

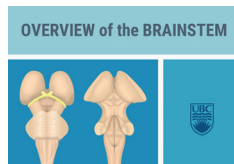
January 12, 2026 - Dr. Stiver ([mikaela.stiver@ubc.ca](mailto:mikaela.stiver@ubc.ca))

### Objectives:

1. Differentiate between the somatic and the visceral peripheral nervous systems and how these two systems are controlled by the CNS. (*MEDD 411 spiral*)
2. Conceptualize the sensory input to the brain: how this information is gathered, sorted, interpreted, prioritized.
3. Conceptualize the motor output from the brain: how is this signal generated, controlled, prioritized.
4. Relate the location and general function of the 12 cranial nerves to the clinical neurological exam.

### Resources

Below are the e-tutorials, videos, and web resources for this lab – click the green buttons to access them.



In today's bootcamp we will review everything from Friday's session and add in a couple of critical concepts. Please make sure you take the time to review materials and 3D brain specimens.



This icon located throughout the lab manual indicates **checklist items!**

**\*\* NOTE:** Interactive PDFs are best viewed on desktop/laptop computers – functionality is not reliable on mobile devices \*\*

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### Somatic Nervous System

The somatic nervous system innervates structures derived from **somites** and is ultimately **controlled by cortical areas**.

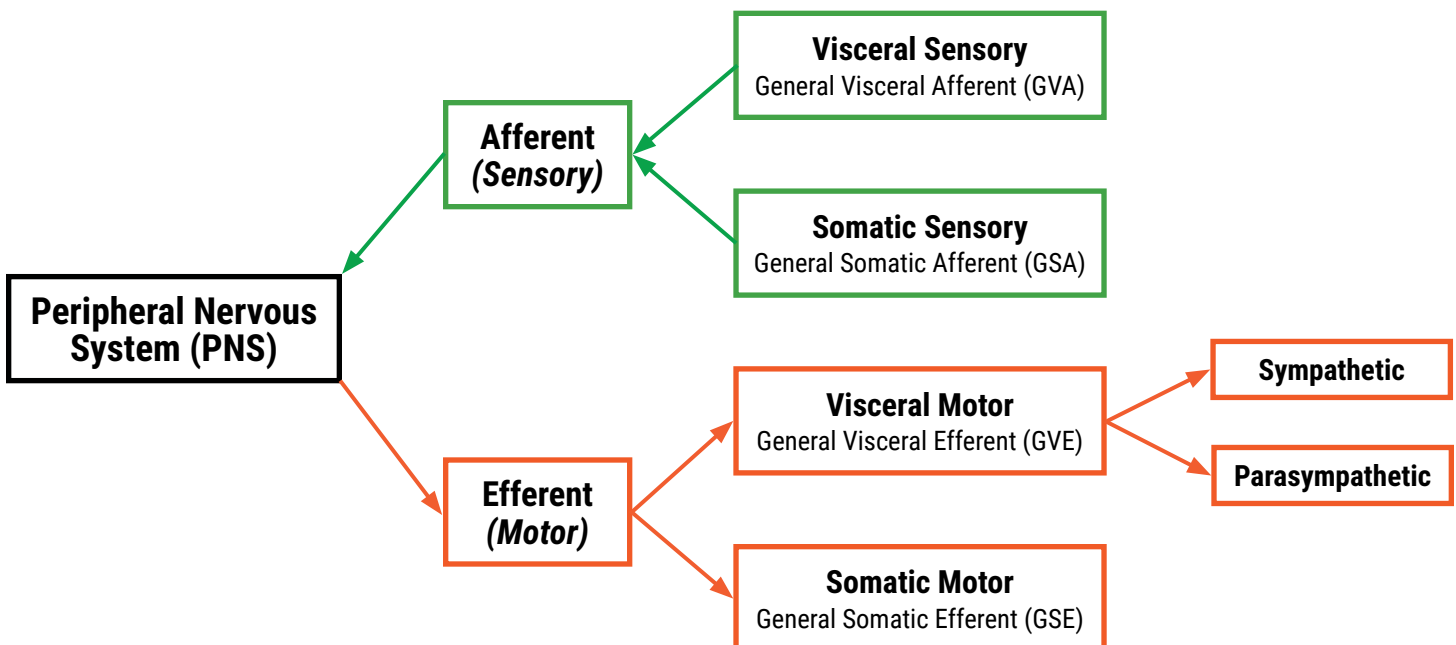
- For the **motor** branch, cell bodies of upper motor neurons are in the primary motor cortex.
- **Sensory** information will terminate in the primary somatosensory cortex.
  - Association areas are closely linked to these areas.

This is a review from MEDD 411. Have a look at this video to refresh your memory:

### Visceral Nervous System

The visceral nervous system innervates the thoracic, abdominal, and pelvic viscera, sweat glands, blood vessels. It is ultimately **controlled by the hypothalamus**.

