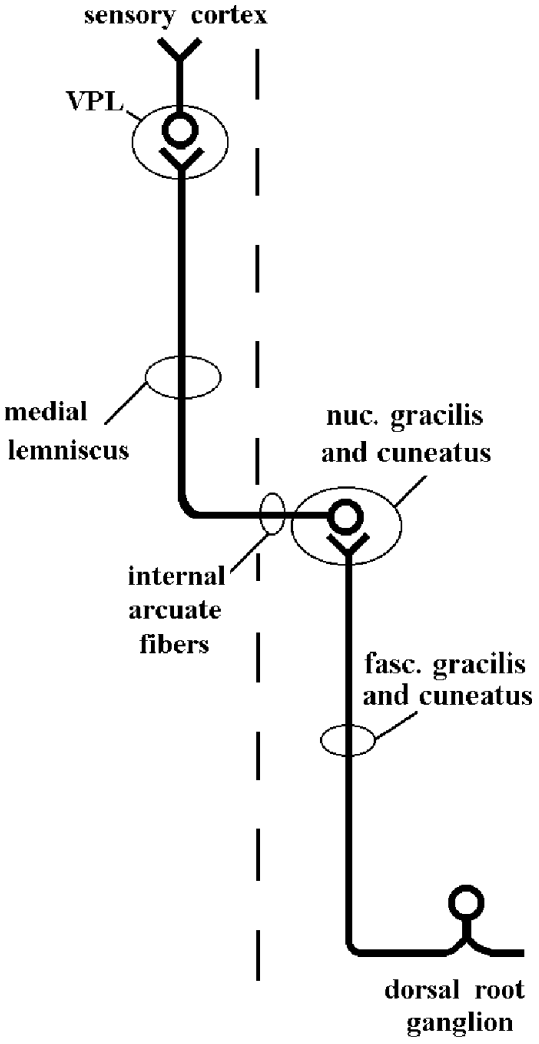
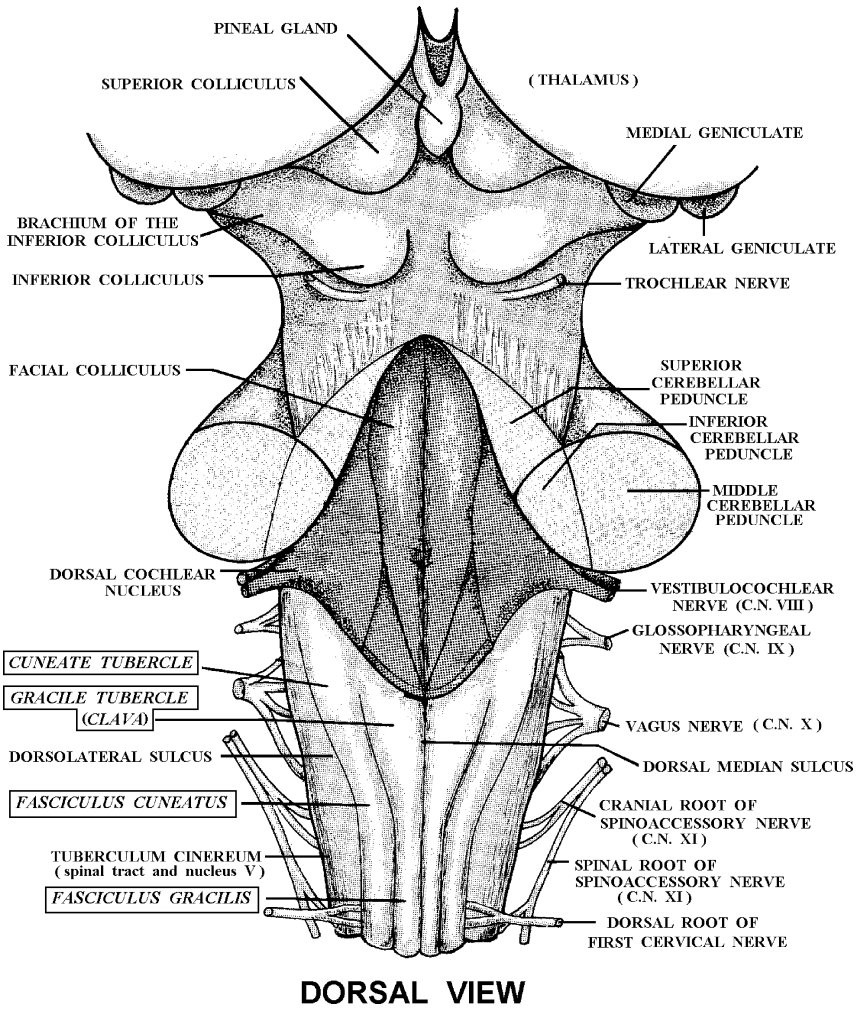
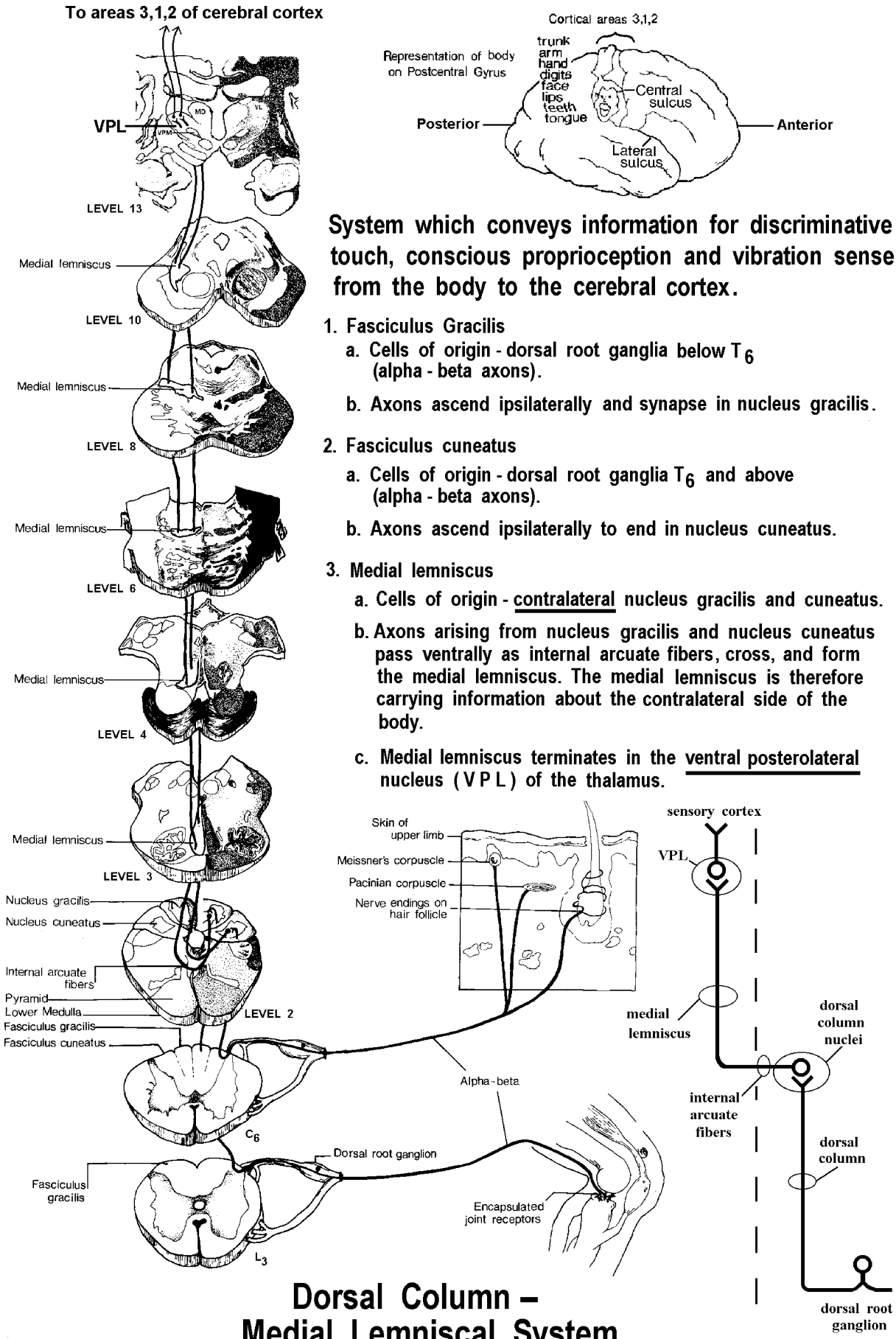
4 NUCLEUS GRACILIS AND CUNEATUS AND THE MEDIAL LEMNISCUS

As you learned in the spinal cord module, axons in the **fasciculus gracilis** and **fasciculus cuneatus** are the **uncrossed** central processes of alpha-beta fibers whose cell bodies lie in the **dorsal root ganglia**. Fibers in fasciculus gracilis end in **nucleus gracilis**, while fibers in fasciculus cuneatus end in **nucleus cuneatus**.

Nucleus gracilis and nucleus cuneatus lie in the dorsal portion of the caudal medulla. These two nuclear groups and their associated fasciculi can be seen as slight swellings on the dorsal surface of the medulla. The bump formed by nucleus and fasciculus gracilis is called the **gracile tubercle** (or **clava**; L., club) and that caused by nucleus and fasciculus cuneatus is called the **cuneate tubercle**. These bumps can be seen on both of the drawings shown below.







System which conveys information for discriminative touch, conscious proprioception and vibration sense from the body to the cerebral cortex.

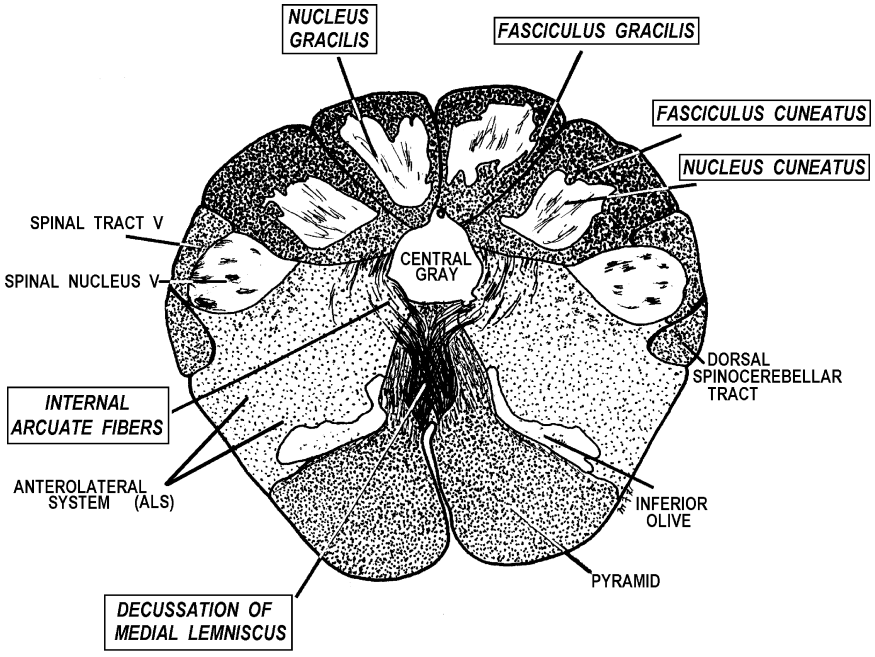
1. Fasciculus Gracilis
   a. Cells of origin - dorsal root ganglia below T_6 (alpha - beta axons).
   b. Axons ascend ipsilaterally and synapse in nucleus gracilis.
2. Fasciculus cuneatus
   a. Cells of origin - dorsal root ganglia T_6 and above (alpha - beta axons).
   b. Axons ascend ipsilaterally to end in nucleus cuneatus.
3. Medial lemniscus
   a. Cells of origin - contralateral nucleus gracilis and cuneatus.
   b. Axons arising from nucleus gracilis and nucleus cuneatus pass ventrally as internal arcuate fibers, cross, and form the medial lemniscus. The medial lemniscus is therefore carrying information about the contralateral side of the body.
   c. Medial lemniscus terminates in the ventral posterolateral nucleus (VPL) of the thalamus.

Dorsal Column – Medial Lemniscal System

Axons of cells within nucleus gracilis and nucleus cuneatus cross as **internal arcuate fibers** and form the **MEDIAL LEMNISCUS**. The medial lemniscus is thus a large ascending bundle of heavily myelinated axons (fast conducting) whose cell bodies lie in the contralateral nucleus gracilis and nucleus cuneatus. The medial lemniscus passes rostrally through the medulla, pons and midbrain to terminate in the **ventral posterolateral** (VPL) nucleus of the thalamus. Cells in the VPL then send their axons to the postcentral gyrus (somatosensory cortex) of the cerebral cortex (areas 3, 1, 2).

The dorsal column-medial lemniscal system carries information from specialized **touch, pressure, vibration, and joint receptors** to the cerebral cortex. While lesions of the dorsal columns (fasciculi gracilis and cuneatus) in the spinal cord result in **IPSILATERAL** deficits, lesions of the medial lemniscus, in the brain stem, result in **CONTRALATERAL** deficits (since its constituent axons have crossed). This is important to understand. Also, do not confuse **VPM** (head, trigeminal, trigeminothalamic) with **VPL** (body, medial lemniscus).

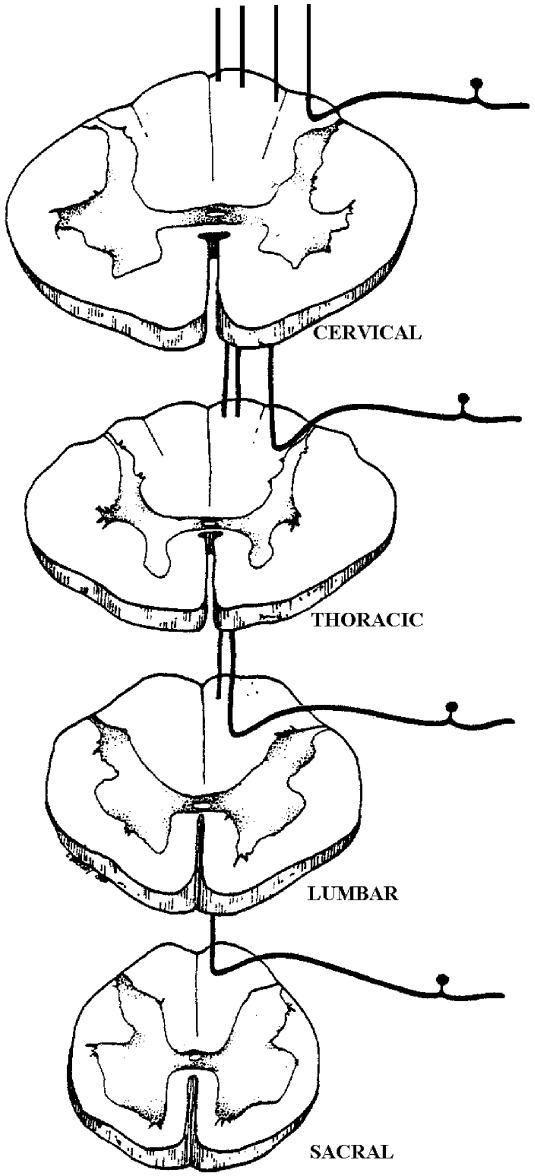
Lesions of the medial lemniscus results in loss of 2 pt. discrimination, vibration and conscious proprioception from the **contralateral** side of the body. Also remember, there is astereognosia, agraphesthesia, and a Romberg sign.



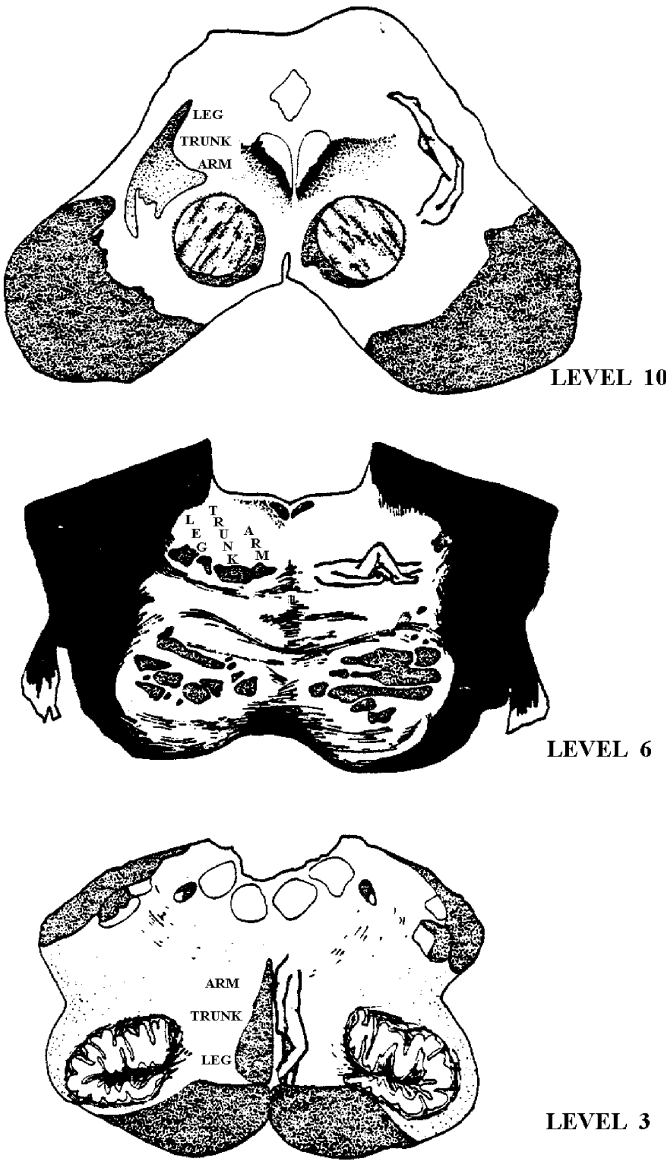
LEVEL 2. DECUSSATION OF MEDIAL LEMNISCUS

You may remember that there is a somatotopic representation of the body in the dorsal columns (fasciculus gracilis and fasciculus cuneatus). **Caudal** (sacral and lumbar) body parts are represented **medially**, while the **rostral** segments (upper thoracic and cervical) are represented **laterally**. Nucleus gracilis receives its input from about T_7 and downward, while nucleus cuneatus receives its input from spinal levels above this.

There is also a somatotopic organization within the medial lemniscus. For instance, in the medullary segment of the medial lemniscus the body is represented in an **upright** position, so that the legs are ventral and the arms dorsal. Within the pontine portion of the medial lemniscus there is a rotation, so that the arms are represented medially and the legs laterally. Finally, in the midbrain portion of the medial lemniscus the arms are represented ventrally while the legs are represented dorsally.

