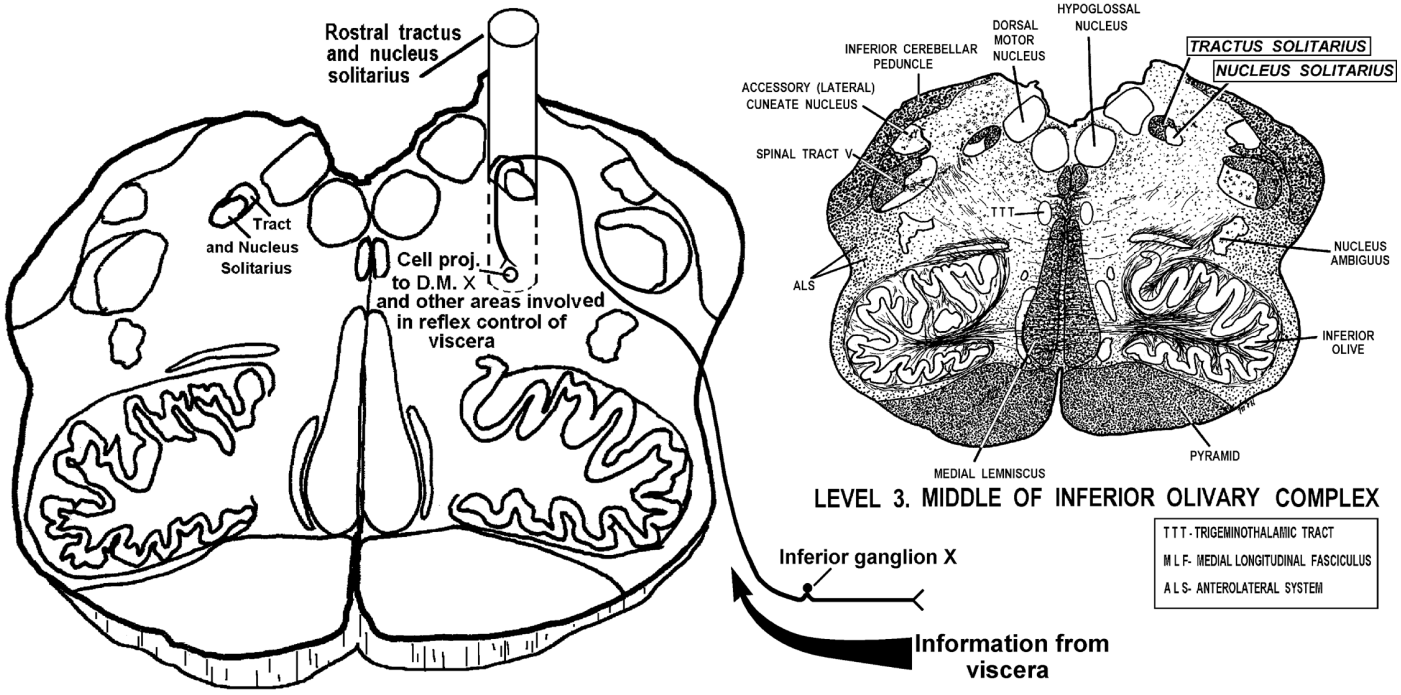
11 NUCLEUS AND TRACTUS SOLITARIUS (VISCEROSENSORY)

The cell group in the brain stem that receives VISCEROsensory information is called NUCLEUS SOLITARIUS. This sad, little, lonely nucleus lies lateral to the dorsal motor nucleus X (pregang. parasym. of C.N. X), and next to (and sometimes surrounds, but this is hard to see in our sections) the solitary fasciculus or tract (somewhat similar arrangement as spinal nuc. and tract V eh?). Only a portion of the entire rostrocaudal axis of the solitary complex can be seen in our series of 10 brain stem sections, and this is on level #3. In order to show the rostral and caudal divisions of the solitary complex (which differ functionally), I have taken some artistic license in the drawing below by schematically illustrating the solitary complex as a column extending above (rostral part) and below (caudal part) level #3. I hope this is not confusing.

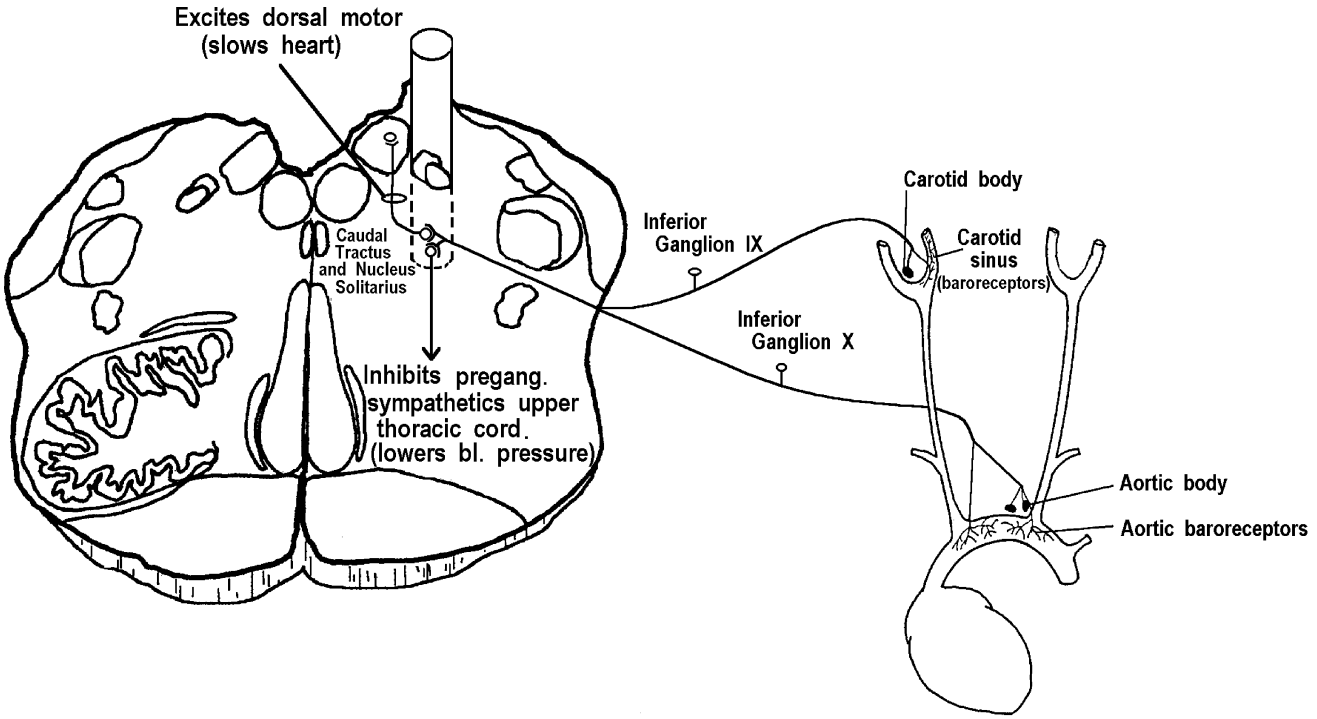
VISCEROSENSORY information reaches the medulla, primarily (but not exclusively) via C.N. X (vagus; the wanderer). We are generally not aware of the viscerosensory information conveyed by the vagus. Most of these messages are related to the status of the viscera (for example, information from receptors in the walls of the viscera, including the entire digestive system to the middle of the transverse colon, and in the respiratory system from the larynx to the pulmonary air sacs of the lung).

The cells of origin of vagal fibers that convey messages from the viscera lie in the INFERIOR GANGLION of X. The peripheral processes of these neurons pass to viscera, while the central processes pass into the medulla to comprise FASCICULUS SOLITARIUS and synapse in the adjacent NUCLEUS SOLITARIUS. As I have mentioned above, the solitary complex is functionally divided into CAUDAL and ROSTRAL portions, and the viscerosensory information that we are talking about now synapses within the CAUDAL NUCLEUS SOLITARIUS. Neurons in the CAUDAL part of nucleus solitarius possess axons that convey information about the status of the viscera to many areas of the brain that are involved in the reflex control of the viscera. For instance, information is sent from the caudal nucleus solitarius to the dorsal motor nucleus X (preganglionic parasympathetic; increases peristalsis) and to the lateral cell column of the spinal cord (preganglionic sympathetic; decreases peristalsis). Messages are also sent to respiratory centers in the brain stem (we will not cover these areas in this course, but you will learn them in Physiology).



VISCEROSENSORY information regarding **blood pressure** is conveyed via both C.Ns. IX and X. For example, information from the **carotid sinus** travels over C.N. IX (cell bodies are in **INFERIOR GANGLION IX**). The carotid sinus is a region near the bifurcation of the internal and external carotids. In this area the wall of the artery is thinner and contains a large number of branching, vine-like endings of C.N. IX. This area serves as a **pressure receptor** (baroreceptor; baros=weight). An **increase** in arterial pressure **increases** the rate of impulses in the fibers of C.N. IX that innervate the carotid sinus, above the baseline ("normal") number of impulses, and this information passes into **caudal** nucleus solitarius. This results in more impulses being sent from **excitatory** neurons in nucleus solitarius to the **DORSAL MOTOR X** (C.N. X). This leads to an increase in the number of impulses sent from dorsal motor X to the heart (of course not directly). This will **SLOW** the heart rate. Cells in nucleus solitarius also project to the preganglionic **sympathetic** neurons in the upper thoracic spinal cord. An **increase** in blood pressure in the carotid sinus will lead to an increase in firing of the fibers of C.N. IX that reach caudal nucleus solitarius. This will result in an increase in firing of **inhibitory** neurons in caudal nucleus solitarius that project to preganglionic **sympathetic** neurons in the thoracic cord. This increase in the amount of inhibition reaching the preganglionic sympathetic neurons will lead eventually to reflex **lowering** of the blood pressure. While similar connections and functions are associated with the baroreceptors in the arch of the aorta, **C.N. X** instead of C.N. IX is involved. You should be able to figure out what happens following a **decrease** in blood pressure in the carotid sinus.

For our **PROBLEM SOLVING EXERCISES, WE WILL EQUATE A UNILATERAL LESION OF CAUDAL NUCLEUS SOLITARIUS WITH AN INCREASE IN HEART RATE** (just like **DORSAL MOTOR X**). Loss of excitation of the dorsal motor nucleus X means dorsal motor X is firing **LESS** and loss of inhibition to the sympathetic neurons in the cord, means that they are firing more. This results in sympathetics dominating = heart rate **UP**!



BARORECEPTOR REFLEX

READ ON ONLY IF YOU ARE INTERESTED. NOT ON EXAM!

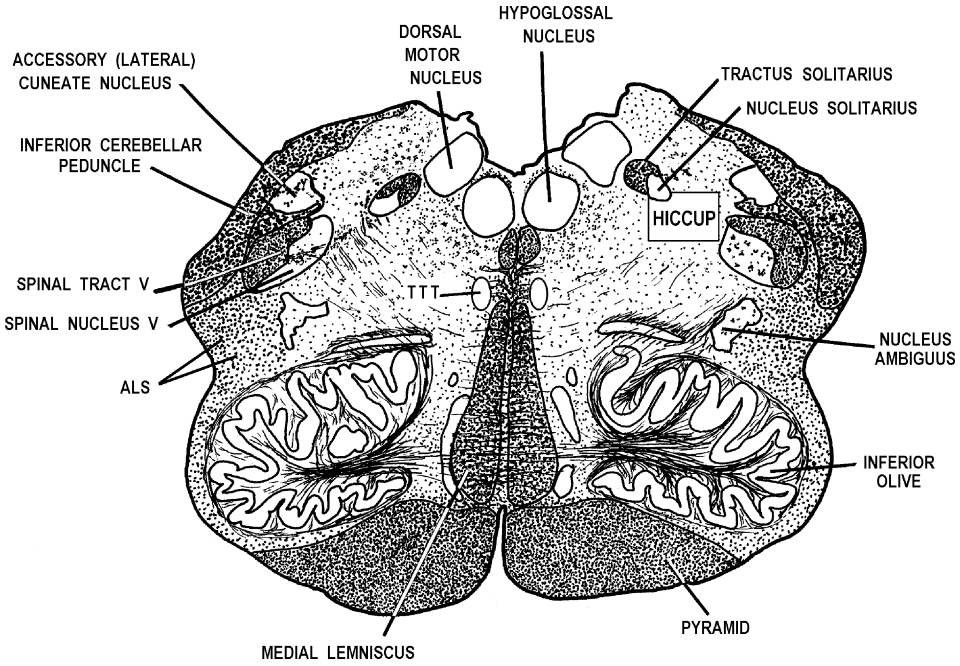
There are **chemoreceptors** in the **carotid and aortic bodies** that affect respiration. The afferent information from the carotid and aortic bodies travel in C.N.s IX (carotid body; cell bodies in **INFERIOR GANGLION** IX) and X (aortic body; cell bodies in **INFERIOR GANGLION** X). The receptors in the carotid and aortic bodies respond to a **decrease** in arterial oxygen tension (P_{O_2}) and an **increase** in arterial carbon dioxide (P_{CO_2}). For instance, an increase in P_{CO_2} will result in an **increase** in the number of impulses traveling over C.N.s IX and X to the caudal nucleus solitarius. Neurons in nucleus solitarius project to the **phrenic nucleus**, which consists of a group of neurons in the ventral horn of the spinal cord from C_3-C_5. Axons arising from the phrenic nucleus comprise the phrenic nerve that innervates the diaphragm. Cells in nucleus solitarius also project to neurons in the spinal cord that innervate the intercostal muscles. Therefore, an **increase** in P_{CO_2} will result in an **increase** in the **depth and rate of breathing**, while a **decrease** in P_{CO_2} will have the opposite effect.

This is **NOT** a course in respiratory or cardiovascular physiology! However, it is extremely important for you to remember that the brain stem, especially the medulla, is an important region for the control of **respiration** and **cardiovascular** functions. **BILATERAL** lesions of the caudal nucleus solitarius will result in major respiratory and cardiovascular problems that result in death.

AN INTERESTING CLINICAL OBSERVATION - KNOW THIS COLD

Clinical case reports mention that lesions of the medulla that involve the area slightly ventral and lateral to nucleus and tractus solitarius result in **HICCUP**. One (of **several**) explanations for this finding is that such a lesion "irritates" descending information from nucleus solitarius to the **phrenic nucleus**. The phrenic nucleus consists of a functionally related group of cell bodies in the ventral horn from C_3-C_5. Axons arising from the phrenic nucleus comprise the phrenic nerve, which innervates the diaphragm. The hiccups result from spasmodic lowering of the diaphragm that causes a short, sharp inspiratory cough.

I want you to remember that brain stem lesions involving the area **ventral and lateral** to nucleus and tractus solitarius (I have cleverly designated it the HICCUP area, but this has not been carefully studied) result in **HICCUP**. **KEEP THIS IN MIND WHEN THE DOING PROBLEM SOLVING EXERCISES**.



TASTE

The **ROSTRAL** portion of the solitary complex is a component of the **TASTE PATHWAY**. The axons within the rostral tractus solitarius are the **central processes** of cells within **THREE** cranial nerve ganglia, the **GENICULATE GANGLION** OF C.N. VII, the **INFERIOR GANGLION** of C.N. IX and the **INFERIOR GANGLION** of C.N. X. These central processes comprise the rostral tractus solitarius and terminate within the **ROSTRAL** or **GUSTATORY** portion of the **nucleus solitarius**. The peripheral processes of these neurons innervate the **TASTE BUDS** of the tongue in the following distribution:

C.N. VII=anterior two-thirds
C.N. IX=posterior one-third
C.N. X=taste buds on epiglottis

Like other ascending sensory pathways, taste information heads for the thalamus (the great **GATEWAY** to the cerebral cortex!), and in particular to the **ventral posteromedial nucleus** (VPM; the nucleus of the **HEAD**!; after all, the tongue is in the head; remember the trigeminal-VPM relationship). The third neuron in the pathway i.e., the thalamic VPM neuron, then sends its axon to the ventral lateral portion of the postcentral gyrus, areas 3, 1, and 2. **UNLIKE OTHER ASCENDING SENSORY PATHWAYS, THE SOLITARIOTHALAMIC TRACT (STT) IS UNCROSSED**, repeat, **UNCROSSED**, repeat, **UNCROSSED**.

SOLITARIOTHALAMIC=UNCROSSED

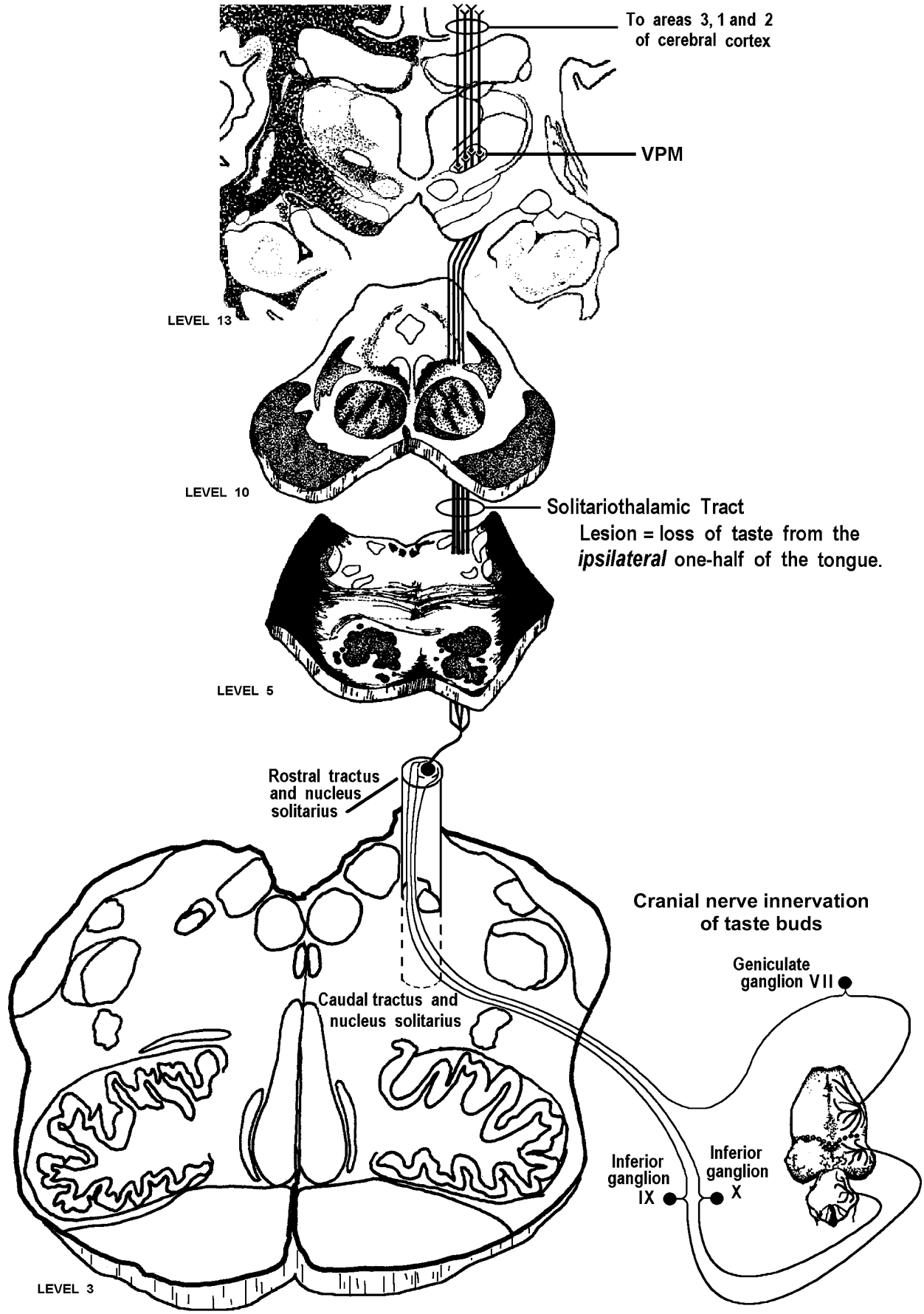
A LESION OF THE ROSTRAL NUCLEUS AND TRACTUS SOLITARIUS WILL RESULT IN THE LOSS OF TASTE FROM THE IPSILATERAL ONE-HALF OF THE TONGUE. SO WILL A LESION OF THE SOLITARIOTHALAMIC TRACT

You should also keep in mind that the interruption of the **solitariothalamic tract** will not result in major problems in respiratory and cardiovascular control, since **most** of the pathways over which the nucleus solitarius controls these functions pass caudally in the brain stem. Let's reserve a loss of **taste** from the ipsilateral side of the tongue for lesions of the solitariothalamic tract. Lesions of the **rostral** nucleus solitarius will also result in loss of taste from the ipsilateral side of the tongue. In contrast, a lesion of the **CAUDAL** portion of nucleus solitarius will result in an **INCREASE IN HEART RATE**.