- The visceral **motor** fibers are either parasympathetic or sympathetic, and both systems are influenced by the hypothalamus.
- The visceral **sensory** information can be divided into two main parts:
  1. Information about **physiological function** will project to specific brainstem nuclei and to the hypothalamus.
  2. Information about **pain** is prioritized and relayed to the cortex, where it often gets confused for somatic pain (look back at your referred pain lecture from MEDD 411).



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### *Spiral From MEDD 411*

*What modalities can be found in a single spinal nerve?*

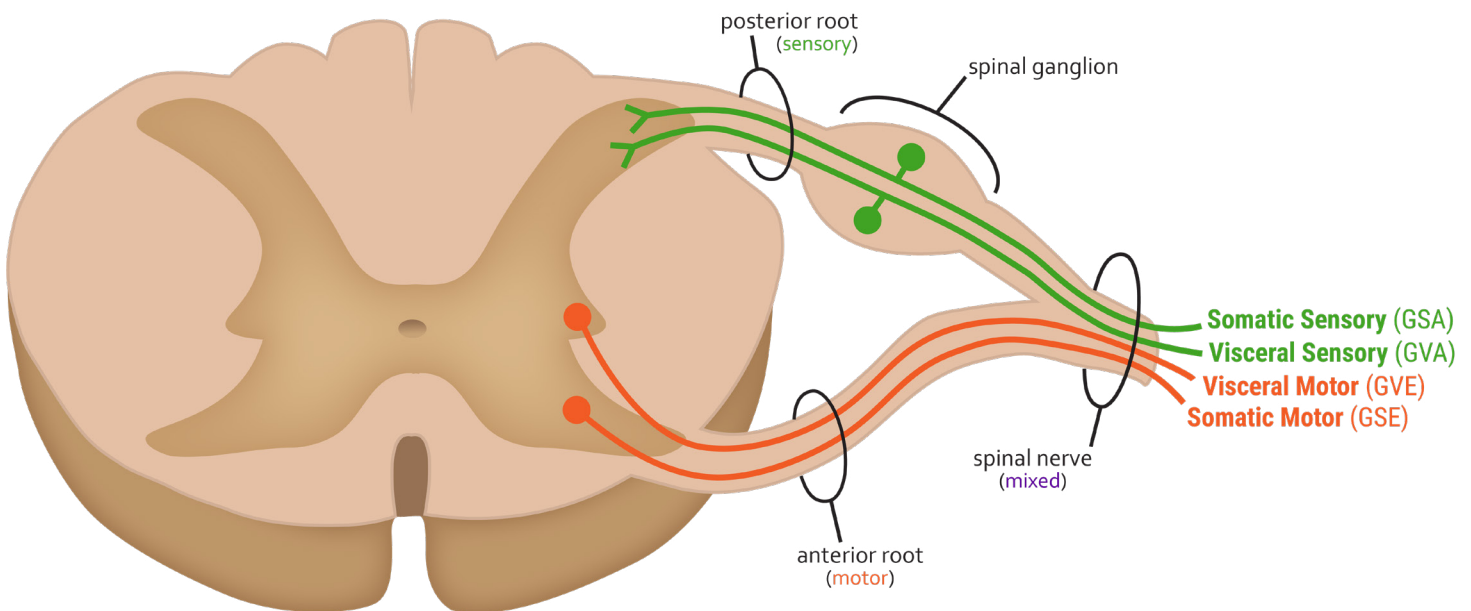
*The bladder has various types of peripheral nerve fibers – what do they do? (Look this up)*

*Which modality will innervate the following muscles? (Look this up, it is meant to set you up for next lab)*

