NBL 356-656 Module 2 Review Q&A

1. *Identify the overall anatomy of the spinal cord. Where and what are the dorsal root ganglia, dorsal horns and dorsal roots, the ventral horns and ventral roots, and general location in the white matter of the lateral and anterior corticospinal tracts, and extrapyramidal tracts? What do the dorsal and ventral horns contain, and what are their general/overall functions? Where do the motor and sensory axons exit and enter the spinal cord? How are the spinal cord gray matter regions (horns) mapped for sensory and motor functions?*

The dorsal and ventral roots lie on the outermost lateral dorsal and ventral regions of the spinal cord, and are the regions where the sensory axons enter (dorsal-posterior) or motor axons exit (ventral-anterior) the spinal cord. The dorsal (posterior) and ventral (anterior) horns are the gray matter regions, which contain neuronal cell bodies, dendrites, synapses, gray matter astrocytes and blood vessels. Gray matter: dorsal/posterior=sensory, ventral/anterior=motor. The white matter includes the ascending/sensory tracts (afferents) and descending/motor tracts (efferents) that contain myelinated axons, oligodendrocytes, white matter astrocytes and blood vessels.

Sensory and Motor Maps in the Spinal Cord: Axons from both the somatic and visceral primary sensory neurons enter the cord through the dorsal (posterior) root. In the spinal cord, the neurons that relay sensory information are also called secondary sensory neurons (or second order sensory neurons). Both somatic and visceral secondary sensory neurons are located in the dorsal horn. (If one is looking at a cross section of the spinal cord with the dorsal horn on the upper side and the ventral horn on the lower side, then the secondary somatic sensory neurons are located in the upper region of the dorsal horn, while the secondary visceral sensory neurons are located in the lower region of the dorsal horn.

Spinal interneurons and somatic motor neurons are located in the ventral (anterior) horn. Spinal interneurons and autonomic motor neurons are located in a region called the lateral horn. Both types of motor axons exit through the ventral root.

1. *What are the five major regions/levels/segments of the spinal cord along the A/P (R/C) axis? What are the functions of the vertebral column and spinal meninges? What are the cervical and lumbar enlargements and why are they enlarged?*

The major regions of the spinal cord (rostral/anterior to caudal/posterior) include cervical, thoracic, lumbar, sacral, and coccygeal. The vertebral column and meninges serve to protect and cushion the spinal cord and allow the flow of CSF around it. The cervical and lumbar enlargements are regions of the spinal cord where the diameter of the spinal cord is larger than in other regions. They contain more gray matter because there are more spinal interneurons, lower motor neurons and secondary sensory neurons in those regions. Those regions of he spinal cord control areas of the body (arms-hands and legs-feet) that have a greater density of innervation.

1. *Upper motor neurons send axons (descending tracts-corticospinal tracts and extrapyramidal tracts) and synapse on two different types of spinal interneurons and two different types of motor neurons in the spinal cord. What are these?*

The two different types of spinal interneurons in the ventral horn are the excitatory (glutamatergic) interneurons and inhibitory (glycinergic-GABAergic) interneurons. The two types of lower motor neurons are the alpha and gamma LMNs, which innervate different types of skeletal muscle. LMNs are cholinergic neurons

1. *The two main types of lower motor neurons are alpha and gamma motor neurons. Where are these neurons located? Where do their axons go? What type of muscles do they innervate? What is the main function of each type of motor neuron?*

Alpha and gamma motor neurons are located in the ventral horn. Their axons exit the spinal cord through the ventral/anterior root. They both travel in spinal nerves to (innervate) skeletal muscles. Alpha motor neurons innervate extrafusal muscle. This type of muscle is involved in muscle contraction that lengthens or shortens muscles that produces force and does work to produce movements. Gamma motor neurons innervate intrafusal muscle (also called the muscle spindle). The muscle spindle is a sensory receptor that sends information about muscle stretch back to the spinal cord. This sensory information ensures that muscle tension is maintained when necessary, and it protects muscles from damage. It is also involved in muscle tone. The gamma motor neurons induce contraction or relaxation of the spindle that maintains the size and tension of the muscle spindle so it can act as an accurate sensor.