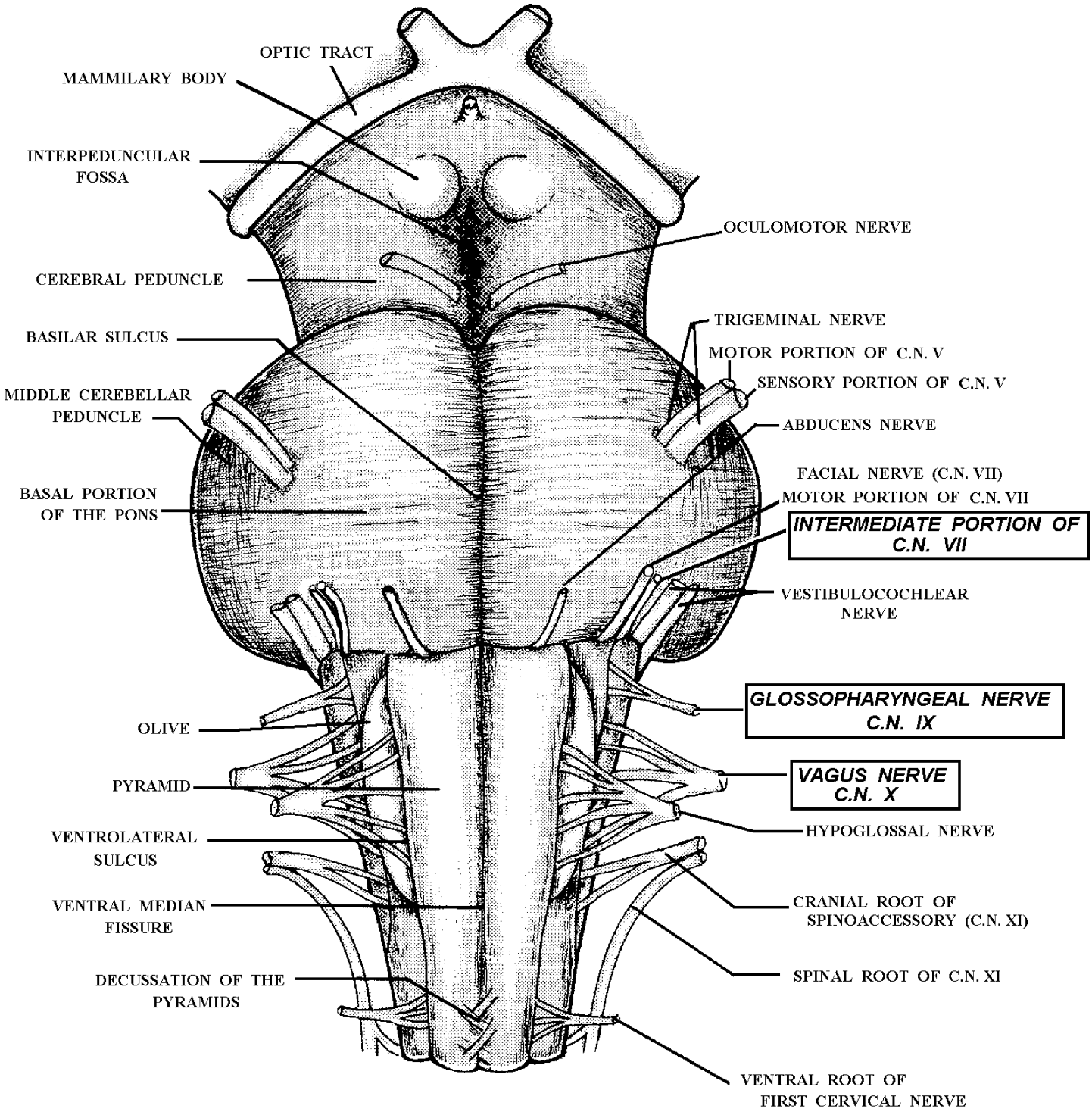
LESION of the ROSTRAL SOLITARIUS=LOSS of IPSILATERAL TASTE

while

LESION of the CAUDAL SOLITARIUS=INCREASE HEART RATE



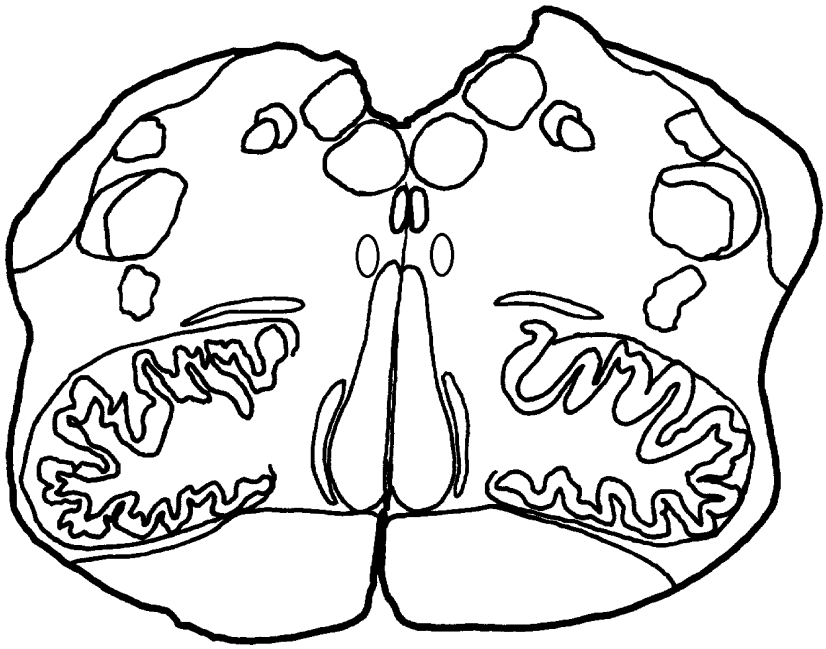
Solitariothalamic tract



VENTRAL VIEW

"TASTE NERVES"

PROBLEM SOLVING

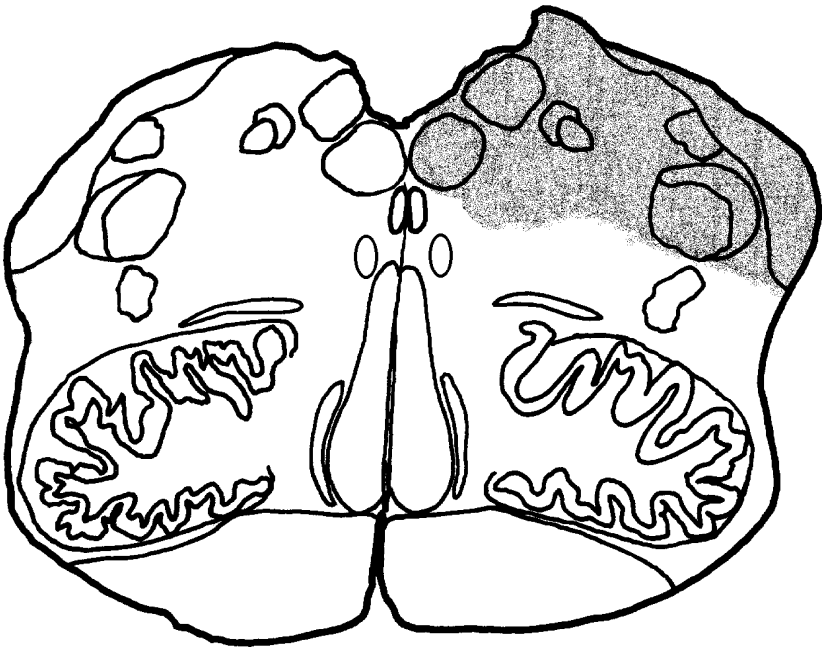


RIGHT LEFT

Shade in the location of a single, continuous, unilateral lesion in the above drawing that will account for the following neurological problems: (consider the part of nucleus and tractus solitarius illustrated to be the ROSTRAL portion)

incoordination of the left arm and leg, loss of taste and pain and temperature from the left side of the tongue, increase in heart rate, fasciculations and atrophy of the muscles on the left side of the tongue, hiccup

PROBLEM SOLVING ANSWER



RIGHT LEFT

PROBLEM SOLVING MATCHING

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

____1. left solitariothalamic tract

____2. left spinal nucleus V

____3. right lateral corticospinal tract

____4. right nucleus ambiguus

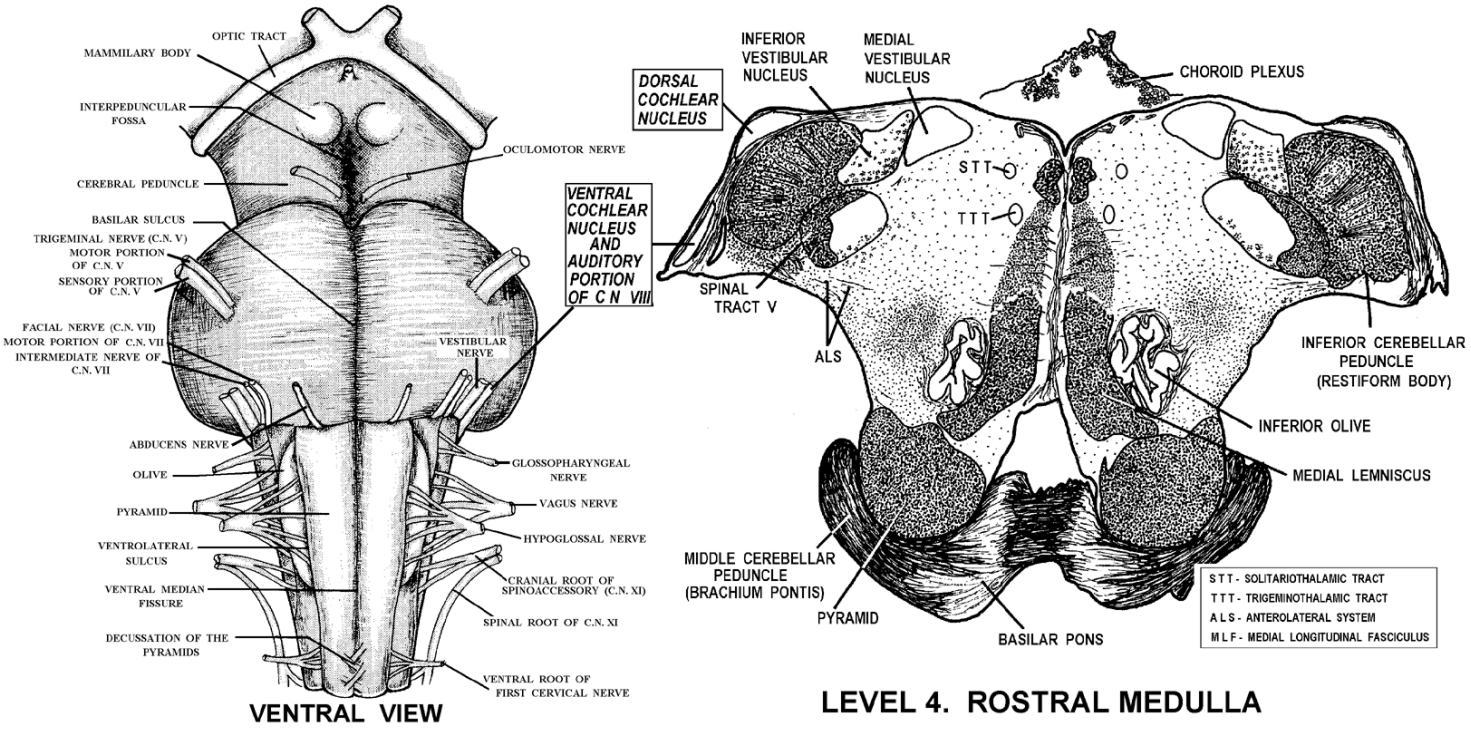
____5. right rostral nucleus solitarius

A. axons arise from cells in the right rostral nucleus solitarius
B. lesion results in the deviation of the uvula to the right
C. lesion results in a loss of taste from the right side of the tongue
D. lesion results in a left Babinski
E. receives input from cells in the right superior ganglion IX
F. terminates in the left VPM
G. receives corticobulbar input from the left motor cortex
H. lesion results in an increased heart rate
I. cells of origin lie in the left motor cortex
J. lesion results in the absence of both direct and consensual gag reflexes upon stimulation of the left side of the pharynx

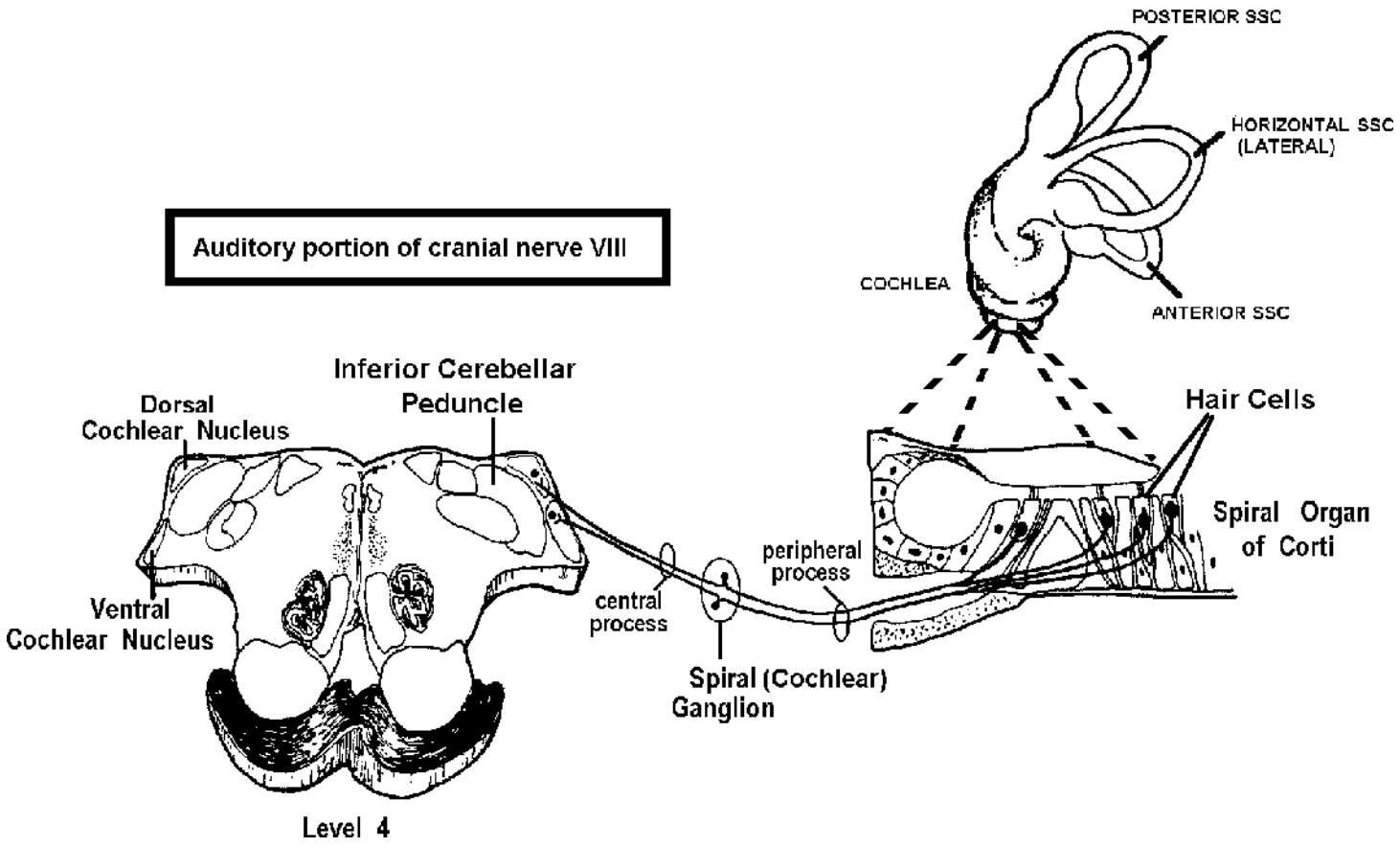
12 DORSAL AND VENTRAL COCHLEAR NUCLEI (A Brief outline of the auditory pathways)

This point contains an extremely cursory outline of auditory pathways. My goal here is to help you identify several auditory nuclei in the brain stem. You will learn more about these pathways later in the course. TRUST ME!!

The two **cochlear** nuclei lie dorsal lateral (dorsal cochlear nucleus) and ventral lateral (ventral cochlear nucleus) to the inferior cerebellar peduncle at the rostral pole of the medulla (they "drape" the inferior cerebellar peduncle). The cell groups related to the other division of C.N. VIII, the **vestibular** nuclei, lie medial to the inferior cerebellar peduncle.

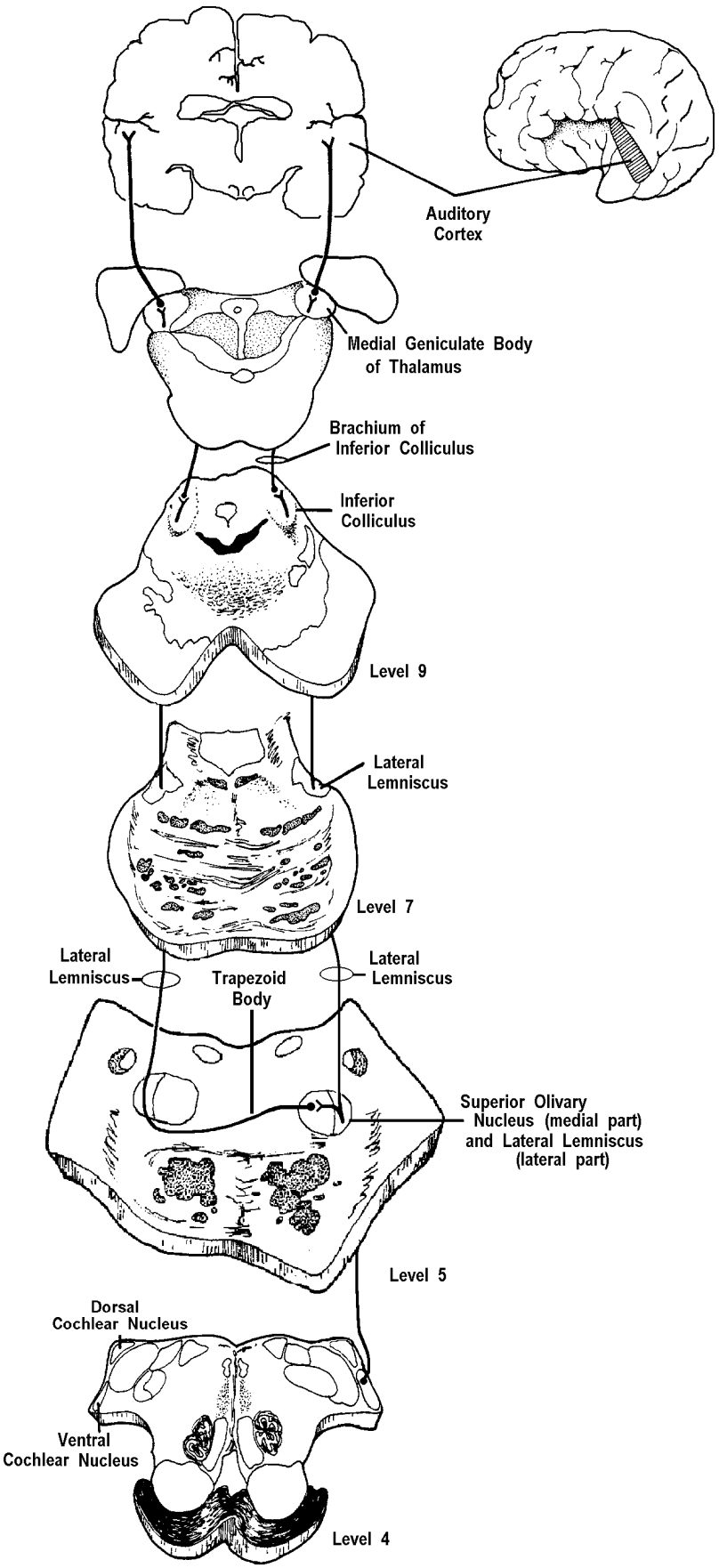


The primary input to both cochlear nuclei is from the auditory portion of C.N. VIII. The axons making up this division of C.N. VIII consist of the central processes of neurons that lie in the spiral or cochlear ganglion (lies in the modiolus [bony core] of the cochlea). The peripheral processes of neurons within the cochlear ganglion end upon the hair cells comprising the organ of Corti. We will not discuss the organ of Corti at this time.



