1 PYRAMIDS - CORTICOSPINAL FIBERS

The **pyramids** are two elongated swellings on the ventral aspect of the medulla. Each pyramid contains approximately 1,000,000 **CORTICOSPINAL AXONS**. As the name suggests, these axons arise from the cerebral cortex and **descend** to terminate within the spinal cord. The cortical cells that give rise to corticospinal axons are called **Betz** cells. As corticospinal axons descend from the cortex, they course through the internal capsule, the cerebral peduncle of the midbrain, and the ventral pons (you will learn about these structures later in the course so don't worry about them now) and onto the ventral surface of the medulla as the pyramids (see below).



At the most caudal pole of the pyramids the corticospinal axons cross over the midline and now continue their descent on the contralateral (to the cell of origin) side. This crossover point is called the **PYRAMIDAL DECUSSATION**. The crossing fibers enter the **lateral funculus** of the spinal cord where they are called the **LATERAL CORTICOSPINAL TRACT** ("corticospinal" is not good enough, you have to call them **lateral** corticospinal; LCST - remember this one??). LCST axons exit the tract to terminate upon neurons in the spinal cord gray matter along its entire length. Some of the spinal cord neurons that receive direct input from lateral corticospinal fibers send axons into the ventral root to innervate striated muscles that move the arms and legs. In such instances only **two** neurons and one synapse are involved (monosynaptic). Lateral corticospinal tract axons also end upon spinal cord neurons that do not directly innervate muscle, but instead synapse upon lower motor neurons. Such cells are called **interneurons**. A pathway involving a lateral corticospinal axon, an interneuron and a lower motor neuron is **disynaptic**.

Let's review! The cells of origin of the corticospinal tract lie in the **ipsilateral** cerebral cortex. In contrast, the cells of origin of the **lateral** corticospinal tract lie in the **contralateral** cerebral cortex. **REMEMBER**—the lateral corticospinal tract is the part of the corticospinal system in the spinal cord. It is important to remember that damage to the corticospinal fibers **rostral** to the pyramidal decussation results in **contralateral** motor deficits, while lesions in the spinal cord (i.e., **caudal** to the decussation) result in **ipsilateral** deficits. Clinicians refer to the motor weakness resulting from a lesion of the corticospinal tract as **hemiplegia** (plegia =stroke or paralysis) or **hemiparesis** (weakness). For instance, they would say that a lesion of the LEFT corticospinal tract at C₁ (in the spinal cord and therefore caudal to the decussation) results in **LEFT** hemiplegia. Since the motor neurons that directly innervated the muscle are OK, there is no muscle atrophy. This is important.

A unilateral lesion of the **lateral corticospinal tract** results in motor deficits **ipsilateral** to the lesion. Interrupting the tract does **NOT** result in muscle atrophy, since the neurons innervating muscle are **NOT** dead. For example, if the **LEFT** lateral corticospinal tract is interrupted at C_1 , then the **LEFT** arm, trunk and leg are affected. This is because the lateral corticospinal tract influences the musculature on the same (ipsilateral) side of the body. If the lesion involves the **LEFT** lateral corticospinal tract at T₃, then only the **LEFT** trunk and leg are affected, since the spinal cord motor neurons that innervate the muscles of the arms still receive corticospinal input.

As mentioned above, the term **hemiplegia** is commonly used by clinicians when discussing the effects of lesions of the **corticospinal tract** anywhere from the cortex to the medulla, and of the **lateral corticospinal tract** anywhere in the spinal cord. Cerebrovascular accidents ("strokes") commonly damage the corticospinal tract in the motor cortex or the posterior limb of the internal capsule (a compact bundle of axons through which almost all neural traffic to and from the cortex passes). The term "stroke" is poorly defined and has different meanings to different users. It generally implies the abrupt onset of neurological deficits as the result of cerebrovascular disease. As such, it includes the manifestations of cerebral **hemorrhage** (Gr. blood bursting forth; may be arterial, venus or capillary) cerebral **infarction** (L. to stuff into; area of tissue undergoes necrosis [death] following cessation of blood supply) and intracranial and extracranial **thrombosis** (Gr. clot condition; when a thrombus is detached from its original site and found in another site it is called a thrombotic **embolis** [Gr. embole=throwing in]).

As you have already learned, immediately following a lesion involving corticospinal or lateral corticospinal fibers, there is a period of flaccid paralysis (**spinal shock**). After a period of days to weeks, muscle tone (spasticity) and muscle reflexes <u>return</u> and <u>increase</u>. There will also be a **Babinski sign**, (extension of the big toe and fanning of the others in response to firmly stroking the sole of the foot; **"the plantar reflexes are extensor"**). There will also be clonus.



