MEDICAL NEUROSCIENCES 731

Medical Neurosciences is aimed at capturing the excitement and relevance of the dynamic field of basic and clinical neuroscience. Our goal is to present an overview of important and timely concepts regarding the structure and function of the nervous system. When you leave our course you should be able to handle the problems (listed in the box below) that the American Academy of Neurology is distributing to lay people to "talk to your physician" about. Every doctor should know the serious implications of these.

Many times in your career, no matter what type of doctor you become, both family members and patients will tell you these things. It is important to have several important diseases in your mind so your patients will avoid serious neurologic dysfunction and you will be known as a competent doctor, instead of one who gives false reassurance. Know the questions to ask and the signs to look for.

1. Dizziness	**7. Unsteadiness**
2. Headache	**8. Tremors/ Twitches**
3. Numbness/Tingling	**9. Head injury**
4. Memory/Concentration loss	**10. Sleep problems**
5. Blackouts/Seizure	**11. Sudden vision change**
6. Muscle weakness/Pain	**12. Slurred speech**

During the semester we will strive to:

- place you in a supportive environment
- give you timely feedback on how well you understand the material
- make the instruction problem-centered
- help you to be active rather than passive learners

We cannot overemphasize how important it is to:

- read the material before coming to lecture
- come to the lecture, relax, and just listen
- answer the practice questions
- attend all small group discussions
- enjoy learning about the nervous system and have a **great** time

MODULE 1

SPINAL CORD

SPINAL CORD CASE HISTORIES

BRAIN STEM

BRAIN STEM CASE HISTORIES

SELF LEARNING WEBSITES FOR MODULE I

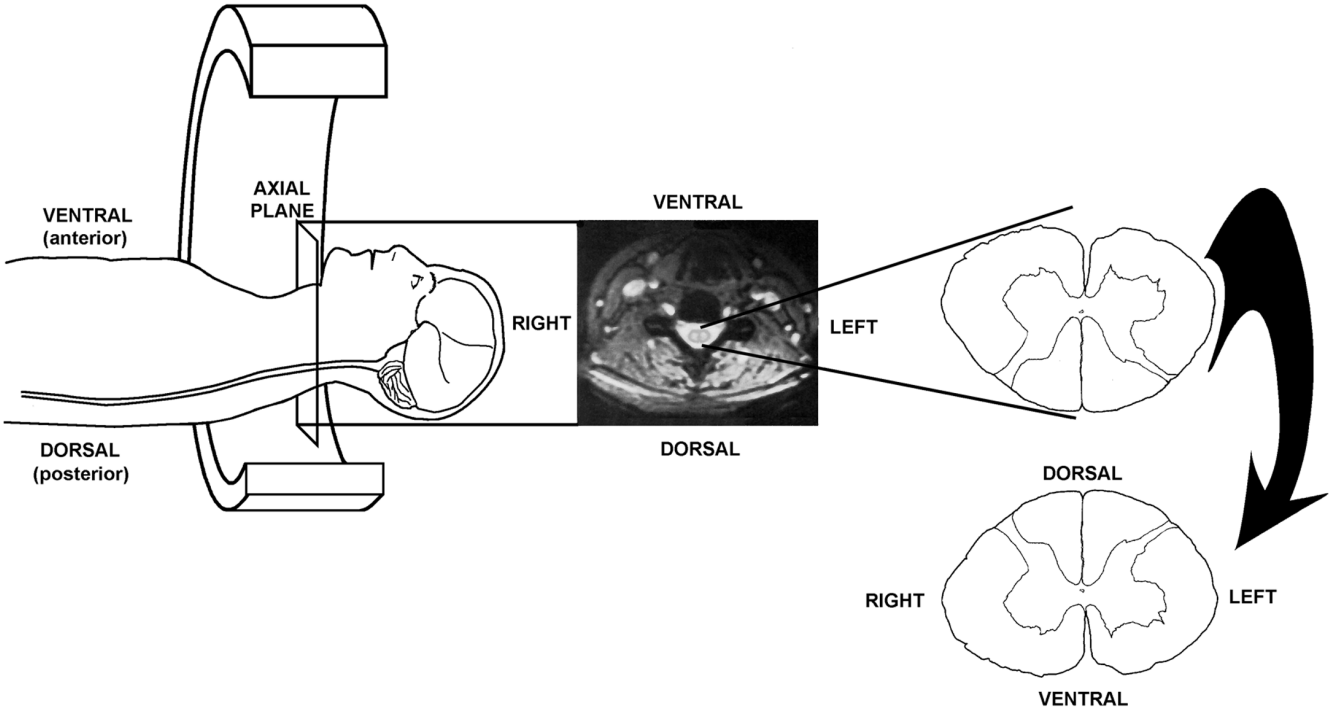
(All can be directly accessed from **www.neuroanatomy.wisc.edu**)

These are relatively short readings that you are to do on your own. There are several rules to this game. First, I cannot help you!! We know that this is 180 degrees different from the way we are presenting the course book material, but you need to do these exercises on your own so as to become independent learners. In other words, we will help you with the basic important facts about the brain and spinal cord so that you are in a position to then learn a little bit more on your own. I suggest that you do not wait till the night before the exam to do this web based learning. The things that you will be tested on from these readings are things **that are related to what you have learned thus far in the course**. The new information should broaden your horizons and pique your interest in clinical neurology, and help to reinforce the topics and concepts that we have stressed.

One final note. As you become more web-based in your learning, you will realize that material in our course book might sometimes differ from what you read on the internet. So the rule is, **go by the course book if there are conflicting data**. I know that this sounds like a cop-out, but as you become doctors you will realize that textbooks (and research and teaching groups) differ in their terminology and interpretations, so ***WHEN IN DOUBT, THE MODULE IS THE LAST WORD*!!**

LET'S GET ORIENTED

Before we can begin to study the organization of the spinal cord, we need to understand how radiologists, neurologists, and neurosurgeons view the central nervous system. You should remember some of this from Gross Anatomy, but I will go slowly. The drawing below shows a person positioned for Magnetic Resonance Imaging (MRI). You can see that the scan is in the axial plane through the rostral portion of the spinal cord. The most ventral (top!) part of the axial "slice" shows some things you should have seen in CT scans in Gross, like the mylo- and geniohyoids, while the dorsal (bottom) shows the back muscles. Somewhere in there you can see the relatively small spinal cord and associated parts of the vertebral column (body, transverse process, and spinous process of the vertebra). This is easy!



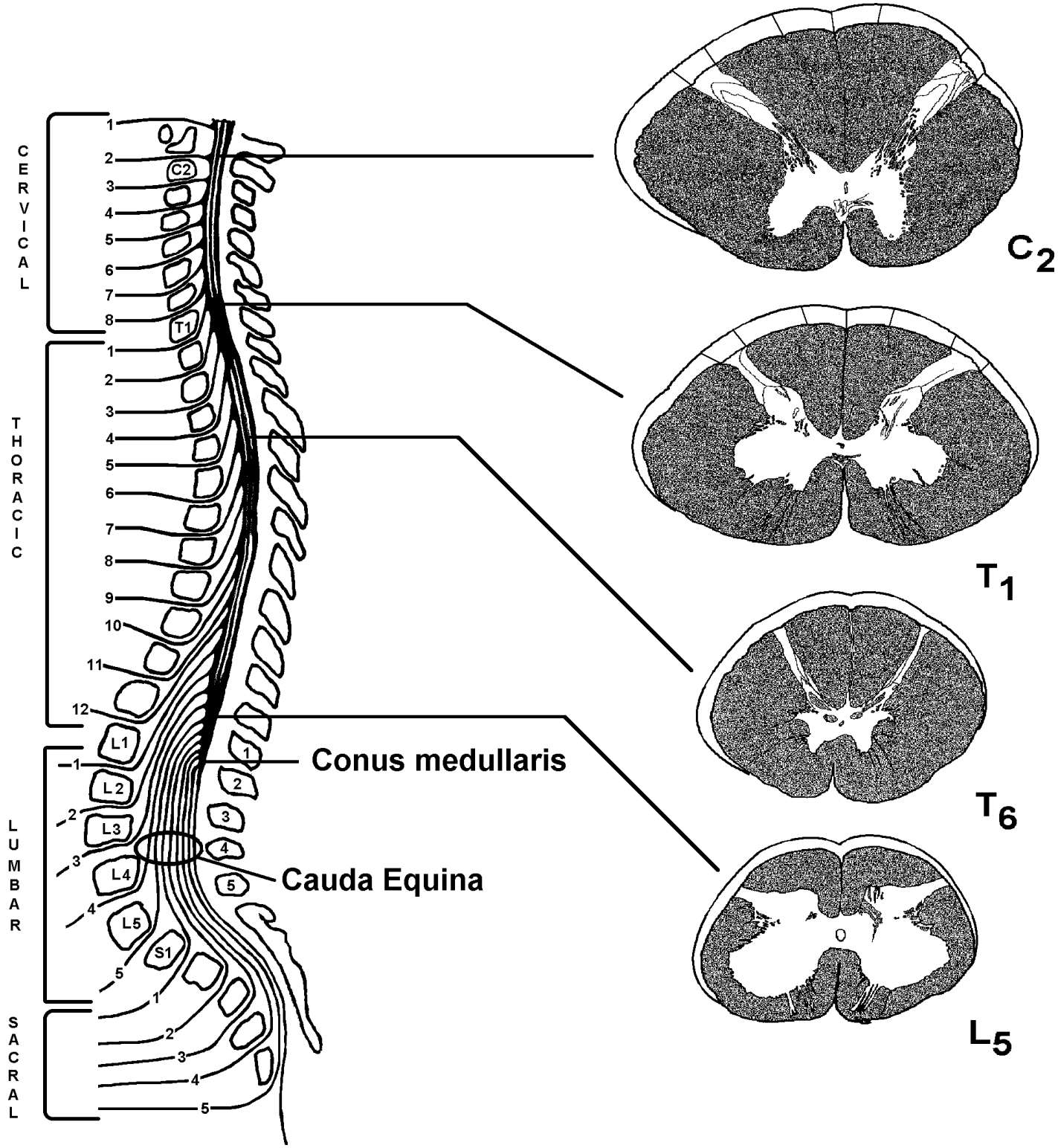
Now, for hard part #1—what is left and right on the MRI!!?? Keep in mind that you, the doctor, are viewing this scan from **the patient's feet.** So the right side of the patient's spinal cord is on your left. Radiologists view the scans this way and this is what we will use in this course. **SO, WHEN YOU LOOK AT A SKETCH OF AN AXIAL CUT OF THE SPINAL CORD, THE SIDE OF THE DRAWING ON YOUR *LEFT* WILL ACTUALLY BE THE *RIGHT* SIDE OF YOUR PATIENT**.