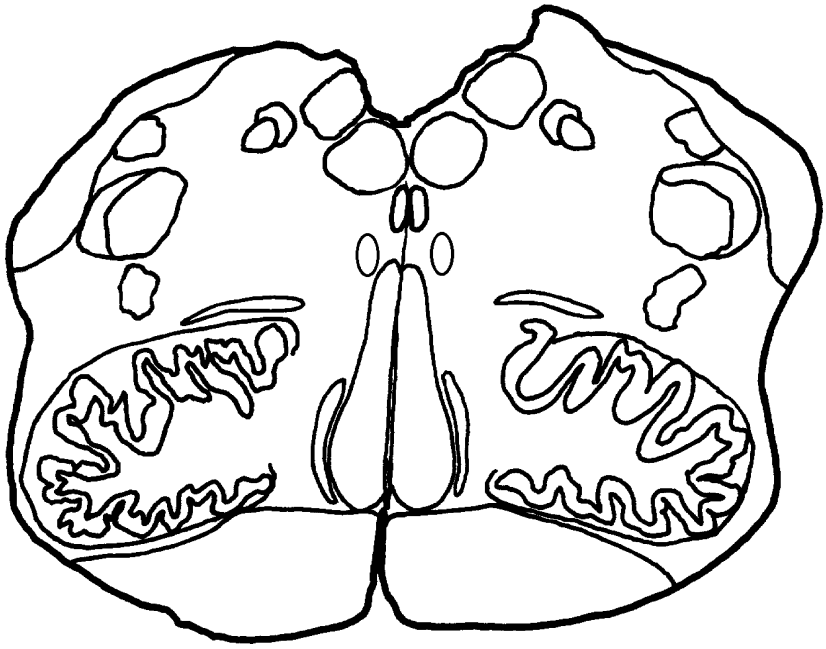


SOMATOTOPIC ORGANIZATION OF THE DORSAL COLUMNS



SOMATOTOPIC ORGANIZATION OF THE MEDIAL LEMNISCUS

PROBLEM SOLVING

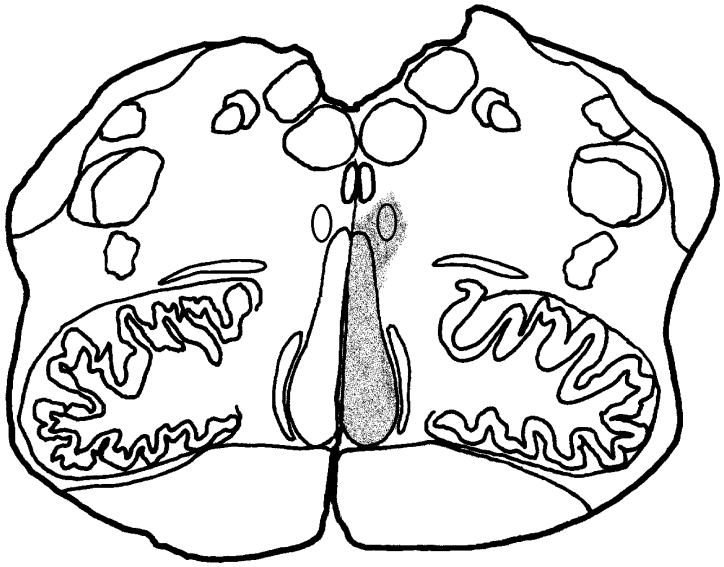


RIGHT LEFT

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

stabbing pain in the right eye followed by the loss of pain and temperature from the right side of the face, loss of vibratory sense from the right arm, loss of vibratory sense from the right leg

PROBLEM SOLVING ANSWER



RIGHT LEFT

5 ACCESSORY CUNEATE NUCLEUS (Lots of hard work for such a TINY nucleus!)

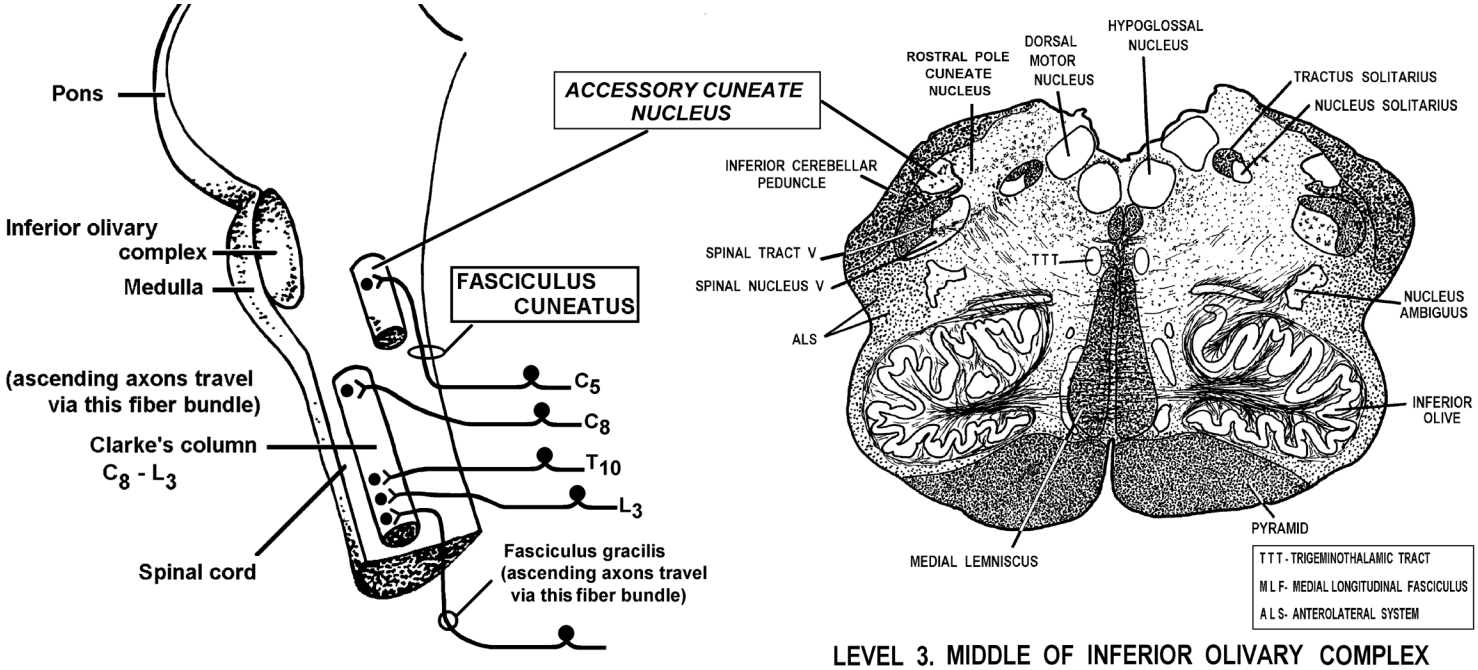
You should recall from the spinal cord module that proprioceptive information from **muscle spindles** (Ia, II) and **Golgi tendon organs** (Ib) reaches the cerebellum via the **dorsal spinocerebellar tract**. The cells of origin of this tract lie in the ipsilateral Clarke's column. This column of cells is present only at spinal cord levels C_8-L_3. Central processes of dorsal root neurons that enter caudal to L_3 have to ascend to reach L_3. Consequently, Clarke's column is quite enlarged caudally. (Clarke's column neurons at L_3 need to serve not only entering fibers at L_3, but all of those entering below L_3.)

Ia, Ib and type II axons of the dorsal root ganglia **rostral** to Clarke's column (C_8) pass rostrally to reach the ipsilateral **caudal medulla**, where they end within the **ACCESSORY CUNEATE** ("wedge-shaped") **NUCLEUS**. This nucleus, which is somewhat difficult to see, lies dorsal to the spinal tract and nucleus V and lateral to the most rostral pole of nucleus cuneatus. Cells in the accessory cuneate nucleus send their axons to the **IPSILATERAL CEREBELLUM** via a fiber bundle called the **INFERIOR CEREBELLAR PEDUNCLE** (together with the dorsal spinocerebellar fibers). This pathway is called the **CUNEOCEREBELLAR TRACT**.

The accessory cuneate nucleus is concerned with relaying proprioceptive information from the arm (and neck) to the cerebellum, and the nucleus can be considered as the rostral equivalent of Clarke's column.

REMEMBER: Accessory cuneate nucleus:

1). lies in the medulla.
2). receives **UNCROSSED** fibers from dorsal root ganglia above C_8.
3). receives the same kind of information that Clarke's column does.
4). projects to the **IPSILATERAL** cerebellum via inferior cerebellar peduncle.
5). is concerned with the arm, while Clarke's column is concerned with the forearm, trunk and lower extremity.

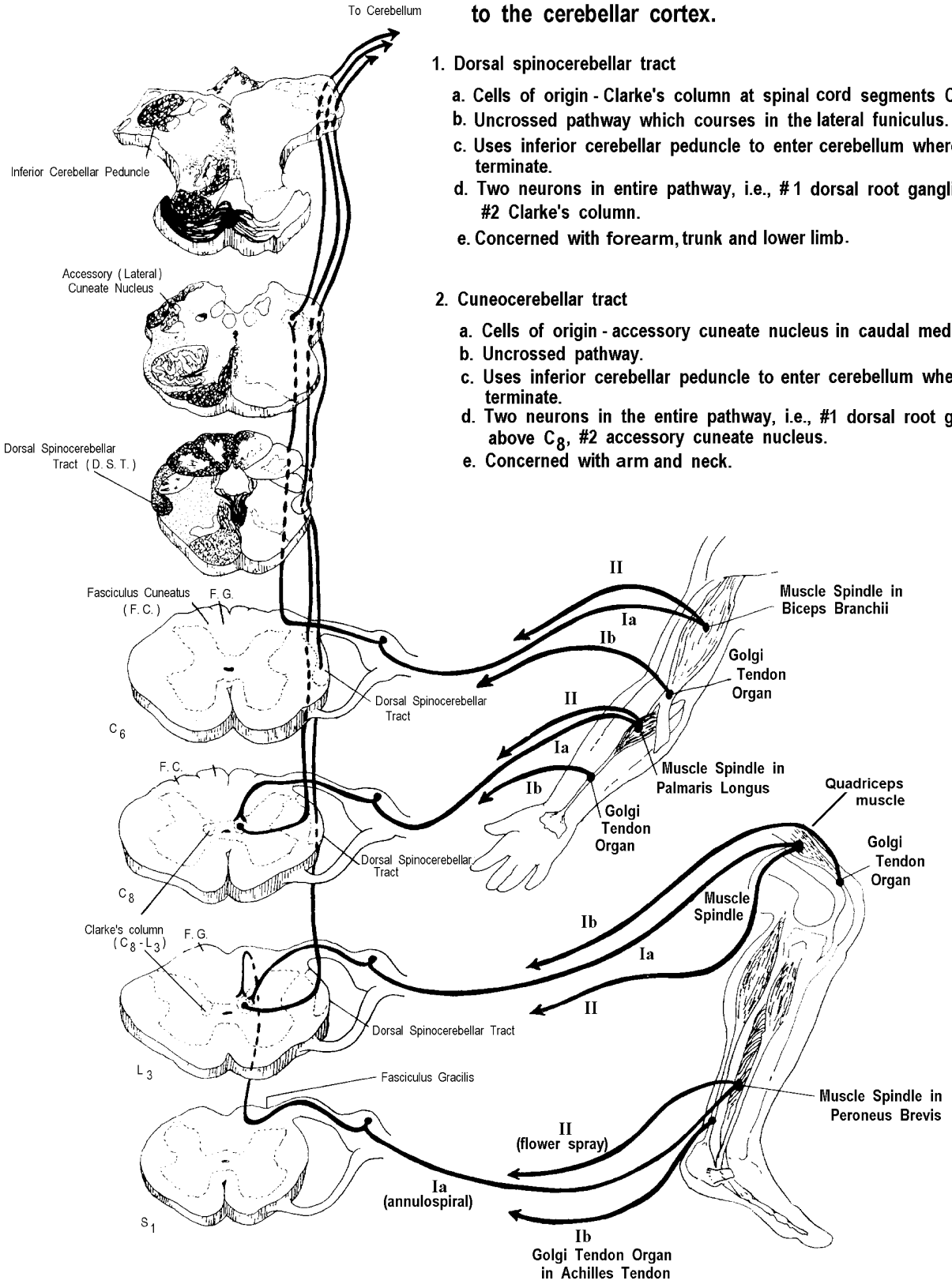


LEVEL 3. MIDDLE OF INFERIOR OLIVARY COMPLEX

Muscle spindle and Golgi tendon organ (unconscious proprioception) pathways to the cerebellar cortex.

1. Dorsal spinocerebellar tract
   a. Cells of origin - Clarke's column at spinal cord segments C_8 - L_3.
   b. Uncrossed pathway which courses in the lateral funiculus.
   c. Uses inferior cerebellar peduncle to enter cerebellum where axons terminate.
   d. Two neurons in entire pathway, i.e., #1 dorsal root ganglia, #2 Clarke's column.
   e. Concerned with forearm, trunk and lower limb.

2. Cuneocerebellar tract
   a. Cells of origin - accessory cuneate nucleus in caudal medulla.
   b. Uncrossed pathway.
   c. Uses inferior cerebellar peduncle to enter cerebellum where axons terminate.
   d. Two neurons in the entire pathway, i.e., #1 dorsal root ganglia above C_8, #2 accessory cuneate nucleus.
   e. Concerned with arm and neck.



DORSAL SPINOCEREBELLAR TRACT

Due to its small size, lesions restricted to the accessory cuneate nucleus are rare. Understanding the laterality (which side) and specific deficits resulting from a lesion of the accessory cuneate requires knowing something about **CEREBELLAR** functions and connections. While this important topic is covered later in this course, we need to do some limited spade work right now to get you prepared for those lectures, and also to let you problem solve on questions regarding lesions of brain stem areas that either project to the cerebellum (like the **accessory cuneate nucleus**, inferior olive and pontine grey [the latter two will be discussed further up the brain stem]) or contain axons leaving the cerebellum (superior cerebellar peduncle).

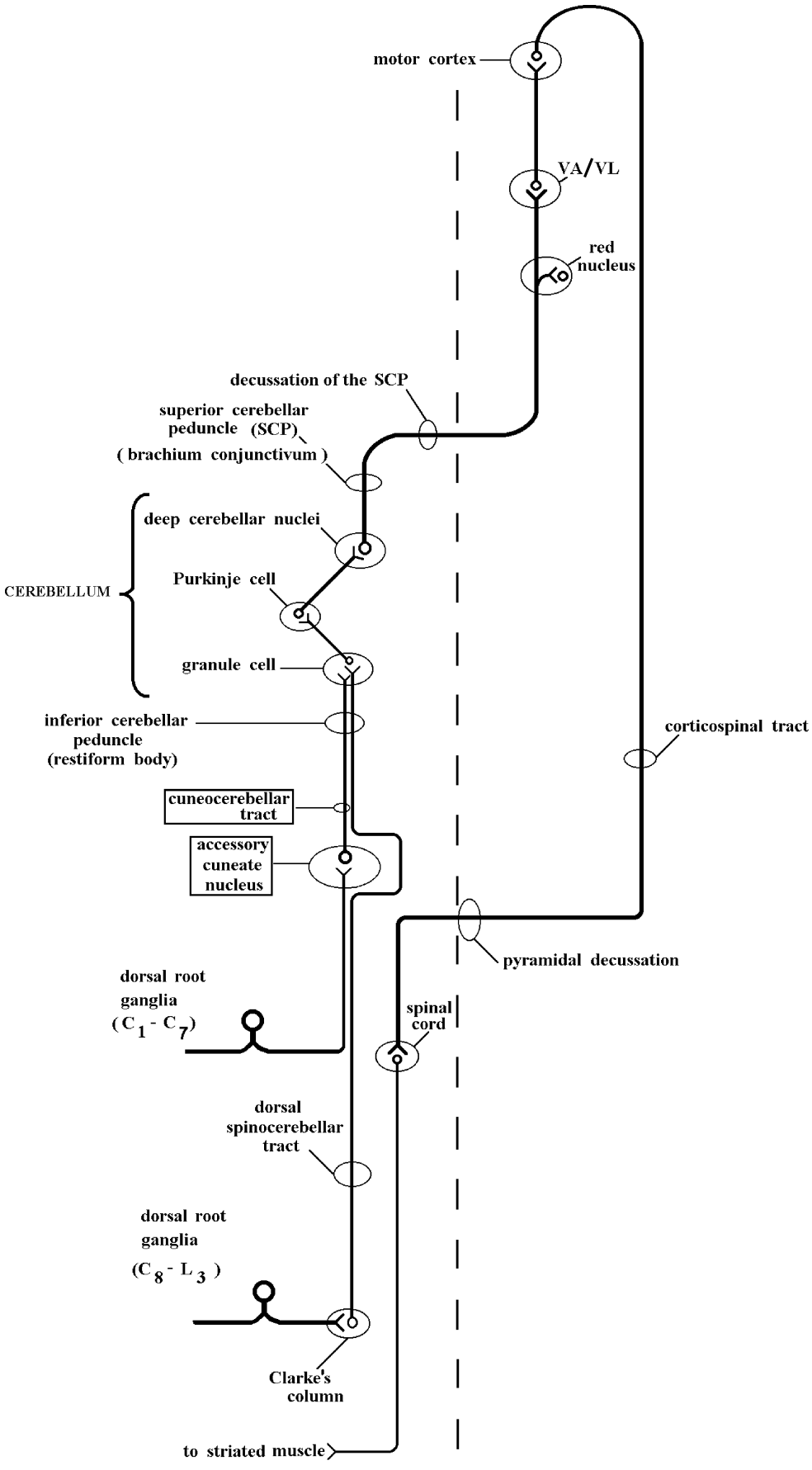
The cerebellum is involved in **motor coordination**. Unlike the cerebrum (i.e., cerebral cortex; cells of origin of the corticospinal tract), the cerebellum has **no** major projections to the spinal cord, but instead regulates movement **indirectly** by projecting to areas of the brain that do project upon the spinal cord. Lesions of the cerebellum lead to defects in the coordination of movements, but **NOT** paresis or paralysis. Such cerebellar defects involve errors in the **rate, range or direction** of voluntary movements. Disturbances following cerebellar lesions are known by a variety of terms such as nystagmus, ataxia, hypotonia, dysmetria, past pointing, rebound, dysdiadochokinesia, asynergy, intention tremor and decomposition of movement (WHEY!).

RIGHT NOW, LET'S JUST CALL THESE DISTURBANCES = **INCOORDINATION/ATAXIA**
AND REMEMBER THAT **INCOORDINATION/ATAXIA** RESULTS FROM LESIONS OF THE CEREBELLUM OR ITS INPUTS (ACCESSORY CUNEATE NUCLEUS) OR OUTPUTS.

In addition to knowing that lesions of the cerebellum and its inputs and outputs result in **incoordination**, we need to know what part of the body is affected (arm, leg) and the laterality of the deficits **(IPSI**. or **CONTRA**.). The important point now is that one side of the cerebellum controls the **SAME OR IPSILATERAL SIDE OF THE BODY**.

OPTIONAL READING

This is due to **TWO DECUSSATIONS** of pathways involved in conveying cerebellar information to the spinal cord. To understand all of this, let's start at the **LEFT** accessory cuneate nucleus, which you now know receives information from the **LEFT** side of the upper extremity. Cells in the **LEFT** accessory cuneate nucleus possess axons that comprise the **LEFT cuneocerebellar** tract and synapse on cells in the **LEFT** cerebellar cortex called **granule** cells. The axons of granule cells synapse on **Purkinje** cells. Purkinje cell axons synapse on cells in the deep white matter of the cerebellum called **DEEP CEREBELLAR NUCLEI**. There are four of these deep cerebellar nuclei on each side of the cerebellum. They are called fastigial, globose, emboliform and dentate. We will **NOT** worry about these nuclei too much at this time, but we need to know that they contain cells whose axons **LEAVE** the cerebellum (efferent; exit) in a large bundle called the **SUPERIOR CEREBELLAR PEDUNCLE** (Point #17). The superior cerebellar peduncle courses rostrally and **CROSSES** in the caudal midbrain **(decussation #1)**. After crossing, axons synapse in the **RED NUCLEUS** (midbrain; we will discuss later in POINT #21; don't worry about it at this time) and in the **ventral lateral** (VL) and **ventral anterior** (VA) nuclei of the thalamus. Cells in VL and VA project to the motor cortex, which of course contains the cells of origin of the **CORTICOSPINAL TRACT**. As you know, the corticospinal tract **CROSSES** in the caudal medulla **(decussation #2)** and innervates spinal cord neurons.


motor cortex
VA/VL
red nucleus
decussation of the SCP
superior cerebellar peduncle (SCP)
(brachium conjunctivum)
deep cerebellar nuclei
CEREBELLUM
Purkinje cell
granule cell
inferior cerebellar peduncle (restiform body)
corticospinal tract
cuneocerebellar tract
accessory cuneate nucleus
pyramidal decussation
dorsal root ganglia (C_1 - C_7)
spinal cord
dorsal spinocerebellar tract
dorsal root ganglia (C_8 - L_3)
Clarke's column
to striated muscle
MIDLINE

This leads to two of the most important "rules" of neurology. That is:
CEREBELLAR PROBLEMS = IPSILATERAL,
CEREBRAL PROBLEMS = CONTRALATERAL

The accessory cuneate nucleus projects to the **IPSILATERAL CEREBELLAR HEMISPHERE** (via the inferior cerebellar peduncle, along with the dorsal spinocerebellar fibers). Therefore lesions of the **accessory cuneate nucleus**, like the cerebellum, result in **IPSILATERAL** deficits. For example, the **LEFT** accessory cuneate nucleus receives input from Ia, Ib and type II fibers of dorsal root ganglia above C_8 on the **LEFT**. The **LEFT** accessory cuneate nucleus projects to the **LEFT** cerebellar hemisphere. Finally the information leaves the cerebellum to eventually influence the **LEFT** arm. Therefore, a lesion of the **LEFT** accessory cuneate nucleus would result in "bad" information reaching the **LEFT** cerebellar hemisphere, and in turn **motor incoordination** of the **LEFT** arm. There is **NO** paralysis or atrophy of these muscles. **REMEMBER**, for our problem solving questions involving the accessory cuneate nucleus let's just focus on **INCOORDINATION/ATAXIA OF THE IPSILATERAL ARM**.