1. *What is a muscle and a muscle fiber? What is the motor unit?*

From Wikipedia: “Muscle cells contain protein filaments of actin and myosin that slide past one another, producing a contraction that changes both the length and the shape of the cell. Muscles function to produce force and motion. They are primarily responsible for maintaining and changing posture, locomotion, as well as movement of internal organs, such as the contraction of the heart and the movement of food through the digestive system via peristalsis. Skeletal muscle is one of three major muscle types, the others being cardiac muscle and smooth muscle. It is a form of striated muscle tissue, which is under the voluntary (and involuntary) control of the somatic nervous system. Most skeletal muscles are attached to bones by bundles of collagen fibers known as tendons.”

A muscle fiber is a muscle cell, also called a myofiber. “Muscle fibers are the individual contractile units within muscle. A single muscle such as the biceps brachii contains many muscle fibers. Individual muscle fibers are formed during development from the fusion of several undifferentiated immature cells known as myoblasts into long, cylindrical, multi-nucleated cells.”

A motor unit consists of a single alpha lower motor neuron (LMN) and all the muscle fibers that it innervates. A single LMN activates the muscle fibers it innervates to simultaneously to contract. The muscle fiber types (slow or fast twitch) in a motor unit are all the same. Groups of motor units often work together to coordinate the contractions of a single muscle; all of the motor units within a muscle are considered a motor pool. Movement is generated when an UMN and spinal interneurons synapse an alpha LMN, which activates a motor unit (group of muscle fibers), causing coordinated contraction. Many movements and reflex arcs involve the coordinated contraction in one muscle and inhibited contraction (relaxation) in the opposing or antagonistic muscle.

1. *What are flexion, flexors, extension and extensors? What does it mean that some muscles work in antagonistic pairs? Explain why some movement would involve coordinated contraction and relaxation?*

Flexor and extensor muscles work in antagonistic pairs. Flexors decrease the angle around a joint (to produce flexion) while extensors increase the angle around a joint (to produce extension). To produce a movement that involves decreasing the joint angle, when the flexor contracts, the opposing extensor muscle is relaxed. Or to produce a movement that involves increasing the joint angle, when the extensors contract, the opposing flexor muscle is relaxed. This ensures that muscles don’t work against each other, prevents muscle strain and injury, and ensures that specific muscle contractions can produce finely tuned precise movements. (Note that not all movements are generated by muscles working in antagonistic pairs.)

1. *What is a motor pool? Describe the general types and organization of neurons in the ventral horn spinal gray matter. Briefly describe the maps of the motor pools in the ventral horn. (Consider the relative locations of neurons that control axial/distal, and flexor/extensor muscles.) Which descending motor tracts control which types of muscle groups? (Try to specify axial/distal and voluntary/involuntary.)*

A motor pool includes all the alpha lower motor neurons that innervate a single muscle. Motor pools exhibit some spread along the anterior/posterior axis. A muscle pool includes LMNs that innervate different types of skeletal muscle fibers including all the types of fast twitch and slow twitch fibers.

Somatic lower motor neurons are located in the ventral horn. For somatic lower motor neurons, the neurons that control proximal muscles in the trunk and midline (axial muscles in trunk) and proximal limb muscles, are located closer to the midline (more medially). LMNs that control distal muscles in the limbs/extremities (arms-hands and legs-feet) are located in the more lateral regions of the ventral horn. Looking again at a cross-section with dorsal up and ventral down, within the flexor/extensor pairs in the ventral horn, neurons that control flexors are located above those that control the extensors, which are located below.

The two lateral spinal cord systems are the corticospinal tract and rubrospinal tract. They synapse on motor neurons located more laterally in the ventral gray matter and control the voluntary movement of distal muscles in the arms-hands and legs-feet.

The four ventromedial spinal cord tracts include three extrapyramidal tracts (reticulospinal, tectospinal, and vestibulospinal) and one corticospinal tract (anterior corticospinal tract). (Apologies about the anatomy names. Ventral = Anterior. For some reason the anterior CST is usually called anterior instead of ventral. And the group of ventromedial systems are called ventromedial instead of anterior-medial.) These axons synapse on motor neurons located more medially in the ventral gray matter and control axial (trunk) and proximal limb muscles.