- Detrusor muscle
- External urinary sphincter

*Which fibers will carry this sensory information? (Look this up)*

- Bladder fullness
- Pain



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### Sensory Input

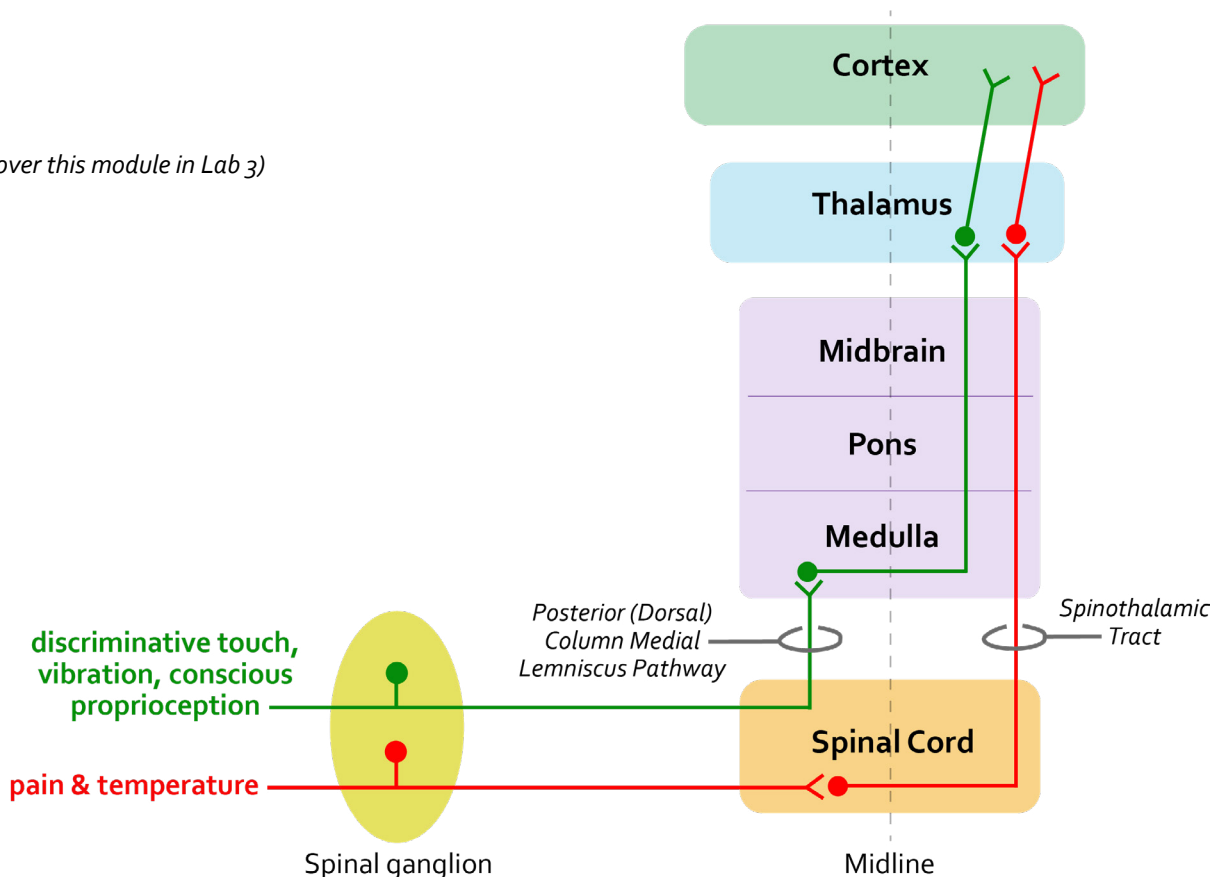
- Sensory information from the body (somatic information) enters the CNS through the **posterior root of a spinal nerve**.
- Sensory information is divided into two major pathways and these have **two anatomically distinct pathways**. This is important because your neurological exam will look at these modalities and help you focus on the diagnosis.

### Pain & Temperature

- In the **spinothalamic tract**
- Enters the **posterior horn** of the spinal cord
- Information is modified in the posterior horn (synapses!)
- Fibers cross the midline in the spinal cord and ascend to the **contralateral thalamus**
- Fibers synapse in the thalamus, then signals are sent to the **primary somatosensory cortex**

**3-neuron chain**  
**Fibers cross in the spinal cord**

(We will cover this module in Lab 3)



How would you approach testing the spinothalamic tract in a clinical neurological exam?