You need to keep in mind that lesions of the corticospinal tract do not destroy motor neurons that DIRECTLY innervate muscle. **THUS, THERE IS NO DEATH OR ATROPHY OF MUSCLE.** Corticospinal tract neurons are referred to as **UPPER MOTOR NEURONS** since they do not innervate muscle **directly**. In contrast, neurons in the ventral horn that **DIRECTLY** innervate muscle are called **LOWER MOTOR NEURONS** (LMN). When these neurons die there **is ATROPHY OF THE MUSCLE**.

In my humble opinion, the corticospinal tract is the most important pathway in clinical neurology. I will ask you about this tract every time I see you, even after you graduate! In other words, if you forget any neuroanatomy, **DO NOT** forget the following points:

- 1. the pathway is **crossed**.
- 2. lesions anywhere **rostral** to the decussation (cortex, internal capsule, cerebral peduncle, basilar pons, rostral two-thirds of pyramid) result in **contralateral** deficits.
- 3. lesions involving this system in the spinal cord (i.e., the lateral corticospinal tract) result in **ipsilateral** deficits **below** the level of the lesion.
- 4. corticospinal neurons are upper motor neurons; and therefore their death does **NOT** result in muscle atrophy.

PROBLEM SOLVING MATCHING

Match the best choice in the right hand column with the pathway or cell group in the left hand column

____1. <u>left</u> pyramid

- A. lesion results in a left Babinski
- B. lesion results in <u>left</u> hemiplegia
- C. cells of origin of axons lie in <u>right</u> motor cortex
- D. axons arise from cells in the left motor cortex
- E. axons terminate on the left side of the spinal cord

Brain stem Pyramids/Corticospinal Tract Problem Solving

PROBLEM SOLVING



Shade in the location of a single, continuous, unilateral (limited to one side) lesion in the above drawing that will account for the following neurological problems:

right hemiplegia, right Babinski

PROBLEM SOLVING ANSWER





2 ANTEROLATERAL SYSTEM (ALS)

Hopefully you remember from the spinal cord module that the cells of origin of **pain** and **temperature** conveying axons in the spinal cord lie in the **dorsal horn**. Axons arising from these dorsal horn cells cross and ascend in the anterolateral portion of the white matter of the spinal cord (hence the name **Anterolateral System**; **ALS**). Thus the cells of origin of the ALS (or spinothalamic tract) lie in the **contralateral** dorsal horn. Axons in the **ALS** are destined for the **ventral posterolateral (VPL) nucleus** of the thalamus. Since the thalamus lies **ROSTRAL** to the midbrain, which is the most rostral part of the brain stem, the **ALS** is present in each of the 10 brain stem slides that you need to learn.

Once the pain and temperature information traveling in the ALS reaches the VPL nucleus of the thalamus (the thalamus is the **GREAT GATEWAY TO THE CORTEX**), it is relayed by a thalamic neuron to the somatosensory cortex (postcentral gyrus; areas 3, 1 and 2). Don't forget: pain and temperature information reaches cells in the dorsal horn via the central processes of dorsal root ganglion cells (delta and C fibers; neuron #1). Dorsal horn cells (neuron #2) project to the contralateral VPL via the ALS. Finally, cells in VPL (neuron #3) project to areas 3, 1 and 2 (somatosensory cortex) for perception of the pain and temperature.

Interruption of the ALS anywhere in the brain stem results in loss of pain and temperature from the **contralateral** side of the <u>body</u> (arms, trunk and legs), but **NOT** the head. We will deal with the head later!

The location of the **ALS** is difficult to see in fiber-stained sections, since most of these axons are either lightly myelinated or non-myelinated (i.e., slowly conducting). You must focus hard on remembering the location of the anterolateral fibers in each of the ten brain stem levels. I will include this system in many of the problem-solving questions in order to help you remember its location. **YOU SHOULD NEVER FORGET THE ALS/SPINOTHALAMIC TRACT**!







- Conveys pain and temperature from the body.
- 1. Cells of origin-neurons in the contralateral dorsal horn.
- 2. Lesions result in contralateral deficits in pain and temperature from the body.
- Axons pass rostrally through the brainstem to reach the ventral posterolateral nucleus of the thalamus (VPL). Neurons in VPL project to the somatosensory cortex (areas 3, 1, 2).



