SPINAL AND CRANIAL NERVE REFLEXES

I. DEFINITION AND OVERVIEW OF REFLEXES

A. **Definition of a reflex** = **stereotyped motor response to a specific sensory stimulus.**

B. **A reflex usually consists of sensory receptors, interneurons and motor neurons:**

1. **sensory receptor** - detects stimulus (termed: **afferent arm** of reflex arc)
2. **interneurons** - receive inputs from sensory receptors and synapse on motor neurons; effects on motor neurons can be excitatory or inhibitory; not present in monosynaptic reflexes.
3. **motor neurons** - (termed **efferent arm** of reflex arc) produce muscle contraction, motor response.

C. Reflexes are **valuable tools for clinical evaluation of nervous system function**. For reflex to occur, all elements must be functional and pathways must be intact. If reflexes are absent, a physician can diagnose where the pathway is interrupted; if reflexes are abnormal, can diagnose where function is compromised.

D. **Reflexes are evaluated according to**
1) **amount (size, magnitude) of motor response**
2) **latency (time to elicit motor response)**

Note: Changes in reflexes are clinical signs - In some disease processes, damage can enhance motor responses (hyper-reflexia = abnormally large reflex responses); responses can also occur with abnormal muscle contractions (ex. Clonus - rapid alternating contraction and relaxation following a single stimulus)

RATING STRETCH (DEEP TENDON) REFLEXES – Decreased = 0,1, Normal = 2, Increased = 3,4

Rating	Characteristics
0	Absent
1	Diminished
2	Normal
3	Brisk, Hyper-reflexic
4	Hyper-reflexic, Pathologic

E. **Some reflexes are protective and relatively constant**; ex. **Pupillary light reflex**

F. **Other reflexes are constant under the same controlled circumstances**; ex. **Stretch reflex (deep tendon reflex)** - **Reflexes can be modulated by the central nervous system**: GET PATIENT TO RELAX.

G. **Other 'reflexes' actually represent triggering of more complex behaviors by sensory signals (ex. 'stepping reflexes' in neonates, infants). Some behaviors (ex. walking) are produced by pattern generators (see below).** Pattern generators are groups of interneurons in the CNS that produce activities in motor neurons and generate rhythmic behaviors. Stepping 'reflexes' in infants may represent triggering of activity in the walking pattern generator.

II. THREE CLASSIC SPINAL REFLEXES - Each reflex has a specific sensory stimulus and motor response

Note: **Terminology** - In describing a reflex:

Homonymous muscle - the muscle that contains or is associated directly with the sense organ producing the reflex
Synergist muscle - muscle that produces a similar motor action (movement)
Antagonist muscle - muscle that produces the opposite motor action (movement)
Contralateral muscle - muscle of opposite limb (leg or arm).

A. **Stretch reflex** (also termed: Monosynaptic Stretch Reflex, Myotatic Reflex, Deep Tendon Reflex)

1. **Stimulus** - fast stretch of muscle; clinically, produced by a brief sharp tap to a muscle tendon. This produces a sudden small lengthening of muscle (not stimulation of tendon receptors).
2. **Sense organ excited** - **Muscle spindle Primary (Group Ia) afferents**; can also produce much weaker discharges of muscle spindle Secondary (Group II) afferents.
3. **Primary response** - **MONOSYNAPTIC** and polysynaptic activation of alpha motor neurons in **muscle that is stretched contracts rapidly**. Monosynaptic reflex is the fastest reflex known, with a central delay of about 1 msec at the synapse.
4. **Effects on synergist and antagonist muscles -** a. **Excite synergist muscles** - Activate muscles with similar action (ex. in arm - biceps spindle sensory neurons excite motor neurons to brachialis muscle). b. **Inhibit antagonist muscles (RECIPROCAL INHIBITION)** - Decrease activity in muscles with opposing action (ex. biceps spindle neurons produce inhibition of triceps motor neurons); these connections are polysynaptic.
5. **Muscle Tonus** - Because the reflex connection is monosynaptic, the ongoing activity in muscle spindles is important determining the level of activity of motor neurons to muscles at rest. Decreases in sensory activity can cause a decrease in muscle tonus (measured by resistance to slow stretch of the muscle). Increased sensory activity can increase muscle tonus.

Note: **Spasticity/Rigidity** – **Increased tonus** (rigidity) occurs after **Upper Motor Neuron lesions** (ex. cortical strokes) due, in part, to **loss of modulation of stretch reflexes** (see below).