Since you will have a series of lectures on the auditory system later in this course I will give you a very **INCOMPLETE** overview of ascending auditory pathways. Let's trace an ascending pathway from the **ventral** cochlear nucleus. The axon courses rostrally to reach the pons where it travels in the **LATERAL LEMNISCUS**. The axon can travel in the lateral lemniscus until it reaches the **INFERIOR COLLICULUS** (midbrain) where it synapses. Cell in the inferior colliculus project to the **MEDIAL GENICULATE BODY** of the thalamus via the **BRACHIUM** (ARM) **OF THE INFERIOR COLLICULUS**. The medial geniculate body projects to **PRIMARY AUDITORY CORTEX** in the temporal lobe.

The ascending axon from the ventral cochlear nucleus can also give off a collateral to a structure called the **superior olive**. Axons of cells in the superior olive then cross in what is called the **trapezoid body**, enter the opposite lateral lemniscus and eventually reach the inferior colliculus. You can take it to auditory cortex from here.



Auditory Pathways

As far as neurological deficits involving lesions of the auditory nuclei and pathways, you should know that a lesion of the auditory portion of the **C.N. VIII (nerve)** results in **deafness in the IPSILATERAL ear**. Also, a lesion involving the **dorsal and ventral cochlear nuclei** results in deafness in the **ipsilateral ear.** Only "subtle deficits" result from unilateral lesions of such structures as the lateral lemniscus, superior olive, inferior colliculus, medial geniculate body and auditory cortex (areas 41 and 42). SO I WILL USE THE TERM **"SUBTLE AUDITORY DEFICITS"** WHEN PROBLEM SOLVING WITH LESIONS INVOLVING AUDITORY STRUCTURES OTHER THAN THE **AUDITORY NERVE** AND **DORSAL AND VENTRAL COCHLEAR NUCLEI**.

REMEMBER:

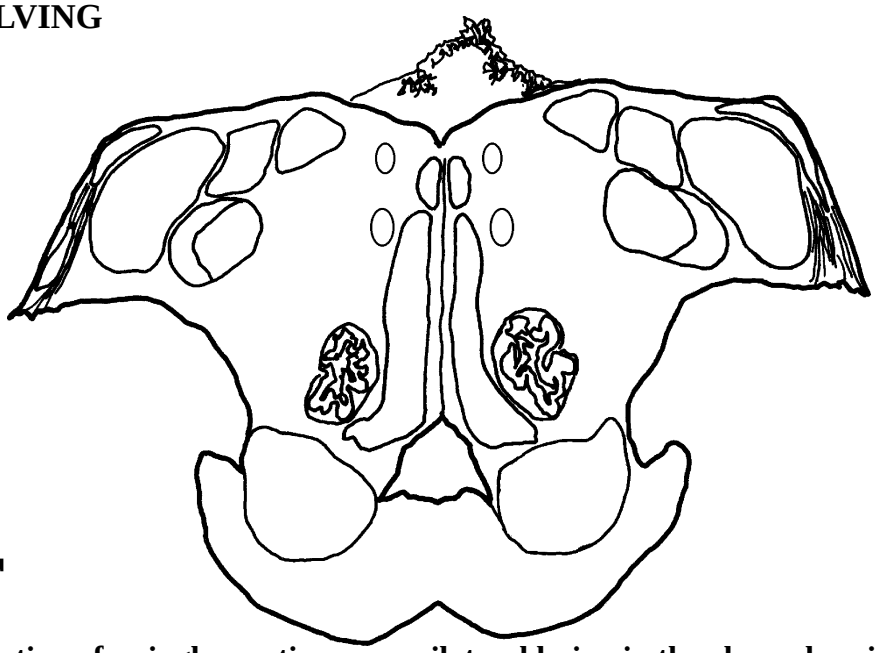
1.) cells of origin of the auditory division of C.N. VIII lie in the spiral or cochlear ganglion.

2.) all axons of the auditory nerve end in the dorsal and ventral cochlear nuclei.

3.) the trapezoid body and superior olive are parts of the auditory system and lie in the pons.

4.) the **lateral lemniscus** terminates in the **inferior colliculus** of the midbrain, while the **brachium of the inferior colliculus** terminates in the **medial geniculate body** of the thalamus.

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:

deafness in the left ear, incoordination of the left arm and leg, and loss of pain and temperature from the left side of the face

PROBLEM SOLVING MATCHING

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

____1. left medial lemniscus

____2. left inferior salivatory nucleus

____3. right pyramid

____4. left dorsal and ventral cochlear nuclei

____5. left inferior colliculus

A. lesion results in deafness in the left ear

B. cells project directly to the secretory cells of the left parotid gland

C. cells of origin lie in the right dorsal root ganglia

D. lesion results in left Babinski

E. cells of origin lie in the right nucleus gracilis and cuneatus

F. lesion results in deviation of the uvula to the right

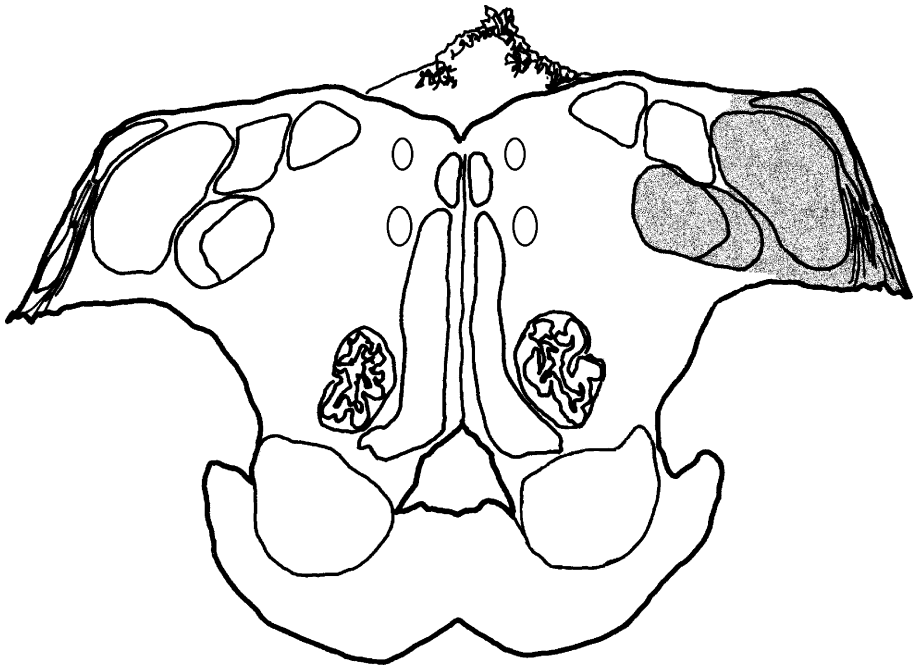
G. source of preganglionic parasympathetic innervation to the left otic ganglion

H. lesion results in subtle auditory deficits

I. lesion results in atrophy of the muscles on the left side of the tongue

J. projects to the right auditory cortex

PROBLEM SOLVING ANSWER



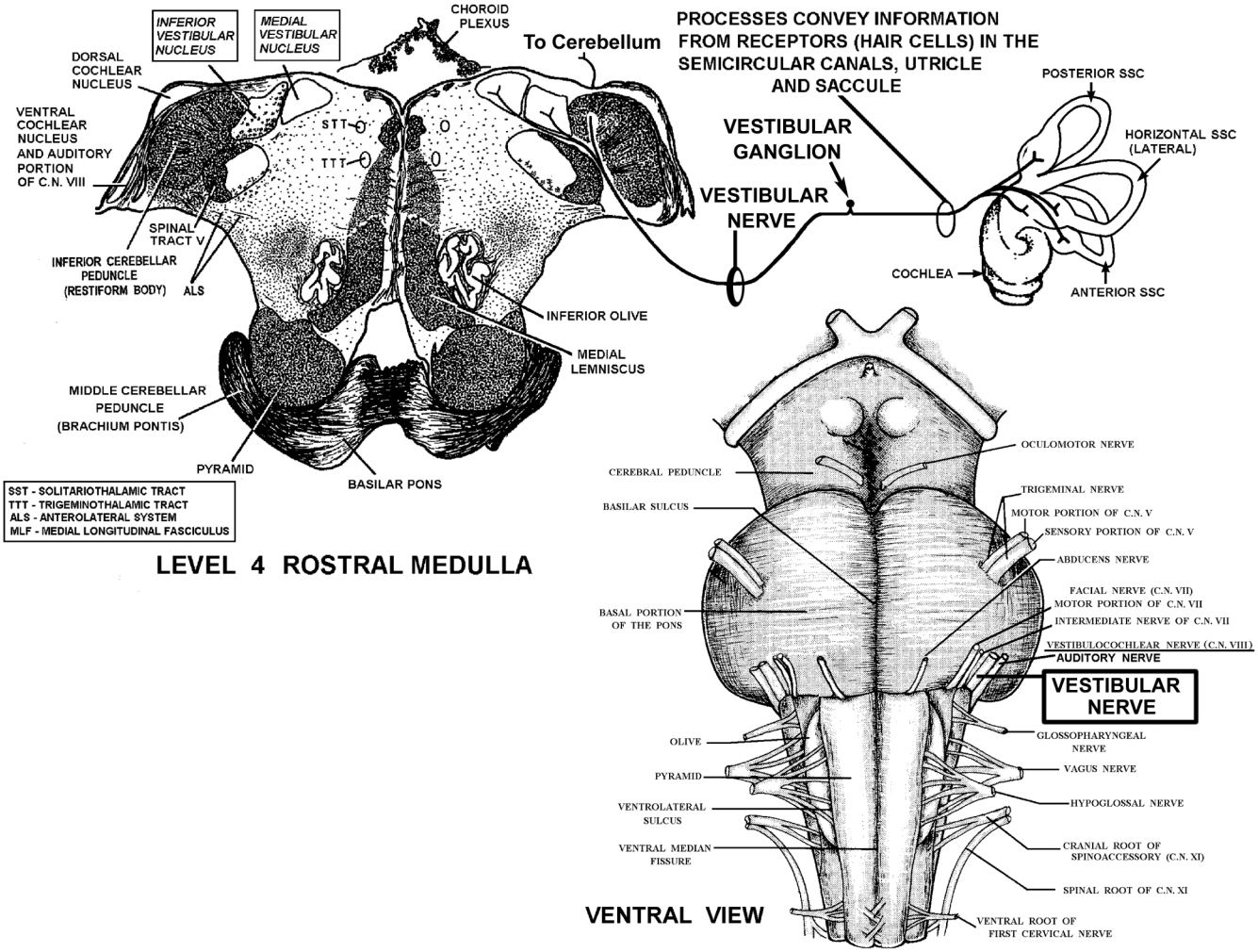
RIGHT LEFT

13 VESTIBULAR NUCLEI AND ABDUCENS NUCLEUS

You are probably wondering why we suddenly have TWO nuclei under ONE point. Well, over the years I've learned that it is much easier to cover them both at the same time because of their close functional association in the control of eye movements. This is a very long and tough point, but hang in there!

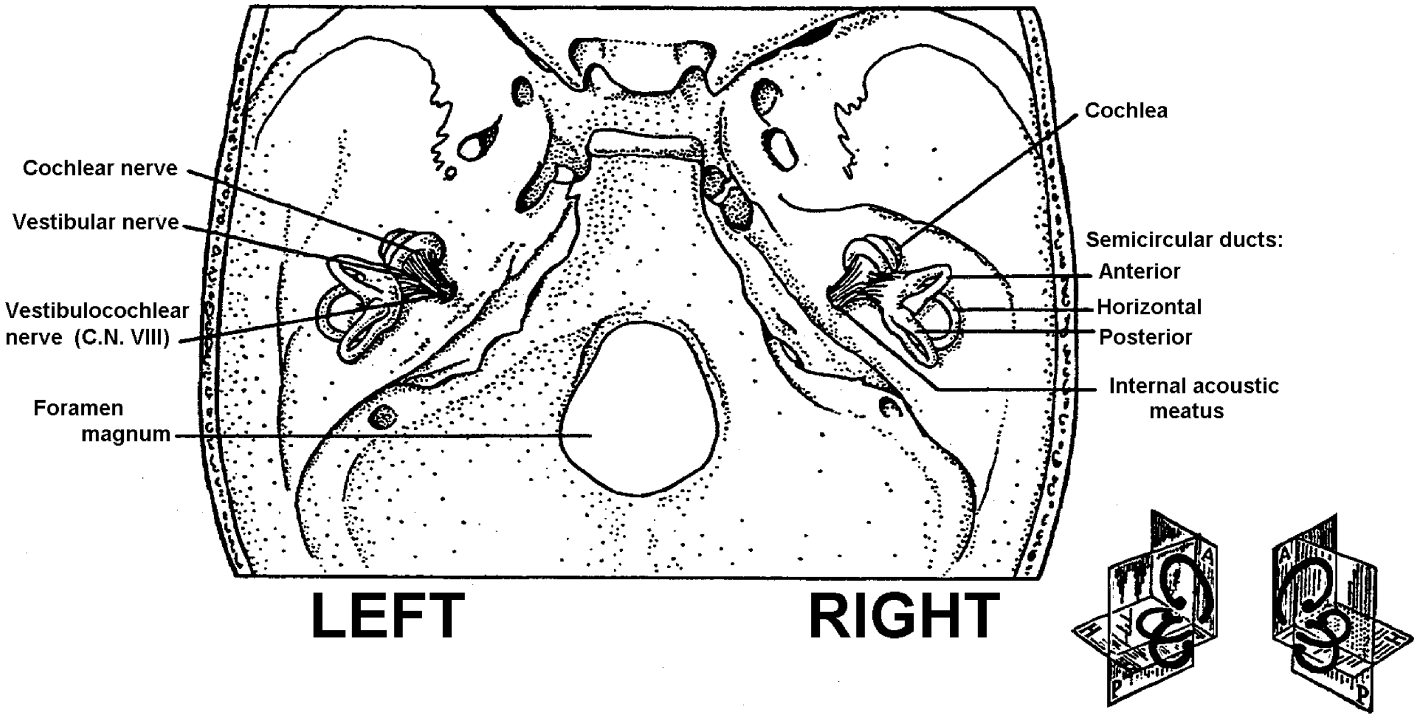
There are four vestibular nuclei within the brain stem (superior, lateral, medial, and inferior). All four can not be seen in the same cross section, since they are present for a considerable rostrocaudal distance from the rostral medulla to the middle of the pons. You only have to be able to identify the **MEDIAL and INFERIOR vestibular nuclei**, both of which are present at level #4 (shown below on the left).

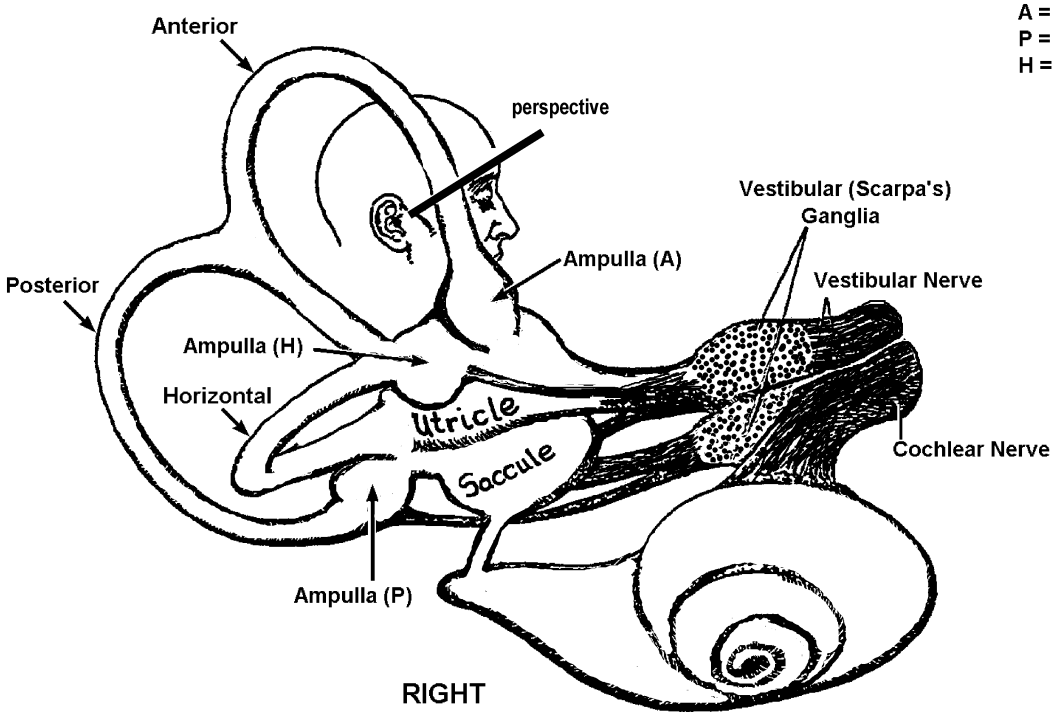
The vestibular nuclei receive their primary input from the vestibular portion of C.N. VIII (vestibular-auditory). The axons in the vestibular nerve are the central processes of neurons that lie in the **vestibular or Scarpa's ganglion** which lies in the internal auditory meatus. These central processes terminate in the vestibular nuclei and the cerebellum. The peripheral processes of these cells receive information from the receptors of the vestibular labyrinth, i.e. **hair cells** located in the **semicircular canals** and the **saccule** and the **utricle (otolith organs)**.