AN INTERESTING CLINICAL **OBSERVATION**

Clinical case reports often comment that lesions involving areas of the brain stem adjacent to the anterolateral system result

in an **IPSILATERAL HORNER'S SYNDROME**. The explanation for this finding is that descending fibers from the **HYPOTHALAMUS** (a major autonomic center) travel close to the ALS in the brain stem (you will not see these descending fibers in the ten brain stem levels). Their

interruption means that the PREGANGLIONIC SYMPATHETIC neurons in the lateral cell column (T_1-L_2) of the spinal cord have lost an important "drive." The most obvious clinical finding would be a **CONSTRICTED PUPIL** (**MIOSIS**) in the ipsilateral eye since the parasympathetic input is now in control. There would also be slight drooping (**PTOSIS**) of the ipsilateral upper eyelid due to the absence of sympathetic "drive" to the superior tarsal (smooth) muscle. There is also lack of sweating (anhidrosis) and vasodilatation (flushed face). Remember, sympathetics innervate sweat glands and constrict blood vessels of the face. All problems are **IPSI** to the brain stem lesion. For our problem solving excercises A HORNER'S WILL BE PRESENT ANY TIME THE LESION INVOLVES THE ALS IN THE BRAIN STEM OR THE AREA OF THE MEDIAL LCST IN THE SPINAL CORD **ABOVE T2! REMEMBER, THE PROBLEMS RELATED TO HORNER'S WILL BE IPSI.**

WILL BE CONTRA.



PROBLEM SOLVING MATCHING

Match the best choice in the right hand column with the pathway or cell group in the left hand column.

- ____1. <u>left</u> pyramid
- _____2. <u>right</u> anterolateral system (ALS) and associated descending pathway
- A. cells of origin lie in the <u>left</u> dorsal root ganglia
- B. axons terminate in the <u>left</u> motor cortex
- C. lesion results in a loss of pain and temp from the <u>right</u> side of the body
- D. lesion results in a left Babinski
- E. cells of origin lie in the <u>left</u> dorsal horn
- F. cells of origin lie in the <u>right</u> motor cortex
- G. lesion results in <u>right</u> hemiplegia
- H. axons terminate on the left side of the spinal cord
- I. lesion results in a <u>dilated</u> pupil in the <u>right</u> eye



PROBLEM SOLVING



RIGHT LEFT

Shade in the location of a single, continuous, unilateral lesion in the above drawing that will account for the following neurological problems:

<u>right</u> hemiplegia, <u>right</u> Babinski, loss of pain and temperature from the <u>right</u> arm and leg, constricted pupil in the <u>left</u> eye

PROBLEM SOLVING ANSWER



RIGHT LEFT

3 SPINAL NUCLEUS AND TRACT OF THE TRIGEMINAL (C.N. V)

The trigeminal nerve is the largest cranial nerve. It contains both sensory and motor fibers. The motor fibers innervate the muscles of mastication and arise from motor neurons in the pons. The general sensory fibers convey information regarding pain, temperature, touch, and conscious and unconscious proprioception. At this time I want to talk about those general sensory fibers that are conveying **PAIN** and **TEMPERATURE** because they are associated with a nucleus and tract in the caudal medulla (where we are now!). We will talk about the other general sensory nuclei and fibers (two point discrimination, conscious proprioception and vibratory sense), as well as the motor fibers associated with the trigeminal, later when we look at sections through the rostral pons.

The cell bodies of the pain and temperature fibers associated with the trigeminal nerve lie in the trigeminal ganglion (located on the cerebral surface of the sphenoid bone in the middle cranial fossa). Just like cells within the dorsal root ganglia of the spinal cord, cells in the trigeminal ganglion possess peripheral and central processes. The peripheral processes of trigeminal ganglion neurons distribute to pain and temperature receptors on the **face**, forehead, mucous membranes of the nose, anterior two-thirds of the tongue, hard and soft palates, nasal cavities, oral cavity, teeth, and portions of cranial dura. The central processes enter the brain at the level of the pons (this is where all trigeminal sensory fibers enter the brain stem and where trigeminal motor fibers leave the brain stem). These central processes of trigeminal ganglion neurons conveying pain and temperature descend in the brain stem and comprise the **SPINAL TRACT V**. Fibers of spinal tract V terminate upon an adjacent cell group called the **SPINAL NUCLEUS V**, which forms a long cell column medial to spinal tract V (spinal tract and nucleus V form a slight elevation on the lateral surface of the caudal medulla called the **tuberculum** [swelling] **cinereum** [ashen or gray]). We are particularly concerned with the caudal-most portion of spinal nucleus V, because ALL of the PAIN and **TEMPERATURE** fibers from the face terminate in this caudal region of the nucleus (other portions of spinal nucleus V will not be discussed). Cells within spinal nucleus V possess axons that curve medially to **CROSS** the midline and course rostrally close to (but not as a part of) the medial lemniscus. These crossed fibers retain their close

association with the medial lemniscus as they ascend in the brain stem and are called the **trigeminothalamic tract** (TTT). Neither the crossing or ascending TTT fibers can be identified in our fiber-stained levels, since they are only lightly myelinated. Fibers in the **TTT** reach the **GREAT GATEWAY TO THE CORTEX**, the thalamus, where they terminate in the **ventral posteromedial** (VPM) nucleus. Neurons in the VPM then project to **somatosensory cortex** (areas 3, 1, 2; postcentral gyrus).