To review: a lesion of the accessory cuneate nucleus results in incoordination/ataxia of the ipsilateral arm. It could not result in a Romberg because it involves a cerebellar afferent (and does not include the lower limbs anyway). A lesion of the DSCT will involve the legs but again, it is a cerebellar afferent and therefore would not give a Romberg sign. What about a lesion of the inferior cerebellar peduncle?

PROBLEM SOLVING MATCHING

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

____1. right pyramid

____2. right anterolateral system (ALS) **and associated descending pathway**

____3. right caudal spinal nucleus V

____4. right nucleus gracilis and cuneatus

____5. left accessory cuneate nucleus

A. lesion results in a loss of vibratory sense from the left arm and leg

B. cells project to the right VPM

C. cells project to the cerebellum via the right inferior cerebellar peduncle

D. axons terminate in the right VPL

E. lesion results in a loss of 2 pt. discrimination from the left arm and leg

F. lesion results in a loss of pain and temp from the right side of the face

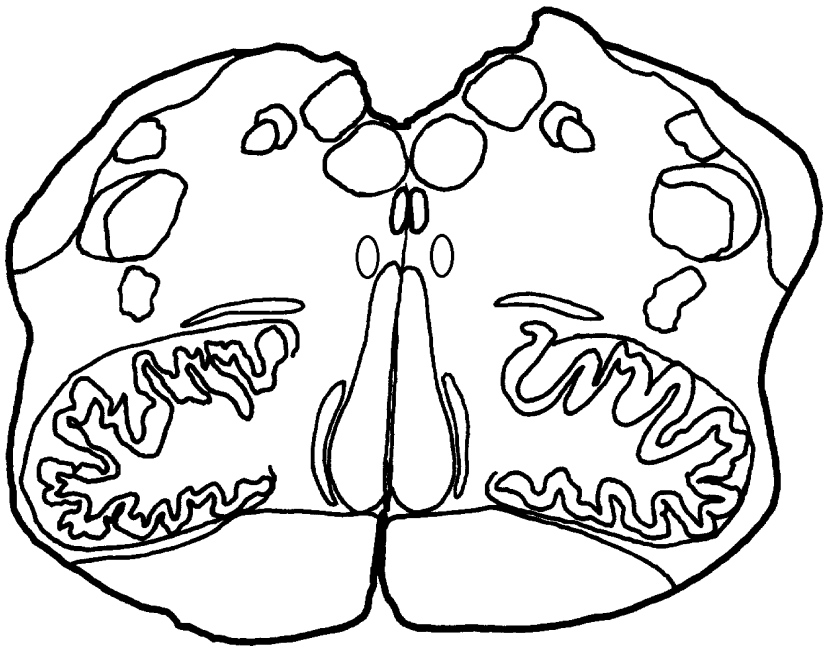
G. lesion results in a dilated pupil in the right eye

H. cells convey 2 pt. discrimination and vibratory information to the left VPL

I. cells project to the left side of the cerebellum

J. lesion results in left hemiplegia

PROBLEM SOLVING

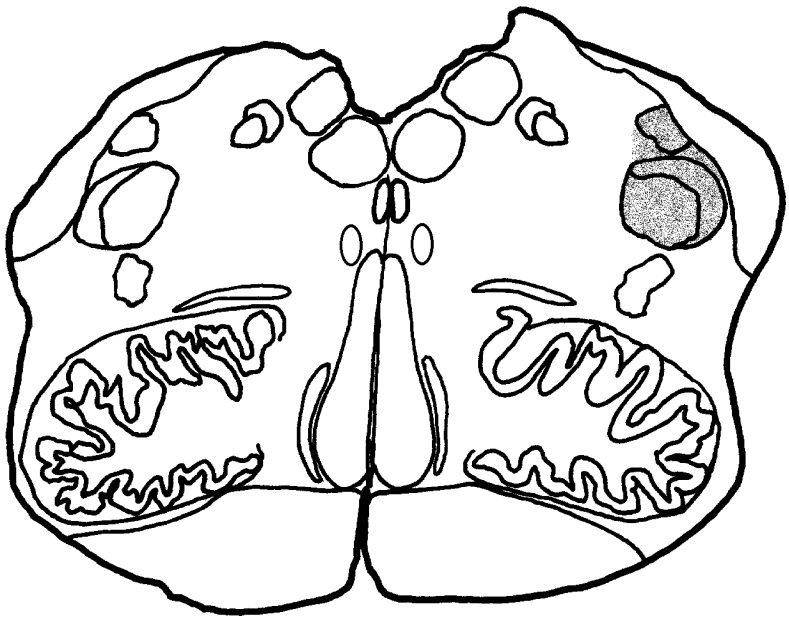


RIGHT LEFT

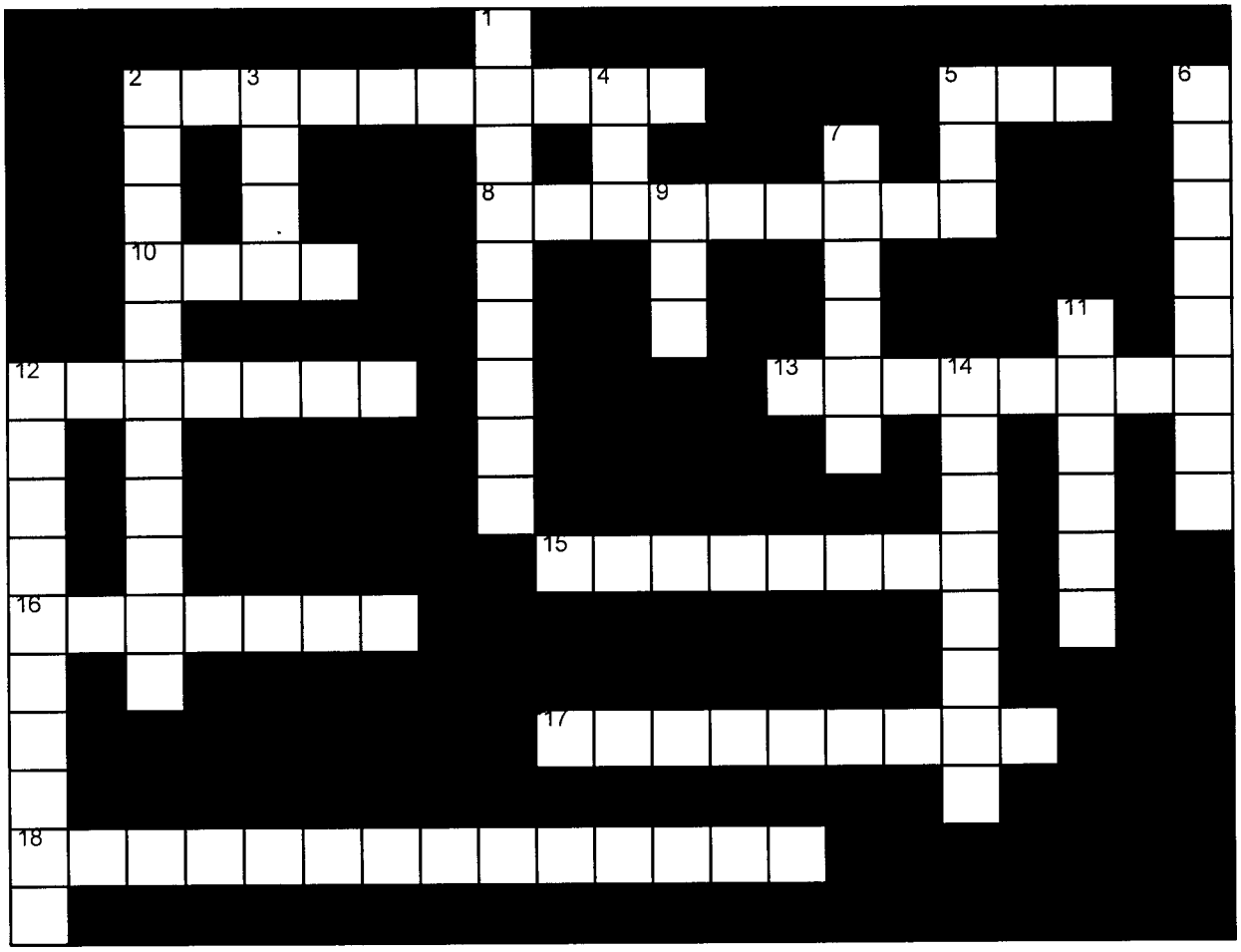
Shade in the location of a single, continuous, unilateral lesion in the above drawing that will account for the following neurological problems:

stabbing pain in the left eye followed by loss of pain and temperature from the left side of the face, incoordination of the left arm

PROBLEM SOLVING ANSWER



RIGHT LEFT



ACROSS

2. three twins
5. termination of ALS
8. _____ body
10. carried by ALS
12. constricted pupil
13. slender (L.)
15. connects brain stem and cerebellum
16. contains corticospinal fibers
17. medial _____
18. result of a lesion of accessory cuneate

DOWN

1. carried by medial lemniscus
2. carried by ALS
3. opposite of contra
4. carries pain and temp
5. target of TTT
6. dorsiflexion of big toe
7. opposite of ipsi
9. projects to VPM
11. C8-L3
12. lesion of corticospinal tract
14. vibration from arm

Problem Solving - ANSWERS POINTS 1-5

ANSWERS TO PROBLEM SOLVING QUESTIONS RELATED TO POINTS 1-5

NOTE: The answers to ALL shade-in questions are illustrated on the back side of the question.

Point #1 Pyramid

Matching D

Point #2 Anterolateral System

Matching G,E

Point #3 Spinal Nucleus and Tract V

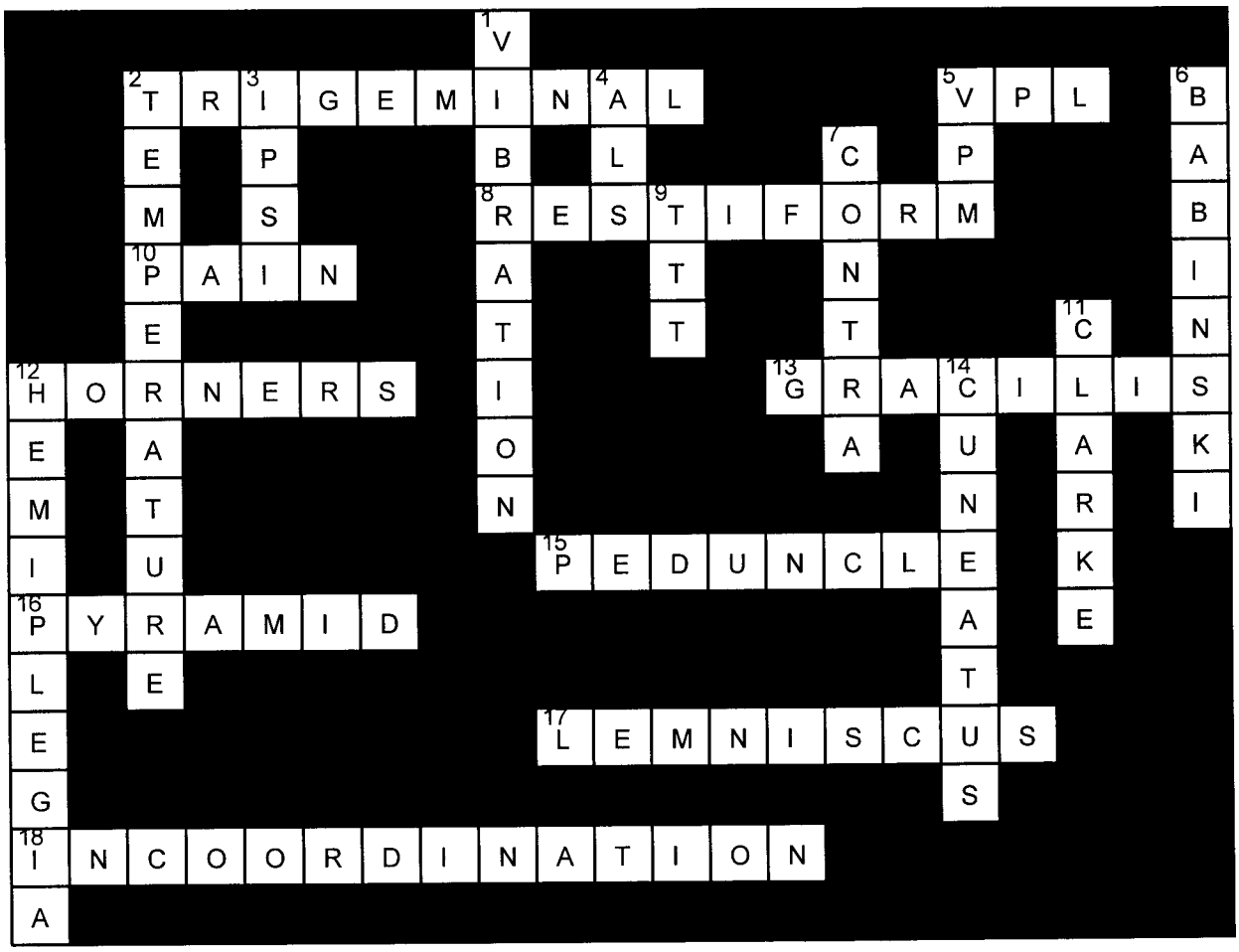
Matching B,H,D

Point #4 Nucleus Gracilis and Cuneatus

Matching D,I,F,J

Point #5 Accessory Cuneate Nucleus

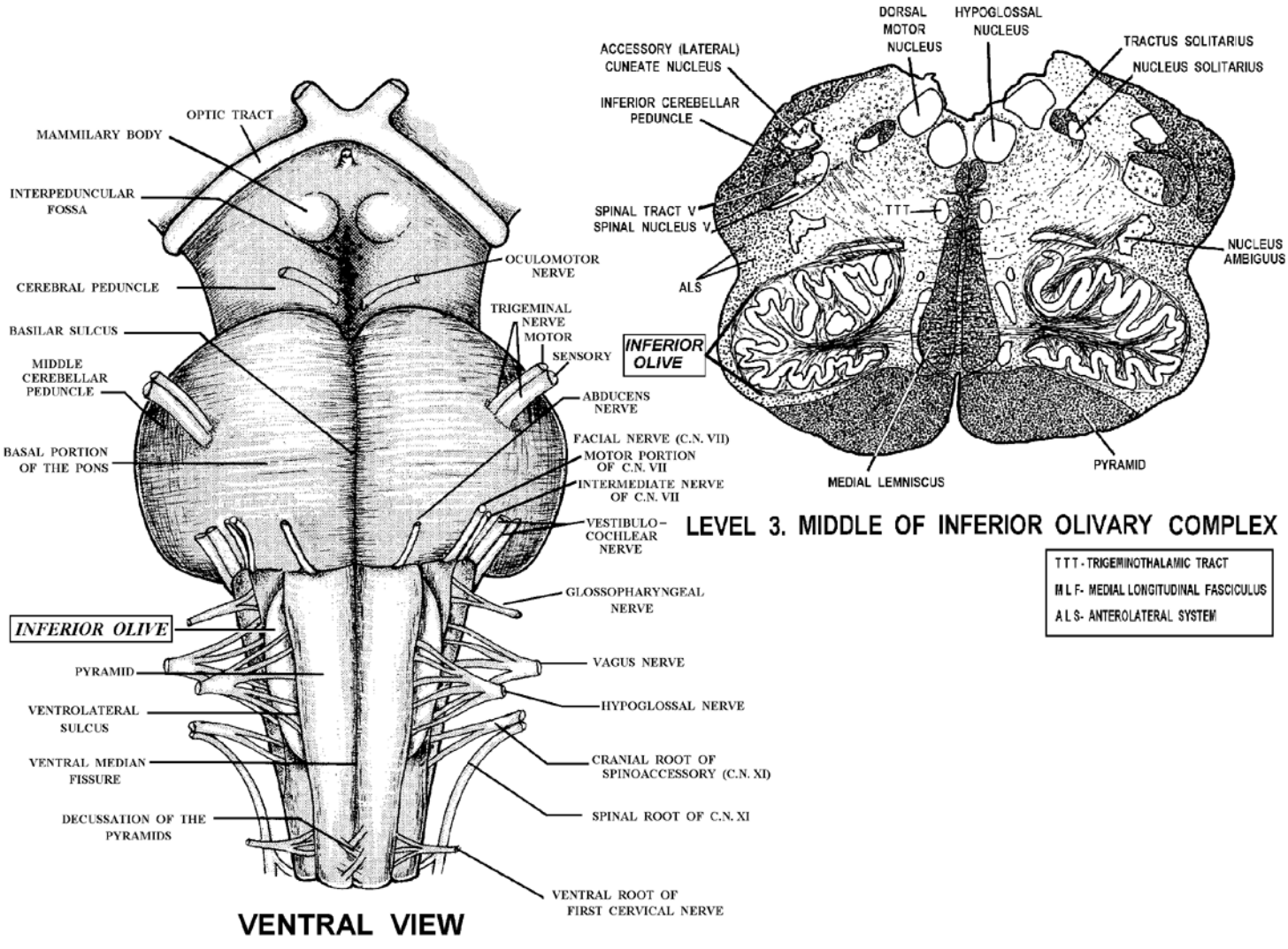
Matching J,D,F,H,I



6 INFERIOR OLIVARY COMPLEX

This is the largest nuclear group in the brain stem. It consists of a convoluted band of cells that lie dorsal to the pyramid. This nucleus is by far the most characteristic and striking feature of the medulla. Sadly, we know little about inferior olivary function(s), but its very intimate association with the cerebellum suggests it is involved in motor coordination and most likely motor "learning".

Cells in the inferior olivary complex project to the **contralateral cerebellum** via the **inferior cerebellar peduncle** (or restiform body). Upon reaching the cerebellum, they end as "**CLIMBING FIBERS**" (they "climb up" the Purkinje cells; more on this later in the course). Climbing fibers arise solely from the inferior olive. Other endings seen in the cerebellar cortex are called **mossy** fibers. Mossy fibers do not arise from the inferior olive, but rather from places like Clarke's column and the accessory cuneate nucleus. Thus, axons in the dorsal spinocerebellar and cuneocerebellar tracts end as "mossy fibers." Inputs to the inferior olive will be discussed during the "Cerebellum" part of the course.