SEMICIRCULAR CANALS

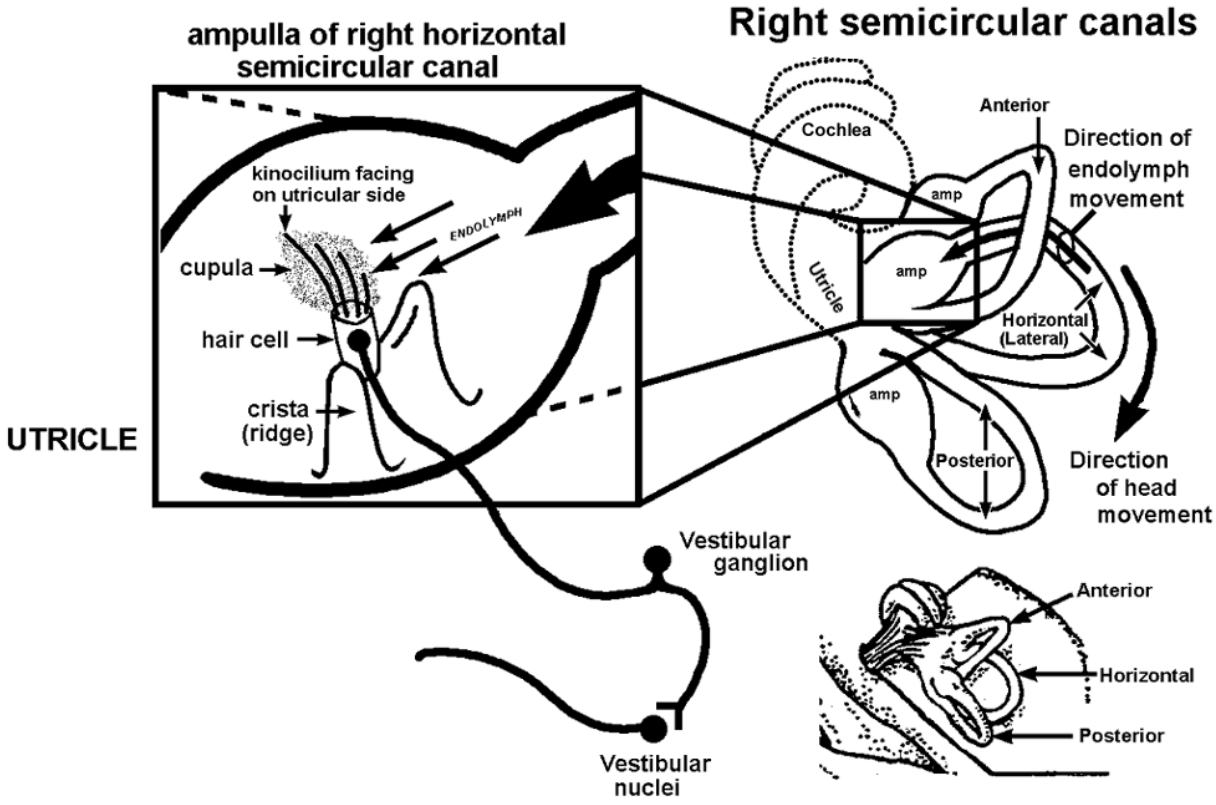
The three (on each side) membranous semicircular canals lie within the bony labyrinth and contain endolymph. As shown in two of the drawings below, the canals, one horizontal and two vertical, lie in three planes that are perpendicular to each other. The **HORIZONTAL** or **lateral** canals on the two sides lie in the same plane, while the plane of each anterior canal is parallel to that of the posterior canal of the **opposite** side. The horizontal semicircular canals communicate at both ends with the **utricle**, which is a large dilation of the membranous labyrinth. The vertical canals (anterior and posterior) communicate with the utricle at one end, and join together at the other end (the common canal communicates with the utricle).

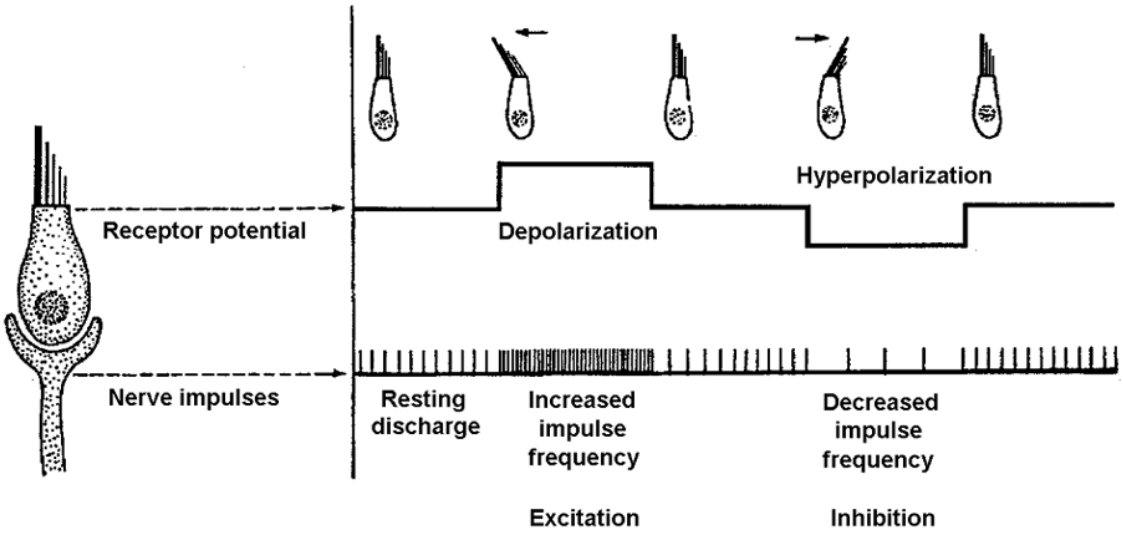




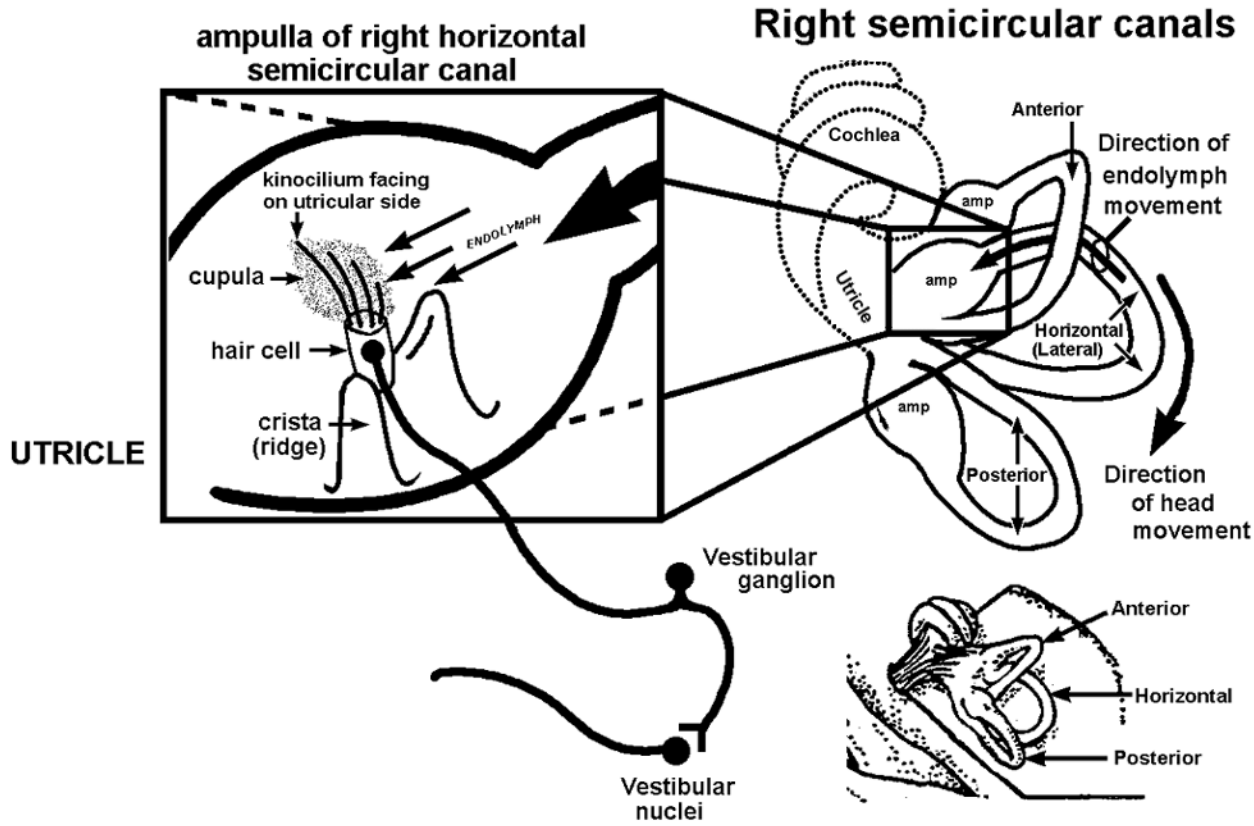
At one end of each semicircular canal is a **dilation** called the **ampulla** (L., little jar, is labeled “amp” in the upper right drawing below). The ampulla of a horizontal semicircular canal has been enlarged in the drawing below (upper left). Each ampulla contains a **crista** (crista ampullaris; ridge), which is a transversely oriented ridge of tissue. The upper surface of the crista contains ciliated sensory hair cells that are embedded in a gelatinous material called the **cupula** (L., little tube). These ciliated sensory hair cells contain vesicles that possess neurotransmitter. When the neurotransmitter is released from the hair cell, the peripheral process of a cell in the vestibular ganglion is turned on. Interestingly, the hair cells release transmitter even when they are not stimulated, so the axons in the vestibular nerve are always firing at a baseline rate.

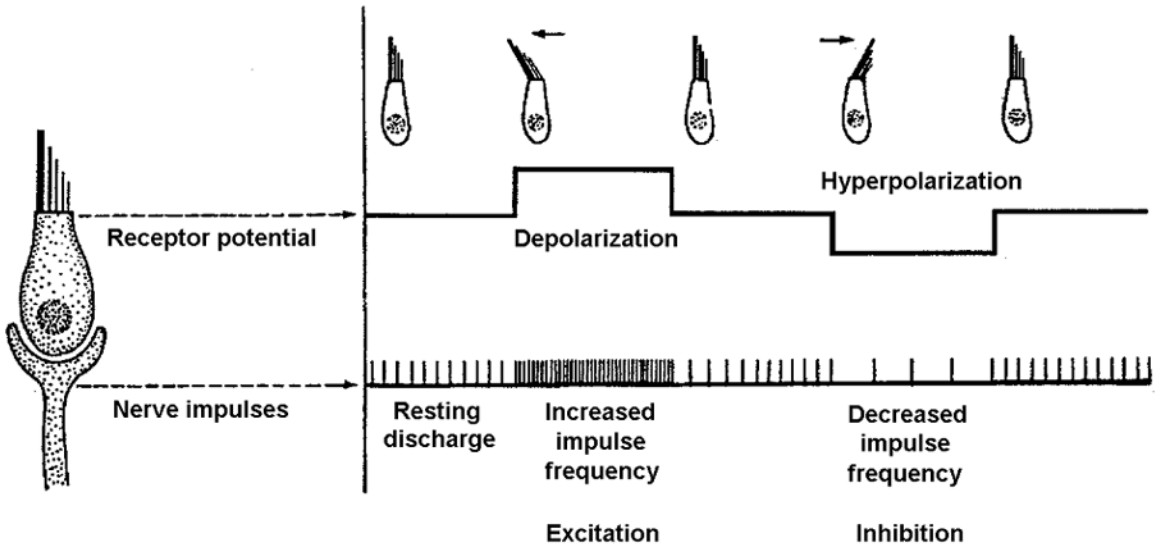
Each hair cell of the crista possesses **several shorter stereocilia** and a **single tall kinocilium** at one margin of the cell as shown in the lower figure. Deflection of the stereocilia **TOWARD** the kinocilium results in an **INCREASE** in the firing rate of the vestibular fiber associated with the hair cell, while deflection **AWAY** from the kinocilium results in a **DECREASE** in the firing rate of the vestibular fiber.





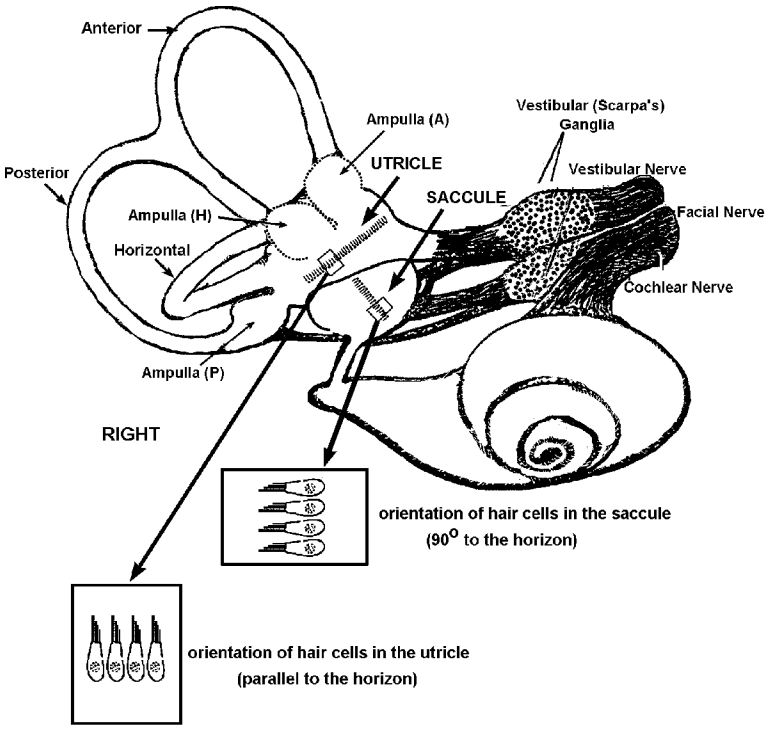
The kinocilia associated with the hair cells in the horizontal semicircular canal lie on the **utricle side** of the ampulla. For instance, rotation of the head to the **RIGHT** will result in **stimulation** of the hair cells in the crista of the **RIGHT** horizontal semicircular canal and **inhibition** of the hair cells in the **LEFT** horizontal semicircular canal. Stimulation of the hair cells in the **RIGHT** horizontal semicircular canal will result in an **increase** in the number of action potentials in the **RIGHT** vestibular nerve which causes increased firing of cells in the **RIGHT** vestibular nuclei. This is easy, **RIGHT** HEAD ROTATION—-**RIGHT** HORIZ. SEMICIRC. CANAL—-**RIGHT** VESTIBULAR NERVE—-**RIGHT** VESTIBULAR NUCLEI TURNED **ON** OR TUNED **UP**. All of this is in response to **ANGULAR ACCELERATION** of the head to the **RIGHT**, the stimulus needed to turn on the hair cells of the **RIGHT** horizontal semicircular canal.



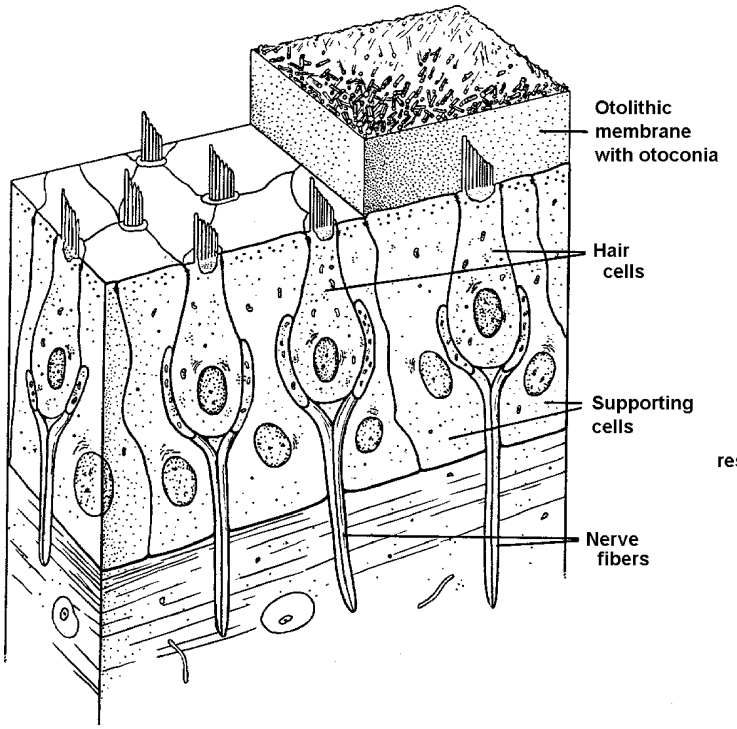


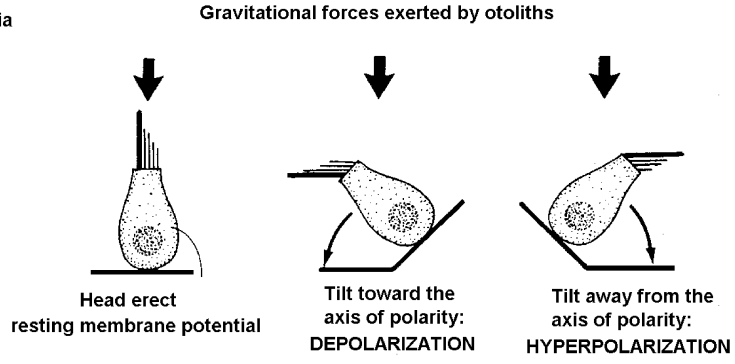
UTRICLE AND SACCULE

While semicircular canals respond to **angular** acceleration in specific directions, hair cells in the utricle and saccule respond to **linear** accelerations. The utricle and saccule are saclike structures that contain a patch of sensory hair cells called the macula (L., spot). The hair cells in the macula, which are similar to those in the cristae, are embedded in the otolith (ear stone) membrane, a gelatinous structure that contains a large number of hexagonal prisms of calcium carbonate called **otoconia** (ear dust). Since the density of the otoconia is greater than the surrounding endolymph, the otolith membrane will be displaced by the force of gravity or other linear accelerations. Such displacement bends the stereocilia and, depending on the polarity of the cell, either causes an increase or a decrease in the number of impulses in the associated vestibular fiber.



Please note, this is a very simplified, schematic representation of hair cell orientation in the vestibule!