6. **Reflexes must be modified during voluntary movements**. Reflexes can be altered by mechanisms of 1) **gamma motor neurons** (reset muscle spindles) 2) **descending inputs from brain** - pre-synaptic inhibition (decrease effectiveness of spindle sensory discharges) and 3) **modulation of motor neuron activities (excitability)**. Some of these changes are produced by activities in neurons of descending motor tracts. 4) **Renshaw cells** - Alpha motor neurons have recurrent processes (axon collaterals); some of these branches make excitatory synapses upon interneurons (Renshaw cells). Renshaw cells can **limit motor neuron firing** and change reflexes (see Dr. Grover's lecture in January).

Note: Changes in stretch reflexes are also symptomatic: **In general, <u>Decrease stretch reflexes can indicate Lower Motor Neuron Disorders</u>, <u>Increase Stretch reflexes can indicate Upper Motor Neuron Syndromes.</u>**

B. **Autogenic Inhibition** (also termed: Inverse Myotatic Reflex, Tendon Organ Reflex or Clasped-

Knife Reflex)

1. **Stimulus** - large force exerted by pulling on muscle tendon (ex. isometric contraction)
2. **Sense organ excited** - Golgi tendon organ (Ib afferent)
3. **Primary response** – motor neurons to muscle are inhibited (hyperpolarized) and muscle attached to tendon **relaxes; effect is polysynaptic.**
4. **Effect on synergist (similar action) and antagonist (opposing action) muscles – all effects polysynaptic -** a. Inhibit synergist muscles - b. Excites antagonist muscles
5. **Function of Autogenic inhibition** - **Regulating muscle tensions.**

Note: **Clasped knife reflex**: In Upper Motor neuron lesions, tonus may increase and resistance of muscle to stretch increases; if sufficient force is applied, **limb resistance suddenly decreases** (like a pocket knife snapping shut); this is thought to be mediated by reflexes of Golgi tendon organs.

Note: The connections for autogenic inhibition are inactivated during walking; Effects of Golgi tendon organs then become excitatory (through other interneurons).

C. **Flexor reflex** - reflex withdrawal from a painful or noxious stimulus; can produce excitation of flexor motor neurons; can also take other forms (exciting muscles with other actions, ex. abductor muscles that pull limb away from midline)

1. **Stimulus** - noxious or painful stimulus to skin
2. **Sense organs excited** - Cutaneous touch receptors, pain (nociceptors)
3. **Primary response** - polysynaptic excitation of motor neurons to flexor muscles.
4. **Other effects - a. Same side – excite flexors, inhibit extensors. b. Opposite side – excite extensors, inhibit flexors.**
5. **Function of Flexor Reflex** - Protective (example: stepping on a nail). The net effect of these connections is that very rapid adjustments are made so that one leg is lifted rapidly and the other supports the weight of the body.
6. **Clinical Changes in Flexor Reflexes** - Flexor Reflexes can change after lesions, disease processes;

ex. **Babinski Sign** - seen after **Upper Motor neuron lesion**; normal response - stroking sole of foot **normally** results in **flexion (plantar flexion) of toes** (not strictly a withdrawal reflex); Babinski sign - direction of movement **changes to extending (dorsiflexing) toes**.

III. REFLEXES OF CRANIAL NERVES

A. Pupillary light reflex (Optic Nerve CNII in; Oculomotor CN III out) - Light shone in the eye causes the pupil to constrict; Stimulus - light; detected by sensory neurons (photoreceptors) in retina; sensory signals in Optic Nerve (Cranial Nerve II); Response - motor signals in Oculomotor Nerve (Cranial Nerve III, innervates pupillary constrictor muscle); Function - limit amount of light; protects photoreceptors in retina; connection present at all times.

B. Corneal reflex (Trigeminal nerve CNV in; Facial nerve CN VII out) - Touching corning of eye causes closing of eyelids. Stimulus - touch detected by sensory neurons (Somatic Sensory) in Trigeminal nerve V (Long Ciliary Nerves V1); Response – Motor signals in Facial Nerve (CN VII) innervate Orbicularis Oculi muscle (muscle of Facial Expression) which closes eyes; Function –

protective of Cornea.

C. Gag Reflex (Glossopharyngeal nerve CNIX in; Vagus nerve CNX out) - Touching pharynx induces gagging. Stimulus - Touch detected by Visceral Sensory neurons in Oropharynx innervated by Glossopharyngeal nerve CNIX; Response - motor signals in Vagus nerve (CNX) cause contraction of pharyngeal constrictor muscles.