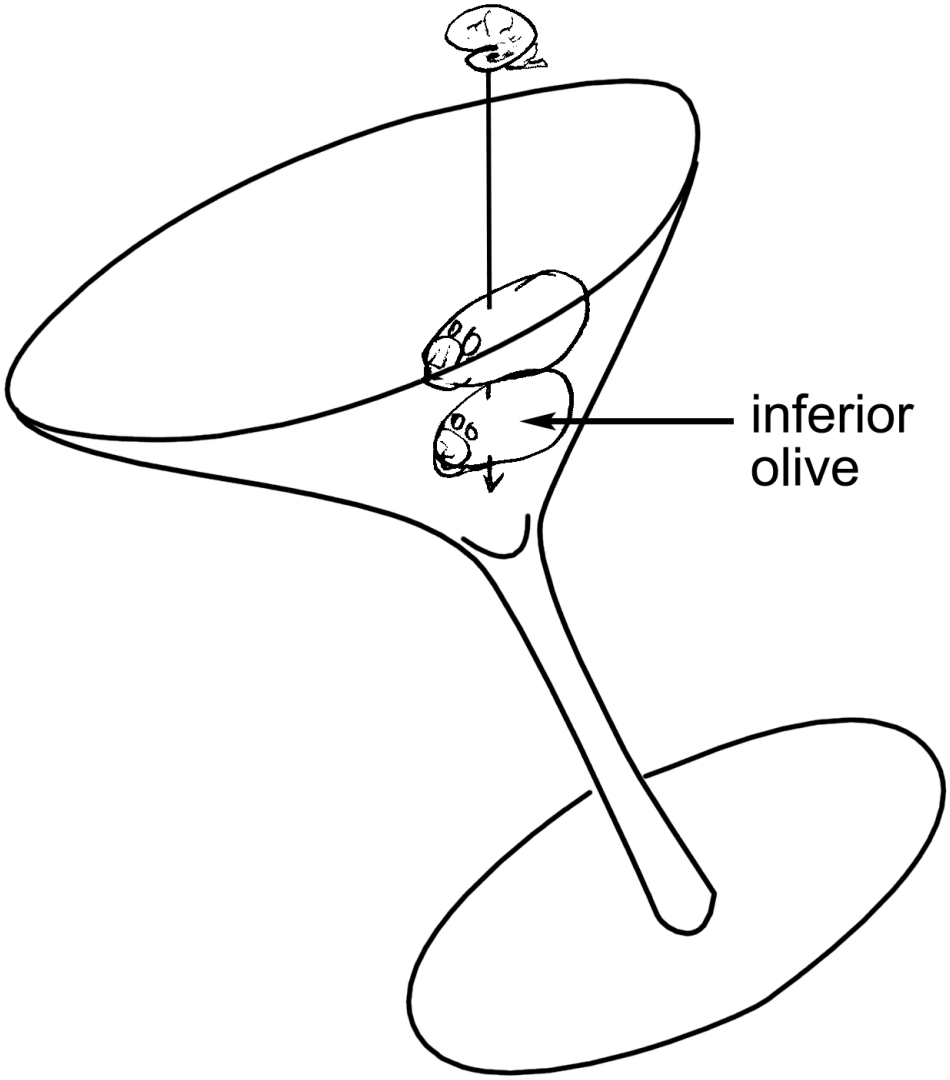
While many questions remain regarding the function(s) of the inferior olive, selective destruction of this nuclear complex in experimental animals has acute effects similar to those following destruction of the entire one-half of the **CONTRALATERAL** cerebellum. Since the inferior olive sends information to the **contralateral** cerebellum, and the cerebellum influences the **SAME** side of the body, then the loss of the **LEFT** olive will mean that the **RIGHT** half of the cerebellum is no longer receiving input from the inferior olive. This will result in incoordination/ ataxia of the **RIGHT** side of the body. Since we will cover cerebellum later in this course, don't worry too much about it right now. We have already discussed that cerebellar deficits involve **incoordination/ataxia** and are **IPSILATERAL** to the side of the lesion. A lesion of the inferior olive will result in incoordination/ataxia of the **CONTRALATERAL ARM AND LEG** (contrast this with a lesion of **ACC. CUNEATE NUC.** = incoordination/ataxia of **IPSI ARM**). What about a Romberg sign? Well, this is like a DSCT lesion. Loss of input from the olive means that you are not going to be able to stand with your feet together to begin with!! So no Romberg! Besides, it's a cerebellar afferent!!

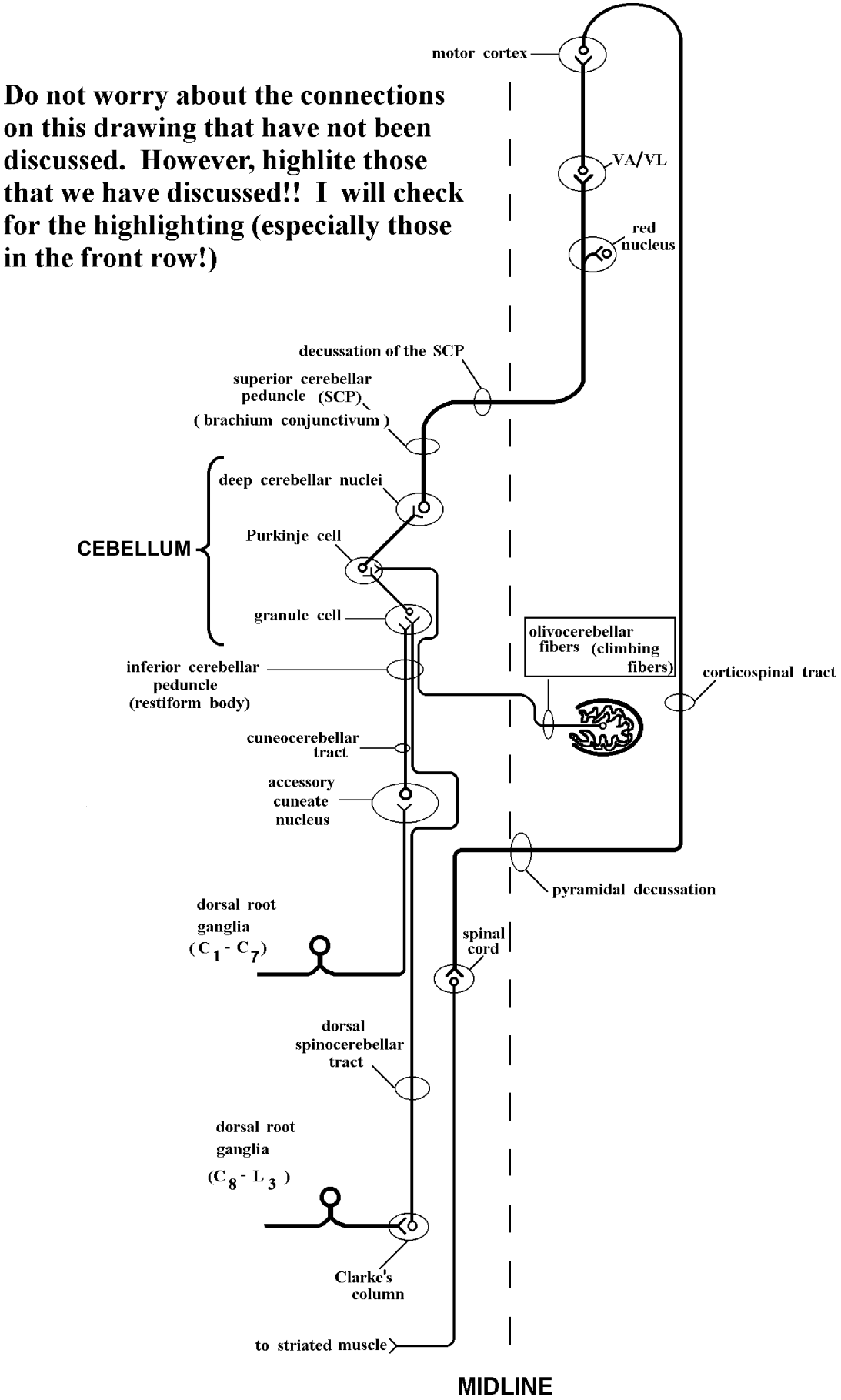
ALSO REMEMBER:

1). the **sole source** of climbing fibers is the inferior olive

2). olivocerebellars **CROSS** and comprise most of the inferior cerebellar peduncle

3). the inferior olive lies in the ventral **medulla**.




Do not worry about the connections on this drawing that have not been discussed. However, highlite those that we have discussed!! I will check for the highlighting (especially those in the front row!)
motor cortex
VA/VL
red nucleus
decussation of the SCP
superior cerebellar peduncle (SCP)
(brachium conjunctivum)
deep cerebellar nuclei
CEBELLUM
Purkinje cell
granule cell
olivocerebellar fibers (climbing fibers)
corticospinal tract
inferior cerebellar peduncle (restiform body)
cuneocerebellar tract
accessory cuneate nucleus
pyramidal decussation
dorsal root ganglia (C_1 - C_7)
spinal cord
dorsal spinocerebellar tract
dorsal root ganglia (C_8 - L_3)
Clarke's column
to striated muscle
MIDLINE

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. right pyramid

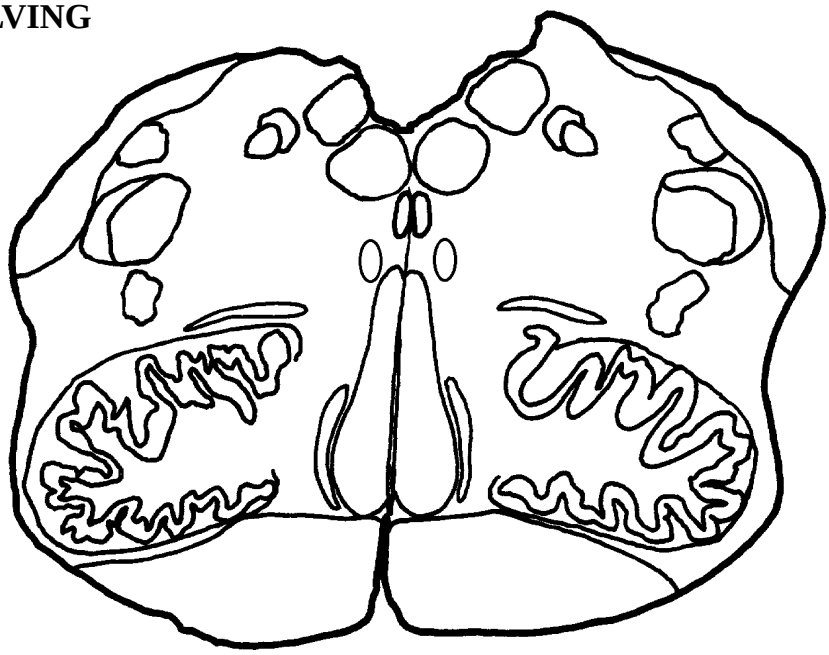
____3. right spinal tract V

____4. left inferior cerebellar peduncle

____5. left inferior olive nucleus

A. lesion results in a loss of vibratory sense from the left arm and leg
B. cells project to the cerebellum via the right inferior cerebellar peduncle
C. cells project to the right VPM
D. contains axons that arise from Clarke's column on the left side of the spinal cord
E. carries fibers from the right accessory cuneate destined for the cerebellum
F. "climbing fibers" from this nucleus terminate in the left side of the cerebellum
G. lesion results in a Babinski sign from the left big toe
H. axons convey pain and temp from the right side of the larynx
I. carries information regarding 2 pt. discrimination from the right side of the body
J. lesion results in a loss of pain and temp from the left side of the body

PROBLEM SOLVING

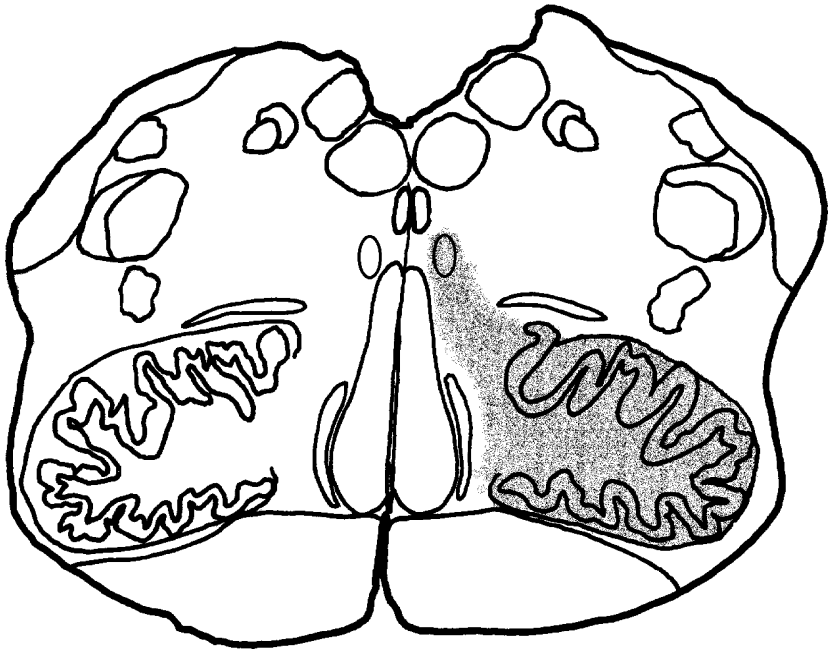


RIGHT LEFT

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

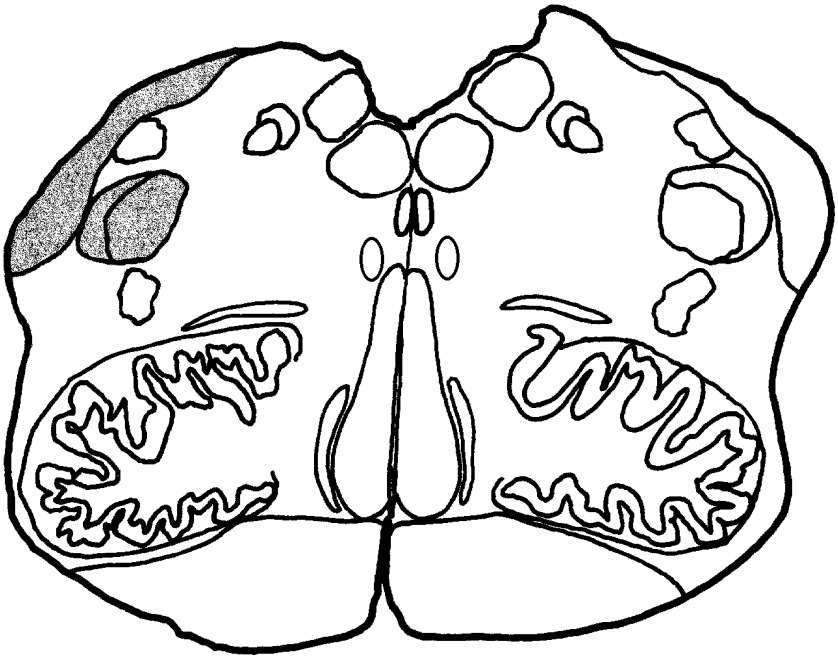
loss of pain and temperature from the right side of the face, incoordination of the right arm and leg

PROBLEM SOLVING ANSWER



RIGHT LEFT

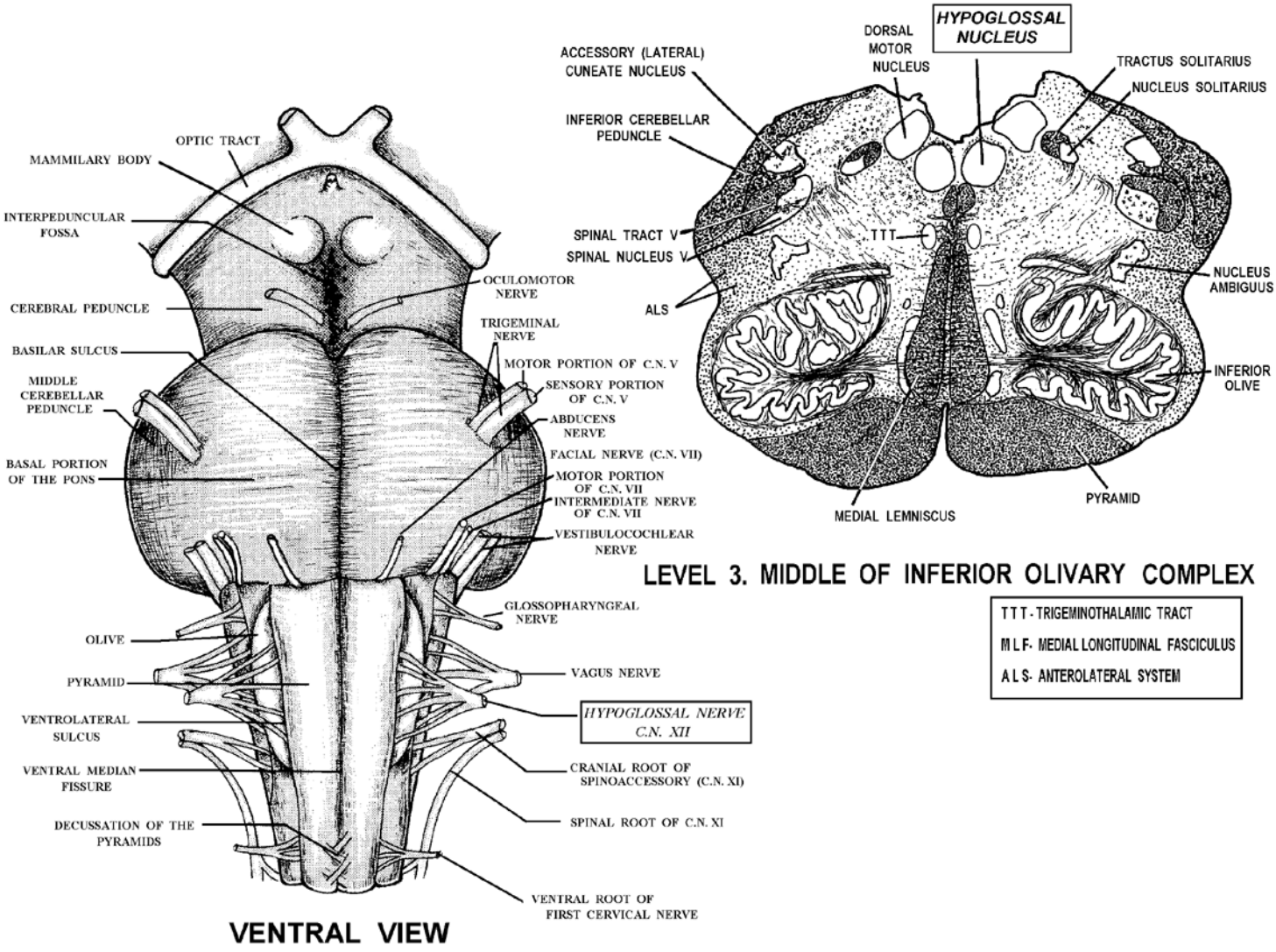
- OR -



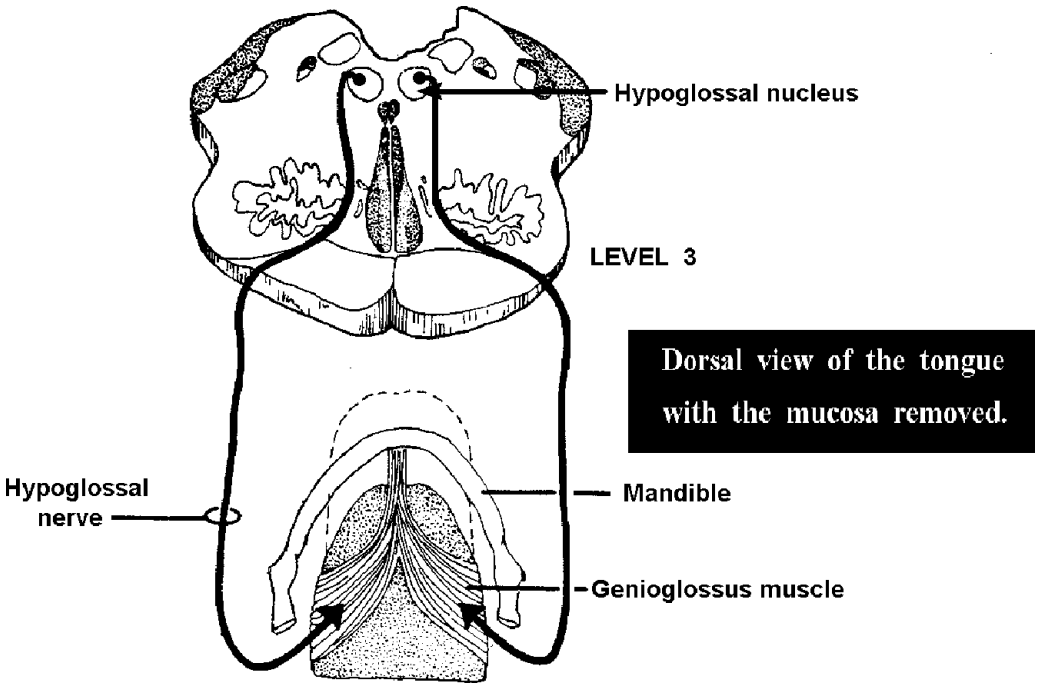
RIGHT LEFT

7 HYPOGLOSSAL NUCLEUS (C.N. XII)

This nucleus lies just off the midline beneath the floor of the fourth ventricle. Axons from cells within the hypoglossal nucleus course ventrally to exit the medulla between the pyramid and the inferior olive. The hypoglossal nerve then passes through the hypoglossal foramen to emerge from the base of the skull. Each nerve innervates the ipsilateral intrinsic and extrinsic muscles of the tongue (you referred to this nerve as **somatomotor** in Gross Anatomy and learned that the palatoglossus is innervated by C.N X; this is tooo detailed for neuro!). These muscles are arranged as paired groups, fused at the midline and oriented in multiple planes that allow the extremely varied and complex movement capabilities of the tongue in speaking, chewing, swallowing, and buccal cleaning processes.