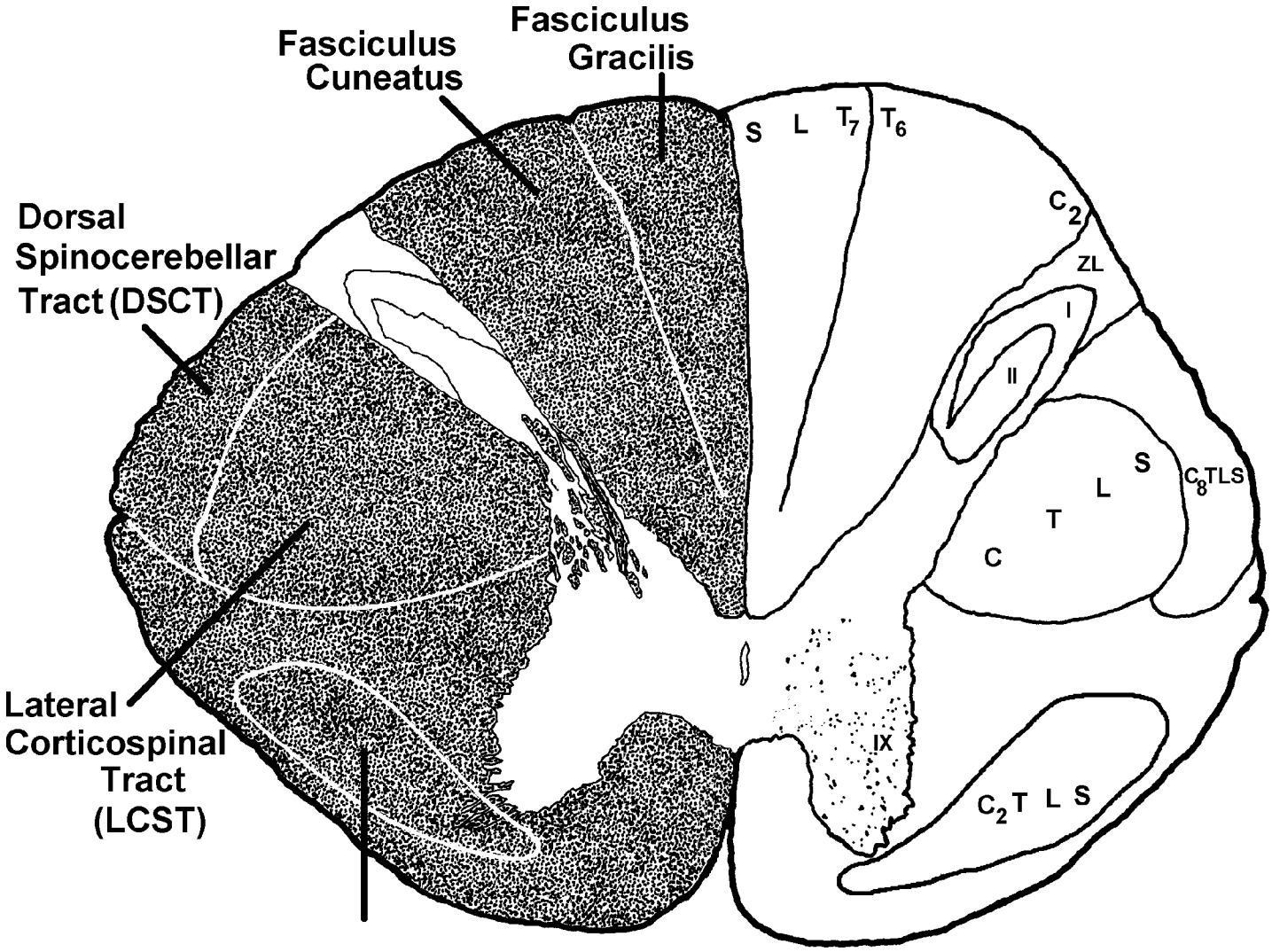
Now, for hard part #2. As you can see from the drawing, in an axial section MRI ventral is "up" and dorsal is "down". This can be seen below in the top drawing of the spinal cord. I am sorry to say that neuroscientists have traditionally viewed and drawn brain sections with dorsal up and ventral down (check out any neuro textbook or the internet). Sooo, we too will flip the section, but remember to keep the right/left designation of the MRI scans.

Now, I know you are really P.O.'ed about all of this flipping around, etc. Well, take my word for it, you will get over it and start flying through the course. As you will find out, I will constantly test you on important concepts and you will not have any trouble with these drawings. To show you how much you already know, here are several practice questions to help alleviate your stress. That is, you will get the questions correct and see that you are in fine shape. Here we go, and don't hang up without doing these.

The spinal cord is an extremely important component of the central nervous system. For the basic science component of this module, I have tried to organize the material in a way that will make you comfortable with the fundamental organization of the spinal cord. This will hopefully prepare you for solving the clinical case problems that the clinicians spring on you!!

I am aware of what you have learned about the spinal cord in Gross Anatomy, Histology and Physiology, and therefore will not dwell on gross structure, meninges or muscle spindles. What I will dwell on *ad nauseum* is the organization of **ascending sensory** pathways and **descending motor** pathways in the spinal cord. **BE SURE TO COMPLETE THESE PRACTICE QUESTIONS!** These questions will help you to evaluate your progress as we move through the various topics and build a more global view of spinal cord organization and function(s). If you get the answers correct, you are doing great**!** Trust me, there are no tricks!!! However, if you miss a few questions, go back and review the material that you have not understood (or that I have not clearly written or discussed??). Then go forward!!!! As you go through the spinal cord material the practice questions build on earlier material (and will therefore be more inclusive and difficult). If you do the questions faithfully, you will have the material well in hand. You will then be ready to problem solve and impress the clinicians.

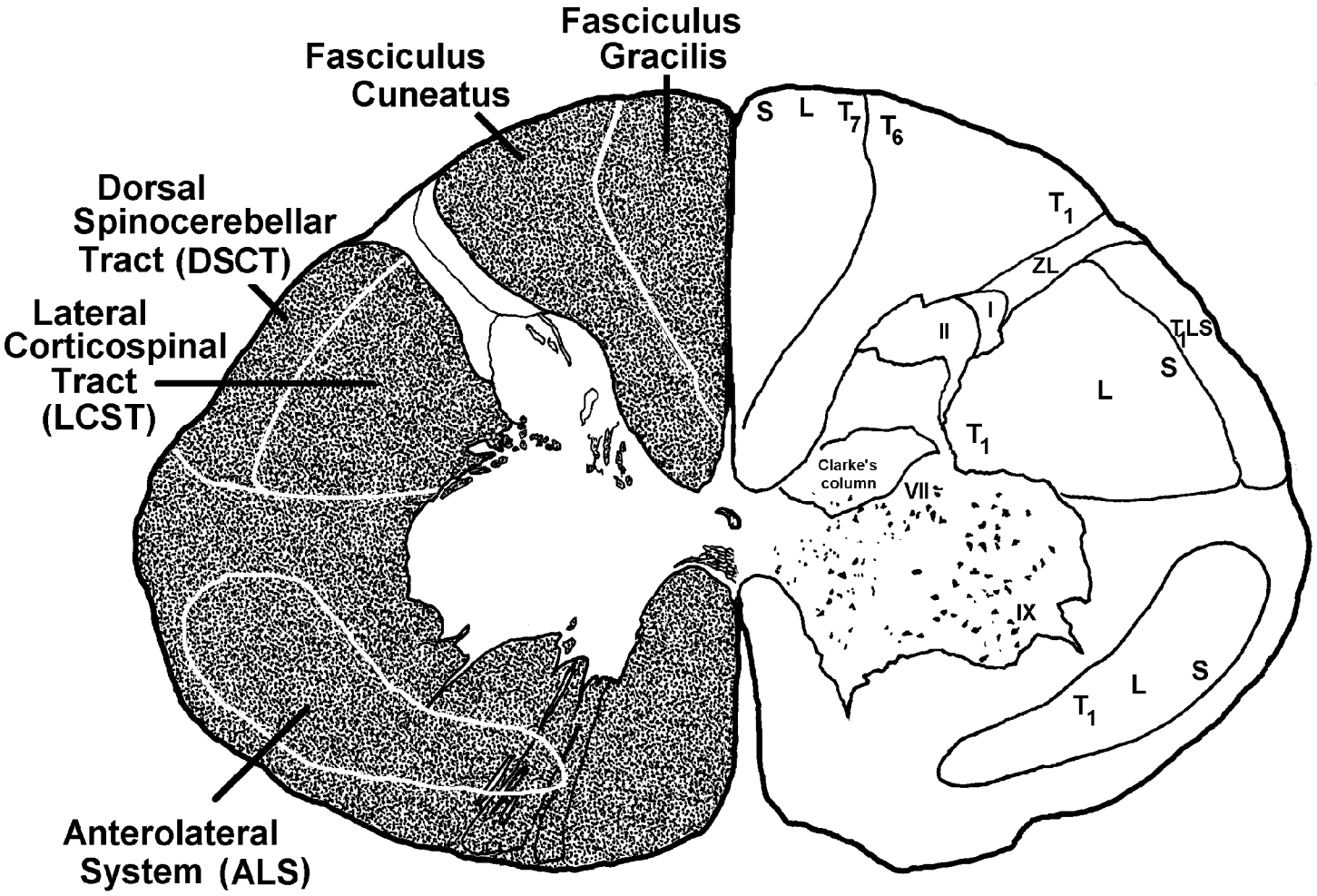

CERVICAL
THORACIC
LUMBAR
SACRAL
C2
T1
L1
L2
L3
L4
L5
S1
Conus medullaris
Cauda Equina
C2
T1
T6
L5



S- sacral L - lumbar T - thoracic C- cervical

ZL - Zone of Lissauer
I - Marginal zone of the dorsal horn
II - Substantia Gelatinosa
IX - Motor Nuclei (includes neurons of spinal accessory C.N. XI)