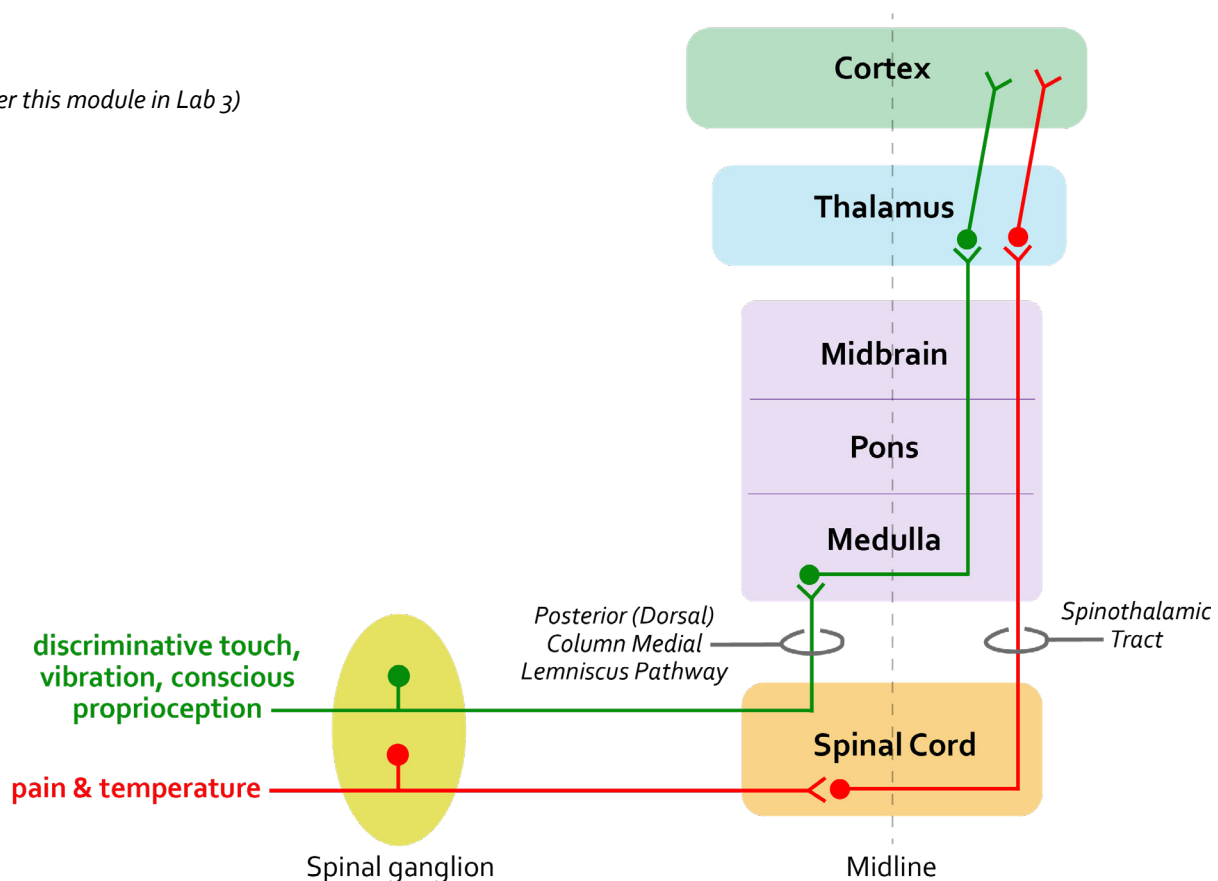
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### Discriminative Touch, Vibration, Conscious Proprioception

- In the **posterior column-medial lemniscus (PCML)** or **dorsal column-medial lemniscus (DCML)** pathway
- Enters the **posterior horn** of the spinal cord
- No modification of the signal in the spinal cord (no synapses!)
- Fibers ascend to the **brainstem**, where they synapse in the medulla
- Fibers then cross the midline and ascend to the **contralateral thalamus**
- Fibers synapse in the thalamus, then signals are sent to the **primary somatosensory cortex**

**3-neuron chain**  
Fibers cross in brainstem (medulla)

(We will cover this module in Lab 3)



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### Thalamus

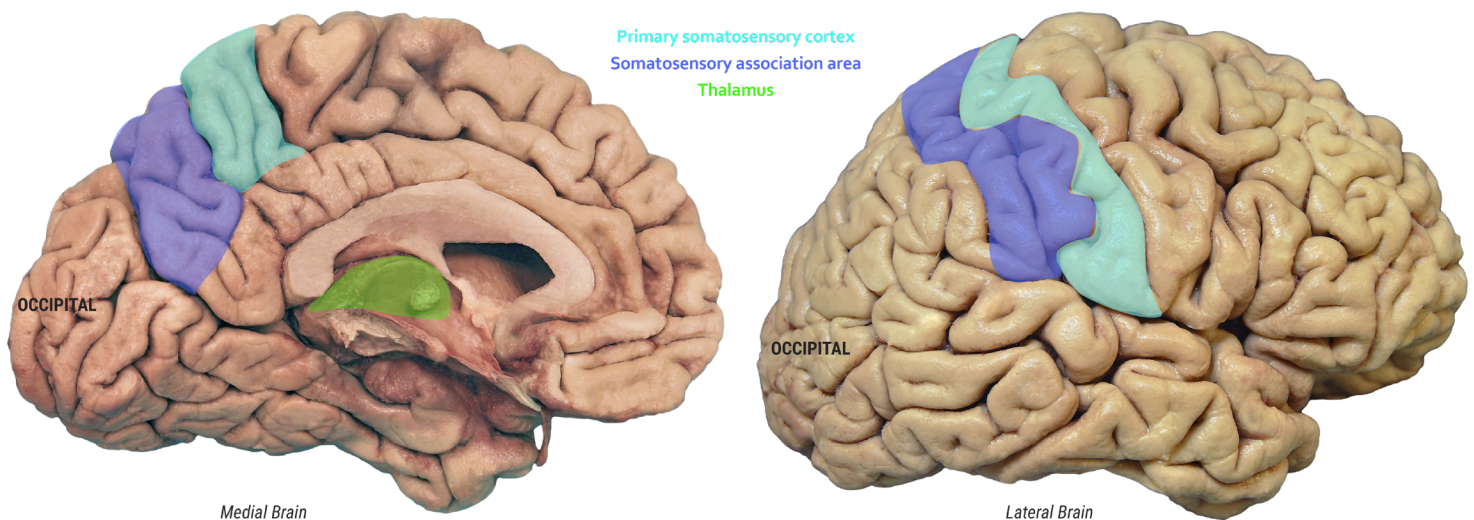
Plays a huge role in regulating which information reaches the cortex – only what is important makes it through. Pain for example will (almost) always be prioritized. The thalamus is an **active gatekeeper** to the cortex.