Following a lesion of the hypoglossal nucleus or nerve, there is **ATROPHY** of the muscles of the **IPSILATERAL** one-half of the tongue. This is a lower motor neuron lesion (the damaged neuron or axon directly innervates skeletal muscle). Upon closer examination, **FASCICULATIONS** (tiny, spontaneous contractions) can be seen. Both **fasciculations** and **atrophy** result from the loss of the normal innervation of the muscle by the lower motor neurons in the hypoglossal nucleus. Upon **protrusion**, the tongue will deviate **TOWARD** the side of the lesion (i.e., same side). This is due to the unopposed action of the genioglossus muscle on the normally innervated side of the tongue (the genioglossus pulls the tongue forward). Remember, the genioglossus arises laterally in the tongue and inserts on the midline of the mandible.



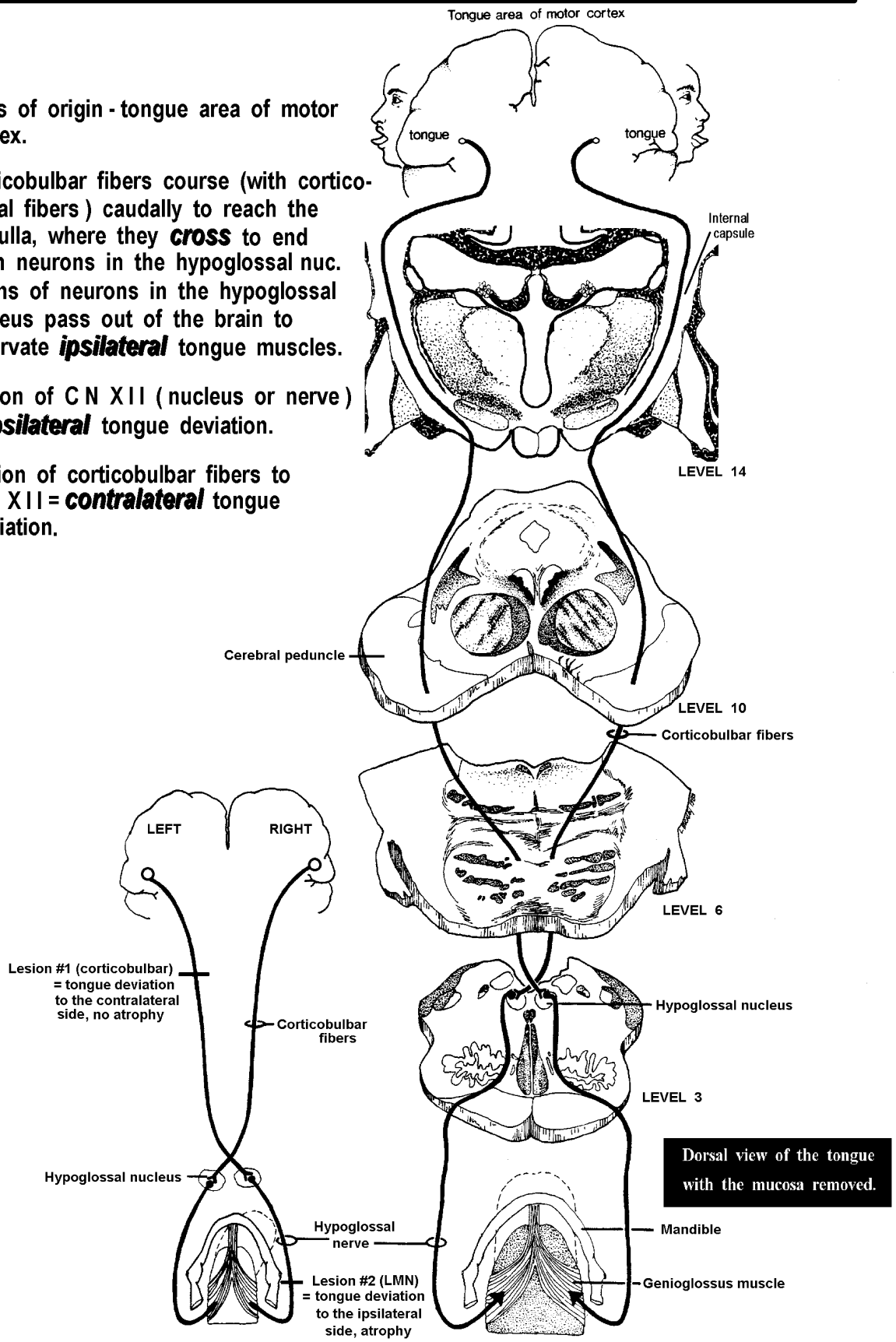
You have already heard of corticospinal axons. By their name, they arise from cortical neurons and end in the spinal cord. Another important group of axons that arise from cortical neurons do not reach the spinal cord. Instead, they end in **motor nuclei of cranial nerves**. These axons are called **CORTICOBULBAR** ("bulb" is a term that some neuroanatomists use when referring to the medulla because of its appearance as a bulb-like expansion of the spinal cord).

The corticobulbar input to the hypoglossal nucleus arises from motor cortex (you can voluntarily move your tongue) and is predominantly **CROSSED**. Thus, a lesion in motor cortex will result in deviation of the tongue toward the opposite side or **CONTRALATERAL** to the lesion. In contrast to the atrophy and fasciculations seen in lesions of the hypoglossal nucleus and nerve (lower motor neuron), **NO** such signs are present after lesions of the corticobulbar tract (remember, the neurons in the hypoglossal nucleus are still alive). A lesion of the corticobulbar input to the hypoglossal nucleus is called a **supranuclear lesion** (i.e., above or rostral to the hypoglossal nucleus). In a lesion of the motor cortex there is also involvement of corticospinal fibers. For example, a lesion in the **LEFT** motor cortex (which involves both corticospinal and corticobulbar axons) would result in a **RIGHT** hemiplegia and deviation of the tongue to the **RIGHT**. There would **NOT** be any atrophy.

Weakness of the tongue manifests itself as a slurring of speech. The patient's tongue feels "thick" and lingual sounds are slurred. This is called **dysarthria** (dys-articulation) and is more apparent in hypoglossal nerve lesions but can occur following supranuclear lesions.

CORTICOBULBARS ARE IMPORTANT - BELIEVE ME, YOU WILL NEVER FORGET THEM!

1. Cells of origin - tongue area of motor cortex.
2. Corticobulbar fibers course (with cortico-spinal fibers) caudally to reach the medulla, where they ***cross*** to end upon neurons in the hypoglossal nuc. Axons of neurons in the hypoglossal nucleus pass out of the brain to innervate ***ipsilateral*** tongue muscles.
3. Lesion of C N X I I (nucleus or nerve) = ***ipsilateral*** tongue deviation.
4. Lesion of corticobulbar fibers to C N X I I = ***contralateral*** tongue deviation.



Corticobulbar input to the Hypoglossal Nucleus

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 spinal nucleus V

____2. right nucleus gracilis

____3. left accessory cuneate nucleus

____4. right inferior olive

____5. left hypoglossal nucleus

A. lesion results in a loss of vibratory sense from the left arm and leg

B. cells project to the cerebellum via the right inferior cerebellar peduncle

C. cells project to the left VPM

D. lesion results in incoordination of the left arm and leg

E. peduncle that carries fibers from the left accessory cuneate nucleus

F. lesion results in deviation of the tongue to the left upon protrusion

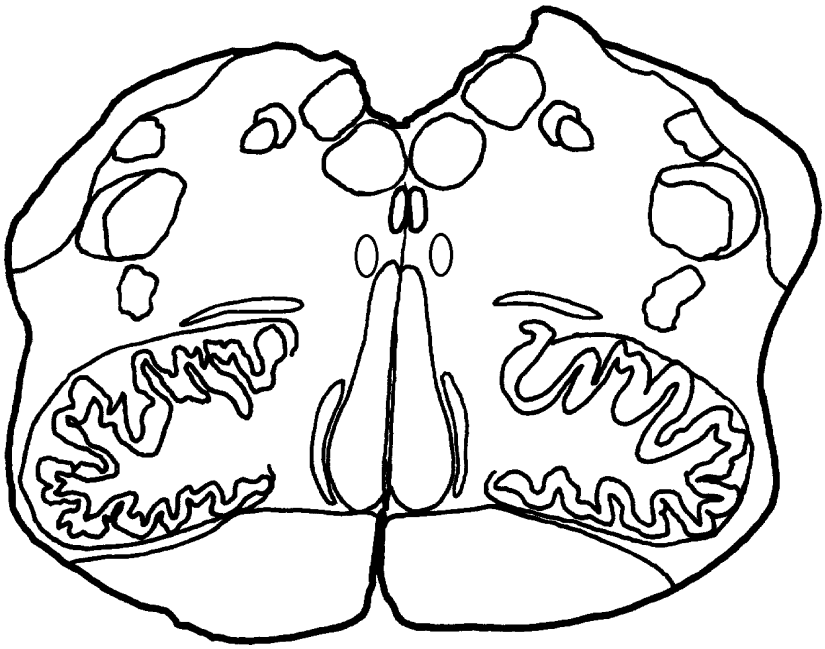
G. lesion results in a loss of vibratory sense from the right leg

H. axons convey pain and temp from the right side of the larynx

I. lesion results in incoordination of **only** the left arm

J. lesion results in a loss of pain and temp from the left side of the face

PROBLEM SOLVING

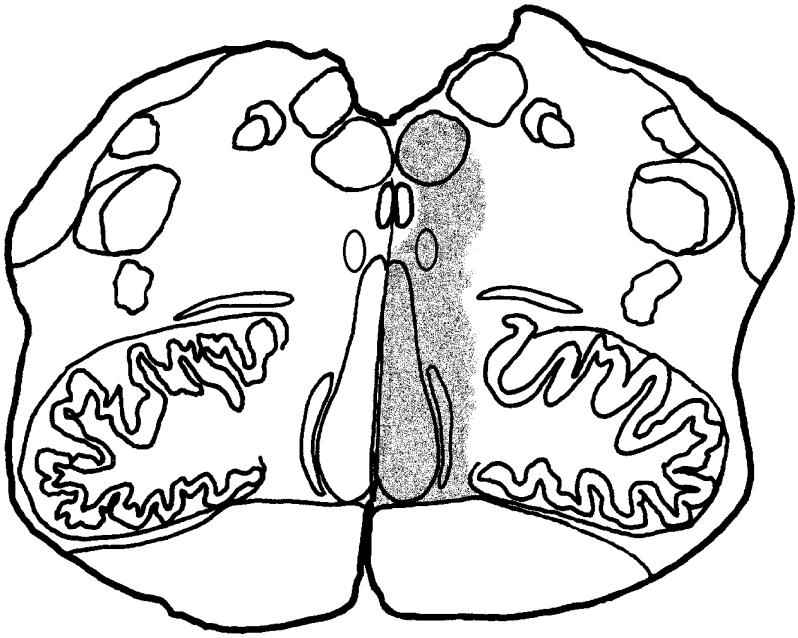


RIGHT LEFT

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

atrophy of the muscles of the left side of the tongue, deviation of the tongue to the left upon protrusion, loss of vibratory sense from the right arm and leg, loss of pain and temperature from the right side of the face

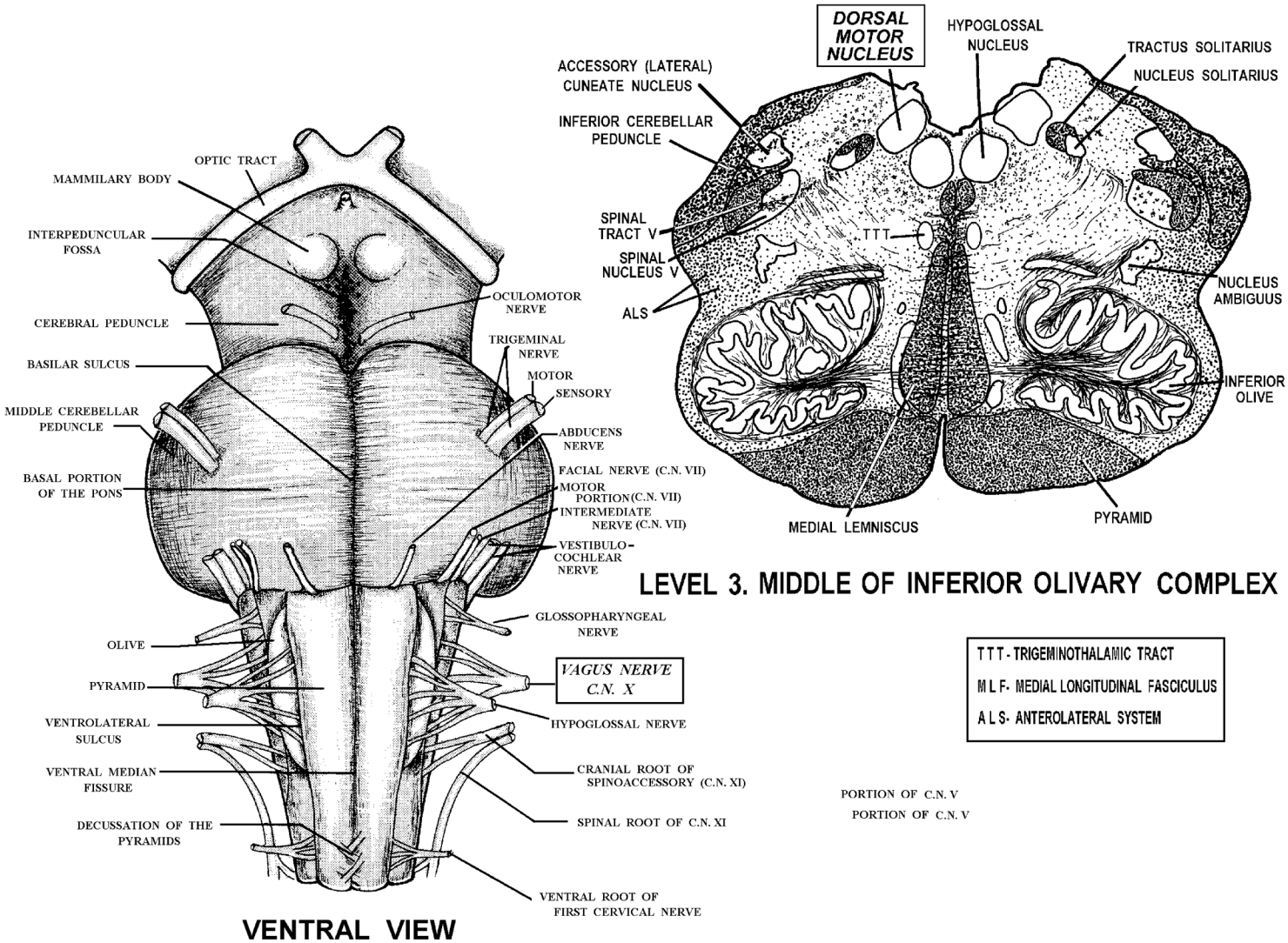
PROBLEM SOLVING ANSWER



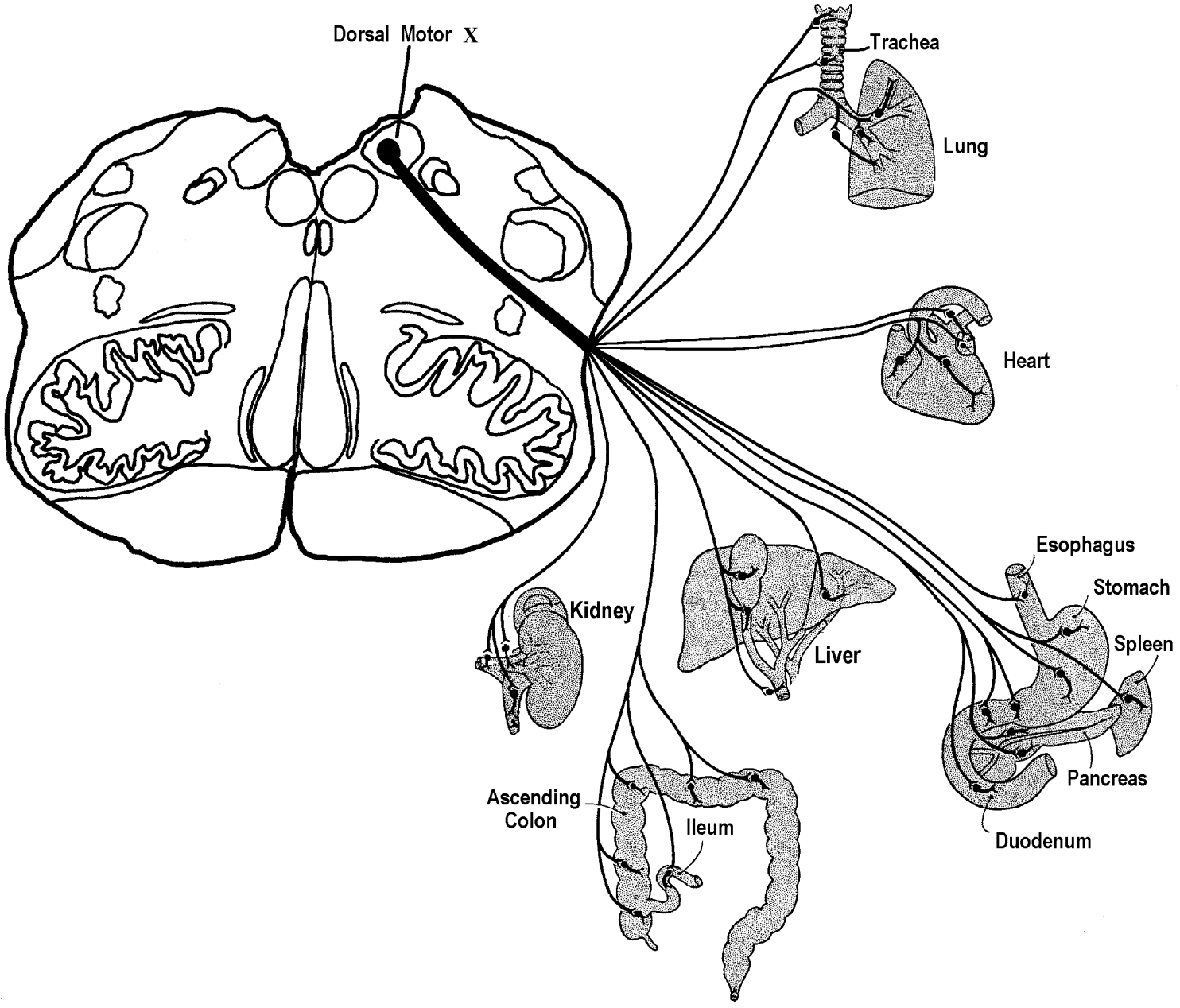
RIGHT LEFT

8 DORSAL MOTOR NUCLEUS OF THE VAGUS (C.N. X)

This nucleus lies slightly dorsal and lateral to the hypoglossal nucleus. Axons arising from cells within the dorsal motor X give rise to **PREGANGLIONIC PARASYMPATHETIC** fibers that course ventral and lateral from the nucleus to exit the brain stem dorsal and lateral to the inferior olive. These axons comprise the **visceromotor** component of the vagus nerve (C.N. X). Preganglionic parasympathetic fibers from the vagus wander all over the place, but eventually terminate in ganglia that contain postganglionic neurons. These terminal ganglia (**NOT** the sympathetic chain!) are located close to, or within the structures innervated by the short postganglionic fibers (do you remember Auerbach's plexus [myenteric] and Meissner's plexus [submucosal] within the gut?).



