem → **bulbospinal animal:** rigid standing, but no balance (destruction of main vestibular nuclei)
 3. Between colliculi → **decerebrate animal:** also rigid, due to ReST and VST
 4. Above brainstem → **midbrain/mesencephalic animal:** animal can right body and head, walk, run, but cannot avoid obstacles
 5. Removal of cerebral cortex → **decorticate animal:** full righting reflexes except visual – can walk, run, climb, but no previously learned behavior]
- c. Motor Cortex
- i. Primary motor cortex, supplementary motor cortex have lowest threshold
 - ii. Somatotopic organization
 1. Primary motor: rostral-to-caudal → lateral-to-medial (head lat, legs med)
 2. Supplementary: rostral-to-caudal → rostral-to-caudal (back = deep)
 3. Homunculus...