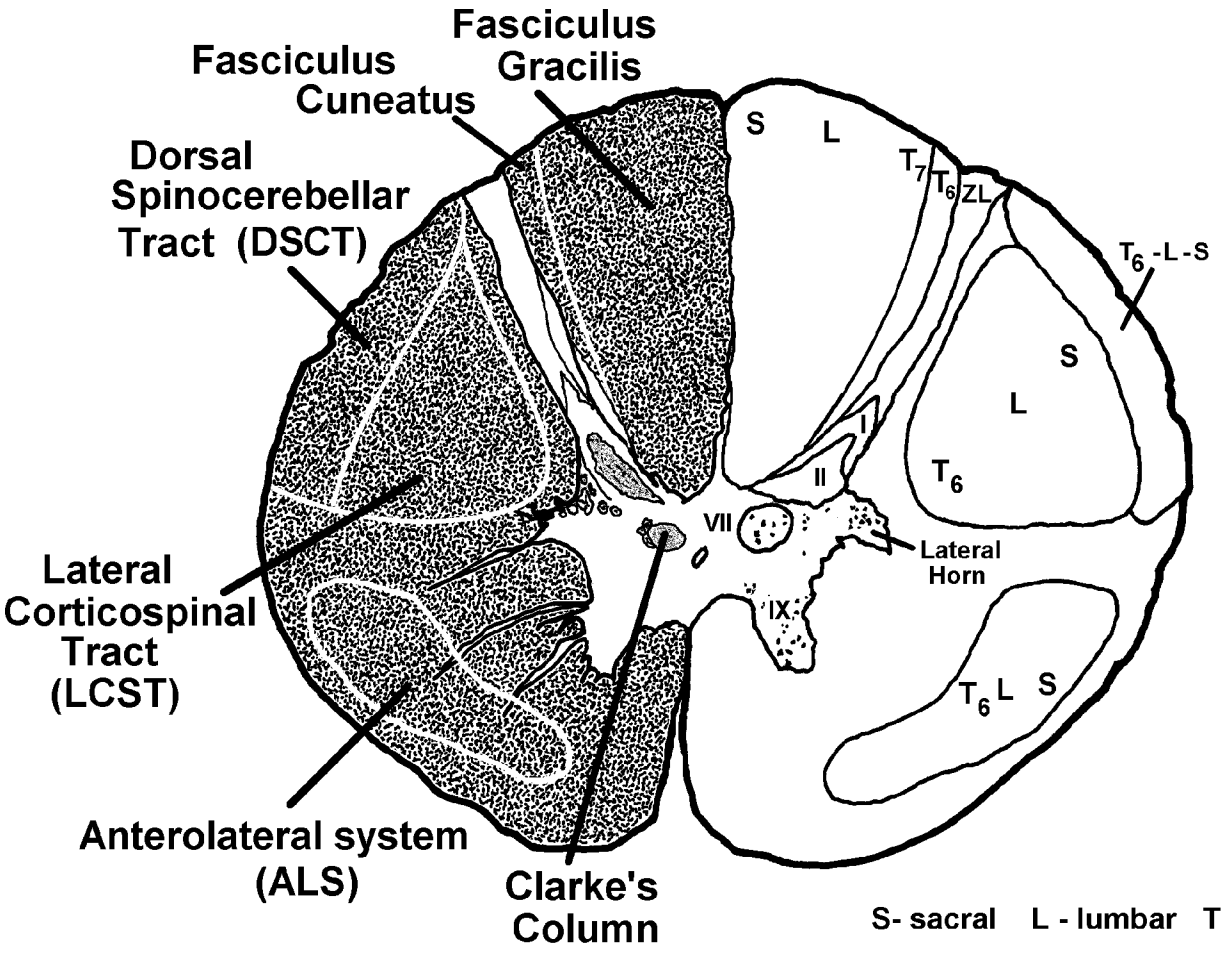
C_2



S- sacral L - lumbar T - thoracic

T_1

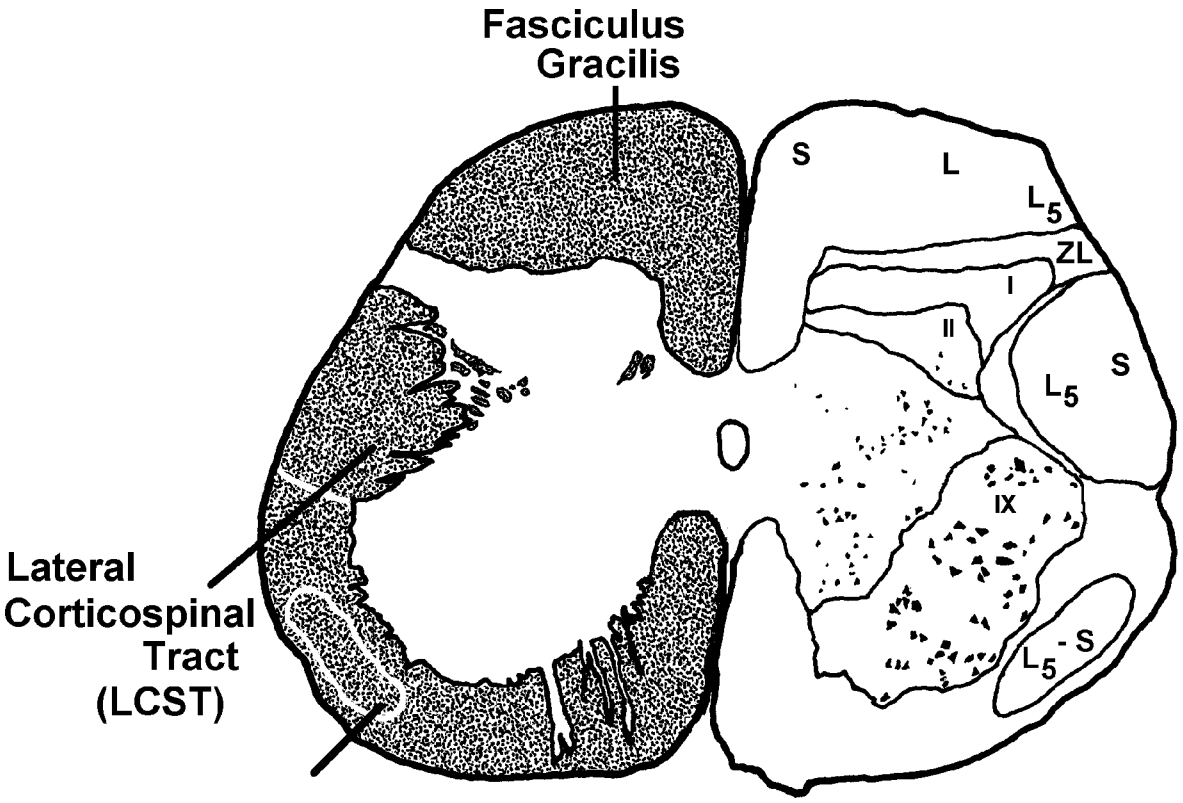
ZL - Zone of Lissauer
I - Marginal zone of the dorsal horn
II - Substantia Gelatinosa
VII - Intermediate zone (Includes neurons of Clarke's column C_8 - L_3)
IX - Motor Nuclei



S- sacral L - lumbar T - thoracic

ZL - Zone of Lissauer
I - Marginal zone of the dorsal horn
II - Substantia Gelatinosa
VII - Intermediate zone (Includes neurons of Clarke's column C_8 - L_3)
IX - Motor Nuclei

T_6



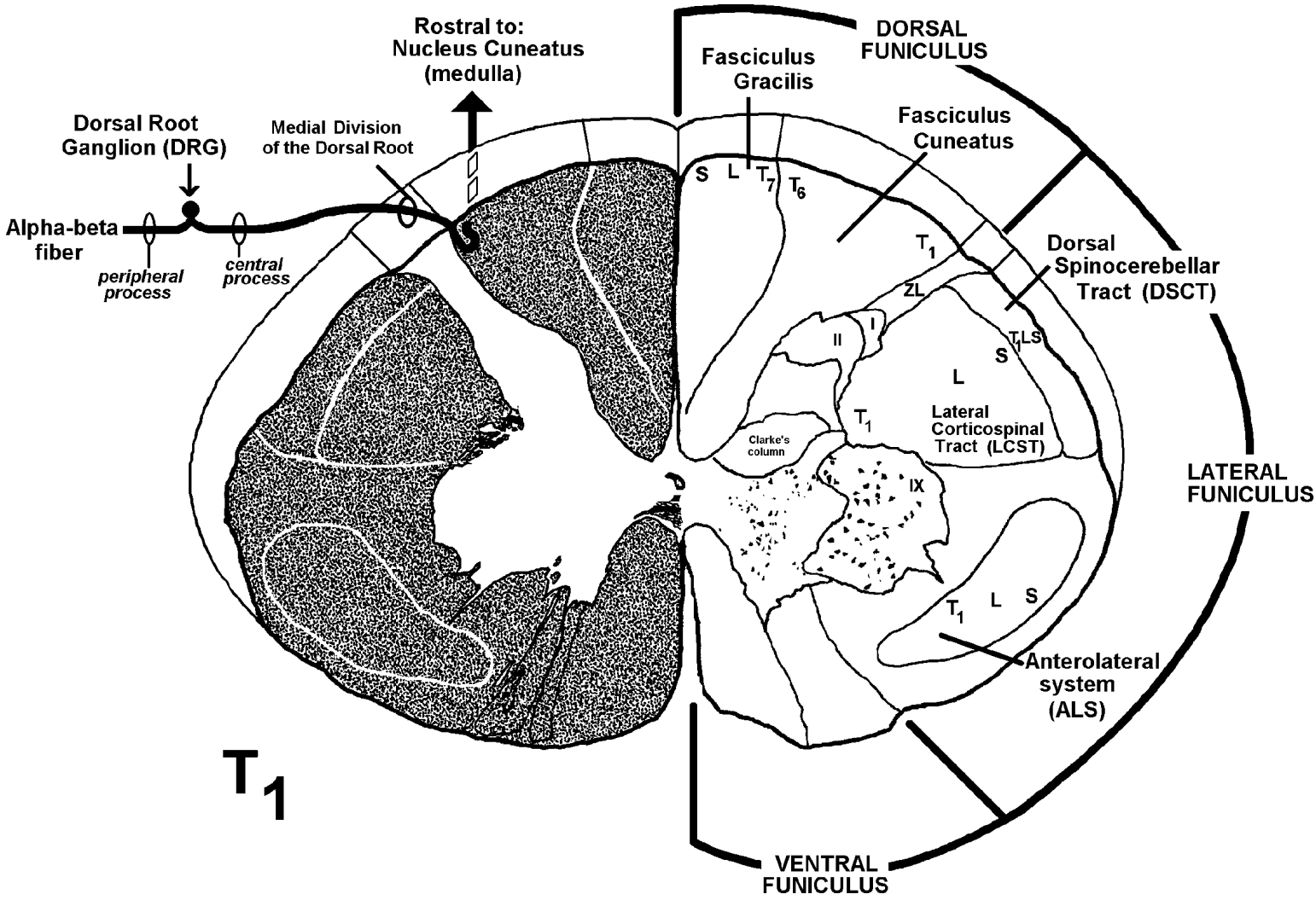
S- sacral L - lumbar

ZL - Zone of Lissauer
I - Marginal zone of the dorsal horn
II - Substantia Gelatinosa
IX - Motor Nuclei