Preganglionic parasympathetic visceromotor fibers from the vagus activate postganglionic neurons in ganglia associated with the pharynx, larynx and esophagus. Short postganglionic parasympathetic fibers in turn innervate **GLANDS** and **SMOOTH MUSCLE** in these structures.



DORSAL MOTOR X

Preganglionic parasympathetic fibers of the vagus

Cardiac branches of the vagus carrying visceromotor fibers synapse on ganglia within the cardiac plexuses (superficial and deep). While some investigators have shown that stimulation of the right and left vagi have different effects upon the heart, I will leave those differences for the cardiovascular section of your physiology course. For now I want you to know that vagal stimulation **slows** heart rate. For our **PROBLEM SOLVING** exercises a unilateral lesion of **either** dorsal motor X (right or left) will result in an **INCREASE IN HEART RATE (TACHYCARDIA)**. This increase in heart rate is the result of losing input from the dorsal motor nucleus, which itself slows the heart (the dorsal motor X is sometimes called the "cardioinhibitory center"). The sympathetic portion of the autonomic nervous system is left in control. The preganglionic **sympathetic** fibers arise from the lateral cell column of spinal cord segments T_1-T_5 and synapse in the three cervical sympathetic ganglia. Postganglionic sympathetic fibers arising from these ganglia pass through the cardiac plexuses and innervate sinoatrial and atrioventricular nodal tissue, conducting tissue and ventricular myocardium. Stimulation of the sympathetics **increases** nodal firing rate, conduction rate and ventricular force. The brain stem input to the lateral cell column will be discussed later under Point #11 (nucleus solitarius).

REMEMBER:
LESION OF DORSAL MOTOR X = INCREASE IN HEART RATE

READ ON ONLY IF YOU ARE INTERESTED. I WILL NOT, REPEAT, WILL NOT ASK YOU ANY QUESTIONS ABOUT WHAT FOLLOWS.

Preganglionic parasympathetic visceromotor fibers from the vagus also activate postganglionic neurons in ganglia associated with the lungs (pulmonary and bronchial plexuses). These postganglionic fibers innervate smooth muscle and glands of bronchioli. Stimulation of the dorsal motor nucleus results in **constriction** of the smooth muscle of bronchioli and **increased** secretion from the bronchial glands. There is mixing of the right and left vagi in the pulmonary plexuses, so a unilateral lesion of one dorsal motor X will be "covered" by the other nucleus and nerve.

The visceromotor fibers from the vagus that reach the stomach and gut end in postganglionic neurons that lie near (gastric plexus) or in (myenteric and submucosal plexuses) the organs. **Stimulation** of the dorsal motor nucleus results in **increased** peristalsis and secretion of gastric and intestinal juices, and **relaxation** of sphincters. You might remember from Gross Anatomy that the right and left vagus nerves exchange fibers on the outer surface of the esophagus. They then enter the abdomen as anterior (or ventral) and posterior (or dorsal) vagal trunks. There is considerable mixing of the right and left vagi as they innervate the stomach and intestine.

Afferent sources (inputs) to the dorsal motor nucleus include the hypothalamus, olfactory system, autonomic centers in the reticular formation, and especially the **nucleus solitarius**. Most of these afferent sources you have never heard of. So, right now remember that the dorsal motor nucleus X plays an important role in various visceral reflexes. Thus, information about the "internal milieu" reaches the dorsal motor nucleus of X via visceral afferent pathways that we will soon talk about. The dorsal motor nucleus X receives these afferent messages and then sends information to the appropriate organ(s) (via the terminal ganglia).

PROBLEM SOLVING MATCHING

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

____1. right trigeminothalamic tract

____2. left anterolateral system **and associated descending pathway**

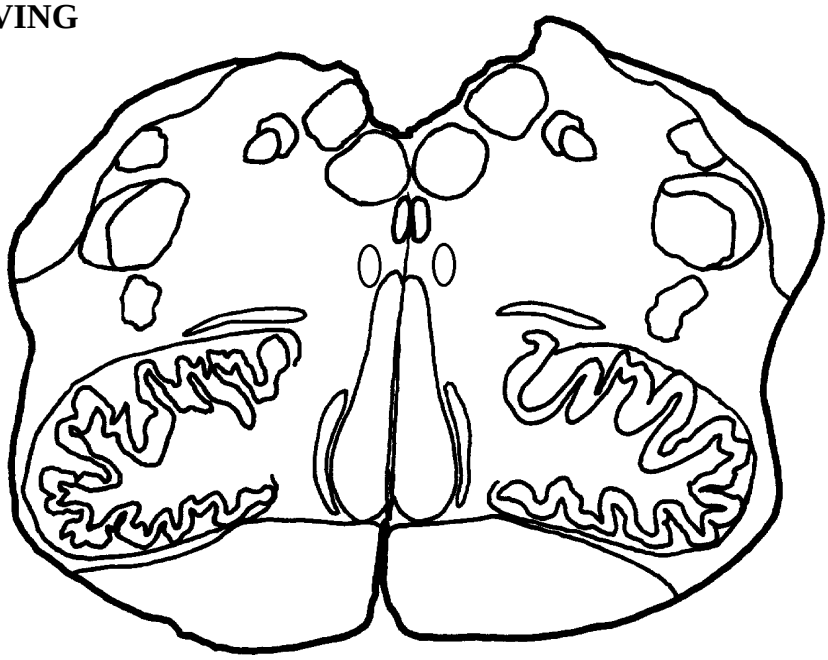
____3. right medial lemniscus

____4. right hypoglossal nerve

____5. left dorsal motor X

A. lesion results in left hemiplegia
B. carries pain and temp information to the left VPM
C. lesion results in bilateral atrophy of the muscles of the tongue
D. lesion results in deviation of the tongue to the left upon protrusion
E. carries pain and temp information to the right VPM
F. lesion results in a constricted pupil in the left eye and ptosis of the left eye lid
G. lesion results in a loss of vibratory sense from the left arm and leg
H. lesion results in atrophy of the tongue muscles on the right side
I. lesion results in an increased heart rate
J. lesion results in a loss of pain and temp from the right side of the face

PROBLEM SOLVING

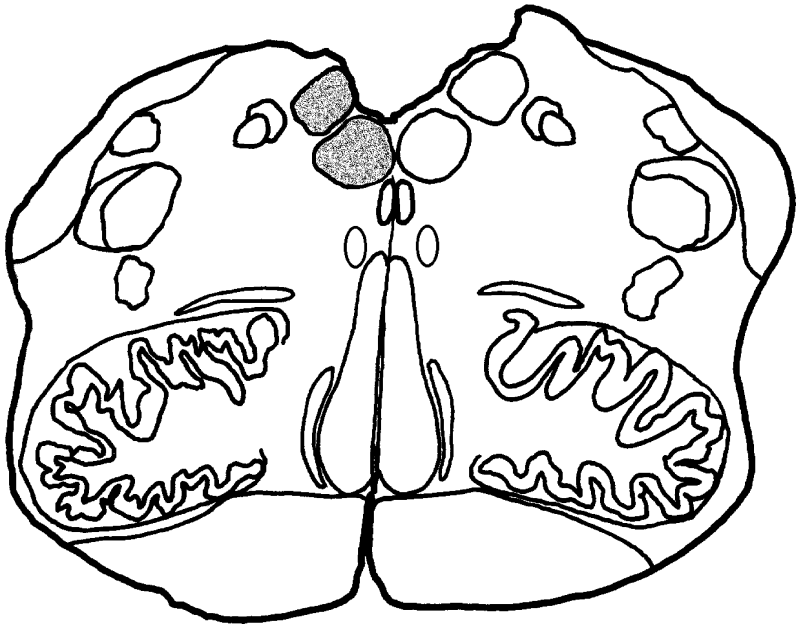


RIGHT LEFT

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

increase in heart rate, atrophy of the muscles of the right side of the tongue, deviation of the tongue to the right upon protrusion and fasciculations of the muscles of the right side of the tongue

PROBLEM SOLVING ANSWER



RIGHT LEFT

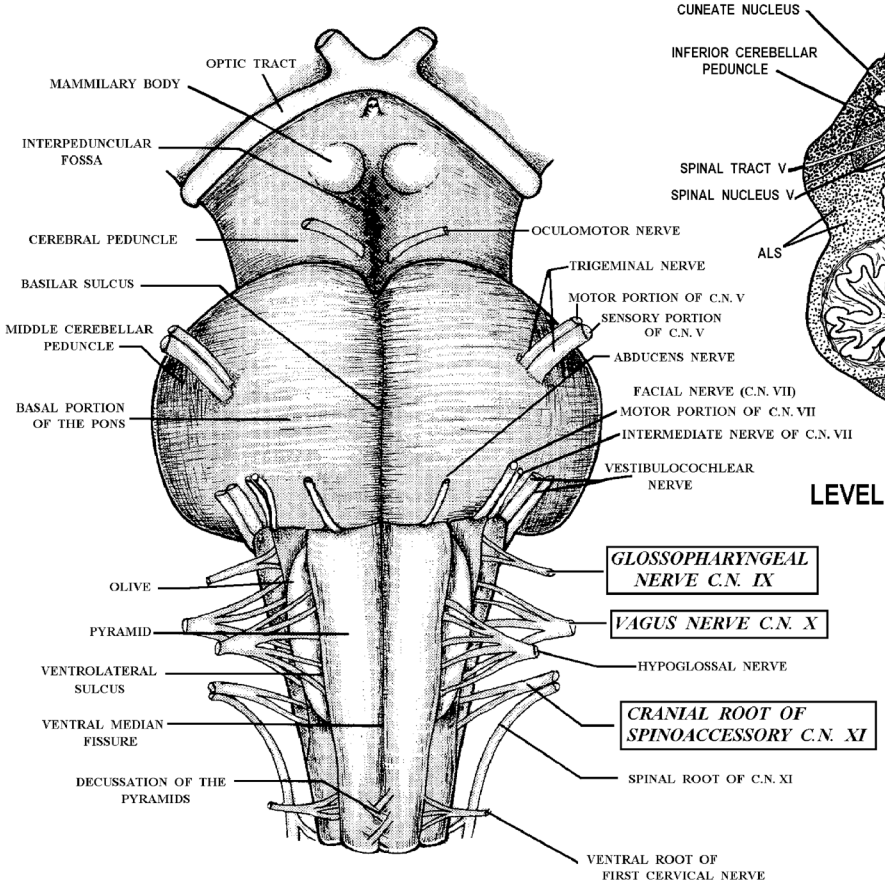
9 NUCLEUS AMBIGUUS

This nucleus lies dorsal and lateral to the inferior olive. Cells in nucleus ambiguus contain motor neurons associated with three cranial nerves (rostral pole =C.N. IX=glossopharyngeal; middle part =C.N. X=vagus; caudal pole =C.N. XI=spinoaccessory). Axons arising from nucleus ambiguus pass laterally and slightly ventrally to exit the medulla just dorsal to the inferior olive. These axons then course with the three cranial nerves--**IX** (glossopharyngeal), **X** (vagus) and **XI** (spinoaccessory)--to innervate the striated muscles of the soft palate, pharynx, larynx, and upper part of the esophagus. Since these muscles have developed embryologically from branchial arches 3, 4 and 5, the cells that innervate them are called **branchiomotor**. Remember, **NUCLEUS AMBIGUUS** is "**SHARED**" by three cranial nerves (IX, X, XI). The general pattern of motor innervation below does **not** have to be memorized.

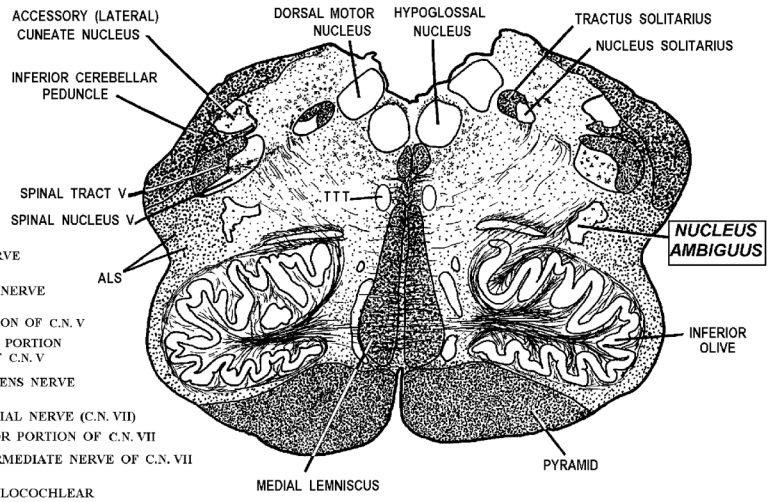
IX stylopharyngeus muscle

X palatal muscles; levator veli palatini (with assistance from V for the tensor veli palatini; of little clinical significance), most of the pharyngeal muscles (with assistance from IX), laryngeal muscles and striated muscles of the esophagus-palatoglossus too!

XI laryngeal muscles (cranial portion)



VENTRAL VIEW



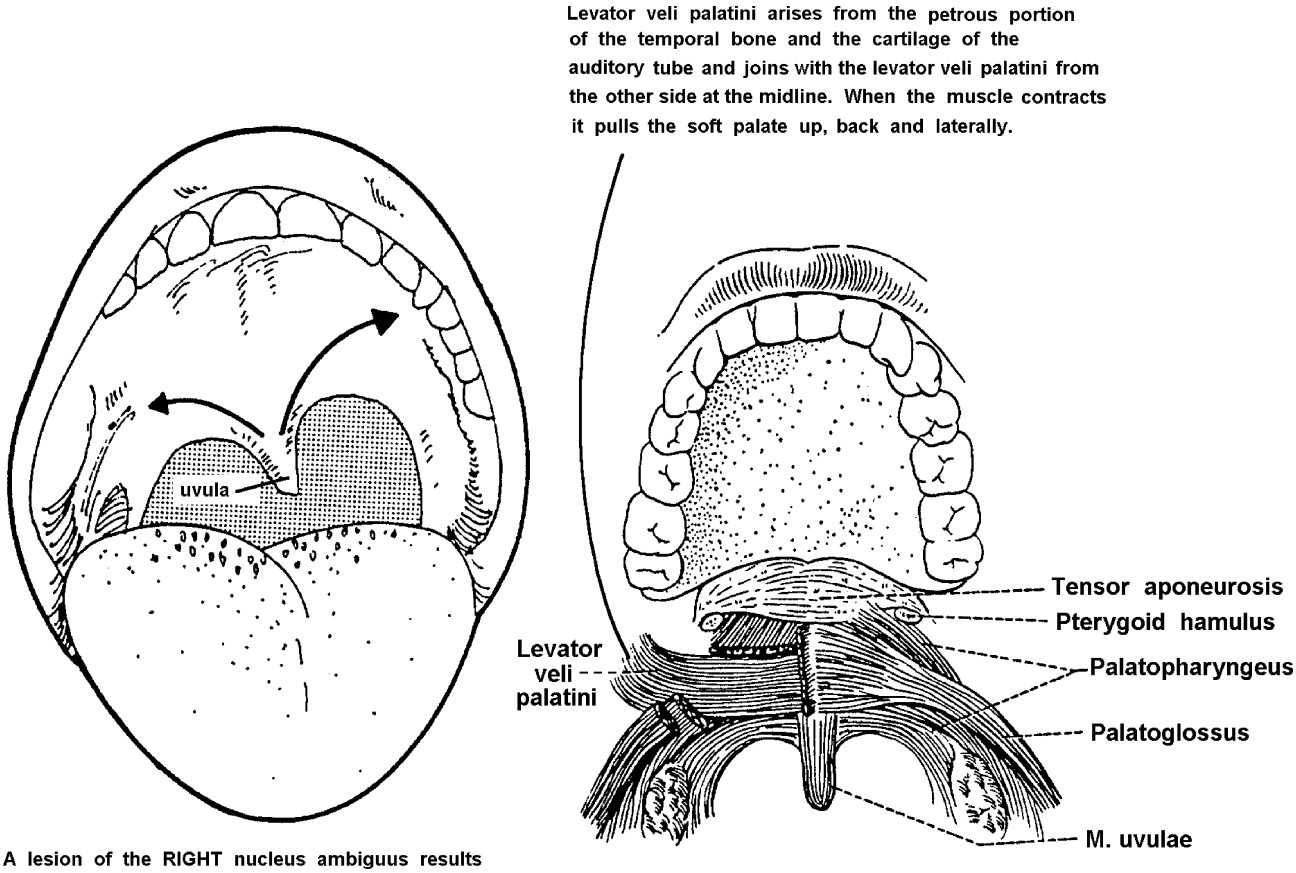
LEVEL 3. MIDDLE OF INFERIOR OLIVARY COMPLEX

TTT - TRIGEMINOTHALAMIC TRACT
MLF - MEDIAL LONGITUDINAL FASCICULUS
ALS - ANTEROLATERAL SYSTEM

THINK SOFT PALATE, PHARYNX, LARYNX

A unilateral lesion of nucleus ambiguus will result in **atrophy** and paralysis of all palatine muscles **ipsilateral** to the lesion, except the tensor veli palatini (C.N. V). Because of the palate paralysis, the patient's speech may be **nasal**. This is because air is allowed to escape into the nose during speaking. Normally, the soft palate elevates in order to reduce the nasopharyngeal aperture during speaking. This elevation of the soft palate detours the air through the mouth, the path of least resistance. Due to the hemiplegic palate the patient may complain of **nasal regurgitation** of liquids since he/she is unable to shut off completely the nasopharynx from the buccal cavity. Moreover, during phonation (say ahhh!) the soft palate is elevated on the normal side and the **UVULA DEVIATES TOWARDS THE NORMAL SIDE** (contralateral to the lesion; contrast this with lesions of the hypoglossal nucleus). Remember from Gross Anatomy that the levator veli palatini raises the soft palate and, in doing so, also pulls it backward. Also, some awkwardness of swallowing, called **dysphagia**, may occur due to the unilateral paralysis of the constrictors of the pharynx. Due to paralysis of the laryngeal muscles, the patient exhibits **dysphonia,** his/her voice being **husky** or **hoarse** (speech requires phonation by the vocal cords; phono=voice, sound).

Bilateral lesions of nucleus ambiguus increase the difficulties I have just described following ipsilateral lesions. Nasal regurgitation is more distressing and permanent. Dysphagia is more pronounced and speech and respiratory disorders may be profound. Respiratory disorders, induced by the paralysis of the abductor muscles (of the larynx) bilaterally may lead to suffocation unless treated by intubation.