Brain stem Spinal Nucleus and Tract V





This diagram shows the middle cranial fossa and the posterior cranial fossa (on the left). Note the large trigeminal ganglion, and the three branches of the nerve (peripheral processes of cells in the trigeminal ganglion) exiting via the superior orbital fissure (ophthalmic) foramen rotundum (maxillary) and foramen ovale (mandibular). The central processes of cell bodies in the trigeminal ganglion enter the pons.

KNOW THE LOCATION OF THE SENSORY AND MOTOR ROOTS OF CN V



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TT

Cortical areas 3,1,2 Representation of trunk bödy on arunk arm digits face lips teeth tongu Postcentral Gyrus entral Conveys pain and temperature sulcus ngue Anterior Posterior information from the head Lateral sulcus 1. Cells of origin of spinal tract V lie in the To areas 3,1,2 of cerebral cortex ipsilateral trigeminal ganglion. 2. Pain and temperature fibers in spinal tract V terminate in caudal spinal nucleus V. 3. Axons of neurons in spinal nucleus V cross the VPM midline and ascend in the trigeminothalamic tract (TTT) to reach the ventral posteromedial (VPM) nucleus of the thalamus. V P M neurons project to areas 3, 1, and 2 (somatosensory cortex). LEVEL 13 Trigeminothalamic 4. There are three neurons in the pathway from tract (TTT) face to cortex; (a) trigeminal ganglion, (b) spinal nucleus V, and (c) VPM. 5. Lesions in the spinal tract result in IPSILATERAL deficits in pain and temperature from the face etc., palate and anterior two-thirds of the tongue. LEVEL 10 -Ophthalmic 6. Lesions of the trigeminothalamic tract result in Maxillary (TTT)CONTRALATERAL deficits in pain and temperature landibula from the area innervated by the trigeminal. to somatosensory cortex Trigeminal LEVEL 6 CN ' Spinal tract V Spinal nuc. trigeminal ganglion LEVEL 5 Spinal tract V Spinal nuc. V (C LEVEL 3 Trigeminothalamic tract (TTT) spinal tract V Spinal tract V spinal nucleus V Spinal nucleus V LEVEL 1

Spinal Tract and Nucleus of the Trigeminal

I am sorry to say that the spinal tract and spinal nucleus V are not exclusively associated with C.N. V. It is also associated with C.N.s VII (facial), IX (glossopharyngeal) and X (vagus). While this makes the story a little confusing, it also makes a lot of sense! The pain and temperature fibers associated with C.N. VII innervate the skin of the external ear, the wall of the external auditory meatus and the outer surface of the tympanic membrane (if it's OK with you, I'll lump these together as "EAR"). These fibers are the peripheral processes of cells that lie in the GENICULATE ganglion (located in the facial canal). The central processes of these neurons enter the brain with C.N. VII (at pontine levels, caudal to the trigeminal), travel in spinal tract V and end in spinal nucleus V. The pain and temperature information is then conveyed rostrally in the TTT (trigeminothalamic tract) to reach the VPM, from which it is relayed to somatosensory cortex (areas 3,1 and 2). Thus the pain and temperature fibers of C.N. VII don't have their "own" central cell group, but instead use that of the trigeminal.

Like C.N. VII, C.N. IX is involved in the pain and temperature innervation of the "EAR". In addition, C.N. IX conveys pain and temperature from the **posterior one-third of the tongue**, the auditory tube and the upper part of the pharynx (touching of the pharyngeal wall results in a gag reflex which we will cover in Point 9: Nucleus Ambiguus). The cell bodies of these pain and temperature axons associated with C.N. IX lie in the relatively small **SUPERIOR GANGLION IX** (located just outside of the jugular foramen; you will hear about the larger inferior ganglion later). The central processes of cells in the superior ganglion enter the brain with C.N. IX (at the lateral medulla), then enter our friend spinal tract V and synapse in the caudal spinal nucleus V. You know the route from here.