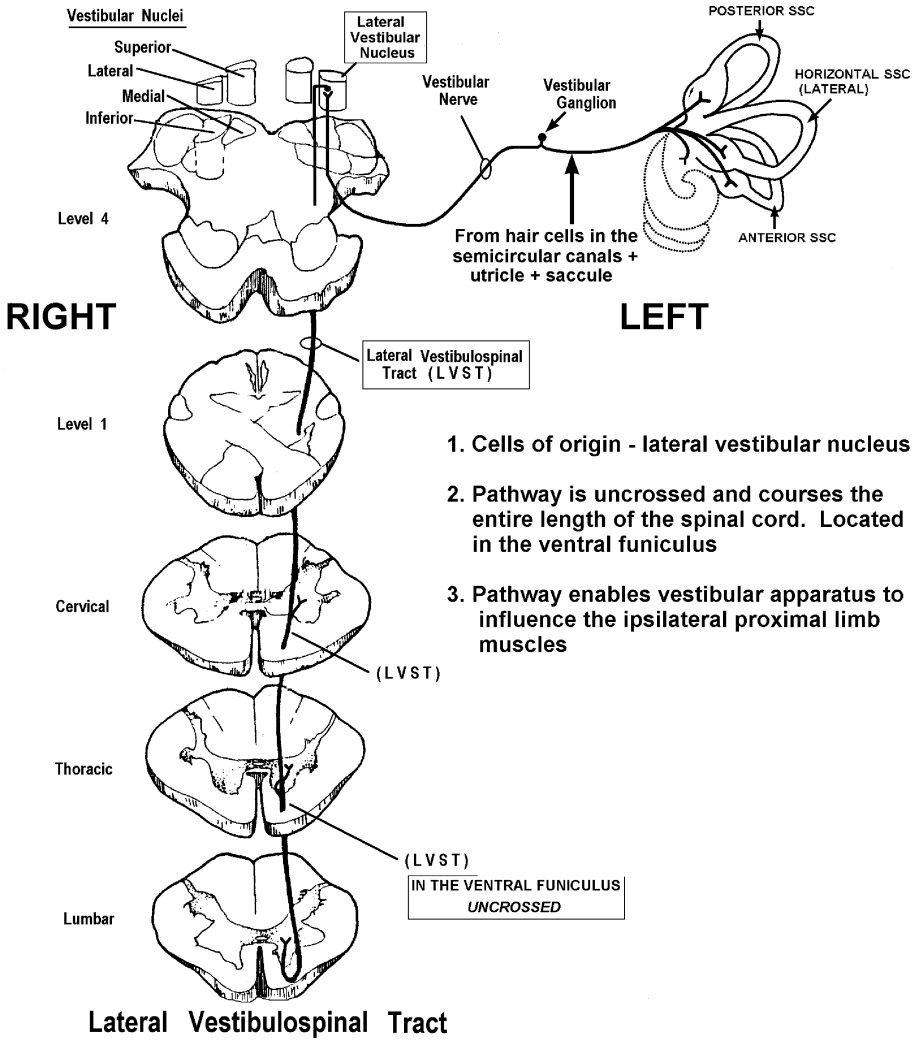




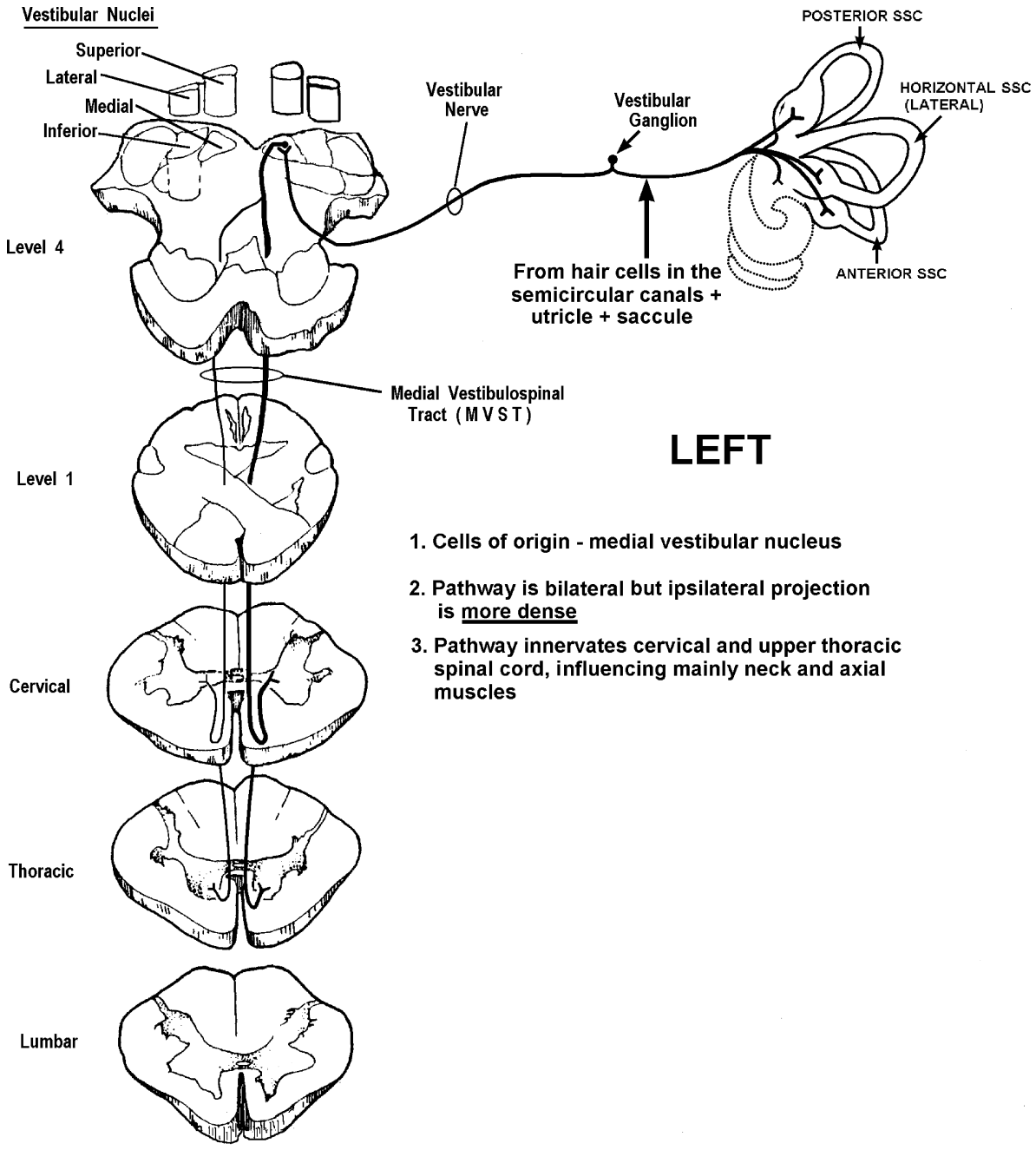
Once vestibular input from the semicircular canals and otolith organs has reached the vestibular nuclei, the information is used to maintain balance and to stabilize the visual image on the retina during head movements. First we will consider only the projections of the vestibular nuclei that reach the spinal cord in order to help us maintain our **BALANCE**. For example, let's say that you are walking to lecture this morning and slip on the icy sidewalk. Your feet fly to the **RIGHT** and your upper body and head fly to the **LEFT** (left ear down). Information coming out of your semicircular canals will be related to the accelerating head. This particular angular acceleration affects several different canals (beyond what you need to know for this course), but what I want you to know is that the **LEFT** vestibular nuclei are **turned on**. Once your head is not moving the information will come from the utricles. Again, you do not have to know the specific pattern from each side, but only that the **LEFT** vestibular nuclei are turned on.

The increased activity in the **LEFT** vestibular nuclei can affect the body musculature via the **LEFT LATERAL VESTIBULOSPINAL TRACT**. This will result in increased activity in the **LEFT** arm and leg in order to right ourselves after slipping. The cells of origin of the lateral vestibulospinal tract lie in the **lateral vestibular nucleus** (you can not see this nucleus in your sections). Axons arising from this nucleus descend through the caudal brain stem (you don't see these fibers on the cross sections) and upon reaching the spinal cord course within the ventral funiculus and innervate neurons for the **ENTIRE** length of the cord. This projection is **UNCROSSED**. Through this tract, the vestibular apparatus—which detects whether the body is on an even keel—exerts its influence on those muscles that restore and maintain upright posture. Such muscles are **proximal** rather than distal.

REMEMBER—LATERAL VESTIBULAR NUC.—LATERAL VESTIBULOSPINAL TRACT—UNCROSSED—ENTIRE LENGTH OF CORD—VENTRAL FUNICULUS—PROXIMAL MUSCLES—MAINTAINS BALANCE BY ACTING MAINLY ON LIMBS



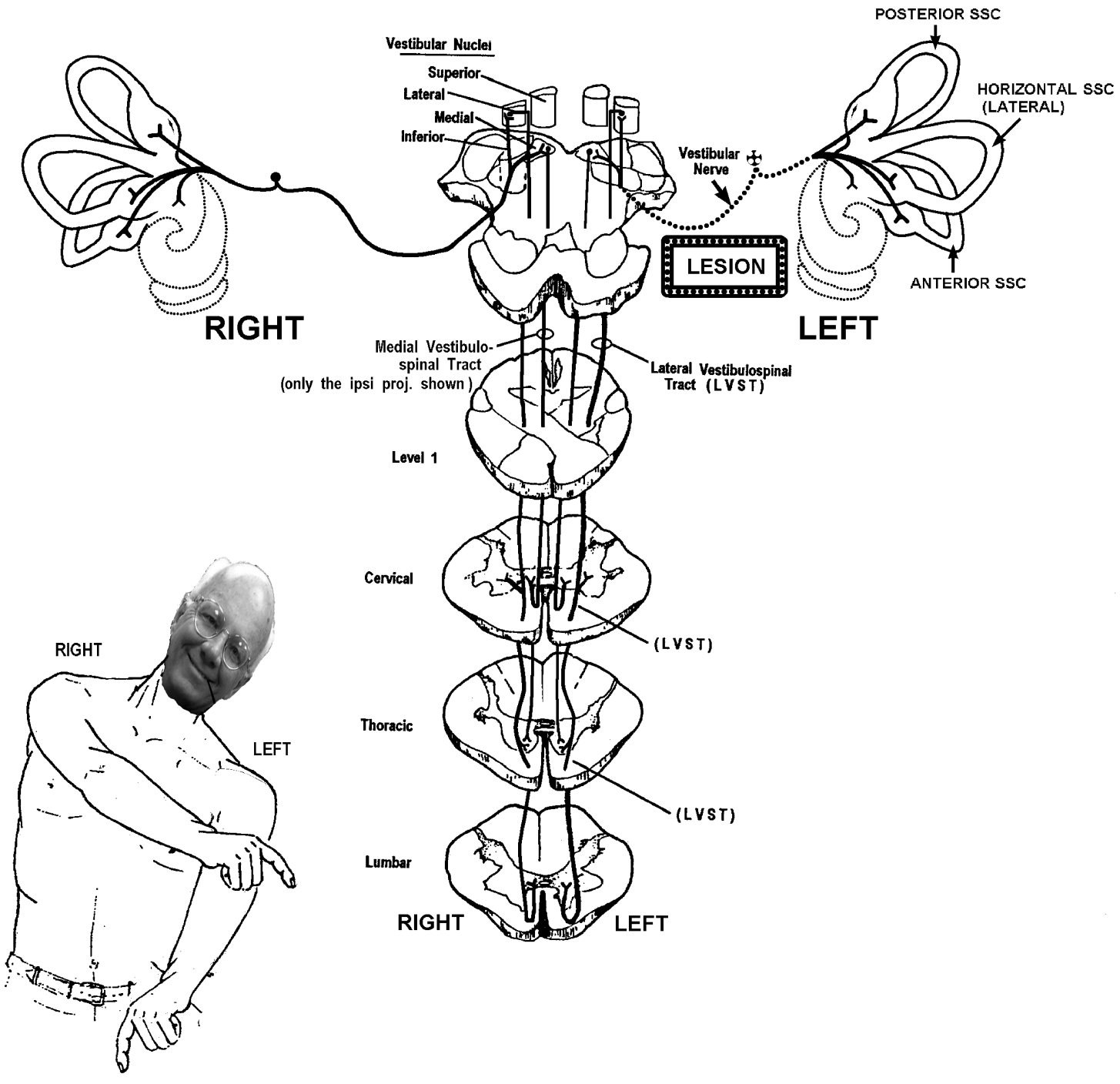
The increased activity in the **LEFT** vestibular nuclei can also affect body musculature via a second, smaller, descending pathway to the spinal cord. This smaller pathway is called the **MEDIAL VESTIBULOSPINAL TRACT** (or descending medial longitudinal fasciculus [MLF]). Cells within the **medial** vestibular nucleus possess axons that descend bilaterally (the ipsilateral projection is **denser**) in a position just off the midline near the dorsal surface of the pons and medulla. These descending axons course caudally and enter the spinal cord, where they lie within the **medial** part of the **ventral funiculus**. This pathway makes connections with **cervical and upper thoracic motor neurons** that play a role in maintaining the normal position of the head via innervation of spinal cord neurons that innervate **neck** musculature. Thus when your head flies to the LEFT, it will reflexively be brought to an upright position via information flowing out of the LEFT medial vestibular nucleus. **REMEMBER—MEDIAL VESTIBULAR NUCLEUS—MEDIAL VESTIBULOSPINAL TRACT—BILATERAL—CERVICAL AND UPPER THORACIC SPINAL CORD ONLY—MAINTAINS HEAD ERECT.**



Medial Vestibulospinal Tract

Now we can do some problem solving. Lesions involving the vestibular nerve, nuclei, and descending pathways will result in problems such as stumbling or falling **TOWARDS THE SIDE OF THE LESION**. Think of the **NORMAL** side as being in control and pushing against the weak side. For example, if you have a patient with a lesion that has destroyed the **LEFT** vestibular nerve, the **LEFT** vestibular nuclei and the **LEFT** lateral vestibulospinal tract are "tuned" down. Meanwhile, the normal **RIGHT** nerve is fine and firing away and thus the **RIGHT** lateral vestibulospinal tract is also in good shape. The two lateral vestibulospinal tracts usually counteract each other functionally, but now the **RIGHT** side takes over. The end result? **STUMBLING AND FALLING TO THE WEAK SIDE, IN THIS CASE TO THE LEFT**.

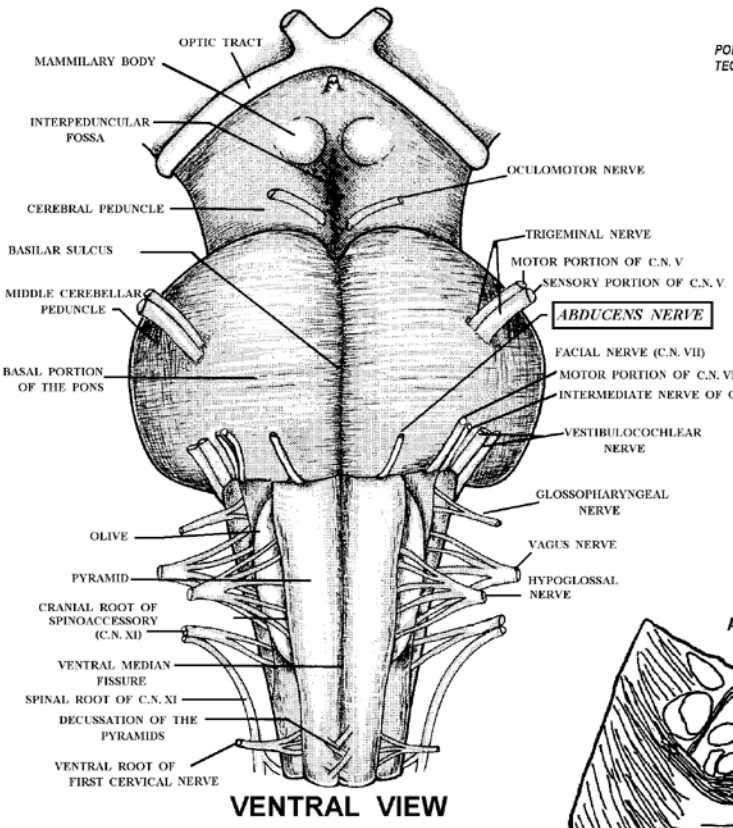
At the very onset of vestibular problems there may be a Romberg sign. Postural instabilities are kept in check by visual inputs however, closing the eyes with the feet together will reveal the unstable condition.



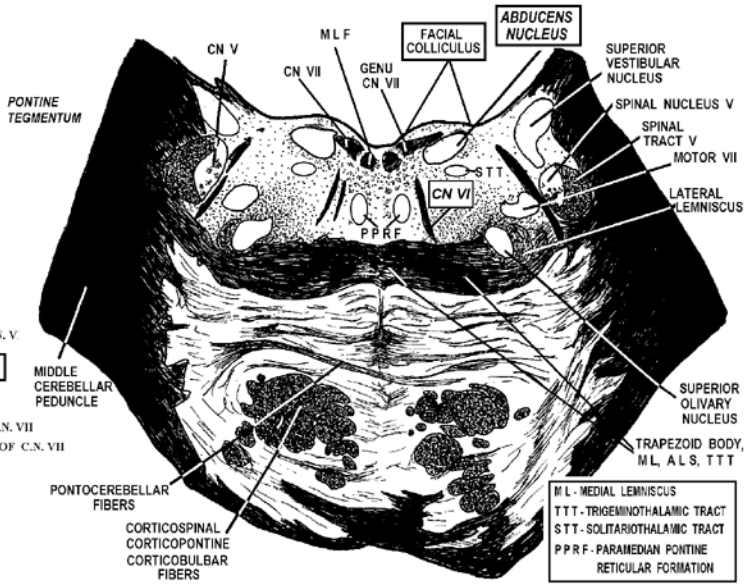
Lesion of the LEFT vestibular nerve = RIGHT side "driving" = stumbling to the LEFT

Imbalance in the vestibulospinal tracts can be demonstrated in healthy medical students. Put a penny on the floor and stand directly over it so that it lies between your feet. Bend your head forward to stare at the penny. While staring at the penny, turn to the **RIGHT** five complete turns. At the end of the five turns stop and try to stand erect and hold your arms straight ahead. Now, the **LEFT** vestibular nerve is dominating, so which way do you stumble? Following this spinning exercise, you might also experience some nausea, autonomic disturbances and **vertigo** (you are spinning, or the room is spinning). In addition, there will be an involuntary to and fro oscillation of the eyes. This is called **nystagmus** and demonstrates the **CONNECTIONS BETWEEN THE VESTIBULAR APPARATUS AND NUCLEI IN THE BRAIN STEM THAT INNERVATE MUSCLES THAT MOVE THE EYES IN THE HORIZONTAL DIRECTION.**

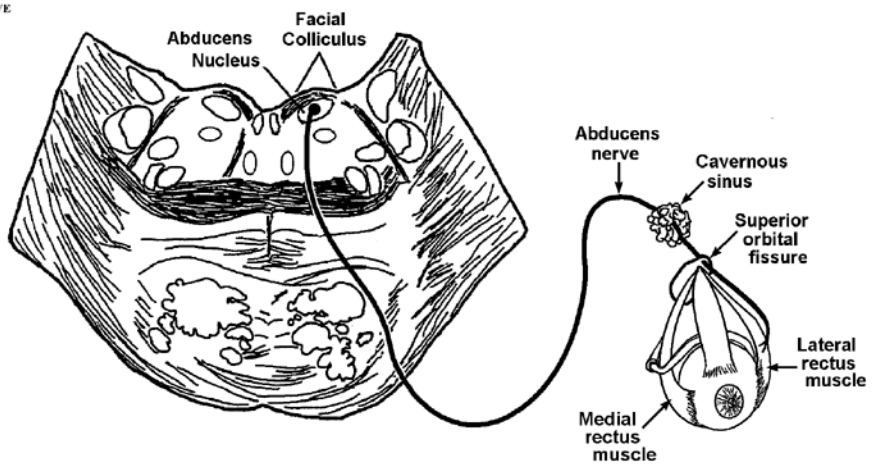
We now need to look at the **ABDUCENS** nucleus. The abducens lies just off the midline within the dorsal part of the pons, just under the fourth ventricle. (Don't let the pons scare you, it's pretty easy!) C.N. VI fibers pass ventrally from the abducens nucleus, exit at the pontomedullary junction and eventually reach the lateral rectus (LR_6). We will evaluate the effects of lesions involving this nucleus and nerve later in this point, but first we need to talk about how the vestibular system influences horizontal eye movements.