### Primary Somatosensory Cortex

Gathers all of the raw data relayed by the thalamus. It's pre-sorted raw data, but still confusing and overwhelming. We need to make sense of it; however, the primary somatosensory cortex can't make sense of the data alone.

### Sensory Association Areas

Help to make sense of the data. They integrate the sensory information about touch, vibration and conscious proprioception with visual and auditory information, with memories, with emotional valence. The association areas help us navigate the world by prioritizing data and putting it into the context of our experience and other input we receive.



### Checklist:

- Thalamus (in half-brains & cross-sections)
- Primary somatosensory cortex
- Somatosensory association areas
- Review the lobes of the brain

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### Motor Input

The motor output from the brain comes through a variety of pathways. We have both **automated** and **voluntary** control of our musculature. We will be going through the various types of motor output both in MEDD 412 and in Year 2. To keep things simple, we will focus on the **corticospinal pathway** today – *the main motor pathway for voluntary motor control of our limb muscles*.

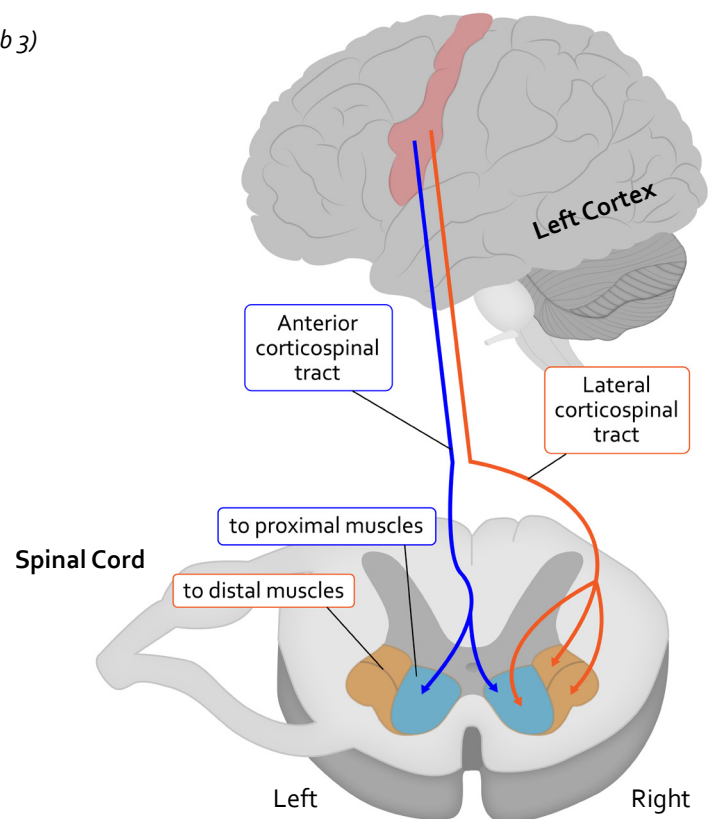
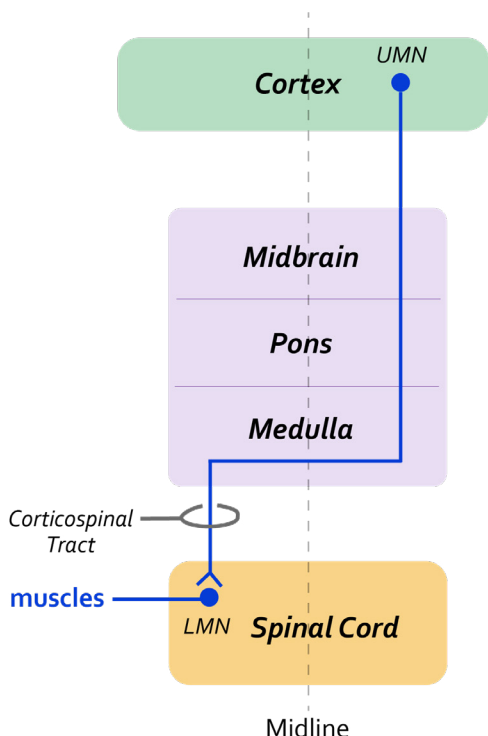
Let's have a closer look at the upper motor neuron that we encountered in the corticospinal tract – it turns out that many of our motor tracts use this principle. Upper motor neurons signal to the lower motor neurons. Lower motor neuron cell bodies are in the CNS (i.e., spinal cord gray matter or brainstem nuclei). Axons of lower motor neurons exit the CNS and form peripheral nerves that will terminate at the motor endplate of a muscle.