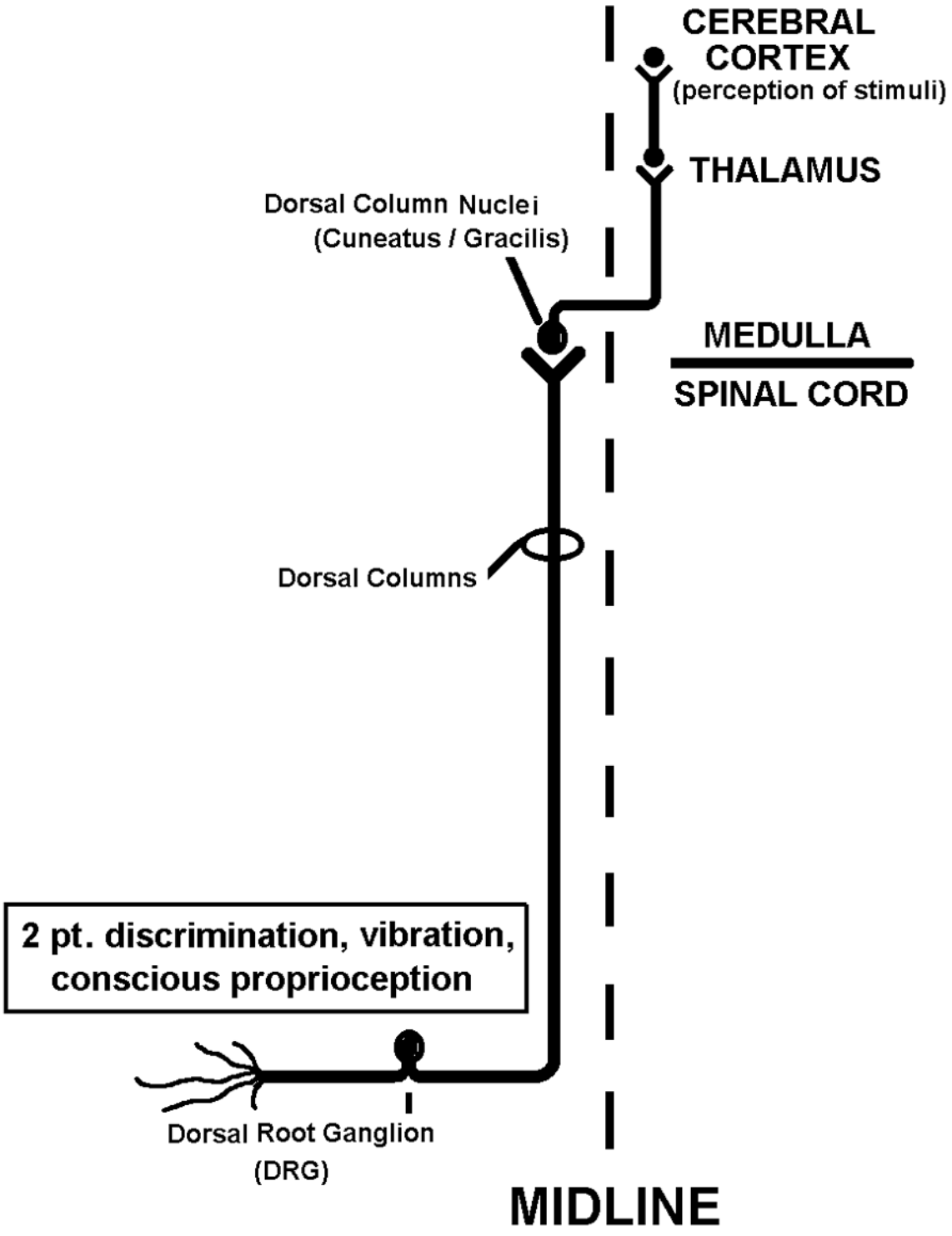
L_5

1 DORSAL COLUMNS (Fasciculus Gracilis and Cuneatus)

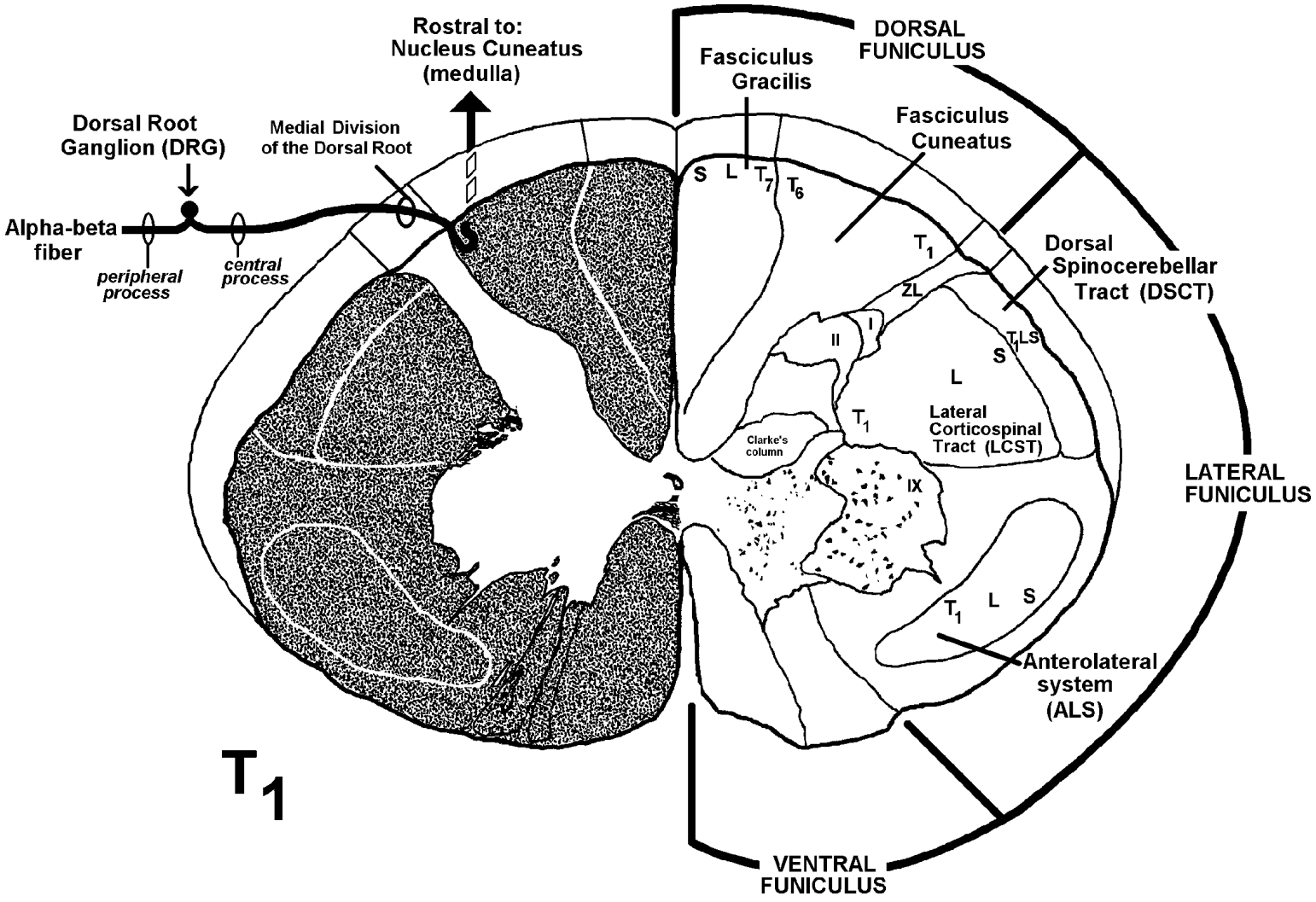
The spinal cord is comprised of an outer zone of white matter and a butterfly-shaped central component of cells and fibers (grey [or gray] matter). The peripherally located white matter consists of three funiculi or columns (**funiculus** = L., little cord) dorsal, lateral and ventral. I want to focus now on the ascending sensory pathways within the dorsal funiculus, or ***dorsal columns***.



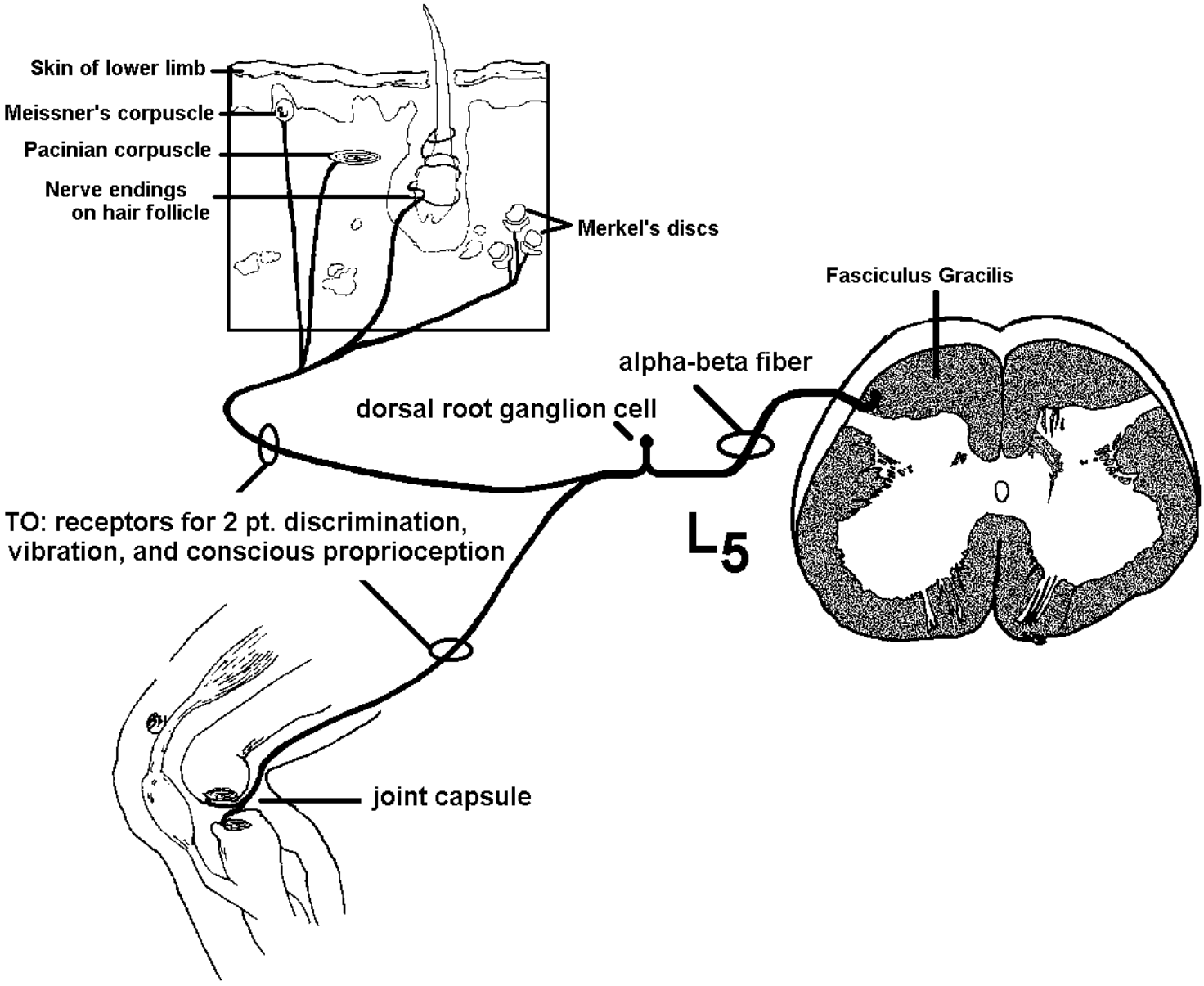
As shown below, the dorsal columns convey 2 point discrimination, vibration and conscious proprioception to nuclei in the medulla. These nuclei then send the information to the opposite side (**contralateral**) thalamus. Cells in the thalamus then project to the cerebral cortex where the perception of stimuli occurs.



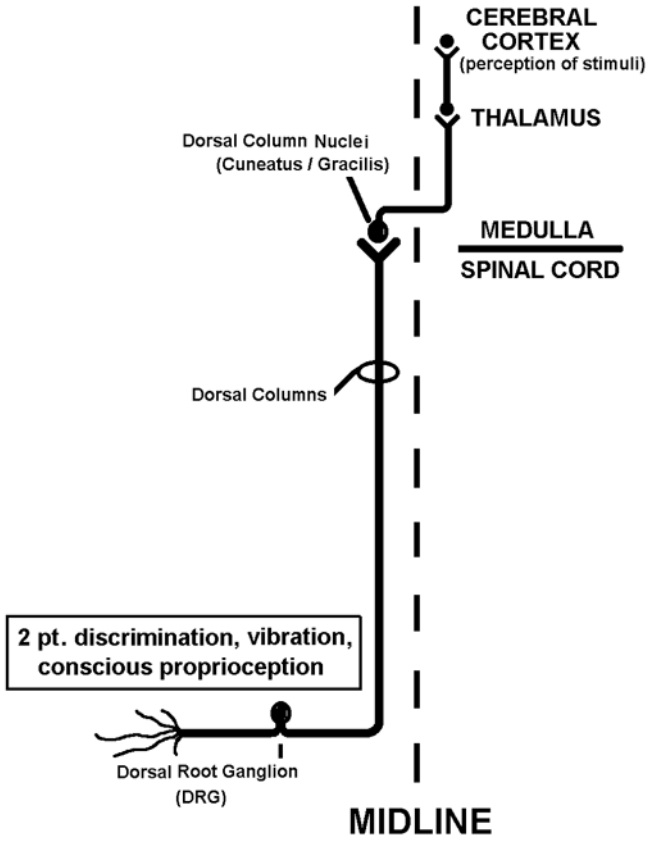
Now that we have an overview of the pathway let's get down to the nitty gritty (before we go to the Kohl Center). All **incoming** (**afferent**) information to the spinal cord is conveyed via the **dorsal root fibers**. Cells in dorsal root ganglia (DRG) possess **two** processes, one that passes **peripherally** to pick up information from a sensory receptor and one that passes **centrally** into the spinal cord. In the case of the dorsal columns, these axons are called **alpha-beta** fibers.