VENTRAL VIEW

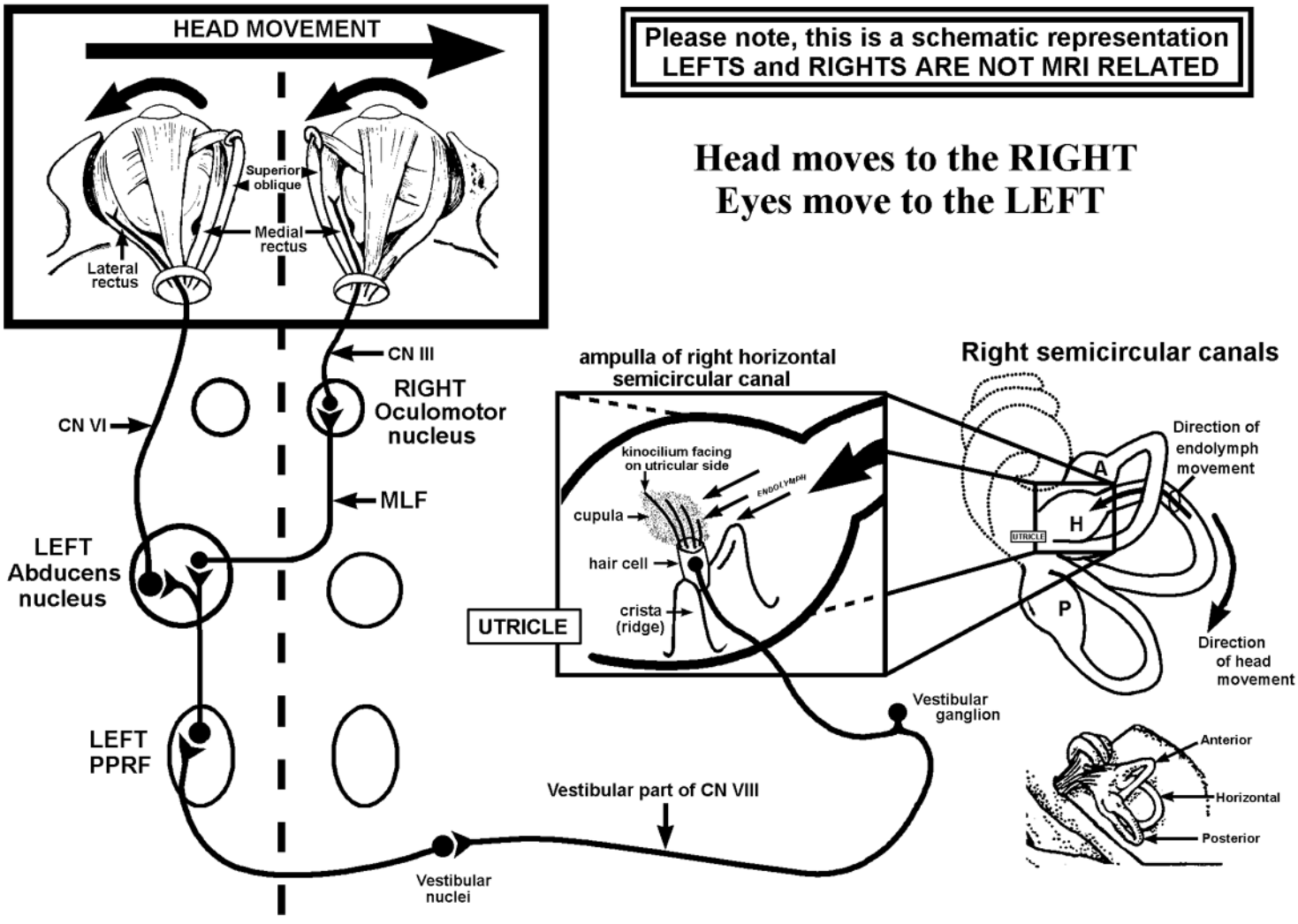


LEVEL 5. MIDDLE PONS - FACIAL COLLICULUS



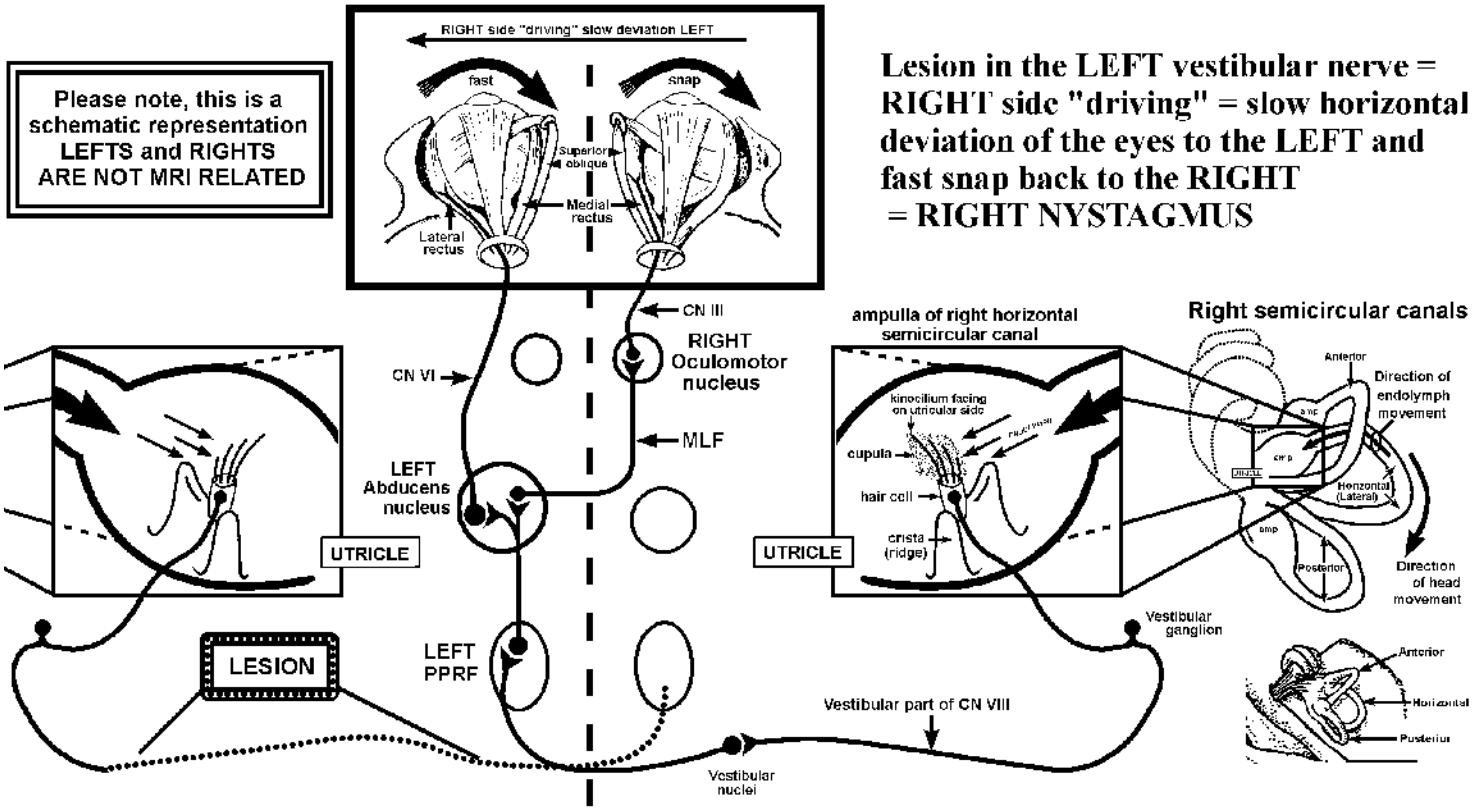
Eye movements induced by the vestibular apparatus are compensatory. That is, they oppose head movements or changes in head position and act to keep the fovea of the retina on an object of interest. For example, a quick turn (or push) of your head to the **RIGHT** will result in a compensatory reflex turning of the two eyes to the **LEFT**. You already know some of the receptors and pathways underlying this reflex. Thus a quick rotation of the head to the **RIGHT** will turn on the hair cells in the **RIGHT** horizontal semicircular canal, increase the firing of the right vestibular nerve and increase the firing of neurons in the **RIGHT** vestibular nuclei.

Now for a new pathway! Cells in the **RIGHT** vestibular nuclei send their axons **across** the midline to the contralateral **PARAMEDIAN PONTINE RETICULAR FORMATION (PPRF)**. The PPRF, which lies within the medial portion of the pontine tegmentum, ventral to the abducens nucleus, is an integrative region involved in the generation of horizontal eye movements. Neurons in the **LEFT PPRF** project to the **LEFT ABDUCENS** nucleus. It contains two types of neurons. The larger motor neurons in this nucleus possess axons that pass ventrally through the pons to exit on the ventral surface of the brain stem (at the pontomedullary junction). Axons of C.N. VI then innervate the ipsilateral **LATERAL RECTUS (LR_6)**. There are also other smaller neurons in the abducens nucleus whose axons do not leave the brain stem, but rather **CROSS AND ASCEND IN THE MEDIAL LONGITUDINAL FASCICULUS (MLF) TO TERMINATE IN THE OCULOMOTOR NUCLEUS (C.N. III). NEVER, I SAID NEVER, FORGET THE MLF!!!** In particular, these crossed axons from the abducens nucleus end only upon those neurons within the oculomotor nucleus that innervate the **MEDIAL RECTUS** muscle. Remember, neurons in the oculomotor nucleus that innervate other eye muscles, as well as the preganglionic parasympathetic neurons that innervate the ciliary ganglion, **do not** receive this crossed input from the abducens nucleus. **Only those neurons that innervate the medial rectus muscle receive this ascending, crossed input.**

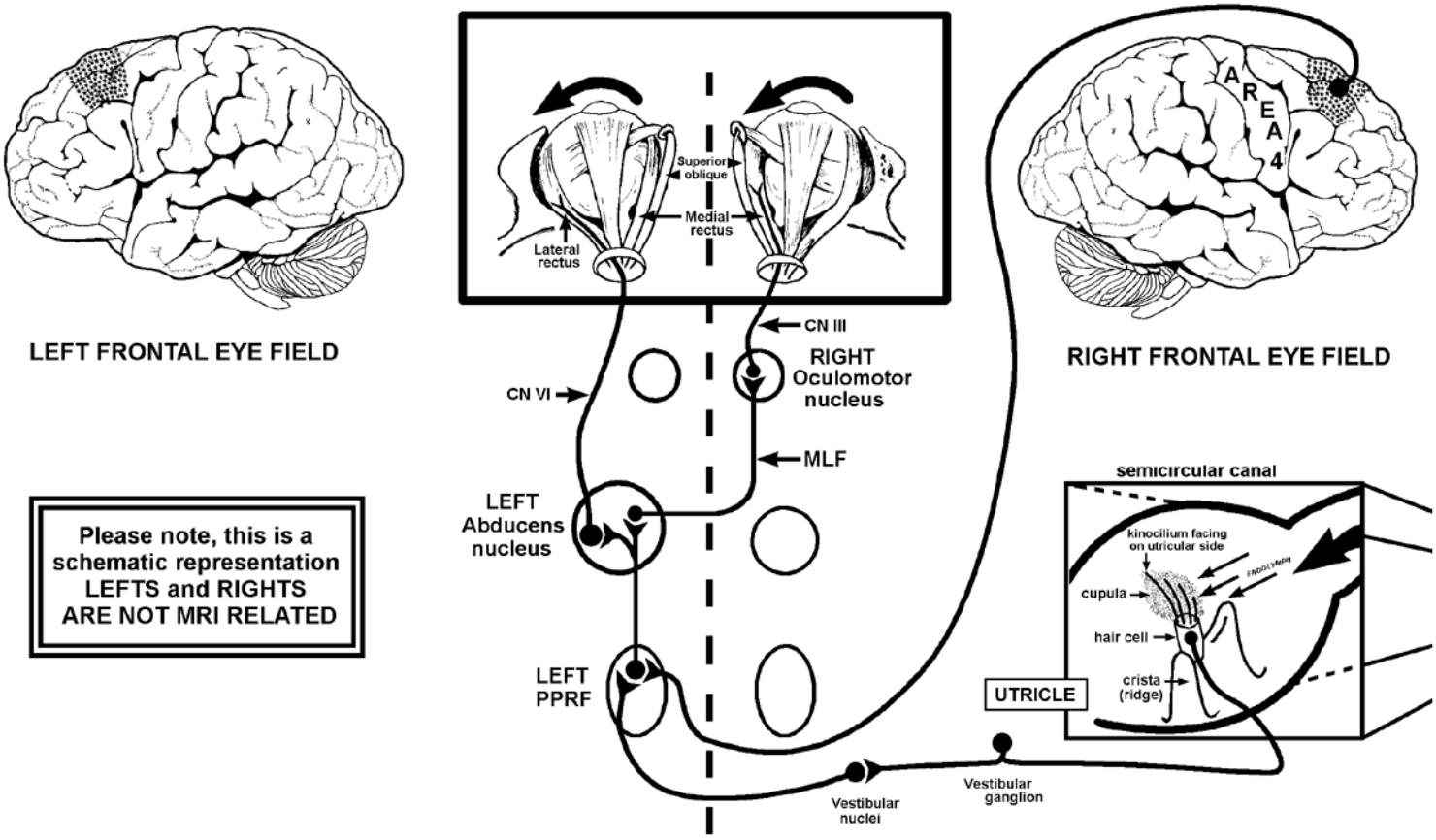


Now you can see how a rotatory movement of the head to the **RIGHT** results in an increase in the discharge of the **RIGHT** vestibular nerve, an increase in firing of the **RIGHT** vestibular nuclei, an increase in firing of neurons in the **LEFT PPRF**, an increase in firing of both small and large neurons in the **LEFT** abducens nucleus and reflex turning of the left eye to the **LEFT** (via **LEFT** lateral rectus; C.N. VI) and the right eye to the **LEFT** (via ascending MLF input to the **RIGHT** medial rectus; C.N. III). This is called the **VESTIBULO-OCULAR REFLEX (VOR)**, which is a critically important reflex for stabilizing visual images in the presence of a continuously moving head.

So; let's examine the results of a lesion in the **LEFT** vestibular nerve on eye movements. Such a lesion puts the **RIGHT** vestibular nerve in "control." This imbalance results in the eyes being pushed slowly to the **LEFT** (**right** vestibular nerve turns on **right** vestibular nuclei, which turns on the **left** PPRF, which turns on the **left** abducens, which turns both eyes to the **left**). When the eyes are pushed as far **LEFT** as possible, they snap back very quickly to the **RIGHT** by mechanisms not fully understood. The eyes then slowly move to the **LEFT** again, and this vicious cycle continues. This nodding back and forth is called **NYSTAGMUS** (to nod). It is named (i.e., right or left) by the **FAST** direction. For instance, a lesion of the **LEFT** vestibular nerve will result in a **RIGHT** nystagmus. Thus, the **RIGHT** (intact) vestibular nerve is "driving" the **LEFT** PPRF and **LEFT ABDUCENS** to move the eyes slowly to the **LEFT**, after which they reflexively snap back to the **RIGHT** (i.e., the direction of the nystagmus).

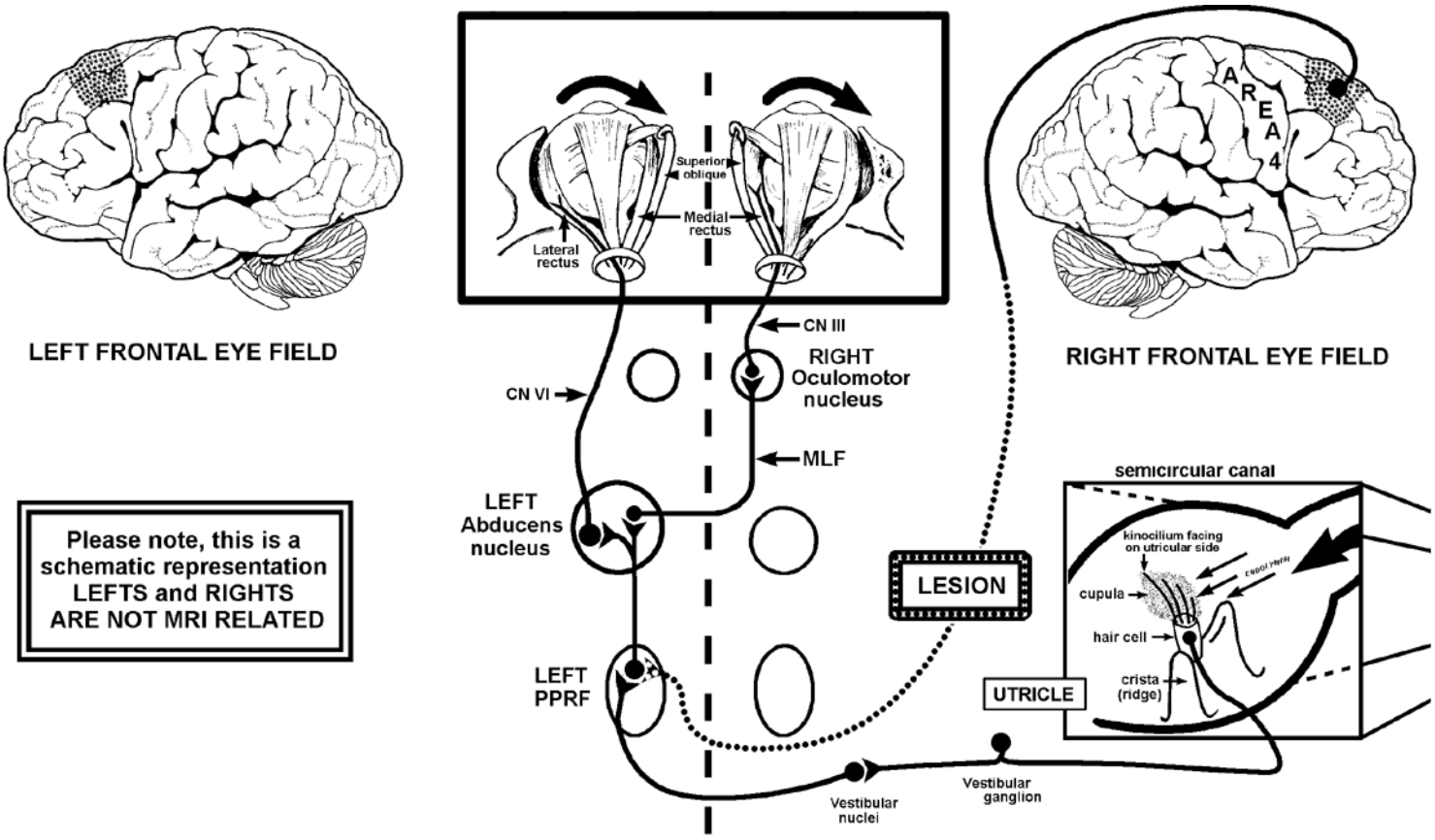


Now we need to consider pathways involved in VOLUNTARILY turning both of our eyes horizontally to the **LEFT** in order to see a new object of interest. This is called a left horizontal **saccade** (jerk). We already know that to do this we need to have the **LEFT** lateral rectus and the **RIGHT** medial rectus contract synchronously. The two eyes will then move together (conjugately) to the **LEFT**. To VOLUNTARILY do this, we use a pathway that begins in the **frontal eye fields** of the cerebral cortex (area 8). This is a cortical area that lies rostral to the primary motor area (area 4; where the corticospinal axons begin). To voluntarily move your eyes to the **LEFT**, information from your **RIGHT** frontal eye fields is conveyed to the **LEFT** (contralateral) PPRF. You should know the rest from here, but I'll help! The **RIGHT** frontal eye field will tell your **LEFT** PPRF to turn on both large and small neurons in the **LEFT** abducens nucleus. Two things will then happen. The **LEFT** eye will turn **LEFT** (laterally) and the **RIGHT** eye will turn **LEFT** (medially). This is a voluntary **LEFT** horizontal saccade.



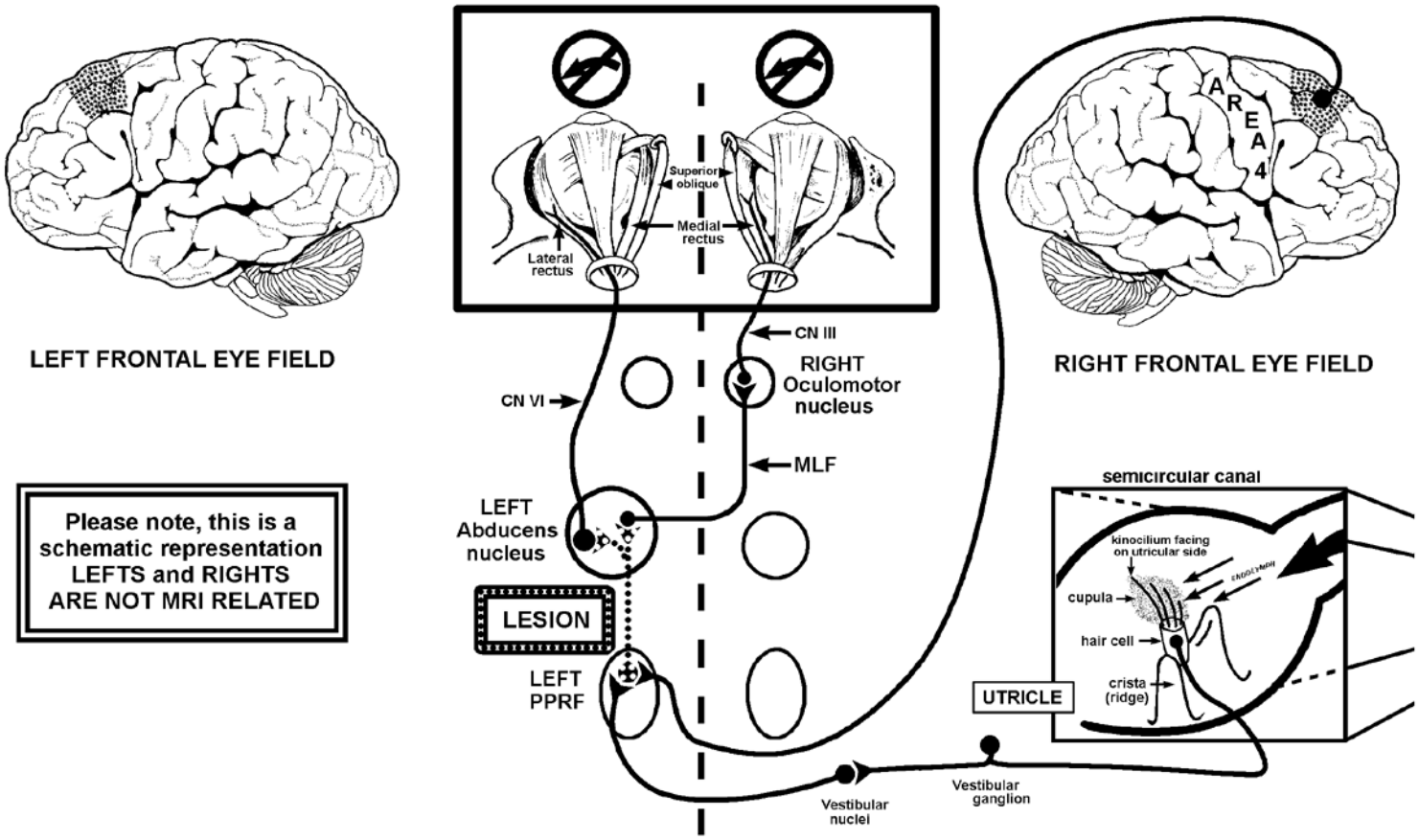
RIGHT frontal eye field turns the eyes to the LEFT

A lesion in the **RIGHT** frontal eye fields will mean that the first part of the circuit involved in voluntarily turning the eyes to the **LEFT** is fouled up. Therefore immediately after such a lesion in area 8 on the **RIGHT**, the intact (**LEFT**) cortex takes over and pushes both eyes to the **RIGHT**. If the cortical lesion also involves area 4 (which is not too far away from area 8) of the **RIGHT** cortex, the hemiplegia (due to damage to the corticospinal tract) will be on the **LEFT**. Thus **THE EYES LOOK AWAY FROM THE HEMIPLEGIA** (the eyes look at the normal intact extremities). This is especially true when the patient is comatose. Once out of the coma, recovery usually occurs and the patient is able to make saccades into the opposite half field. However, saccades are less frequent in such patients. You should know by now that there is **NO** atrophy of any eye muscles following a cortical lesion. Also there is **NO** diplopia (no double vision due to the misalignment of the two eyes; we will cover this later). Remember, the motor neurons in the abducens and oculomotor nuclei are not dead and the eyes are turned **conjugately** to the right just after the lesion!