### Corticospinal Tract

- Cell bodies of **upper motor neurons (UMNs)** are in the **primary motor cortex**
- Axons of UMNs descend through the internal capsule
- Through the midbrain (cerebral peduncles)
- Through the pons (anterior pons)
- Through the medulla (pyramids)
- Cross the midline (*aka* decussate) in the caudal medulla at the pyramidal decussation
- Descend in the spinal cord to the appropriate spinal level
- Synapse with cell bodies of **lower motor neurons (LMNs)** in the **ventral / anterior horn** of the spinal cord

**2-neuron chain (UMN, LMN)**  
**Fibers cross in caudal medulla**

(We will cover this module in Lab 3)





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### Control of Movement

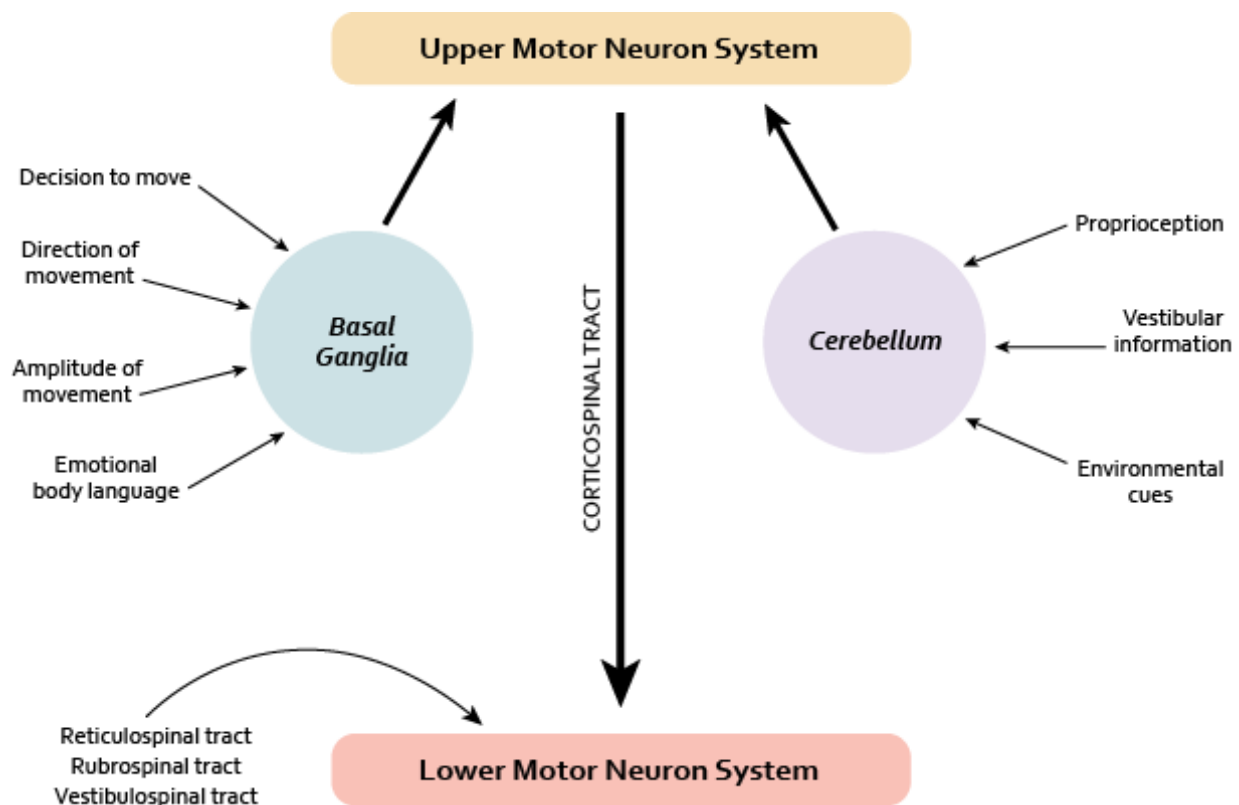
How do the **upper motor neurons** “know” when to signal, how to signal, and why to signal? Upper motor neurons are controlled by many systems in the CNS:

- the **supplementary motor area** is an important association area that helps to generate motor plans and patterns
- the **cerebellum** gives precise signals, adjusts the firing pattern for fine control and coordination, takes care of error control, and it can predict the future (*more to come in MEDD 4.2.1*)
- the **basal ganglia** (*aka* basal nuclei) also influence the firing pattern of the upper motor neuron, they integrate the sum of everything that is on our mind into one motor output, or one behaviour (*more in MEDD 4.2.1*)

Since the corticospinal tract travels through the pyramids, this system is often referred to as the **pyramidal system**, a lesion to this tract is sometimes called a pyramidal lesion.