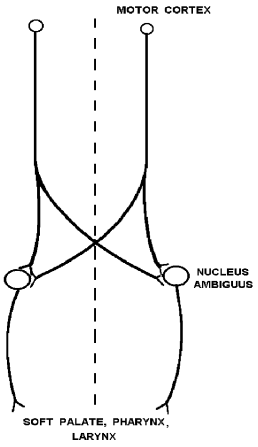


1. **Cells of origin - soft palate, pharynx and larynx area of motor cortex.**

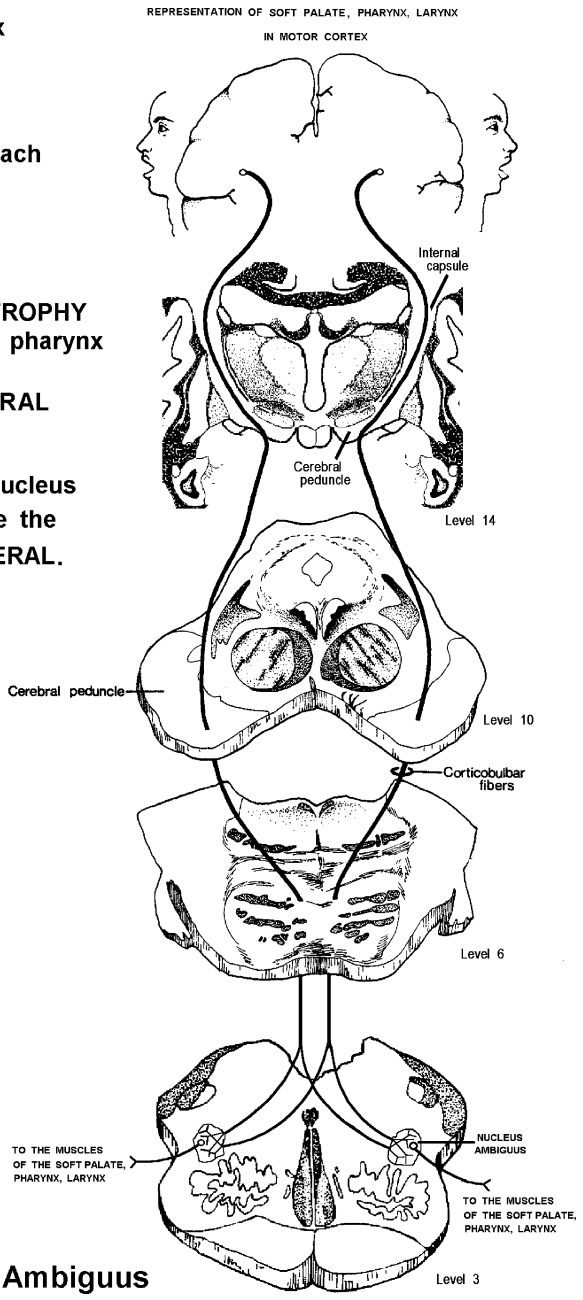
2. **Corticobulbar fibers course (with corticospinal fibers) caudally to reach the medulla, where they project BILATERALLY to both Ambiguus nuclei.**

3. **Lesion of Nucleus Ambiguus = ATROPHY of the muscles of the soft palate, pharynx and larynx (uvula DEVIATION = CONTRALATERAL to the side of the lesion).**

4. **Lesion of corticobulbar input to nucleus ambiguus = NO PROBLEM because the input from motor cortex is BILATERAL.**



Bilateral Corticobulbar = ambiguus = good no problem !!!!



Corticobulbar input to Nucleus Ambiguus

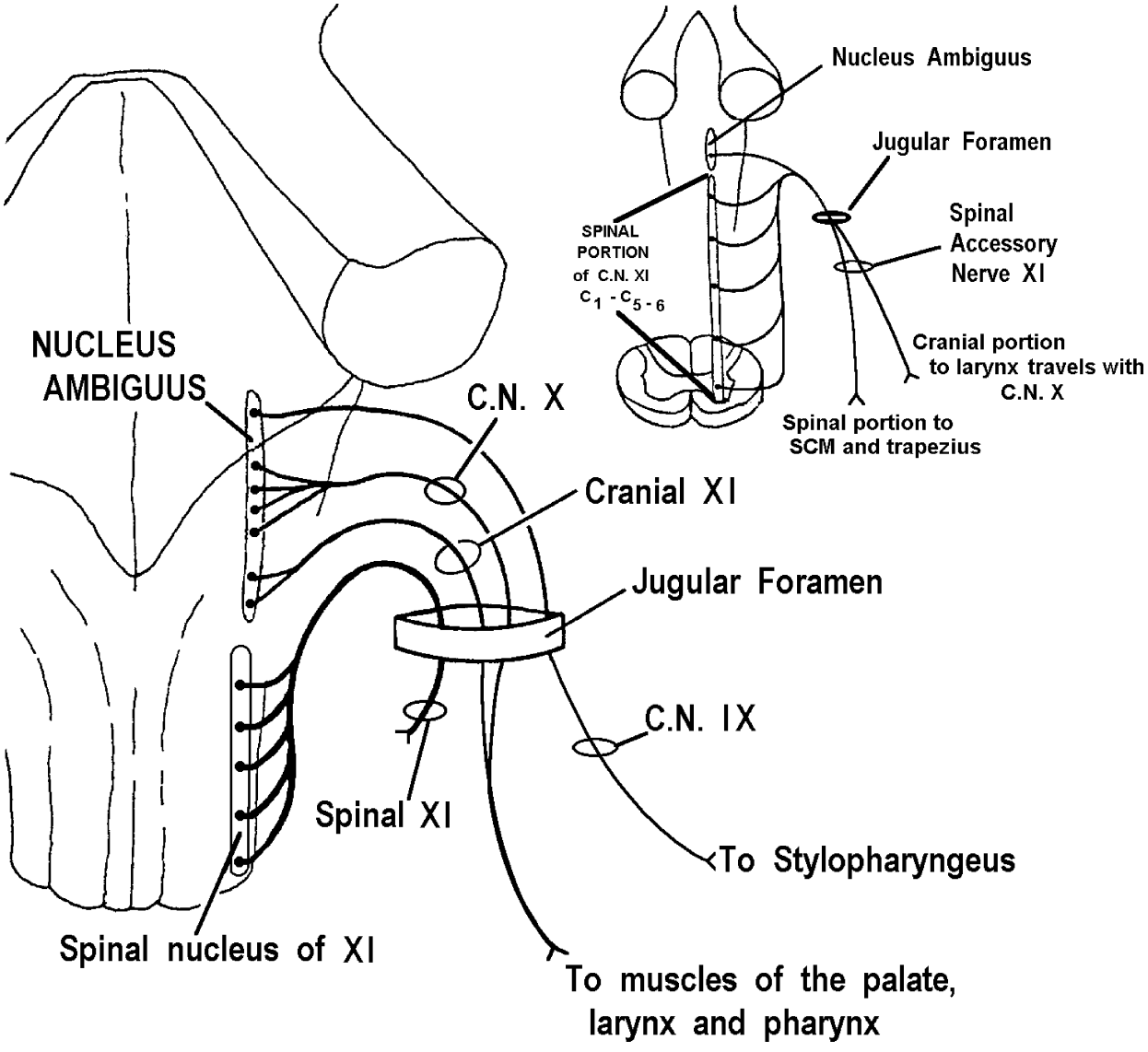
Corticobulbar fibers (you can voluntarily swallow!) to nucleus ambiguus are **BILATERAL** (both crossed and uncrossed). Therefore, muscles supplied by the nucleus ambiguus are **NOT** noticeably weakened in the event of unilateral lesions of the corticobulbar system (i.e., in the motor cortex). This means that there is **NO** deviation of the uvula following cortical lesions. **Don't confuse the results of lesions of the corticobulbar projection to nucleus ambiguus with lesions of nucleus ambiguus!** Also don't confuse the bilateral corticobulbar input to nucleus ambiguus with the primarily **CROSSED** corticobulbar input to the **HYPOGLOSSAL** nucleus. What it boils down to is that **BILATERAL** corticobulbar input is **GREAT** for you as students, since you don't have to remember which way something deviates following its interruption. It is only those corticobulbar projections that are not equally bilateral (so far only that to the **HYPOGLOSSAL**, but more to come) that you need to worry about.

Motor fibers of C.N. XI that arise from the nucleus ambiguus join the vagus outside of the skull and innervate muscles of the larynx (recurrent [inferior] vagus). These fibers comprise the **CRANIAL** branch of C.N. XI. **REMEMBER: CRANIAL XI=AMBIGUUS**. In contrast, the **SPINAL** portion of C.N. XI consists of motor axons whose cell bodies lie in the lateral part of the ventral horn of the first five or six cervical **SPINAL CORD** segments. The axons of these cells pass dorsal and laterally (that is they **do not** exit via the ventral root), leave the spinal cord between the dorsal and ventral roots and unite to ascend in the spinal canal to enter the skull via the foramen magnum. They then exit the skull via the jugular foramen along with cranial nerves IX and X and eventually innervate the **sternocleidomastoid** and the upper fibers of the **trapezius**. **REMEMBER: CAUDAL XI=SPINAL CORD**.

Lesions involving C.N. XI fibers to these two muscles result in **atrophy** of the muscles. Since the **RIGHT** sternocleidomastoid rotates the head to the **LEFT** (opposite), a lesion of the **RIGHT** C.N. XI will result in the chin being turned slightly to the **RIGHT** (paralyzed) side, especially when the head is flexed. The same **RIGHT** side lesion will result in paralysis of the **RIGHT** upper trapezius and slight sagging of the **RIGHT** shoulder.

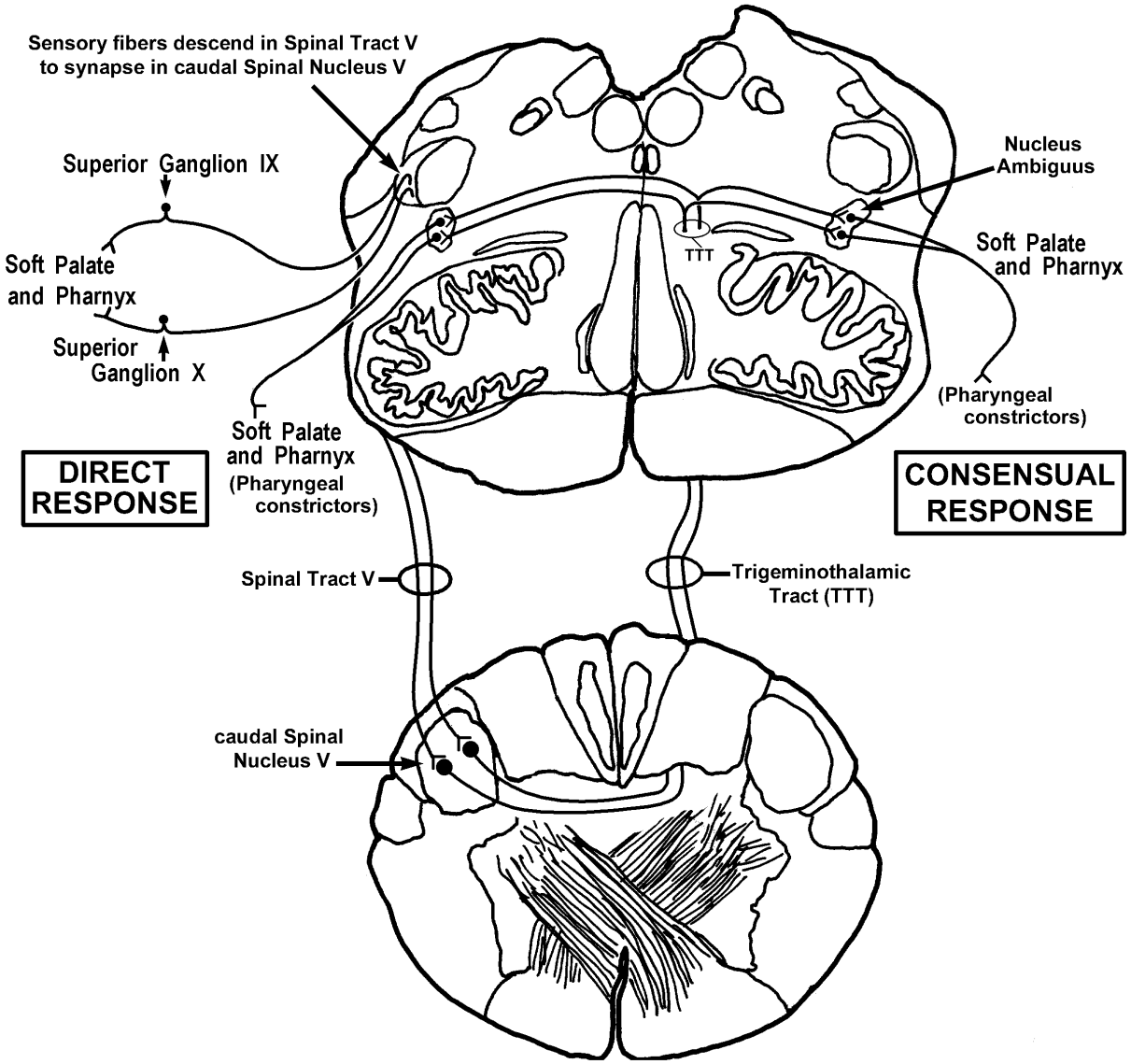
As for cortical input to the cells of origin of the spinal part of XI, you are lucky since it is **bilateral**.

CORTICOBULBAR TO SPINAL PART XI=BILATERAL=GOOD=TAKE A BREAK!!!!



The normal **gag reflex** is a mass contraction of **both** sides of the posterior oral and pharyngeal musculature and an indication by the patient of an unpleasant experience. Sensory information (painful) comes into the brain stem over C.N.s IX and X (cell bodies in superior ganglia), enters the spinal tract V and terminates in caudal spinal nucleus V. Cells in spinal nucleus V then project **bilaterally** to nucleus ambiguus (we cannot identify these axons in our sections, but they travel over the TTT). The contractions of the pharyngeal musculature ipsilateral to the stimulus is called the **DIRECT response**, while the contractions of the musculature contralateral to the stimulus is called the **CONSENSUAL response** (consensus=agreement). Don't forget that with a lesion of nucleus ambiguus the efferent or motor part of the **GAG REFLEX** is lost **IPSILATERAL** to the lesion. Sensory stimulation from the soft palate and pharynx can reach spinal nucleus V (via C.N.s IX and X; superior ganglia), and, via the TTT, both nuclei ambiguui. However, there is contraction of only the muscles innervated by cells in the "alive" nucleus ambiguus. Look at the diagram and contrast the effects of lesions involving (1) C.N.s IX and X, (2) caudal spinal nucleus V and (3) nucleus ambiguus. Also, do the practice questions on the next page. Good luck!

GAG REFLEX



Touching of the soft palate and pharynx sends information to the ipsilateral caudal spinal nucleus V. Cells in spinal nucleus V project bilaterally to Nucleus Ambiguus resulting in elevation of the palate and gagging. The DIRECT response is on the same side as the stimulation, while the CONSENSUAL response is on the side opposite to the stimulus.

FYI and enjoyment

The **cortex** gets its name from the Latin word for "bark" (of a tree).
The word "**hypnosis**" comes from the Greek word meaning "sleep."
The term "**homo sapiens**" comes from the Latin words meaning "wise man."
In 1891, Wilhelm von Waldeyer coined the term "**neuron**."
The word "**genu**" comes from the Greek word gonia meaning "angle/corner." (thanks PM)

Charles Scott Sherrington coined the term "**synapse**" in 1897.

The dura mater is the outermost covering of the brain. The term "**dura mater**" comes from Latin meaning "hard mother."

Written about 1,700 B.C., the Edwin Smith surgical papyrus contains the first recorded use of the word "**brain**."

The word "**glia**" comes from the Greek word meaning "glue."

The word "**carotid**" (carotid artery) comes from the Greek word karotis meaning "deep sleep." This is because it has been known for a long time that pressure on the carotid arteries causes animals to become sleepy.

The term "**dendrite**" was introduced by C. Golgi in about 1870. (From Afifi, A.K. and Bergman, R.A., Functional Neuroanatomy, New York: McGraw-Hill, 1998.)

The part of the brain called the "**amygdala**" gets its name from the Greek word for "almond" because of the similarities in shape.

The word "**axon**" comes from the Greek word meaning "axle" or "axis."

The cerebral cortex makes up about 77% of the total volume of the human brain. (Statistic from *Trends in Neuroscience*, November 1995.)

The cerebral cortex is composed of six layers of cells.

The Neanderthals brain was larger than a humans

Bicycle helmets reduce the risk for head injury by as much as 85% and reduce the risk for brain injury by as much as 88%. (Statistics from the Center for Disease Control.)

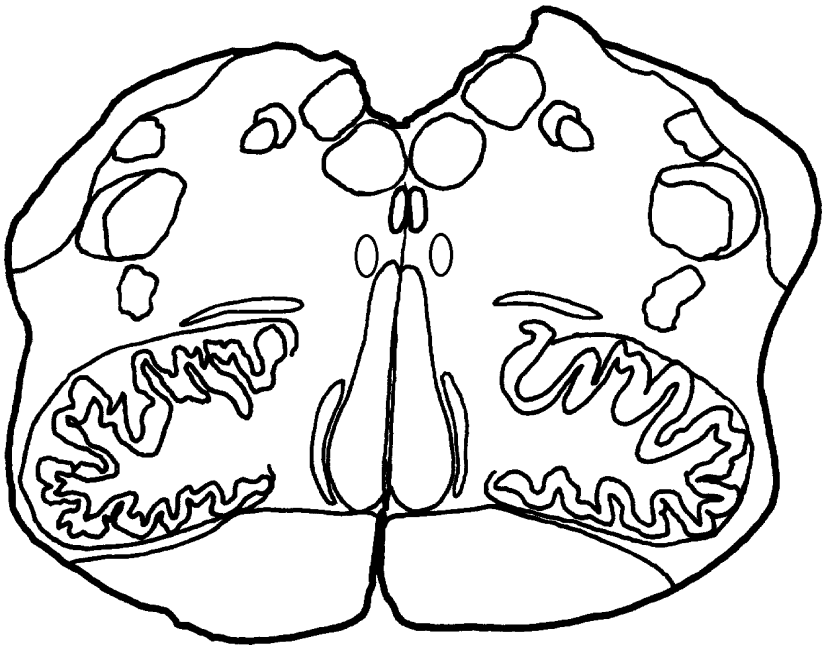
Head injuries account for 62% of bicycle-related deaths. (Statistic from *Morbidity and Mortality Weekly Report*, Feb. 17, 1995.)

Each year in the United States, about 200,000 people require hospitalization for head injury and 52,000 people die due to head injuries. Another 1.74 million people have mild traumatic brain injury that requires them to visit a doctor or disables them for at least one day. (Statistics from *Traumatic Brain Injury*, edited by D.W. Marion, 1999, page 9 and 11.)

Humans experience a biological urge to fantasize every 90 minutes.

Scientists think that every time you have a new thought or memory, you are making a new brain connection.