D. Jaw Jerk Reflex (Stretch Reflex of Muscles of Mastication (ex. Masseter) (Trigeminal nerve V in; Trigeminal nerve V out) - Tap down on mandible induces contraction of muscles that elevate mandible (close mouth). Stimulus – detected by Muscle spindles in muscles of mastication (ex. jaw closer, Masseter) contained in Trigeminal nerve (CNV); Response - motor neurons also contained in Trigeminal nerve cause. contraction of jaw closer muscle

IV. PATTERN GENERATORS IN SPINAL CORD AND BRAINSTEM

A. Spinal cord contains networks of interneurons that generate patterned motor activities (networks are called Pattern Generators; see Dr. Grover's lecture, Neuronal Integration, next block).

B. ex. Walking - Walking is thought to be produced by pattern generators. In addition, after spinal cord lesion, rear limbs of animals and legs of humans can walk on treadmills (if body weight is supported). This has led to new therapies for patients with spinal cord injuries (ex. Christopher Reeve, actor who played 'Superman')

Note: Stepping reflexes in infants probably represent activation of the pattern generator for walking. Infants don't learn to walk; they learn to maintain balance while walking.

SPINAL REFLEXES AND DISORDERS

REFLEX	STIMULUS/SENSE ORGAN(S) EXCITED	NORMAL RESPONSE	UPPER MOTOR NEURON DISORDERS
Stretch (Myotatic, Deep Tendon) Reflex – Compensatory maintain position (ex. riding on moving bus)	Rapid Stretch of muscle (test: tap on muscle tendon) Excites Muscle Spindle Primary (Ia) and Secondary (II) sensory neurons (NOT Golgi Tendon Organ)	Stretched muscle contracts rapidly (monosynaptic connection); also Excite synergist and Inhibit antagonist Note: Gamma motor neurons can enhance stretch reflexes, tell patient to relax before test	Hyperreflexia - (increase) - characteristic of Upper Motor Neuron lesions (ex. spinal cord injury, damage Corticospinal tract); note: Clonus = hyperreflexia with repetitive or sustained contractions to single stimulus
Autogenic Inhibition - Limits Muscle Tension	Large force on tendon excites Golgi Tendon Organ Ib (test: pull on muscle when resisted)	Muscle tension decreases; Also inhibit synergist muscles; excite antagonist muscles	Clasped Knife Reflex - occurs in Upper Motor Neuron lesions - forceful stretch of muscle is first resisted then collapses
Flexor Reflex - Protective avoidance reflex	Sharp, painful stimulus, as in stepping on nail; Excites - Cutaneous and pain receptors (test: stroke foot with pointed object)	Limb is rapidly withdrawn from stimulus; protective reflex; also inhibit extensors of same limb and excite extensors of opposite limb (Crossed Extensor Reflex)	Babinski sign- toes extend (dorsiflex) to cutaneous stimulus of sole of foot (normally plantar flex); characteristic of Upper Motor Neuron lesion

REFLEXES OF CRANIAL NERVES

REFLEX	STIMULUS	SENSORY	RESPONSE	CLINICAL
Pupillary Light Reflex (II to III)	Test: Shine light in eye	Light detected by Optic Nerve	Excite Constrictor of pupil of eye (III Short Ciliary nerves (Ciliary Ganglion, parasympathetic)	Extensively used to check CN II; Absence of Pupillary Light Reflex can indicate catastrophe (brain herniation)
Corneal Reflex (V to VII)	Touch cornea of eye with cotton	Touch detected by Long Ciliary nerves (V1), Somatic sensory	Close eye (VII to Orbicularis Oculi muscle) Branchiomotor	Absence of Corneal Reflex; Test for damage to V1 sensory, VII motor
Gag Reflex (IX to X)	Test: Touch posterior tongue, oropharynx;	Excites Visceral Sensory endings in Glossopharyngeal N. (IX)	Excite muscles of pharynx, palate; Vagus N. (X), Branchiomotor	Other symptoms of Vagus damage (X); Patient Say's Ahh: soft palate not elevated on ipsilateral side (paralyze Levator Palati); uvula deviated away from side of lesion
Jaw Jerk Reflex Stretch (Deep Tendon) Reflex (V to V)	Test: tap down on mandible; Stretch muscles of mastication (ex. Masseter)	Excites Muscle Spindle sensory neurons in Trigeminal nerve (V)	Contract muscles that elevate mandible Motor - V3	Hyporeflexia - indicates Trigeminal nerve damage