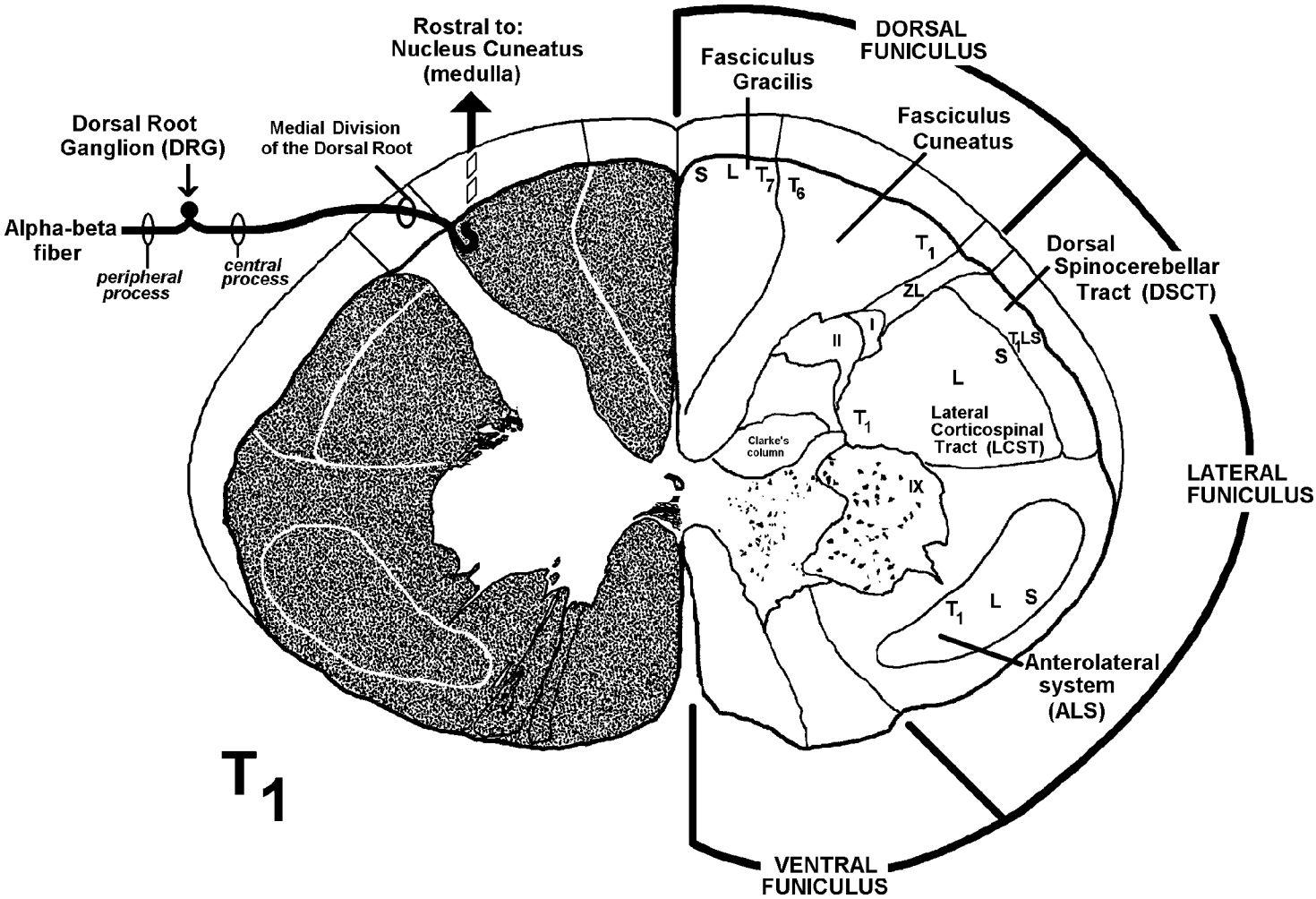


Alpha-beta axons are myelinated and measure from 6-12μm in diameter. Their peripheral processes possess specialized receptors such as Meissner's corpuscles, Merkel's (tactile) discs and Pacinian corpuscles. Meissner's corpuscles are primarily velocity detectors (movement across the skin) while Merkel's discs are primarily touch pressure receptors (how close the two points of a caliper are). Pacinian corpuscles are velocity detectors and sense vibration. The above three receptors lie in the skin and can account for **two point discrimination and vibration**. 2 pt. discrimination is our ability to tell how close two points are on our skin. It can also be called discriminative touch. **Conscious proprioception** (L. proprius = one's own; ceptor = a receiver) is the ability to tell the position of one's limb (is the arm bent or straight??) with the eyes closed. Ruffini corpuscles within joint capsules might convey such information.



So, here comes the information about two point discrimination, vibration and conscious proprioception (very important stuff!!) over the central process of the alpha-beta axon. As the central process of the alpha-beta axon approaches the spinal cord it travels in what is called the **medial division of the dorsal root** (this medial group of fibers will be contrasted with other central processes that lie laterally in the dorsal root [Point 2]). Once in the dorsal funiculus, the alpha-beta axon takes off for the medulla, where it synapses. The medulla is the most caudal part of the brain stem (midbrain, pons, medulla), and it lies immediately rostral to the spinal cord. Remember—there has been no synapse in the dorsal root OR spinal cord. **Also, the axon does NOT CROSS IN THE SPINAL CORD!!** It terminates in the medulla on the same side (IPSILATERAL; ipsi = L., same; latus = side) as its cell body. Cells in nucleus gracilis and cuneatus project to the thalamus. The information is then relayed to somatosensory cortex for perception. More on this later!! Let's stick to the spinal cord for now.