**The systems controlling the upper motor neuron – including the basal ganglia and the cerebellum – are referred to as the extrapyramidal system.**

These terms are not very precise from a neuroscience perspective, since we know how integrated these systems are, but they are useful clinically since the pyramidal lesion symptoms (usually associated with UMN lesions) are distinct from extrapyramidal symptoms (usually associated with basal ganglia lesions).





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Upper Motor Neuron Lesions (often referred to as Pyramidal clinically)	Lower Motor Neuron Lesions	Basal Ganglia or Cerebellum Lesions (often referred to as Extrapyramidal clinically)
Weakness	Weakness	No weakness
Spastic paralysis	Flaccid paralysis	No paralysis Slowed (hypokinetic) or increased (hyperkinetic) involuntary movement ( <i>Basal Ganglia</i> ) Ataxia ( <i>Cerebellum</i> )
Hypertonia (increased muscle tone)	Hypotonia (decreased muscle tone)	Normal muscle tone ( <i>Basal Ganglia</i> ) Abnormal muscle tone ( <i>Cerebellum</i> )
Hyperreflexia (increased tendon reflexes)	Hyporeflexia (decreased tendon reflexes)	Normal reflexes ( <i>Basal Ganglia</i> ) Abnormal reflexes ( <i>Cerebellum</i> )



### Checklist:

- Supplementary motor area
- Cerebellum
- Basal ganglia (collectively as a group of deep nuclei)
- Brainstem:
  - Cerebral peduncles (midbrain)
  - Anterior pons
  - Pyramids (medulla)
  - Pyramidal decussation (medulla)

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### Introduction to the Cranial Nerves

A cranial nerve is defined as a **peripheral nerve that passes through a hole in the skull**. Not all of the 12 cranial nerves are strictly speaking nerves – for example, the optic nerves are derived from the diencephalon and are therefore technically "tracts" (white matter in the CNS) rather than "nerves" (white matter in the PNS).

It is important to know the function of the 12 cranial nerves, and their associated nuclei in the brainstem. We will be reviewing all of them in more detail in the upcoming labs.

In a **neurological exam** you will test all 12 cranial nerves. This is very important for a couple of reasons: for one, they innervate structures in the head and neck and a clinical exam will reveal any deficits in these important areas. Most cranial nerves have their nuclei in the brainstem. The tracts we talked about in objectives 2 & 3 also travel through the brainstem and a deficit seen in one of the tracts can indicate a problem in the brainstem, but it's hard to know where exactly – this is where the cranial nerves will be helpful: the combination of a deficit associated with a tract and a deficit associated with the cranial nerves will help you pinpoint where the lesion is. This is so important, because even a small lesion in the brainstem can have devastating consequences and every clinician needs to be able to put clinical symptoms together for a rapid and precise referral.

*Today we will look at this general overview of the 12 cranial nerves and then add to this over the next labs.*

### Where to find the cranial nerves:

Region	Cranial Nerve	Localization
<b>Inferior surface of brain</b>	CN I	bipolar olfactory receptor cells, olfactory bulb, olfactory tract
	CN II	optic nerve, optic chiasm, optic tract
<b>Midbrain</b>	CN III	emerges anteriorly in interpeduncular fossa, on medial side of cerebellar peduncles
	CN IV	emerges posteriorly, just caudal to inferior colliculus
<b>Pons</b>	CN V	attached to basal pons laterally at junction with middle cerebellar peduncle
	CN VI	in groove between medulla and pons medially
	CN VII	in groove between medulla and pons laterally (i.e., cerebellopontine angle)
	CN VIII	in groove between medulla and pons laterally (i.e., cerebellopontine angle)
<b>Medulla</b>	CN IX	emerge from medulla, posterior to the olive
	CN X	
	CN XI*	multiple rootlets emerge laterally along the cervical spinal cord
	CN XII	emerges from medulla, between the olive and pyramid