1. *Upper motor neurons send axons (descending tracts-corticospinal tracts and extrapyramidal tracts) and synapse on two different types of spinal interneurons and two different types of motor neurons in the spinal cord. What are these?*

The two different types of spinal interneurons in the ventral horn are the excitatory (glutamatergic) interneurons and inhibitory (glycinergic-GABAergic) interneurons. The two types of lower motor neurons are the alpha and gamma LMNs which innervate different types of skeletal muscle. Alpha and gamma motor neurons are located in the ventral horn. Their axons exit the spinal cord through the ventral/anterior root. They both travel in spinal nerves to (innervate) skeletal muscles. Alpha motor neurons innervate extrafusal muscle. This type of muscle is involved in muscle contraction that lengthens or shortens muscles that produces force and does work to produce movements. Gamma motor neurons innervate intrafusal muscle (also called the muscle spindle). The muscle spindle is a sensory receptor that sends information about muscle stretch back to the spinal cord.

1. *Alpha lower motor neurons receive inputs from different types of neurons. What are these? What is the “Final Common Pathway?”*

Alpha LMNs receive inputs from upper motor neurons of the corticospinal spinal tracts or upper motor neurons of the extrapyramidal tracts, as well as inputs from excitatory and inhibitory interneurons. LMNs can also receive direct inputs from sensory neurons (called 1a sensory neurons) that innervate muscle spindles. From Purves: “The cell bodies of upper motor neurons are located either in the cortex (pyramidal tracts) or in brainstem centers (extrapyramidal tracts). The axons of the upper motor neurons typically contact the local circuit neurons in the brainstem and spinal cord, which, via relatively short axons, contact in turn the appropriate combinations of lower motor neurons. The local circuit neurons also receive direct input from sensory neurons, thus mediating important sensory motor reflexes that operate at the level of the brainstem and spinal cord. Lower motor neurons, therefore, are the final common pathway for transmitting neural information from a variety of sources to the skeletal muscles.”

1. *What specifically is the neuromuscular junction (NMJ)? What neurotransmitter and type of receptor is used at the NMJ? Briefly describe the major stages (presynaptic and postsynaptic) of synaptic transmission at the NMJ. What is the end plate potential, where is it produced, what type of response is it, and what is its purpose? (Include how an action potential in the motor neuron produce an action potential in the muscle cell.)*

The neuromuscular junction (NMJ) is the synapse between the presynaptic motor axon and the postsynaptic muscle fiber (myofiber) at a region called the motor end plate. The lower motor neurons (both alpha and gamma) are cholinergic neurons that release acetylcholine at the NMJ. The receptors on the postsynaptic muscle cell motor end plate are nicotinic acetylcholine receptors, a type of excitatory ionotropic receptor.

The presynaptic motor neuron AP depolarizes the presynaptic membrane potential → this depolarization opens voltage gated (VG) Ca2+ channels that conduct the flow of Ca2+ inside the presynaptic terminus → Ca2+ induces the NT vesicle containing ACh to fuse with the presynaptic membrane by exocytosis, releasing ACh into the synaptic cleft → postsynaptic nicotinic ACh receptors at the motor end plate bind the ACh, which opens the channel region of these ionotropic receptors, and conducts the inward flow of Na+ and outward flow of K+ → this produces the end plate potential (EPP), which is a large (~50 mV) depolarization → the EPP spreads/travels along the muscle plasma membranes → the depolarization activates VG Na+ and VG K+ channels located in the plasma membrane adjacent to the motor end plate → this induces an AP in the muscle fiber that is conducted along the muscle plasma membrane and along the T-tubule. The AP in the T-tubule then causes an increase in Ca2+ in the muscle cytoplasm (sarcoplasm) that activates muscle contraction (see next.)

1. *How does the AP in the muscle T tubule lead to an increase in Ca2+ in the sarcoplasm? Include the roles of the DHPR voltage gated Ca2+ channels, sarcoplasmic reticulum, ryanodine receptor SR Ca2+ channels, and SERCA Ca2+ pump. (The lecture summary slide, the YouTube video, and this paragraph on excitation-contraction coupling in the Wikipedia article on muscle contraction may be helpful:* [*https://en.wikipedia.org/wiki/Muscle\_contraction#Excitation-contraction\_coupling*](https://en.wikipedia.org/wiki/Muscle_contraction#Excitation-contraction_coupling)*.)*