FINALLY (whew!) C.N. X innervates the "**EAR**" (along with C.N.s VII and IX). In addition, pain and temperature from the **lower pharynx, the larynx and the upper esophagus** are conveyed by C.N. X. The cell bodies of these pain and temperature axons lie in the **SUPERIOR GANGLION X** (located just outside the jugular foramen). The central processes of cells in the superior ganglion X pass into the brain at the lateral medulla, enter our old friend spinal tract V and synapse in the caudal spinal nucleus V. I know you can take it from here up to cortex via the thalamus!





Spinal Tract and Nucleus of the Trigeminal

KEY THOUGHT: only **ONE** ganglion to worry about with C.N. VII (**GENICULATE**). **SUPERIOR** ganglia of **BOTH IX and X=somatic** afferents (inferior ganglia IX and X will be discussed later, but if you are interested, they are related to **visceral** afferents; ahhh, those donuts on Friday during Gross **tasted** great!)

As mentioned above, pain fibers end only in the **caudal spinal nucleus V**. You should also be aware of the fact that not much is known about the other parts of the spinal trigeminal nucleus (nucleus oralis and interpolaris). The important fact is that the great majority of the pain and temperature fibers pass to the most **caudal** portion of spinal nucleus V. This means that lesions anywhere along the rostrocaudal extent of the spinal tract V, as well as in the caudal spinal itself, will result in deficits in pain and temperature from the ipsilateral face etc. Finally, you have probably heard of **tic douloureux or trigeminal neuralgia**, which is a disorder characterized by brief attacks of severe pain within the distribution of one or more divisions of the trigeminal nerve. Sometimes, section of the spinal nucleus and tract V in the caudal medulla relieves these symptoms.

For our **PROBLEM SOLVING**, you must know that a lesion of **spinal tract V** along its entire course (the pain information never gets to the caudal spinal nucleus) results in **IPSILATERAL** deficits in **pain** and **temperature** from the face etc. Interruption of the **trigeminothalamic tract**, which is comprised of axons that have **crossed** the midline, results in deficits in **pain** and **temperature** on the **contralateral** side of the face etc.

REMEMBER:

1. spinal tract and nucleus V are present in the pons and medulla

- 2. the cells of origin of spinal tract V lie in the trigeminal ganglion
- 3. axons in spinal tract V terminate in the spinal nucleus V
- 4. axons of cells within spinal nucleus V project to the contralateral VPM

5. spinal tract and nucleus V = pain and temp. from face etc. (ear, tongue, palates, pharynx, larynx, upper esophagus)

AN INTERESTING CLINICAL OBSERVATION

Reports in the clinical literature note that vascular lesions that interrupt the blood supply to the spinal nucleus and tract V in the medulla (for example a thrombosis of the posterior inferior cerebellar artery) sometimes are immediately followed by sharp stabbing **pain** in and around the eye and on the ipsilateral face (hyperalgesia; algesia; Gr., = sense of pain). A possible explanation for this paradox is that the pain fibers are highly **irritated** before they die (spontaneous pain also sometimes occurs on the **contralateral** side of the body immediately following a lesion of the anterolateral system). **Please remember that lesions of spinal tract and nucleus V can result in PAIN in the face** (in addition to loss of pain and temperature later). What a paradox!

PROBLEM SOLVING



Shade in the location of a single, continuous, **unilateral** lesion in the above drawing that will account for the following neurological problems:

<u>left</u> hemiplegia, <u>left</u> Babinski, loss of pain and temperature from the <u>right</u> arm and leg, sharp pain in the <u>left</u> eye followed later by loss of pain and temperature from the <u>left</u> side of the face, constricted pupil in the <u>left</u> eye

PROBLEM SOLVING MATCHING

Match the best choice in the right hand column with the pathway or cell group in the left hand column

____1. <u>right</u> pyramid

2. <u>right</u> anterolateral system (ALS) and associated descending pathways

____3. <u>right</u> caudal spinal nucleus V

A. axons terminate on <u>right</u> side of the spinal cord B lesion results in a <u>left</u> Babinski

- C. lesion results in the loss of pain and temp from the <u>right</u> side of the body
- D. lesion results in the loss of pain and temp from the <u>right</u> side of the face
- E. cells of origin lie in the <u>right</u> dorsal horn
- F. cells of origin lie in the <u>left</u> trigeminal ganglion
- G. lesion results in <u>right</u> hemiplegia
- H. lesion results in a constricted pupil in the <u>right</u> eye
- I. axons arise from cells in the <u>left</u> motor cortex
- J. cells project to the right VPM

PROBLEM SOLVING ANSWER