*\*This is a weird one – we will discuss further in class!*

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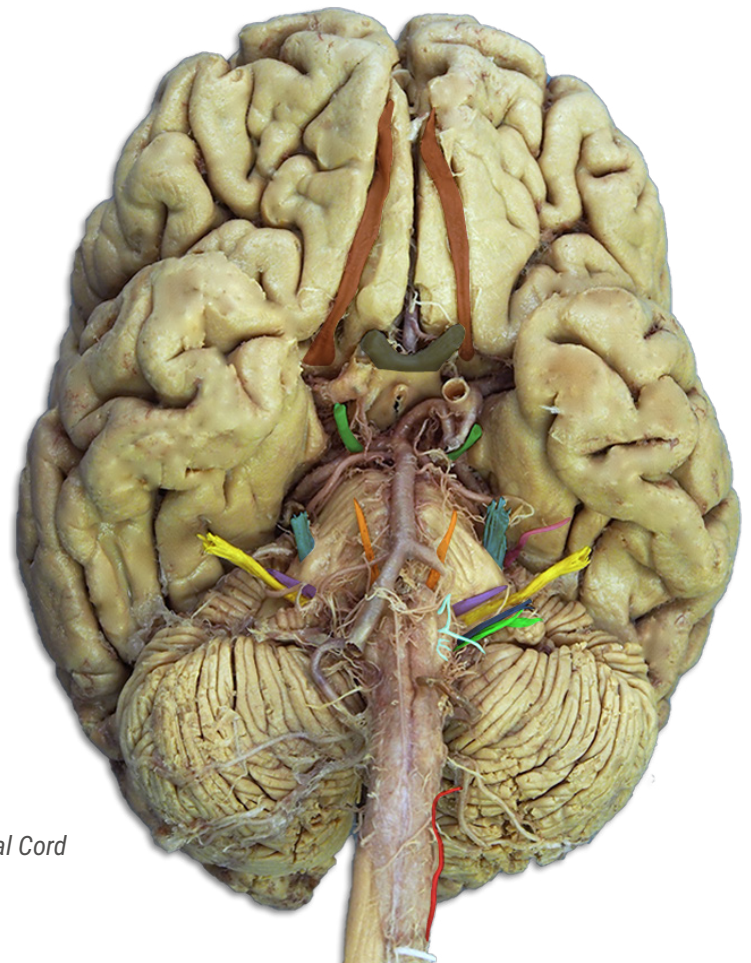
## *Important Surface Markings and Cranial Nerves*

*Anterior View*

*Posterior View*

*Anterior Brainstem*

*Posterior Brainstem*



*Ventral (Inferior) View of Brain and Spinal Cord*

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### Cranial Nerve Functions

Again, this is an overview – we will be reviewing all of the cranial nerves in more detail in upcoming labs.

Cranial Nerve	Function	Evaluation
CN I <i>Olfactory</i>	Smell	Not routinely evaluated
CN II <i>Optic</i>	Vision, pupillary light reflexes	Pupillary light reflexes (afferent), visual acuity
CN III <i>Oculomotor</i>	Parasympathetic to pupil, motor to most extraocular muscles (medial / superior / inferior rectus, inferior oblique)	Pupillary light reflexes (efferent), eye movements
CN IV <i>Trochlear</i>	Motor to superior oblique muscle	Eye movements
CN V <i>Trigeminal</i>	Sensory to face, motor to muscles of mastication	Sensation to face, muscles of mastication
CN VI <i>Abducens</i>	Motor to lateral rectus muscle	Eye movements
CN VII <i>Facial</i>	Motor to muscles of facial expression, parasympathetic to lacrimal gland, taste	Blink, various facial movements, tear production
CN VIII <i>Vestibulocochlear</i>	Balance, hearing	Body posture, eye movements, hearing, vestibulo-ocular reflex
CN IX <i>Glossopharyngeal</i>	Sensory & motor to pharynx	Gag reflex, swallowing
CN X <i>Vagus</i>	Parasympathetic to viscera, sensory & motor to pharynx	Gag reflex, swallowing, palatal elevation
CN XI <i>Accessory</i>	Motor to trapezius & sternocleidomastoid muscles	Shoulder / neck muscle tone, mass & movement
CN XII <i>Hypoglossal</i>	Motor to tongue muscles	Tongue movement & tongue bulk

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### RESOURCES

#### Websites:

Neuroanatomy | Entrada

#### Recommended Textbooks:

##### Lippincott Illustrated Reviews: Neuroscience

By: Claudia Krebs, Joanne Weinberg, Elizabeth J. Akesson, Esma Dilli

Lippincott Williams & Wilkins

ISBN 978-1-4963-6789-1

##### Neuroanatomy Through Clinical Cases

By: Hal Blumenfeld

Sinauer

ISBN 978-0-8789-3613-7

##### Neuroanatomy in Clinical Context: An Atlas of Structures, Sections, Systems, and Syndromes

By: Duane E. Haines

Wolters Kluwer Health

ISBN 978-1-4511-8625-3

### ACKNOWLEDGEMENTS

#### Artwork & Design:

The HIVE, UBC Faculty of Arts

Instructional Design: Monika Fejtek

Medical Illustration Lead: Paige Blumer

Academic Lead: Claudia Krebs

Prosector: Lien Vo

Content Review: Esma Dilli