The AP travels along the muscle fiber plasma membrane via propagation and along and down the T-tubules, which contain DHP receptors (DHPRs) which are L-type voltage gated Ca2+ channels (VGCC) → Depolarization induces a conformational change in DHPR, activating them. The Ca2+channel opens and allows the inward flow of Ca2+. However, this influx of Ca2+ is not actually necessary→ The DHPR are bound (by protein-protein interactions) to and so they are physically coupled to the ryanodine receptor (which are a type of Ca2+ channel that are located in the SR membrane). Importantly, the voltage sensitive conformation change in the DHPR (that is there to open the Ca2+ channel region) is also transmitted to and produces a conformational change in the RyR located on the SR membrane, and opens them, allowing Ca2+ to flow out of the SR into the sarcoplasm. The SR contains a large Ca2+ store, because of the SERCA pumps that fill the SR with Ca2+ → When the coupled RyR are activated by the DHPR, they open and this allows Ca2+ to flow out of the SR and into the sarcoplasm. That released Ca2+ from the SR further promotes the release of more Ca2+ from neighboring RyR, since they are also activated by just the binding of Ca2+. This produces a feed-forward release of Ca2+ from the SR. The released Ca2+ activates the muscle contraction by the sliding filament mechanism (see below). Lastly, Ca2+ is rapidly transported inside the SR to stop muscle contraction via the SERCA pump. Ca2+ can also be transported out of the muscle cell by plasma membrane Na+/ Ca2+ exchanger and Ca2+ ATPase/pump.

1. *A category of drugs called dihyrdopyridines (DHP) are blockers of voltage gated Ca2+ channels (they block the movement of Ca2+ through the channel) and are used to treat high blood pressure (hypertension). Why don’t the DHP drugs cause paralysis or other mental problems?*

The activation of the DHPR/VGCC by depolarization of the T tubule membrane leads to an activation of the RyR in the SR membrane, since these two channel proteins are bound to each other. In response to the depolarization, the DHPR undergoes a conformation change that is transmitted to the RyR which is a Ca2+ channel and it is opened. Then Ca2+ flows out of the SR into the muscle cytoplasm where it will stimulate muscle contraction. Although the DHPR/VGCC functions as a Ca2+ channel, the movement of Ca2+ through the channel is not actually required for it to activate the RyR in the SR. So although dihydropyridines block the movement of Ca2+ through the channel, it doesn’t block the conformation change in the DHRPR that leads to activation of the RyR and release of Ca2+ from the SR. Why does it not affect mental function? Though in the CNS presynaptic NT release requires VGCCs and Ca2+ influx, the Ca2+ channels that mediate NT release are a different isoform of VGCC (they are the N and T types in the presynaptic neuron) that don’t bind and are not sensitive to DHPs. Also DHPs don’t cross the blood-brain barrier very well.

1. *Briefly describe the “sliding filament theory” (what is the mechanism whereby increased Ca2+ in the sarcoplasm (muscle cytoplasm) leads to muscle contraction.)*

A. Before contraction, when the muscle is in the relaxed but active state: Myosin binds ATP and hydrolyzes the ATP to ADP and Pi. This leads to a large conformational movement of the myosin head, and the myosin with ADP and Pi bound is now in what is called the “cocked” state. The actin filaments have troponin bound to them, which covers the myosin binding sites.

B. The muscle AP leads to Ca2+ release from the SR (described above).

C. Ca2+ binds to troponin. This releases troponin from the actin filament, and exposes the myosin binding sites on the actin filament.

D. Myosin heads bind to the actin filament.

E. While bound to actin, the myosin head releases the Pi, and this induces the myosin head to move/pivot (it undergoes a large conformational change), producing the “power stroke,” and since the myosin head is bound to the actin filament, this results in the sliding of the actin filament along the myosin filament.

F. After moving/pivoting, the myosin head then releases the bound ADP, but the myosin is still bound to actin. This is the “rigor state.” (Some models show the myosin releasing both Pi and ADP at the same time, leading to the power stroke and then the myosin is bound to actin only.) (If there is no ATP in the cell, the myosin stays bound to the actin in the rigor state.)

G. The myosin head can now bind a new molecule of ATP, and when it binds ATP, that releases the myosin from the actin.

H. The myosin head then hydrolyzes the bound ATP to ADP and Pi, inducing the movement of the myosin head back into the “cocked state,” with ADP and Pi bound. When Ca2+ levels decrease, the troponin rebinds the actin filament (blocking the myosin binding site), and the filament is back to the relaxed but active state.