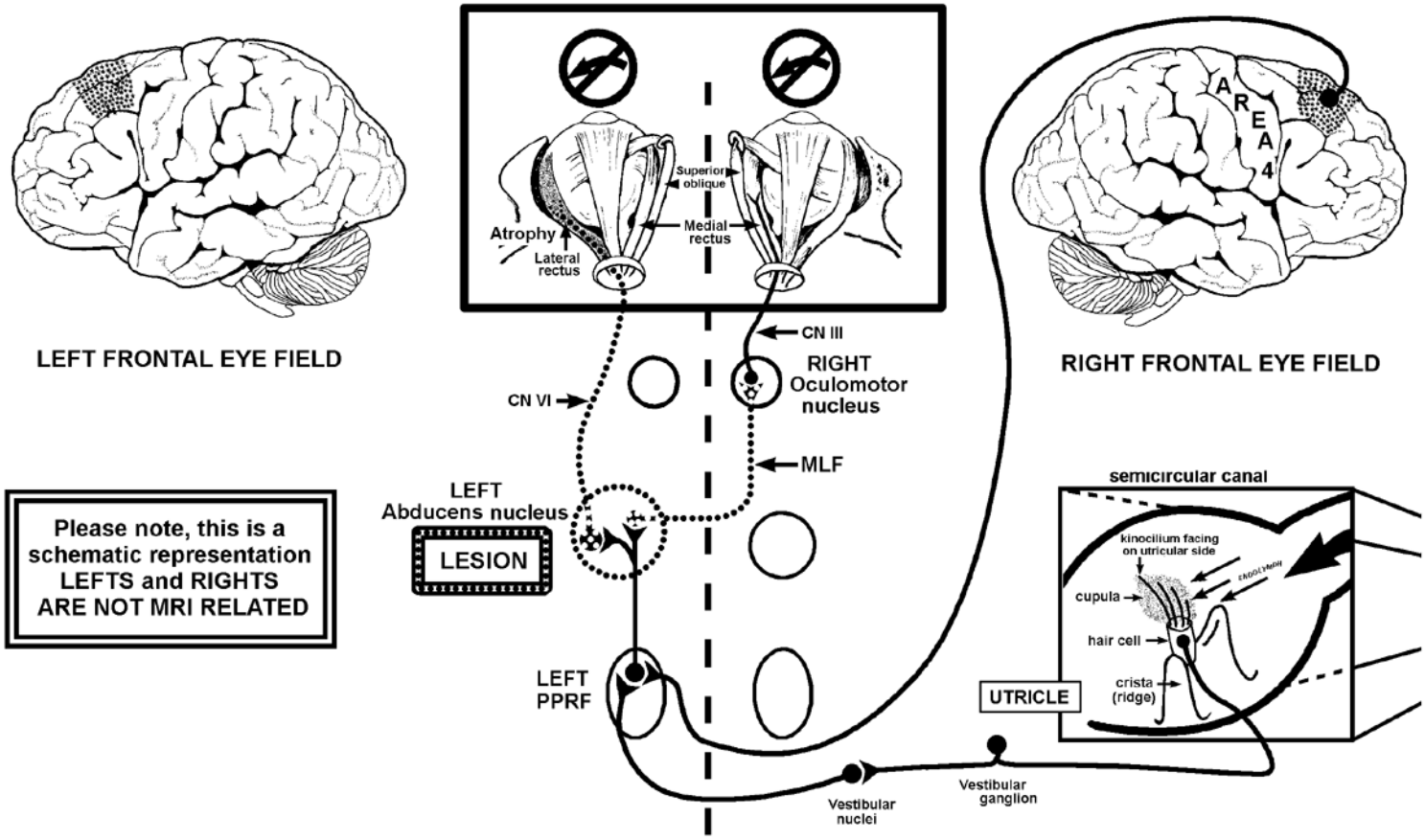
Lesion in the RIGHT frontal eye field = tonic deviation of the eyes to the RIGHT (LEFT side "driving"), No atrophy or diplopia

Now for a lesion in the **PPRF**. A lesion of the **LEFT** PPRF will result in the inability to make a **VOLUNTARY** saccade that moves the eyes to the **LEFT** of the midline. There is no atrophy of the lateral rectus or diplopia (no misalignment of the two eyes). Sometimes the eyes will be deviated to the **RIGHT** due to the unopposed normal circuitry for making **RIGHT** horizontal saccades. If a lesion in the pons is big enough to also involve the corticospinal fibers on the same side, the deviating eyes will **LOOK TOWARDS THE HEMIPLEGIA**.



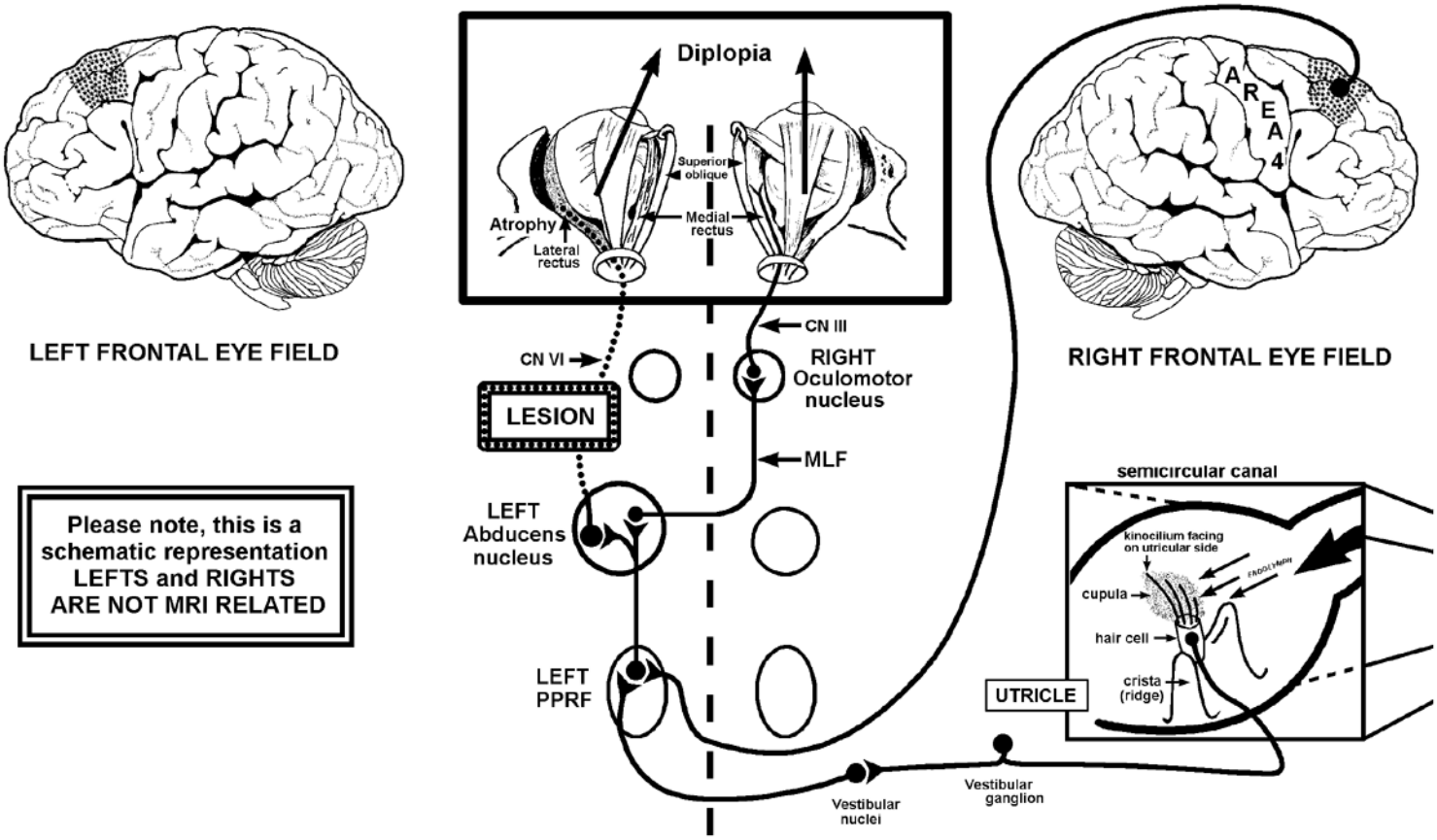
Lesion in the LEFT PPRF = inability to voluntarily move both eyes past the midline to the LEFT, No atrophy or diplopia

A lesion of the **LEFT ABDUCENS NUCLEUS** will result in atrophy of the **LEFT LATERAL RECTUS** and the inability to turn the **LEFT** eye laterally. Also, the small cells in the **LEFT** abducens nucleus are dead, so the ascending input to the **RIGHT** (contralateral) oculomotor nucleus, and in particular to the neurons innervating the medial rectus, is lost. This results in the inability to turn the **RIGHT** eye medially when attempting to look to the **LEFT**. THERE IS **NO** ATROPHY OF THE **RIGHT** MEDIAL RECTUS. WHY? BECAUSE THE NEURONS INNERVATING THE **RIGHT** MEDIAL RECTUS ARE NOT DEAD. THEY HAVE JUST LOST AN INPUT TELLING THEM TO FIRE DURING A **LEFT** HORIZONTAL SACCADE. They will fire for example during convergence (simultaneous contraction of both medial recti). In such a case the neurons in the right medial rectus receive an input from a convergence center located rostral to the oculomotor complex. The left lateral rectus has atrophied and the neurons innervating the right medial rectus have only lost an input. Thus the left eye will deviate further to the right then the right eye. Thus to ameliorate double vision the patient will **ROTATE THEIR HEAD** to the left (toward the abducens nucleus lesion).



Lesion in the LEFT Abducens nucleus = inability to voluntarily move both eyes past the midline to the LEFT (atrophy of the LEFT lateral rectus but not the RIGHT medial rectus), Diplopia

What about a lesion of the **ABDUCENS NERVE**? Let's examine the results of a lesion of the **LEFT C.N. VI** (for instance in the cavernous sinus). Such a lesion will result in **atrophy** of the **LEFT** lateral rectus muscle. Due to the unopposed action of the **LEFT** medial rectus, the **LEFT** eye will be deviated **MEDIALLY**. This will result in double vision, since the two eyes are not aligned (the fovea are not looking at the same point in space). There is further information regarding diplopia on the next page.

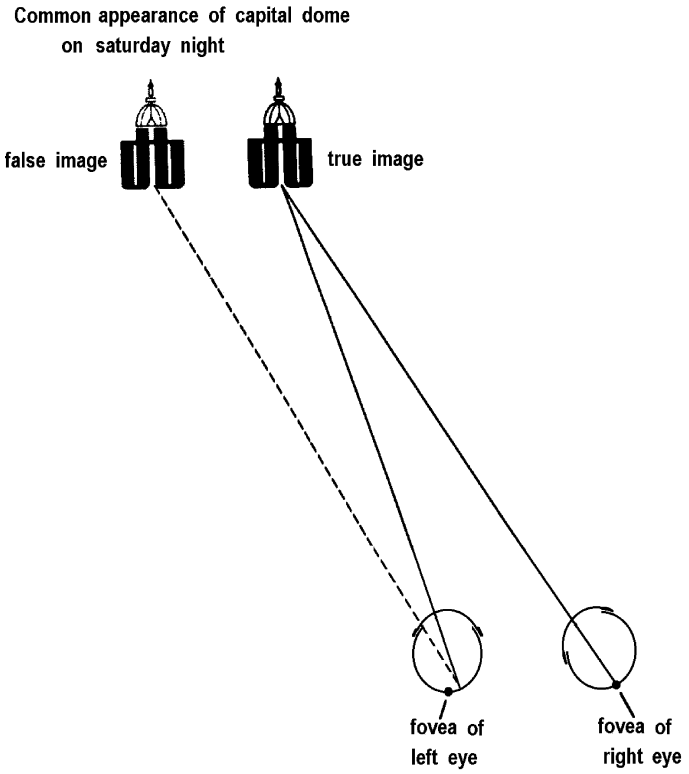


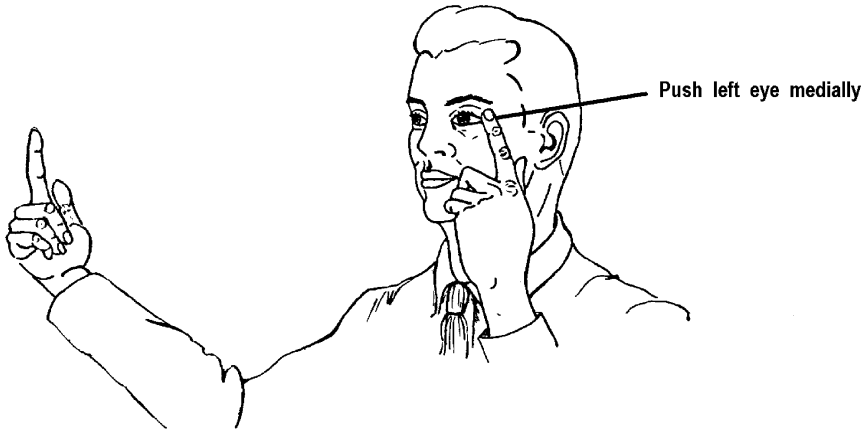
Lesion of the LEFT Abducens nerve = atrophy of the LEFT lateral rectus, LEFT eye deviated medially, Diplopia

DIPLOPIA

(Gr., diplous=double + ope=sight)

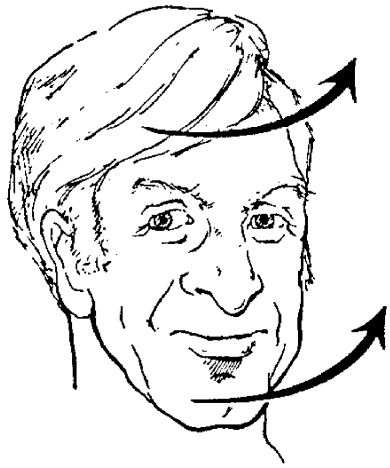
When looking at an object (such as your finger, as in the drawing below), its image falls upon the fovea of both retinae. The fovea lies at the posterior pole of the retina and is the part of the retina where visual acuity is greatest. Misalignment of the visual axes causes the image to fall on **non-corresponding** areas of the two retinae and two images are seen instead of one. For instance, hold your **RIGHT** finger out in front of you and place your **LEFT** index finger upon your **LEFT** lateral canthus (see drawing below). Press gently with your **LEFT** finger and you should obtain diplopia. If the pushing deviates your **LEFT** eye **medially**, (as in a lesion of the **LEFT LR**) you will see the false image to the **LEFT** of the true image. You will notice that the false image moves, and is not as clear as the true image. Also, you will notice that the further you move your **RIGHT** finger to the **LEFT** (towards the bad eye in the case of a **LEFT LR** lesion) the greater the separation of the two images. This is called **horizontal** diplopia. **Vertical** diplopia in which the images are separated in the vertical (up and down) axis.





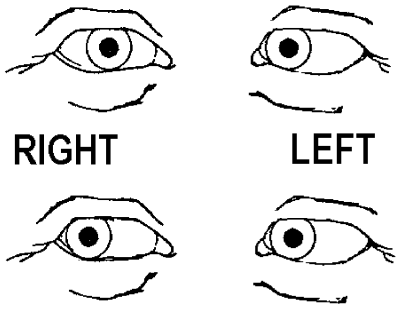
You know that following a lesion of the **LEFT** lateral rectus the **LEFT** eye will be deviated **medially** or to the **RIGHT**. As a way of avoiding the diplopia (which is greatest on **LEFT** gaze), the patient will turn their head **TOWARD** the side of the **paralyzed** muscle (**LEFT** in this example). This alleviates the double vision.

Head rotated to the LEFT (towards the paralyzed LEFT L R)



RIGHT LEFT

Lesion in LEFT L R
LEFT eye deviated medially
Horizontal diplopia



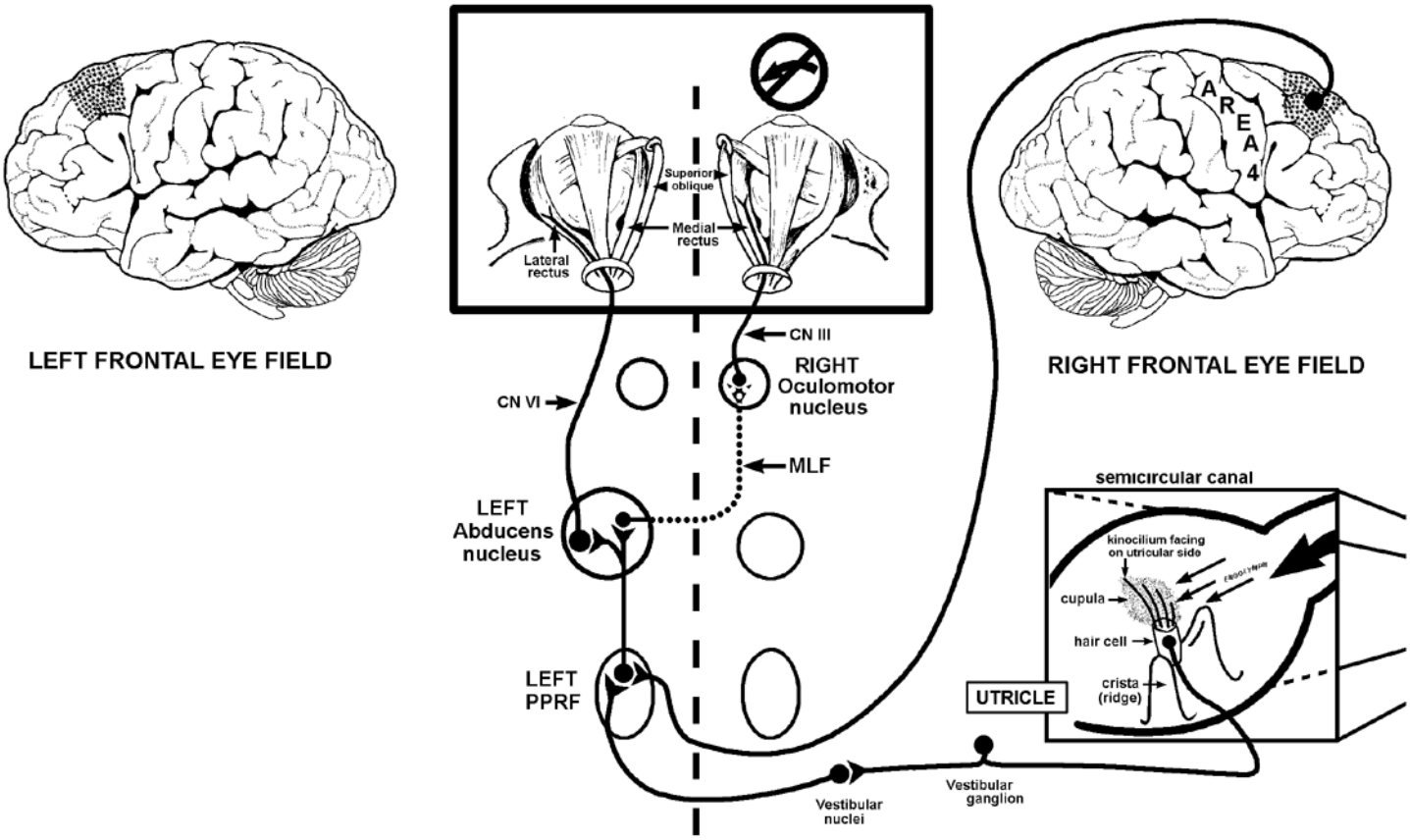
RIGHT LEFT

Rotating the head to the LEFT causes the RIGHT eye to abduct so that the visual axes are aligned. No diplopia.