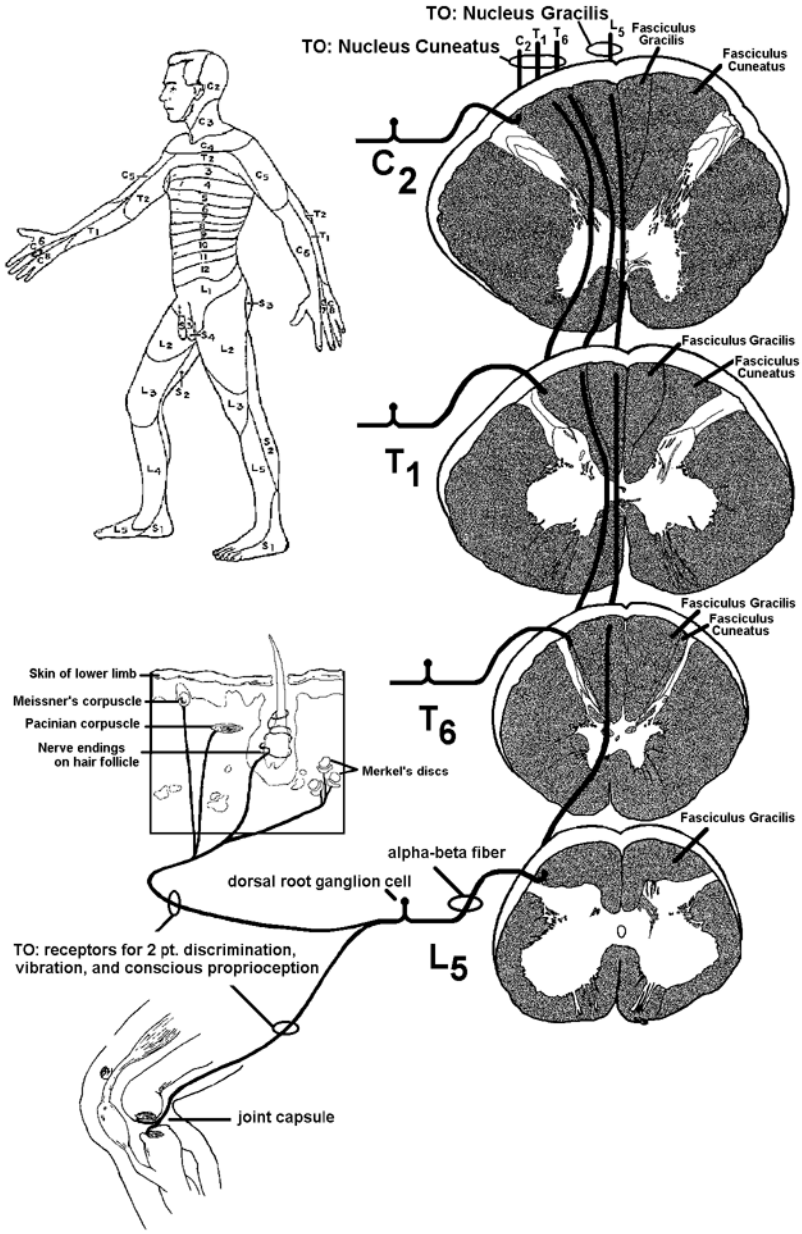




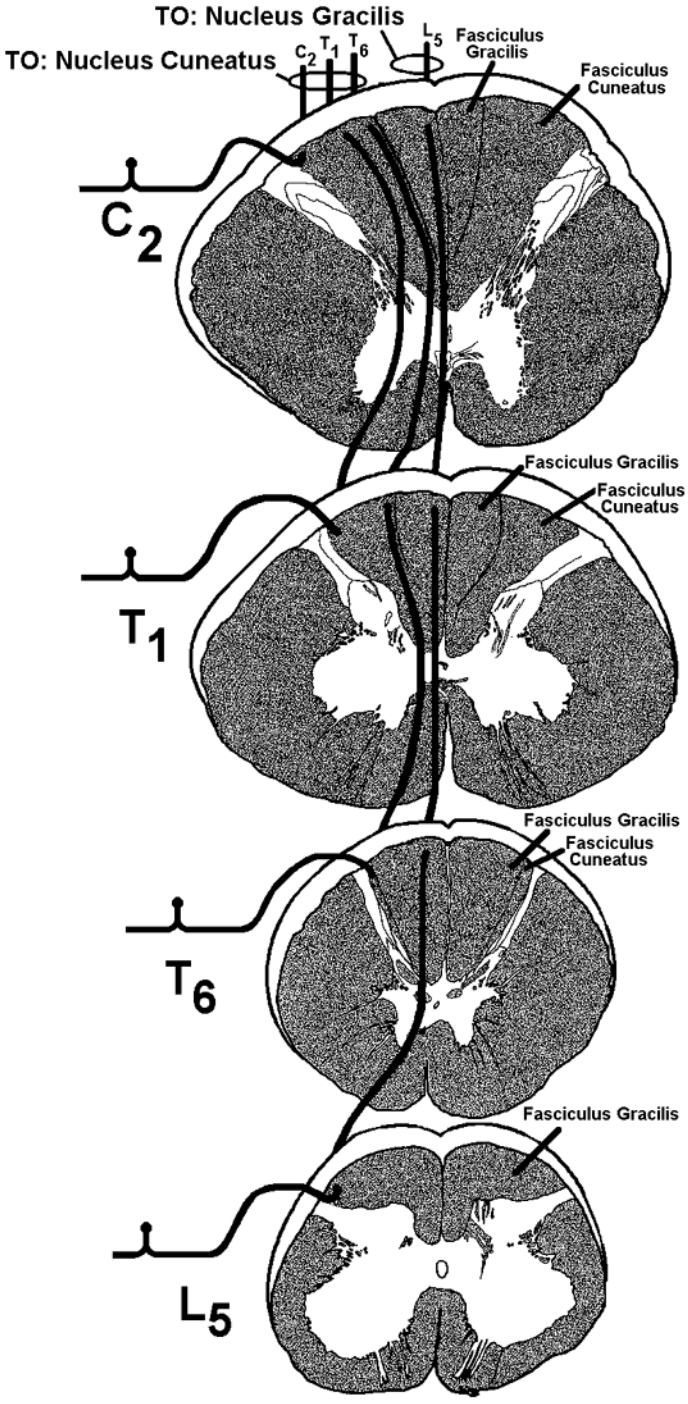
There are two components to the dorsal columns, called **fasciculus gracilis and fasciculus cuneatus** (fasciculus = L., little bundle; gracilis = slender; cuneatus = wedge). The central process of the alpha-beta fiber travels within the fasciculus gracilis if it arises from dorsal root ganglia **T7 and below.** In contrast, if the central process of the alpha-beta fiber arises from cells in dorsal roots **T6 and above** (toward your head), it is part of **fasciculus cuneatus.**

CUNEATUS = "ARM" = T6 and up
GRACILIS = "LEG" = T7 and down

Fasciculus gracilis and fasciculus cuneatus are thus comprised of the alpha-beta axons whose cell bodies lie in **IPSILATERAL DORSAL ROOT GANGLIA**. That is, the cell bodies are on the **SAME SIDE** as the fasciculi. I have mentioned that fibers in the dorsal columns **DO NOT CROSS** in the spinal cord and eventually synapse in the medulla. While we will cover the medulla later in the course, you might like to know that axons in fasciculus gracilis terminate in the ipsilateral (to the fasciculus) **nucleus gracilis,** while fibers in fasciculus cuneatus synapse in ipsilateral **nucleus cuneatus** (big surprise).



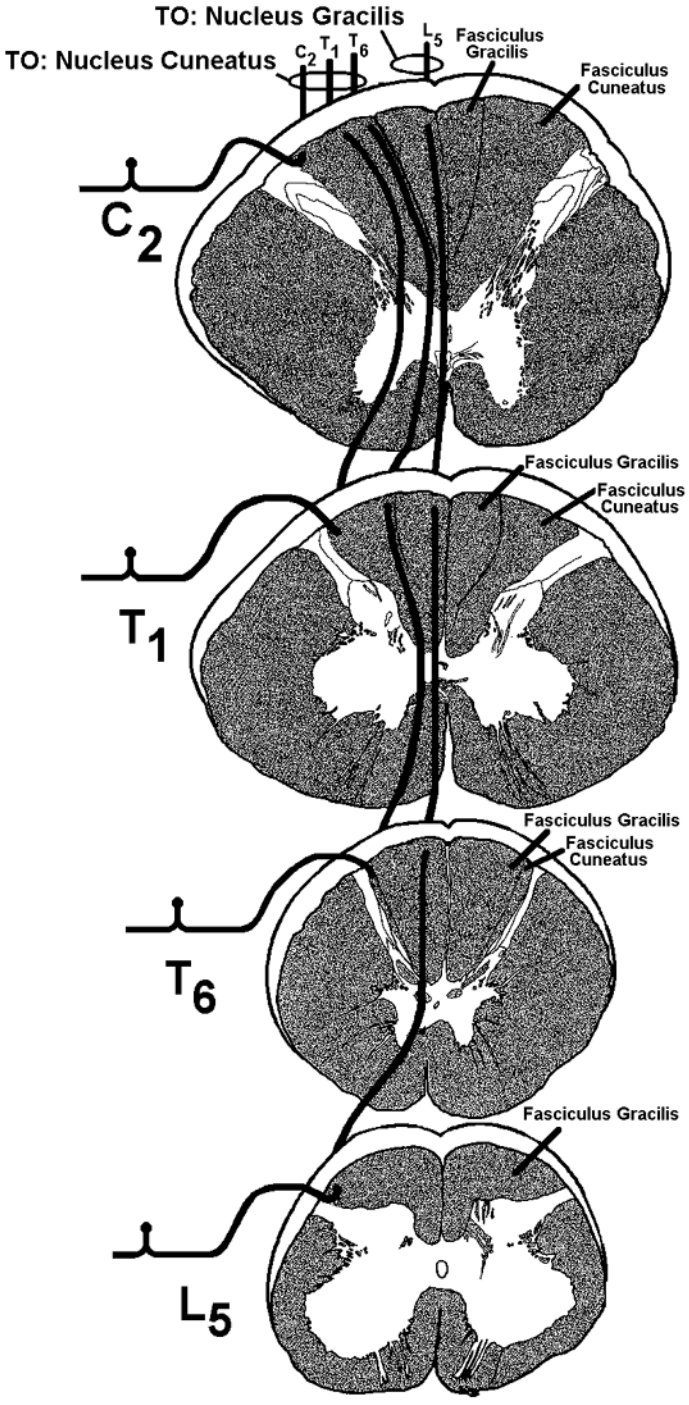
The **fasciculus gracilis** contains fibers from spinal cord levels lower than fasciculus cuneatus, and fasciculus gracilis lies **MEDIAL** to fasciculus cuneatus. This lower = medial spatial relationship is an example of **somatotopic organization** and holds not only for the two fasciculi, but also for the individual fibers in each fasciculus. For example, the most **medially** placed fiber in **fasciculus gracilis** arises from the **coccygeal** dorsal root and the most **laterally** placed arises from the **T7** dorsal root. In the **fasciculus cuneatus**, the most **medially** placed fiber arises from dorsal root **T6** and the most lateral arises from dorsal root **C2** (remember from Gross Anatomy that C1 is purely motor, and therefore does not have a dorsal root ganglion?!!).



What happens when there is a lesion anywhere in the system involving the peripheral processes of the dorsal root neurons, the fasciculus gracilis, the fasciculus cuneatus, the nucleus gracilis and the nucleus cuneatus? Let's take the peripheral processes first and include all alpha-beta fibers which can carry **TWO POINT DISCRIMINATION, VIBRATION, AND CONSCIOUS PROPRIOCEPTION.** Such a lesion would result in interruption of the information from the region of the body innervated by that dorsal root. This is called a **dermatome**. While there is overlap of adjacent dermatomes, don't worry about that now. Think about the distribution of the peripheral process of each dorsal root.

Following a lesion of the dorsal root the resulting deficits are manifest on the same side as the lesion = **IPSILATERAL.**

Deficits that result from a lesion in the dorsal column system (i.e. in the spinal cord) would differ depending on the precise location of the lesion. For instance, following a lesion at spinal cord level C2 which damages both fasciculi, information from the entire ipsilateral side of the body (and the back of the head, which is innervated by C2) would not reach the nucleus gracilis and nucleus cuneatus, and thus we would never feel the sensations (they don't reach consciousness via pathways from the medulla to the cerebral cortex). If the spinal cord lesion involves only fasciculus gracilis at spinal segment **C2**, only the information from spinal segments **T7** and below (all the way down) is lost. Information from the arm is **OK** because fasciculus cuneatus is fine. If the lesion lies at **S1**, then only the ascending information from spinal segments **S1** and below are affected. Information coming in above **S1** (toward the head) gets in OK and ascends to the caudal medulla.

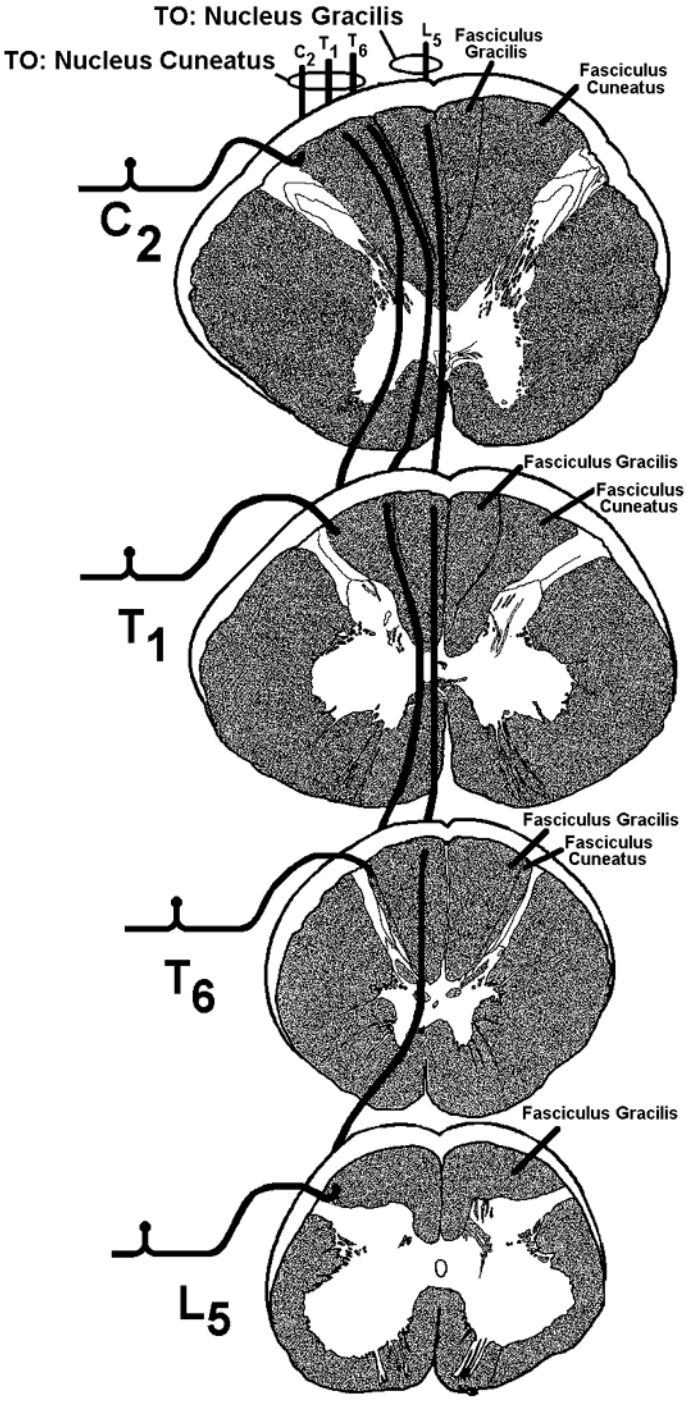


In addition to the loss of **2 pt. discrimination, vibration and conscious proprioception**, you should know that dorsal column lesions result in **astereognosia** (Gr. steros = solid, gnosis = recognition), which is the inability to recognize objects or forms by touch. Put a key in your hand with your eyes closed and you can identify it as a key. Another problem is called **agraphesthesia** (inability to recognize letters, numbers, etc., drawn on the skin). In the upper extremity, these sensory losses result in clumsiness or **ataxia**. Finally, damage to the dorsal columns sometimes presents as **paresthesia** (Gr.- para = abnormal, aisthesis = sensation) which is **tingling and numbness**. Think of this as resulting from irritation of the fibers as they die.