1. *What features of the NMJ ensure that it is an efficient and reliable synapse (an AP in the alpha motor neuron always produces muscle contraction)? Why do you think the muscle resting membrane potential is so negative (-90 mV)?*

One muscle fiber receives inputs from only a single motor neuron, hence the activity of the single alpha motor neuron determines the activity/contraction of the muscle fiber. There is no integration of inputs at the muscle fiber. The entire control of muscle contraction and relaxation occurs at the level of the control of the alpha motor neuron in the spinal cord (or brainstem).

The NMJ is a very large synapse with postsynaptic folds that maximize the surface area, which can incorporate many nicotinic ACh receptors. The presynaptic region of the motor axon has many active zones and each active zone has lots of synaptic vesicles. Therefore when an action potential arrives, a large amount of acetylcholine is released from the presynaptic motor axon. And because of the junctional folds there are a lot of nicotinic ACh receptors located on the muscle membrane at the motor end plate. Activation of many nAChRs produces a large end plate potential (EPP), on the order of ~ 50 mV. This means that the muscle depolarizes from its resting potential (-90 mV) to about -40 mV. As the depolarization (the EPP) spreads/travels outside the end plate along the membrane, it is always large enough to meet threshold (-55 mV) to activate the nearby VG Na+ channels and an EPP will always produce an AP in the muscle membrane. Therefore, an AP in the presynaptic motor axon always produces an AP in the muscle membrane, which then always leads to muscle contraction.

Two mechanisms ensure that muscle contraction occurs only when there is a LMN AP. The resting membrane potential is very negative (-90 mV), so if there is any spurious small amount of release of Ach (in the absence of a LMN AP) it will not produce an EPP that will be large enough to trigger an AP in the muscle. Also, the presence of acetylcholine esterase (AchE) ensures that if there is any spurious small release of Ach (in the absence of a AP) from the presynaptic motor neuron, it will be degraded rapidly to prevent any unwanted muscle contraction.

1. *What two major factors influence the strength and speed of muscle contraction? What are the three main types of muscle fibers, where are they located and why do muscles need both fast and slow twitch fibers? (Briefly explain the size principle for muscle recruitment.)*

Factors: the type of muscle fibers and the motor neuron activity. Types of muscle fibers: slow, fast fatigue resistant and fast fatigable. (More types have been identified including intermediate.) All human muscles contain all types of fibers, but different ratios of the three/four types. Need three/four types because they match the physiological properties of different motor unit types with the different amounts of force required to complete tasks. Fast fatigable allow for rapid quick movements such as sprinting, jumping and other force heavy movements. Fast fatigue resistant and intermediate are important for intermediate contractions such as carrying objects and jogging. Slow allows for lower force contractions that need to be used for a long periods of time, such as slow walking, and to maintain posture.

Movement is generated when an UMN and spinal interneurons synapse an alpha LMN, which activates a motor unit (group of muscle fibers), causing coordinated contraction. A motor unit consists of a single alpha lower motor neuron (LMN) and all the muscle fibers that it innervates. A single LMN activates the muscle fibers it innervates to simultaneously to contract. The muscle fiber types (slow or fast twitch) in a motor unit are all the same. Groups of motor units often work together to coordinate the contractions of a single muscle; all of the motor units within a muscle are considered a motor pool. A motor pool includes all the alpha lower motor neurons that innervate a single muscle, including all the types of fast twitch and slow twitch fibers. Size principle (from Wiki) “Henneman's size principle states that under load, motor units are recruited from smallest to largest. In practice, this means that slow-twitch, low-force, fatigue-resistant muscle fibers (which are smaller) are activated before fast-twitch, high-force, less fatigue-resistant muscle fibers (which are larger).” The ratio of the main types of fibers in a muscle is not fixed, but can be changed by use and different types of activity.

1. *What is the rate code and what suggests that motor neurons use the rate code to convey information?*

The rate code is a type of neural code where the information from the presynaptic neuron is encoded in the frequency (rate) of action potentials. Experiments show that for a given muscle fiber there is a direct correlation (linear relationship) between action potential rate and muscle contraction/force. (However there is a plateau since there is a ceiling effect, with a maximal amount of force that any muscle can produce even when maximally stimulated.)