"SPEED PLAY"

Diplopia is always due to a lesion in the brain stem or a peripheral station (nerve/ neuromuscular junction/muscle) but NOT cortex. A gaze palsy (impairment of both eyes in one direction, yet both eyes move conjugately and remain aligned) is due to a lesion in cortex (frontal eye field) or brain stem (PPRF) but not in the periphery.

A lesion in the **RIGHT MLF** involving the ascending axons of the neurons in the **LEFT** abducens will result in the inability to turn the **RIGHT** eye medially when attempting to look to the **LEFT**. This is called **INTERNUCLEAR OPHTHALMOPLEGIA** (INO; between the nuclei [6+3], paralysis of eye muscles). Because the **RIGHT** medial rectus has lost its drive (from the **LEFT** abducens) the **RIGHT** eye will deviate a little to the **RIGHT** when looking straight ahead and **there will be diplopia**. Turning the head to the <u>left</u> will ameliorate the diplopia. A final interesting finding in INO is that following a lesion of the (for example) <u>right</u> MLF, when the patient attempts to look left, the left eye will exhibit nystagmus. There are several hypotheses regarding this condition, but we will not delve into them at this time. Think about why this might happen.



Lesion of the RIGHT MLF = inability to turn the RIGHT eye past the midline to the LEFT = INTERNUCLEAR OPTHALMOPLEGIA (INO), <u>No</u> atrophy of the RIGHT medial rectus, Diplopia when attempting to look LEFT of the midline, also there is nystagmus of the LEFT eye

We can produce nystagmus in people with normal vestibular circuitry. For example, you can elicit vestibular nystagmus by seating a subject in a darkened room (it is dark so the patient cannot fixate on objects to reduce the nystagmus, like a figure skater), and rotating him/her in one direction. For example, if you rotate the subject to the **RIGHT**, the eyes will move to the **LEFT** and snap back to the **RIGHT** (a **RIGHT** NYSTAGMUS). Motion to the **RIGHT** turns on the hair cells in the **RIGHT** horizontal semicircular canal and I know you can take it from here! Realize that when the subject spins to the **RIGHT**, he/she will initially have a **RIGHT** nystagmus, but after rotation at a constant speed for a while, the endolymph will catch up and the nystagmus will cease. When the subject is brought to an abrupt halt the hair cells in the **LEFT** horizontal semicircular canals will now be turned on. Like those in the **RIGHT** ampulla of the **RIGHT** horizontal semicircular canal, the hair cells are polarized towards the utricle. This will make the **LEFT** side the driving side, thus pushing the eyes slowly to the **RIGHT** after which they snap back to the **LEFT** (i.e., there is a **LEFT** nystagmus).

DON'T FORGET THE DESCENDING VESTIBULOSPINAL PATHWAYS. THINK ABOUT THE DRIVING SIDE CAUSING THE ARMS AND LEGS TO BE SO ACTIVE THAT THEY PUSH YOU TOWARDS THE OPPOSITE SIDE. THUS A PERSON WITH A LEFT NYSTAGMUS (LEFT SIDE IS DRIVING) WILL FALL OR STUMBLE TO THE RIGHT.

You also need to be aware of **caloric nystagmus**. If a subject tilts his or her head back (so that the horizontal canals are oriented vertically), and one ear is irrigated with either warm or cold water, nystagmus will result. For instance, irrigation of the **RIGHT** ear with **WARM** water will turn on the receptors in the **RIGHT** horizontal canal (because the endolymph flows **towards** the utricle). This will result in the **RIGHT** side "driving" the system. **RIGHT** side dominance means that the eyes will go slowly to the **LEFT** and then snap back to the **RIGHT** (a **RIGHT NYSTAGMUS**; warm=nystagmus same side). Cooling the **RIGHT** ear would give the opposite results. Just remember **COWS** = **cold opposite, warm same**. You should understand **COWS** under normal conditions. I **WILL NOT, REPEAT, WILL NOT**, ask you questions on the exam regarding the results of caloric testing following the various lesions I have just presented. This is beyond the scope of this course.

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

____1. right hypoglossal nucleus

____2. right rostral nucleus solitarius

____3. right spinal tract V

____4. left abducens nucleus

____5. left vestibular nerve

A. receives input from cells in the left geniculate ganglion

B. lesion results in right nystagmus

C. lesion results in a loss of taste from the left side of the tongue

D. cells project to the right VPM

E. lesion results in stumbling to the right

F. lesion results in the inability to turn both eyes past the midline to the left

G. lesion results in a loss of pain and temp from the anterior two-thirds of the right side of the tongue

H. projects to the left VPM

I. nucleus receives corticobulbar input from the left motor cortex

J. lesion results in a decreased heart rate

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

____1. left PPRF

____2. left MLF

____3. right vestibular ganglion

____4. right oculomotor nucleus

____5. left frontal eye field (FEF)

A. cells project to the left vestibular nuclei

B. receives input from the left vestibular ganglion

C. lesion results in a loss of taste from the left side of the tongue

D. terminates in the right VPM

E. lesion results in stumbling to the right

F. lesion results in inability to turn both eyes past the midline to the left

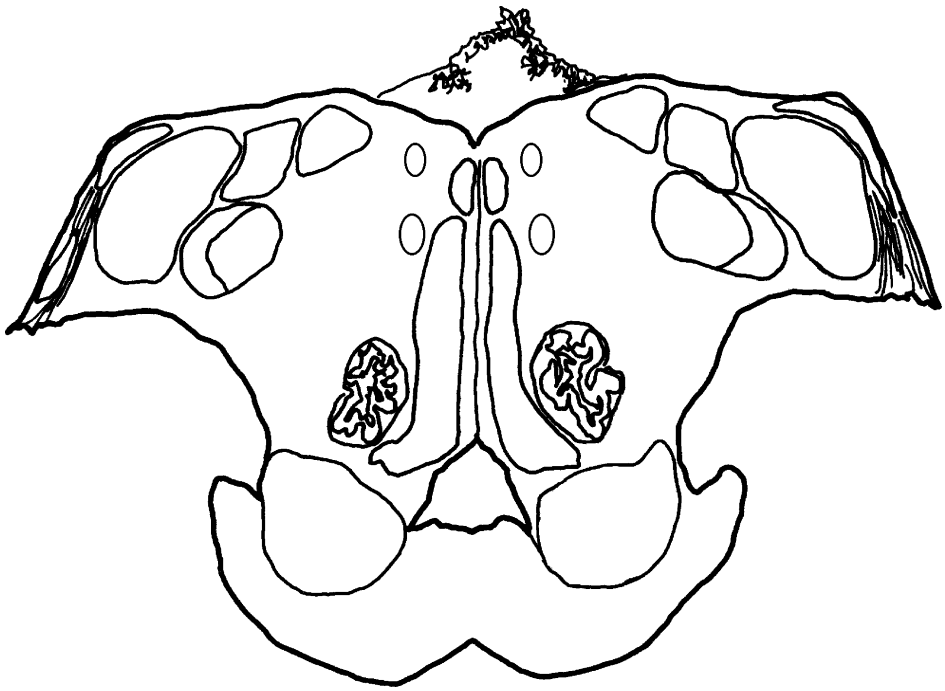
G. lesion results in loss of pain and temp from the anterior two-thirds of the right side of the tongue

H. contains cells that project to the right PPRF

I. receives input from cells in the left abducens nucleus

J. lesion results in the inability to turn the left eye past the midline to the right (medially)

PROBLEM SOLVING

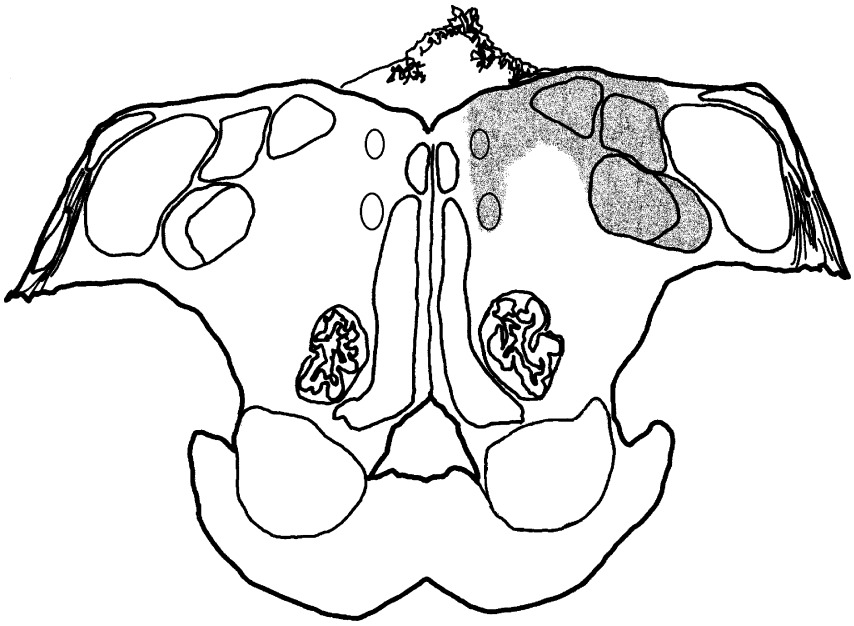


RIGHT LEFT

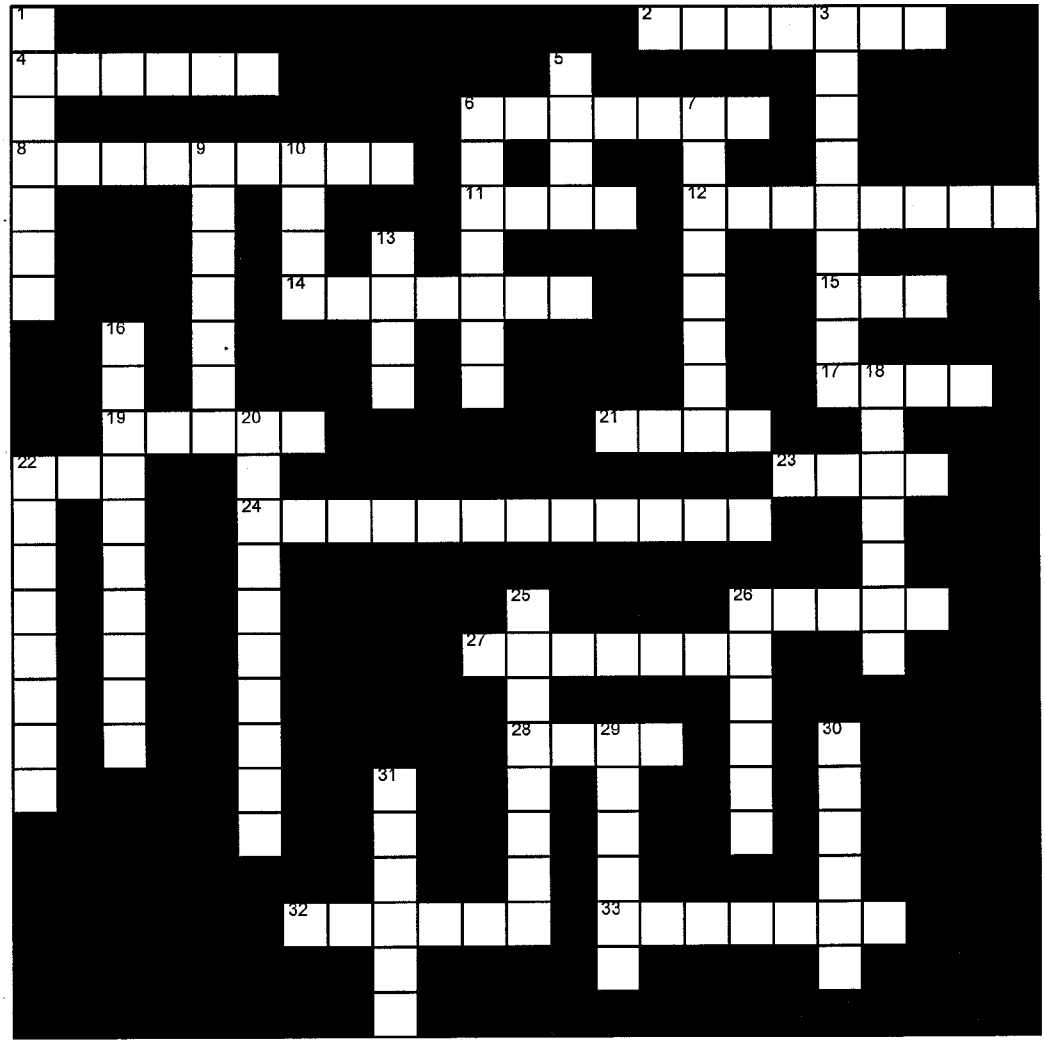
Shade in the location of a single, continuous, unilateral lesion in the above drawing that will account for the following neurological problems:

dizziness, nausea, stumbling to the left, loss of taste from the left side of the tongue, bilateral loss of pain and temperature from the face and tongue

PROBLEM SOLVING ANSWER



RIGHT LEFT

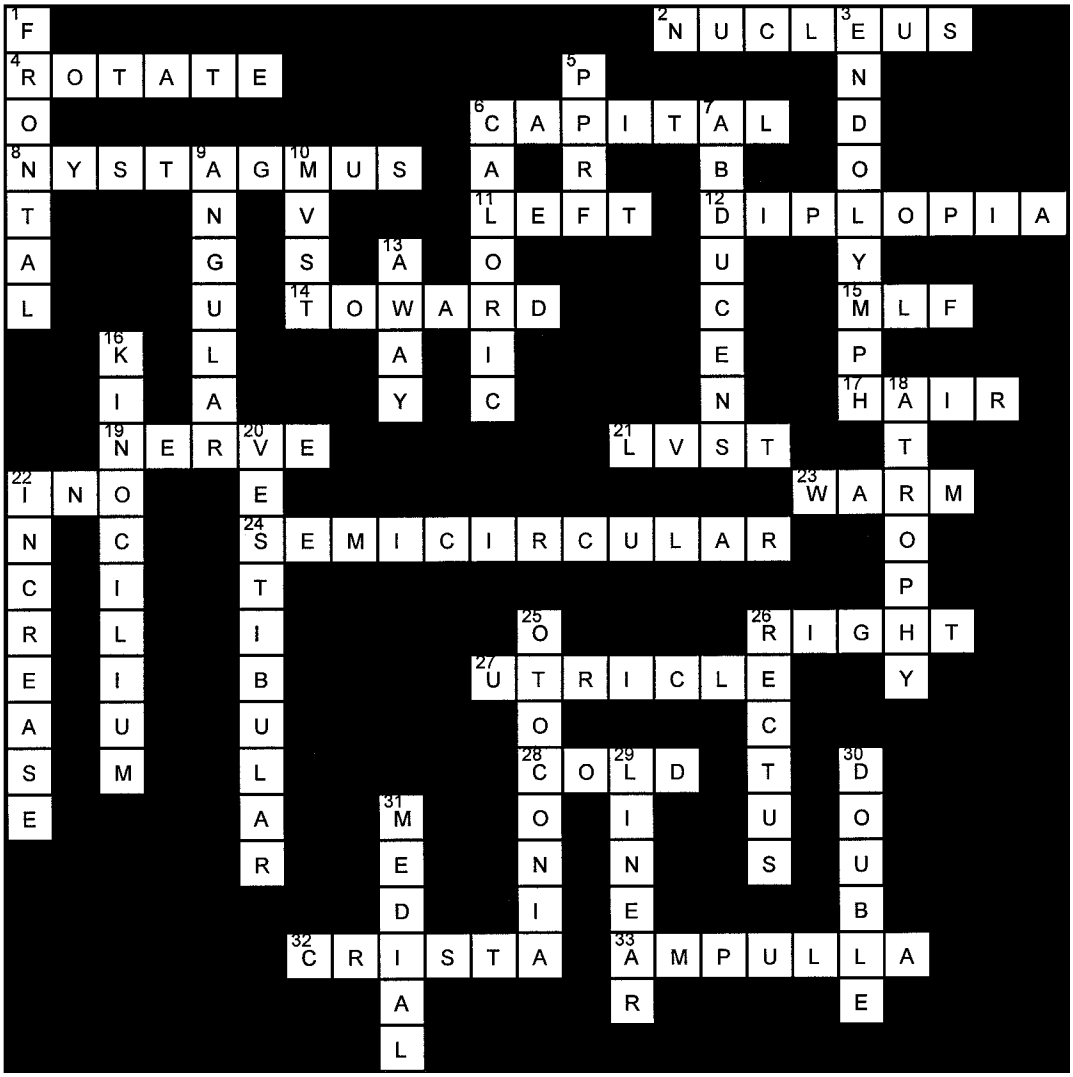


ACROSS

2. aggregation of cells with a common function
4. what you do with your head following a lesion of LR
6. what image you see two of _____dome
8. problem with eyes seen following lesions of C.N.VIII (vestibular part) and vestibular nucleus
11. direction of nystagmus when right ear irrigated with cold water
12. present following a lesion of the abducens nerve, but not after a lesion of the PPRF
14. kinocilia in horizontal semicircular canal face _____ the utricle
15. lesion of reduces the drive on the medial rectus neurons in the oculomotor complex, but does not result in atrophy of the medial rectus
17. _____cells in the SSCs (JKH lacks this)
19. lesion of abducens _____ results in diplopia
21. arises from lateral vestibular nucleus
22. results from a lesion of the MLF
23. temperature of water irrigated into the left ear to cause stumbling to the right
24. _____ canals
26. direction of left eye following a lesion of the left abducens nerve
28. temperature of water that causes right nystagmus when irrigated into the left ear
32. found in ampulla of semicircular canal; (L.-ridge)
33. dilation, swelling; (L.- little jar)

DOWN

1. _____eye field
3. fluid inside semicircular canal
5. projects to ipsi abducens, receives from contra vestibular nucleus
6. tests for integrity of vestibular apparatus; _____test
7. projects to both LR muscle and oculomotor nucleus
9. type of acceleration when you rotate your head
10. bilateral vestibulospinal tract
13. following a lesion of the frontal eye field and adjacent motor cortex, the eyes look _____(from) the hemiplegia
16. the big cilium
18. what happens to the lateral rectus following a lesion of the abducens nerve
20. one division of C.N.VIII
22. what happens to firing of the right C.N. VIII fibers (vestibular part) following head rotation to the right
25. ear dust or stones
26. second half of name of muscle that is innervated by MLF; (L. straight)
29. type of acceleration that occurs to body when the elevator suddenly plunges down the shaft
30. L.-diplous
31. direction of eye deviation following a lesion of the abducens nerve


1 F
2 N U C L E U S
3 N
4 R O T A T E
5 P
O
6 C A P I T A L D
7
8 N Y S T A G M U S A R B O
9
10
T N V 11 L E F T 12 D I P L O P I A
A G S 13 A O U Y
L U 14 T O W A R D C 15 M L F
16 K L A I E P
I A Y C N 17 H 18 A I R
19 N E R 20 V E 21 L V S T T
22 I N O E 23 W A R M
N C 24 S E M I C I R C U L A R O
C I T P
R L I 25 O 26 R I G H T
E I B 27 U T R I C L E Y
A U U O C
S M L 28 C O 29 L D T 30 D
E A 31 M O I U O
R E N N S U
D I E B
32 C R I S T A 33 A M P U L L A
A R E
L