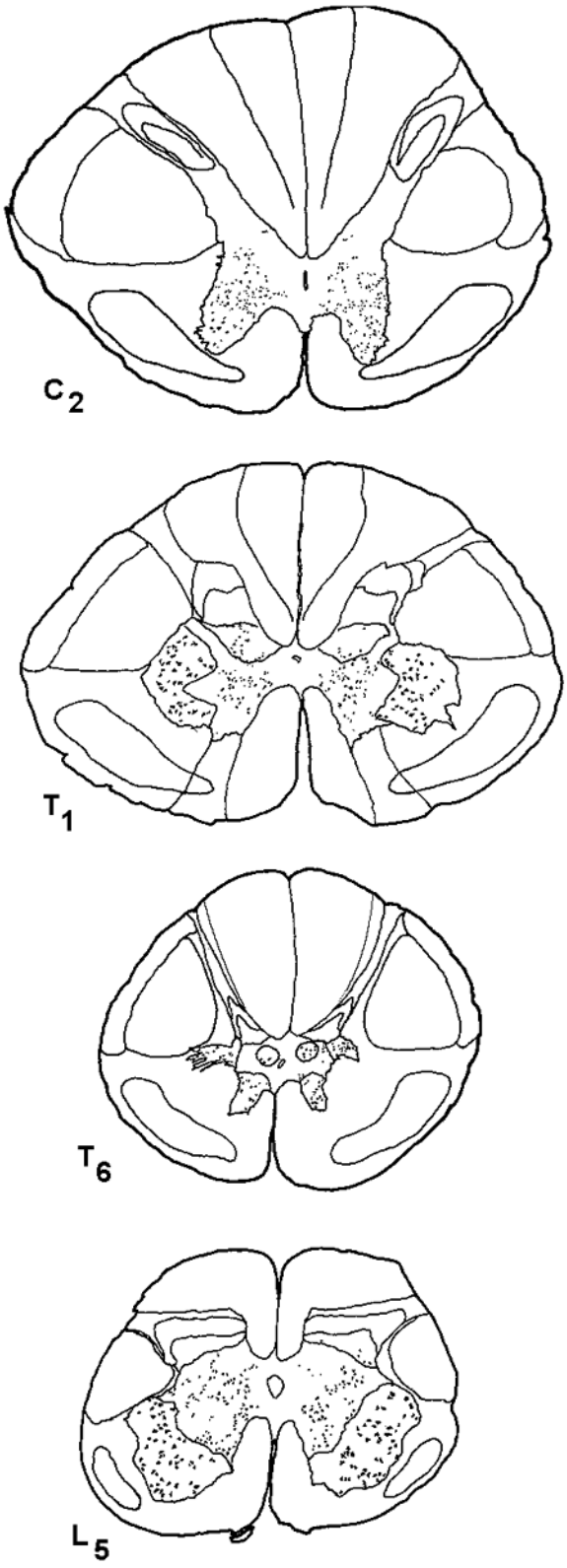
The ataxia (lack of order or incoordination) that results from lesions of the dorsal columns is due to the loss of proprioceptive information regarding the position of our limbs. If the fasciculi gracili are involved, the patient will exhibit a **Romberg sign**. To test for this sign the patient is asked to stand with their feet together and their eyes open. If closing the eyes causes the patient to sway then there is a Romberg sign. That is, the patient has lost the sensory proprioceptive input and once visual inputs are eliminated the deficit becomes apparent.

When a patient with a dorsal column lesion steps forward the legs are flung abruptly forward, often being lifted higher than necessary. This is seen in **tabes** (wasting) **dorsalis** or **neurosyphilis**. There is an audible sound as the foot stamps the ground (they are not sure when it hits) and since they usually have a cane it is refered to as a "stick and stamp."

Finally, patients with dorsal column disease in the cervical region exhibit a **Lhermitte's sign**. This is described as the sensation of an "electric shock" that runs down the vertebral column and permeates the arms and legs. These sensations are set off by flexion at the neck which stretches the dorsal columns in the cervical region. This stretching causes demyelinated axons in the dorsal columns to send "funny" messages to the cortex.



Use the diagram below to draw your own lesions (hopefully you will be more creative than me!) and think about the resulting deficits



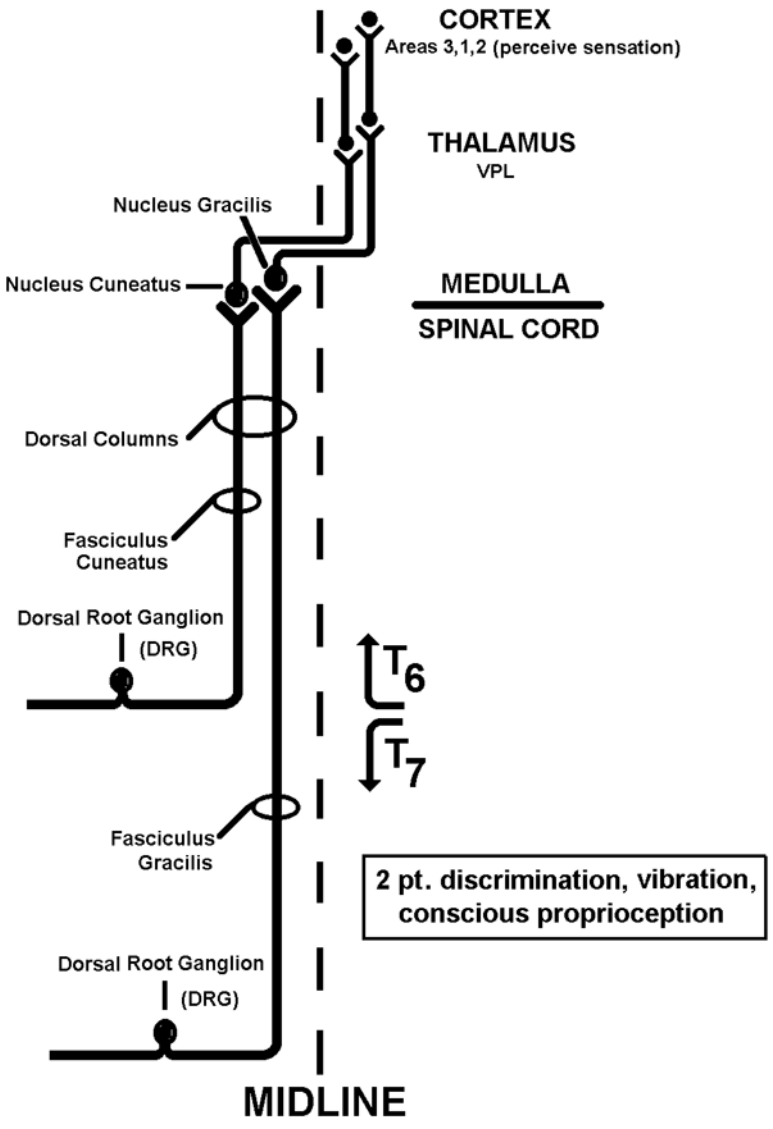
LET'S REVIEW THE DORSAL COLUMNS

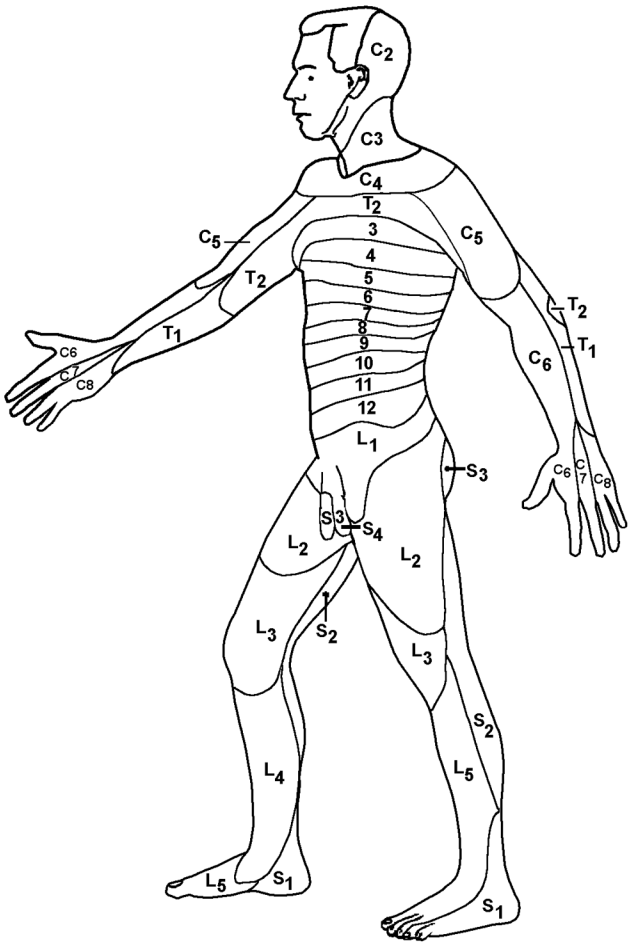
1. **CELLS OF ORIGIN** = ipsilateral dorsal root ganglia(alpha-betas; 6-12μ)
1. fasciculus **gracilis** = T7 and below ("LEG")
2. fasciculus **cuneatus** = T6 and above ("ARM")

2. **LOCATION** = dorsal funiculus

3. TERMINATION = ipsilateral nucleus gracilis and nucleus cuneatus (medulla)

4. LESION DEFICIT(S) = ipsi 2 point discrimination, vibration, conscious proprioception, astereognosia, agraphesthesia and ataxia





You will find this dermatome chart useful in the problem solvings. We will provide more details later in the course.