1. *If muscle cells only receive excitatory inputs (at the motor end plate from alpha motor neurons), how is muscle contraction inhibited to produce muscle relaxation in the antagonist muscle?*

Muscle contraction is inhibited (and relaxation produced) through the activation of inhibitory interneurons in the spinal cord. Inhibitory interneurons receive inputs from upper motor neurons, sensory neurons and other spinal interneurons. They synapse on and control alpha motor neurons and inhibit them from firing action potentials. When action potentials in the alpha motor neuron are inhibited, the muscle will relax.

1. *What is the function of gamma motor neurons?*

They are motor neurons that innervate intrafusal muscle fibers (also called muscle spindles). Muscle spindles have a sensory function to relay the amount of stretch or movement in a muscle. Gamma motor neurons don’t produce force like alpha motor neurons but instead function to control the contraction of the muscle spindle and adjusting the sensitivity of muscle spindles, so they can provide the correct amount of sensory information.

1. *What is a “reflex?” What are the two main types of reflexes?*

A reflex is an involuntary and nearly instantaneous movement in response to a stimulus. A reflex does not require higher brain function. Another definition: a stereotyped response to a specific sensory stimulus that integrates sensory input with motor output and does not require higher brain function once activated. Reflex arcs are found in the spinal cord and brainstem. The types of neurons involved in a reflex are sensory (afferent) neurons, interneurons and lower motor neurons (efferent) of the spinal cord or brain stem. The two main types of reflexes are somatic and autonomic. Somatic reflexes involve a sensory neuron and control of skeletal muscle. Somatic reflexes include spinal and cranial nerve reflexes, and developmental reflexes in infants such as grasping and sucking, and other reflexes such as the gastrocolic reflex. Autonomic reflexes involve a visceral sensory neuron and control of visceral tissues or organs through regulation of smooth or cardiac muscles or glands. Autonomic reflexes include the baroreflex, sweating, flushing of the skin, and the accommodation reflex of eye.

Spinal reflexes are a type of somatic reflex that includes 1. Stretch (muscle) 2. Golgi tendon (muscle-tendon) and 3.Withdrawal (skin) (The stretch and GTO reflexes are mainly involved in proprioception with some function in protection of muscles from over-compression; the withdrawal reflex is involved in limiting injury.)

1. *What are stretch reflexes and what do they involve? What is proprioception and what are the two types?*

Stretch (myotatic) reflexes involve muscle spindles, which sense/detect the amount of muscle stretch, spinal interneurons and alpha motor neurons. From Wikipedia: “The stretch reflex (myotatic reflex) is a muscle contraction in response to stretching within the muscle. It includes a monosynaptic reflex, which provides automatic regulation of skeletal muscle length, and a disynaptic component to regulate the opposing muscle. When a muscle lengthens, the muscle spindle is stretched and its nerve activity increases. This increases alpha motor neuron activity, causing the muscle fibers to contract and thus resist the stretching. A secondary set of neurons also causes the opposing muscle to relax. The reflex functions to maintain the muscle at a constant length.”

Proprioception is also called kinesthesia; it is the sense of self-movement and body position. Proprioception is mediated by proprioceptors, which are types of mechanoreceptors located within muscles, tendons, and joints. Proprioceptors are activated during distinct behaviors and encode distinct types of information: limb velocity and movement, load on a limb, and limb limits. The central nervous system integrates proprioception and other sensory systems, such as vision and the vestibular system, to create an overall representation of body position, movement, and acceleration. The two types are conscious and unconscious (non-conscious). Conscious proprioception involves the transmission of information to the somatosensory cortex for conscious perception of body and limb position and movement. Unconscious (or non-conscious) proprioception involves the transmission of information mainly to the cerebellum for the unconscious control of posture and balance, coordination, and feedback for pattern movements such as walking.

1. *What is the patellar tendon/ligament (knee-jerk) reflex and what two functions is it involved in? Describe the circuitry.*

Intrafusal muscle fibers are skeletal muscle fibers that serve as specialized sensory organs (proprioceptors) that detect the amount and rate of change in length of a muscle. They constitute the muscle spindle and are innervated by three axons, two sensory (Ia and II) and one motor (from gamma motor neuron). The knee jerk reflex is the reflex caused when the muscle spindle is abruptly stretched in the quadriceps muscle, which can be induced by striking the patellar tendon/ligament. The muscle stretch causes the 1a sensory neurons to increase firing. When the primary sensory neuron axons enter the spinal cord, they branch and one branch synapses directly on the alpha motor neurons in the dorsal horn, which directly activate the quadriceps muscle to contract. This is a monosynaptic connection. Another 1a branch synapses on an inhibitory interneuron that inhibits the alpha motor neuron of the opposing muscle (the antagonist muscle, the hamstring) causing it to relax. This is a disynaptic connection. This reflex is ipsilateral since it affects muscle contraction and relaxation on the same side of the body where the sensory afferents detect info and send it into the spinal cord. It is important in maintaining balance and posture. It is part of the righting response when one loses ones balance, and it is also though to be part of the central pattern generator involved in locomotion (walking).