PROBLEM SOLVING

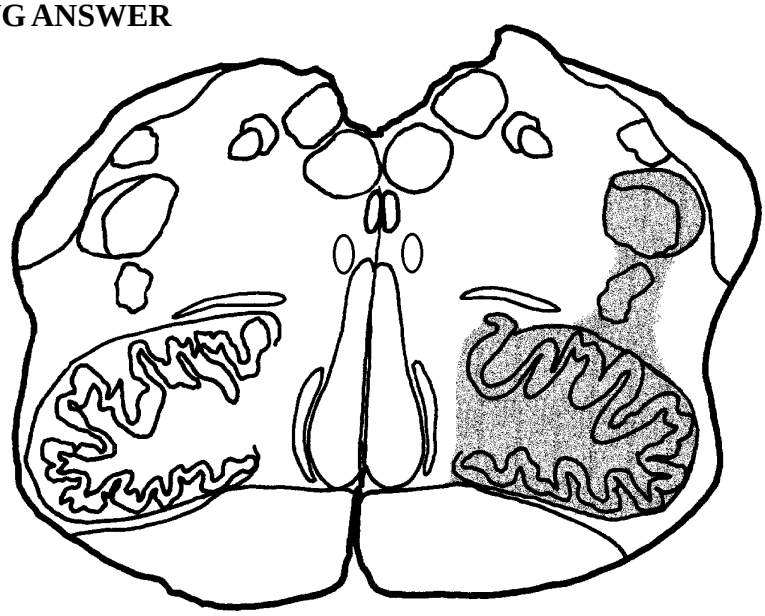


RIGHT LEFT

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

dysphagia, dysphonia, incoordination of the right arm and leg, atrophy of the pharyngeal constrictors on the left, deviation of the uvula to the right upon saying “ahhhh”, and loss of pain and temperature from the left side of the face

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. right trigeminothalamic tract (TTT)

____2. left anterolateral system
and associated descending pathway

____3. left nuclei gracilis and cuneatus

____4. right dorsal motor X

____5. left nucleus ambiguus

A. receives only crossed corticobulbar input

B. carries pain and temp information to the left VPM

C. lesion results in bilateral atrophy of the muscles of the tongue

D. lesion results in deviation of the uvula to the left

E. arises from cells in the left caudal spinal nucleus V

F. lesion results in a loss of pain and temp from the right side of the face

G. lesion results in a loss of 2 pt. discrimination from the left arm and leg

H. lesion results in atrophy of pharyngeal constrictors on the left side

I. lesion results in an increased heart rate

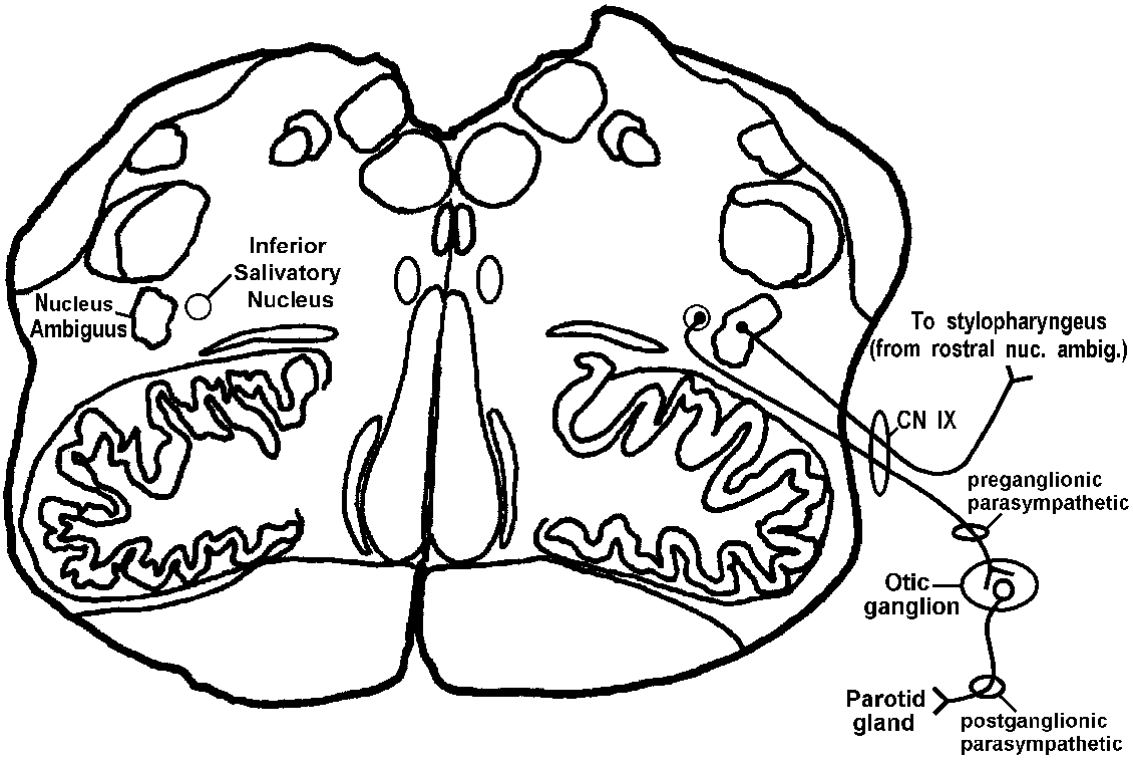
J. lesion results in a constricted pupil in the left eye and slight ptosis of the left eye lid

10 INFERIOR SALIVATORY NUCLEUS

We have already discussed one **PREGANGLIONIC PARASYMPATHETIC** (visceromotor nuclei) nucleus, the dorsal motor nucleus X. There are three other areas in the brain stem that contain preganglionic parasympathetic cell bodies. Unlike the dorsal motor nucleus X, **these nuclei cannot be seen in your brain stem sections**. The most caudal of these cell groups is the **INFERIOR SALIVATORY** nucleus of **C.N. IX**. This nucleus lies in the medulla just **MEDIAL** to the **nucleus ambiguus**. Preganglionic parasympathetic axons arising from cells in the inferior salivatory nucleus end within the **OTIC GANGLION**. Postganglionic axons then pass to the **parotid** gland where they stimulate secretion.

For our problem solving exercises you need to remember that the **inferior salivatory nucleus lies medial to nucleus ambiguus** in the medulla. Remember, the inferior salivatory nucleus is the visceromotor component of C.N. IX. Cells within this nucleus possess preganglionic parasympathetic axons that pass out of the brain stem just dorsal and lateral to the inferior olive and eventually synapse in the otic ganglion. **A lesion involving the inferior salivatory nucleus will result in a loss of SALIVATION** from the ipsilateral parotid gland.

ANYTIME IN THE PROBLEM SETS THAT THE LESION INVOLVES NUCLEUS AMBIGUUS AND THE AREA OF THE MEDULLA MEDIAL TO THE NUCLEUS AMBIGUUS, YOU MUST ASSUME THAT BOTH NUCLEUS AMBIGUUS AND



THE INFERIOR SALIVATORY NUCLEUS ARE INVOLVED.

Keep in mind that a lesion of the inferior salivatory nucleus does not result in a **complete** loss of salivation because the opposite inf. sal. nuc. is OK and C.N. VII is OK. C.N. VII (via the Superior Salivatory Nucleus; to be discussed later) is involved in innervating the submandibular ganglion. Postganglionic neurons in this ganglion innervate the submandibular and sublingual glands (more on this later!).

PROBLEM SOLVING MATCHING

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

____1. right inferior cerebellar peduncle

____2. left corticobulbar pathway

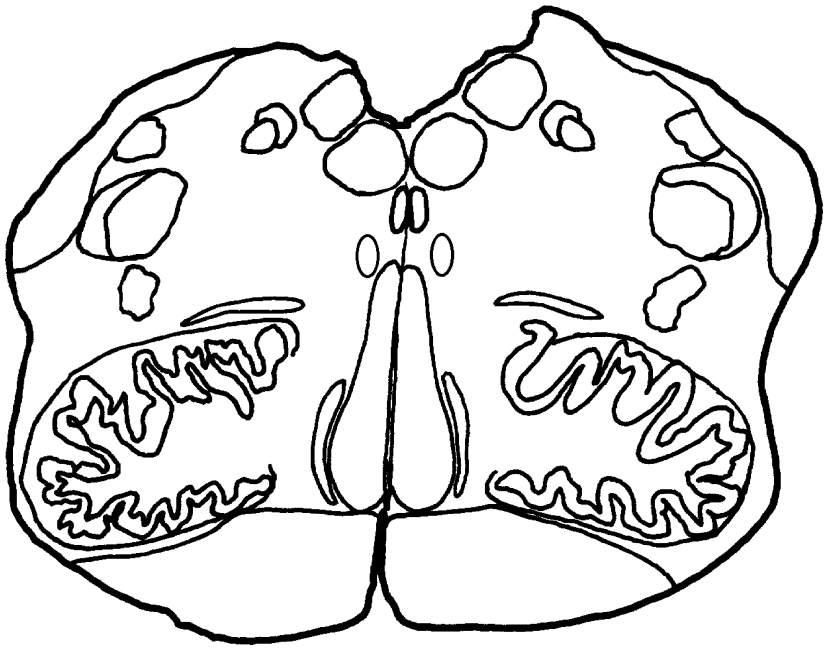
____3. left nucleus ambiguus

____4. dorsal motor X

____5. right inferior salivatory nucleus

A. axons project to the left side of the cerebellum
B. lesion results in deviation of the uvula to the left
C. cells of origin lie in the right accessory cuneate nucleus, right Clarke's' column and left inferior olive
D. lesion results in bilateral atrophy of the muscles of the soft palate, pharynx and larynx
E. cells contribute preganglionic parasympathetic input to the heart and lungs
F. cells project directly to the right otic ganglion
G. lesion results in deviation of the uvula to the right
H. lesion results in bilateral atrophy of the muscles of the tongue
I. lesion results in deviation of the tongue to the right upon protrusion
J. lesion results in a loss of pain and temp from the left side of the pharynx

PROBLEM SOLVING

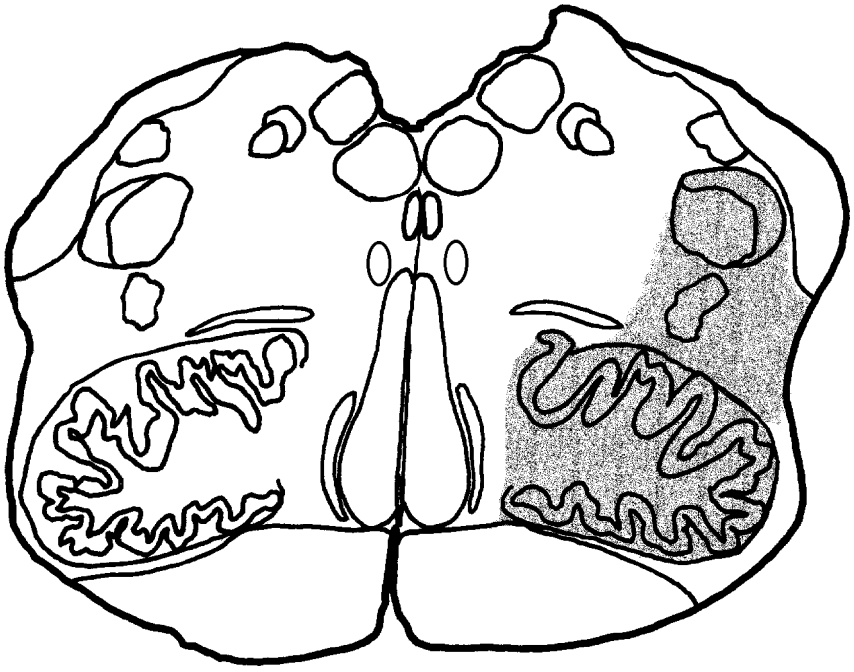


RIGHT LEFT

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

decrease in saliva, incoordination of the right arm and leg, hoarseness and dysphagia, ptosis of the left eyelid and constriction of the left pupil, pain in the left eye followed by loss of pain and temperature from the left side of the face, and pain in the right arm and leg followed by loss of pain and temp from the right arm and leg

PROBLEM SOLVING ANSWER



RIGHT LEFT

ANSWERS TO PROBLEM SOLVING QUESTIONS RELATED TO POINTS 6-10.

NOTE: The answers to ALL shade-in questions are illustrated on the back side of the question.

Point #6 Inferior Olive

Matching I,G,H,D,B

Point #7 Hypoglossal

Matching J,G,I,D,F

Point #8 Dorsal Motor X

Matching E,F,G,H,I

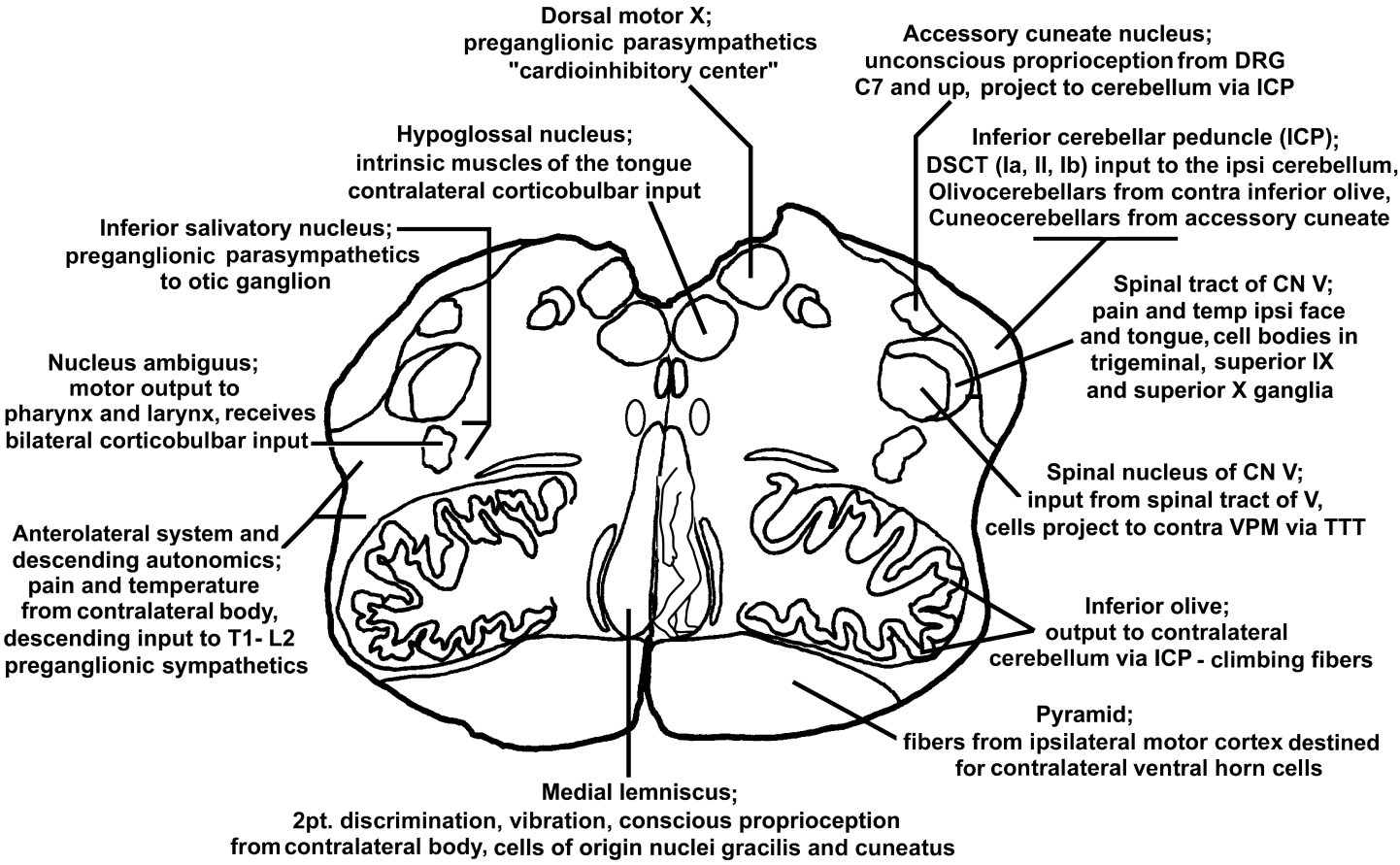
Point #9 Nucleus Ambiguus

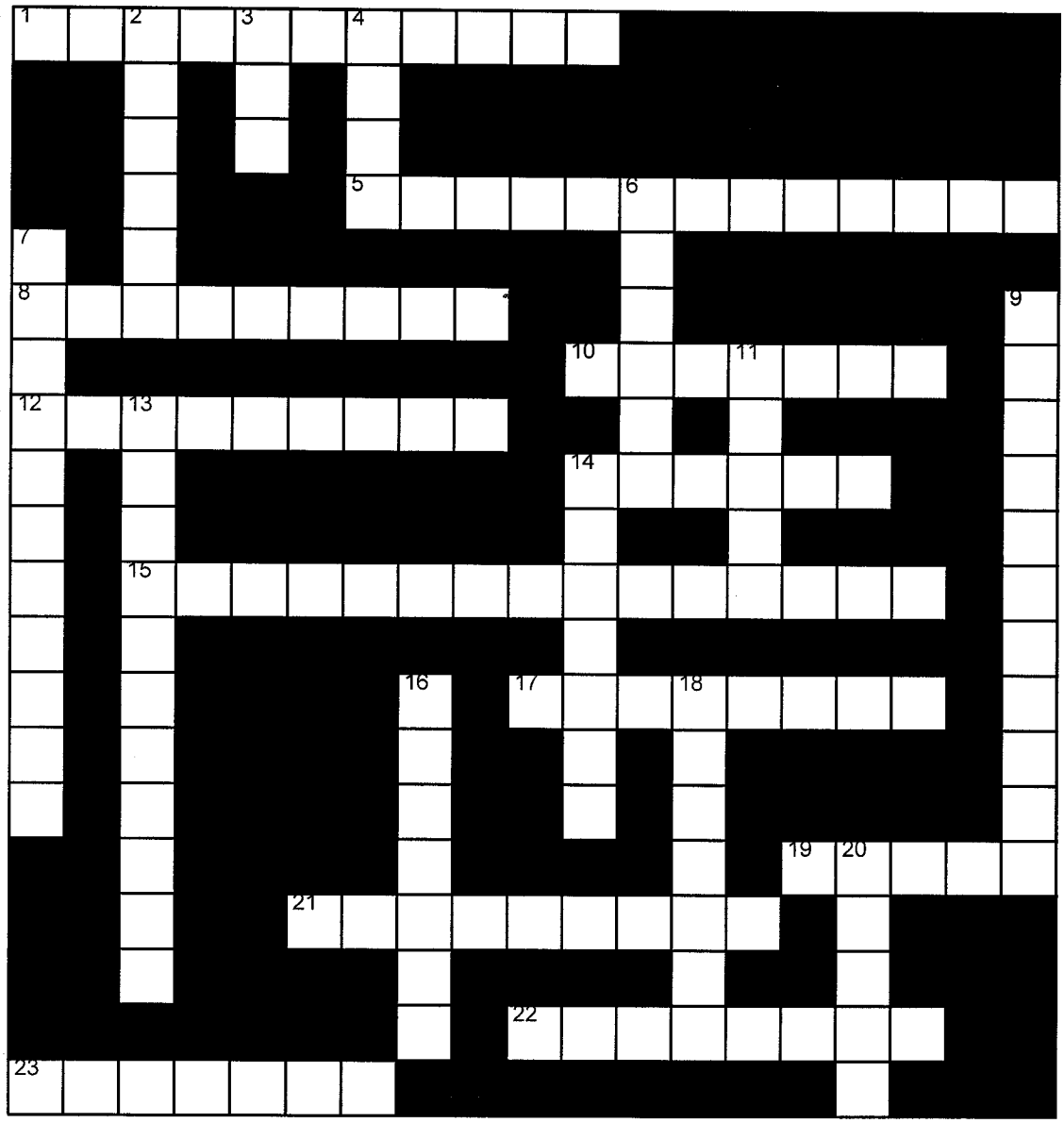
Matching E,J,G,I,H

Point #10 Inferior Salivatory

Matching C,I,G,E,F

Let's recap what we know about Level 3 so far





ACROSS

1. tongue nerve
5. crossed to C.N. XII
8. _____body
10. lesion involving LMN
12. difficulty swallowing
14. contains lysozyme (thanks GJR)
15. dorsal motor X
17. sole source of these fibers is inferior olive
19. innervated by ambiguus
21. hoarseness
22. not clear
23. located near the ear

DOWN

2. drooping
3. to retch
4. _____ganglion
6. side of deficit following lesion of inferior olive
7. results from parasympath. domination of heart rate
9. sympathetic domination of heart rate
11. martini
13. dominates following lesion of dorsal motor X
14. muscles involved controlled by nucleus ambiguus
16. what corticospinal tract does in pyramidal decussation
18. olive
20. the wanderer

Brain stem
Inferior Salivatory Nucleus
Problem Solving