1. *What is the loading reflex and its function? Describe the circuitry.*

Another type of myotatic/stretch reflex is the loading reflex. It maintains limb position in response to a load; for example, it allows one to hold a cup and have someone fill it without spilling. Stretch reflexes also allow for us to judge the precise amount of muscle force to generate especially when the “load” changes. This is important for stable movement. Stretch reflexes also been proposed to act as a minor protective measure for the muscles, to prevent muscle tearing. The biceps muscle becomes stretched, and the 1a sensory neuron in the spindle is activated, it synapses on alpha motor neuron that innervates the same/biceps muscle and causes it to contract, thus resisting the load. The 1a sensory neuron also synapses on an inhibitory interneuron that controls the alpha motor neuron that innervates the opposing/antagonist muscle, the triceps muscle, causing it to relax.

1. *What is the Golgi tendon organ and what does it do? Describe the circuitry of the Golgi tendon reflex.*

The GTOs are sensory organs that are located at the junction of muscles and tendons. They detect muscle tension and are relatively insensitive to passive muscle stretch. A Golgi tendon reflex is when muscle contraction compresses the collagen fibrils and compression/activation of the 1b sensory receptor. They are important because they prevent over-contraction of muscle, and allow fine control of tension when grasping delicate objects. It is similar to the disynaptic component of the patellar tendon/ligament reflex, since it involves sensory neurons that synapse on interneurons in the spinal cord, which then control alpha motor neurons, and is ipsilateral (on the same side). However, it produces the opposite type of response in that activation of the 1b sensory neuron causes the muscle, which produced the original contraction response, to relax. And simultaneously through activation of an excitatory interneuron, it induces the opposing/antagonist muscle to contract. In the GTO, both responses are disynaptic.

1. *What is the withdrawal (flexion-crossed extension) reflex, what components are involved and what is its function? Describe the circuitry.*

The W/FCE reflex is a reflex in which an extremity is withdrawn in response to a painful/noxious stimulus. It is not involved in proprioception like the stretch reflex but is involved in protection of tissues from damaging stimuli. Components: nociceptor in skin, muscle doing the contracting, cell body and axon of afferent neuron, cell body and axon of motor/efferent neuron, and interneuron to transmit signal from sensory afferent axon to efferent motor neuron located in spinal cord. Flexion is stimulated by a painful stimulus on the ipsilateral side, with numerous flexor muscles stimulated. Extension is stimulated on the contralateral side with numerous extensors stimulated. The flexion-crossed extension reflex is important to limit damage to a limb caused by the painful stimulus. It also ensures that the limb on the contralateral side are regulated to ensure stability.

From Wikipedia: The crossed extensor reflex is a withdrawal reflex. When the reflex occurs, the flexors in the withdrawing limb contract and the extensors relax, while in the other limb, the opposite occurs. An example of this is when a person steps on a nail, the leg that is stepping on the nail pulls away, while the other leg takes the weight of the whole body. The crossed extensor reflex is contralateral, meaning the reflex occurs on the opposite side of the body from the stimulus. To produce this reflex, branches of the afferent nerve fibers cross from the stimulated side of the body to the contralateral side of the spinal cord. There, they synapse with interneurons, which, in turn, excite or inhibit alpha motor neurons to the muscles of the contralateral limb. In the ipsilateral leg (the one which steps on the nail), the flexors contract and the extensors relax to lift the leg from the ground. On the contralateral side (the one that bears all the weight), the flexors relax and the extensors contract to stiffen the leg since it must suddenly support the entire weight of the body. At the same time, signals travel up the spinal cord and cause contraction of the contralateral muscles of the hip and abdomen to shift the body’s center of gravity over the extended leg. To a large extent, the coordination of all these muscles and maintenance of equilibrium is mediated by the cerebellum and cerebral cortex.