PROBLEM SOLVING MATCHING

Match the best choice in the **right** hand column with the pathway or cell group in the **left** hand column. **There might be deficits that are not included in the responses.**

_____1. right fasciculus gracilis at C2

_____2. left fasciculus cuneatus at C2

A. lesion results in deficit in vibration sense from left leg

B. axons arise from dorsal root ganglia **T7** and below on the right

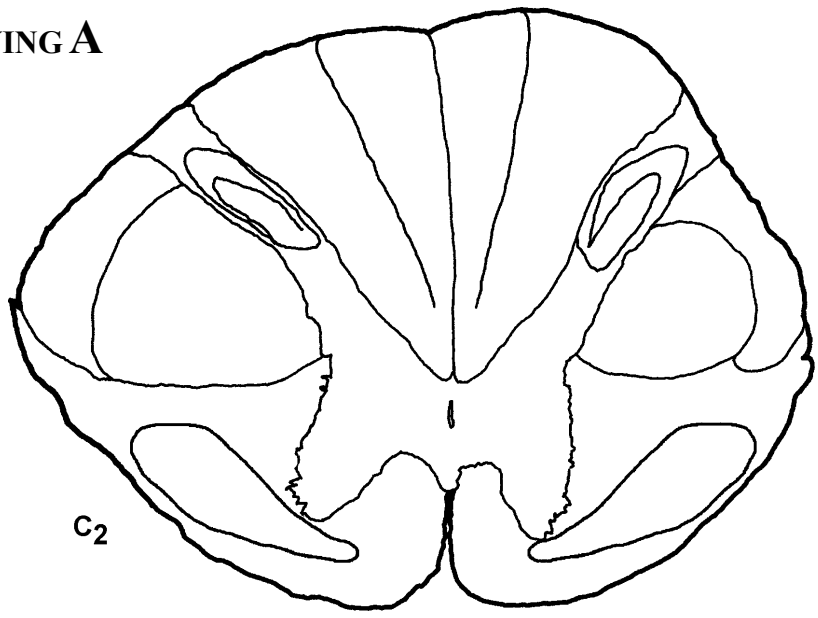
C. axons terminate in right nucleus cuneatus

D. lesion results in deficit of 2 pt. discrimination from the right hand

E. lesion results in deficit in conscious proprioception from the left elbow

Refer to Table of contents for Problem Solving ANSWER sets.

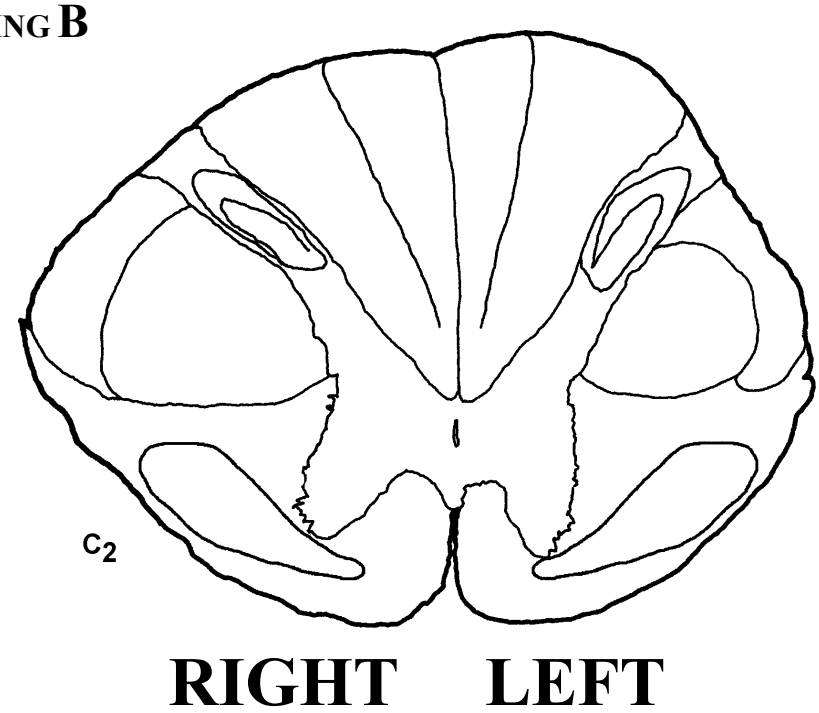
PROBLEM SOLVING A



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

deficit in conscious proprioception, vibration, and two point discrimination from **only** the right foot (be careful)

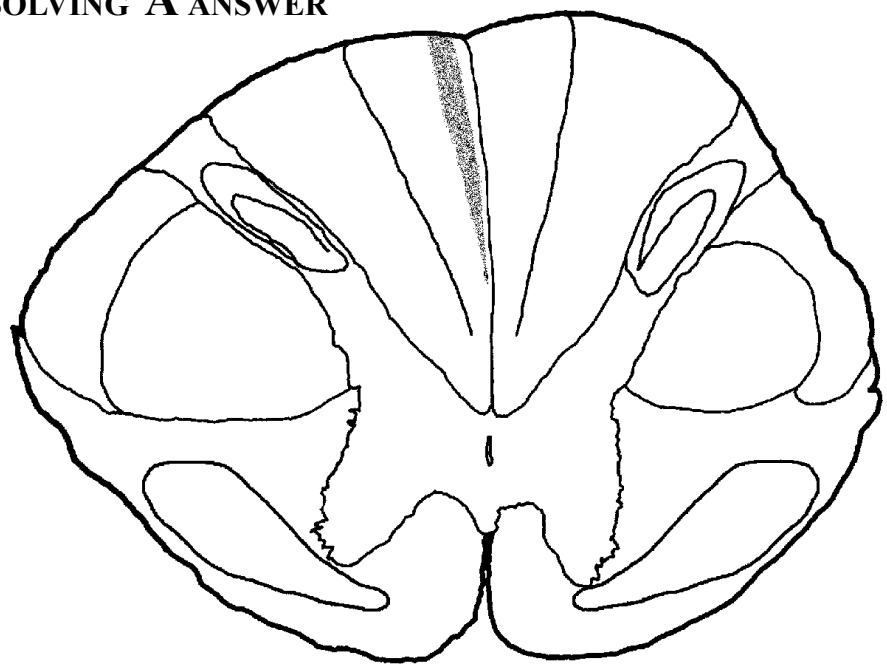
PROBLEM SOLVING B



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

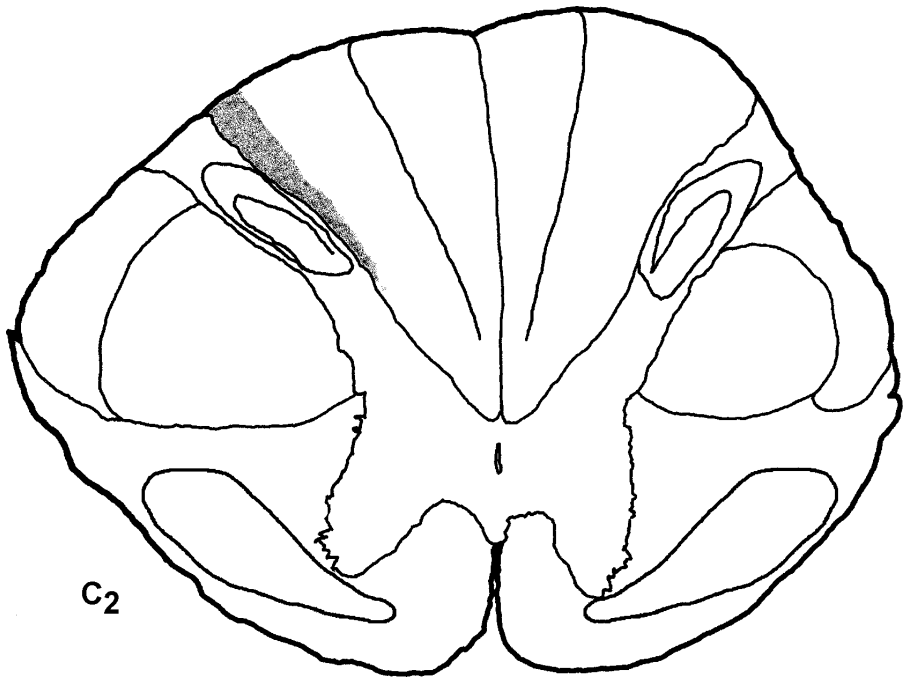
deficit in conscious proprioception, vibration and two point discrimination from **only** the right side of the neck (be careful!!)

PROBLEM SOLVING A ANSWER



RIGHT LEFT

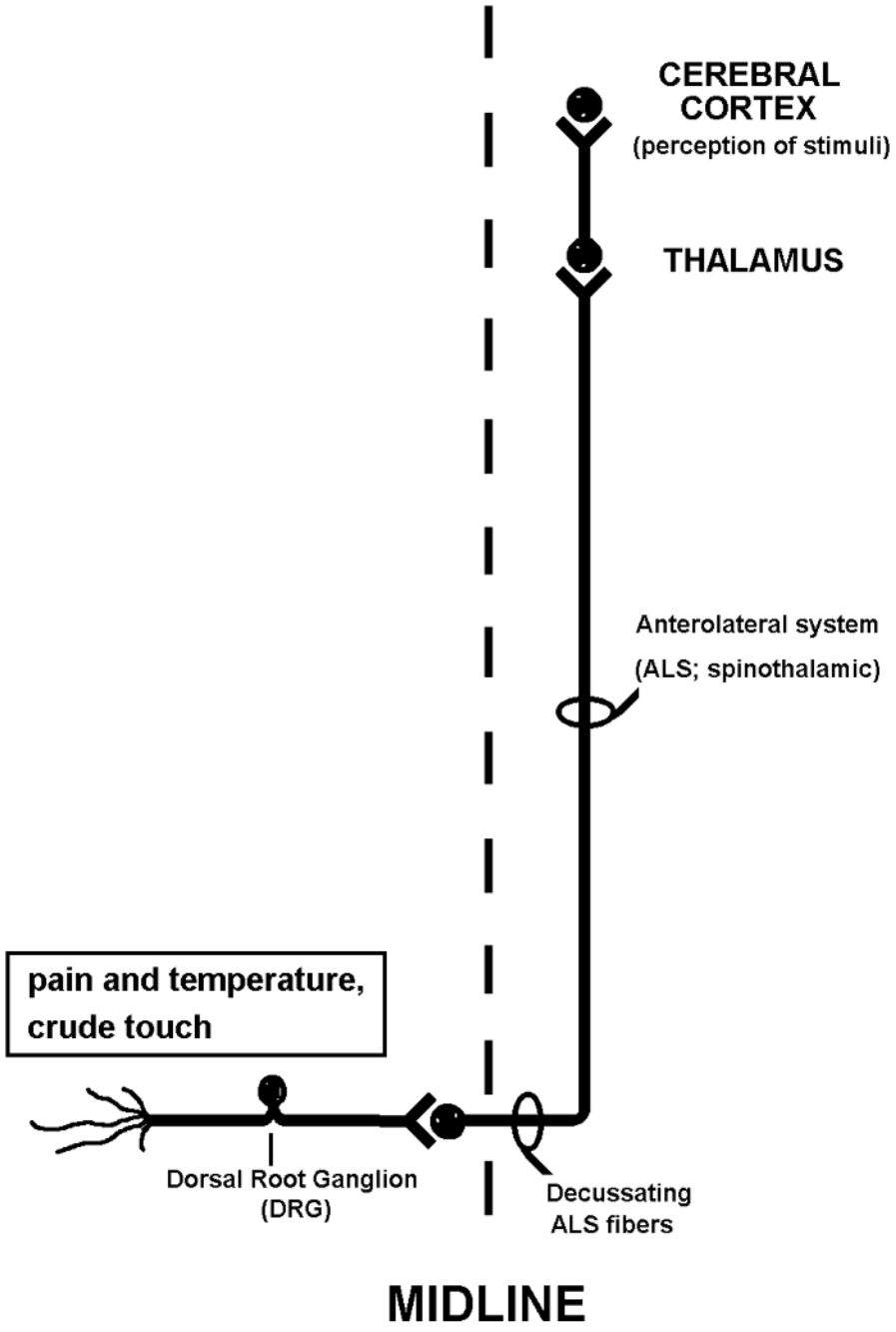
PROBLEM SOLVING B ANSWER



RIGHT LEFT

2 ANTEROLATERAL SYSTEM (ALS) or Spinothalamic Tract

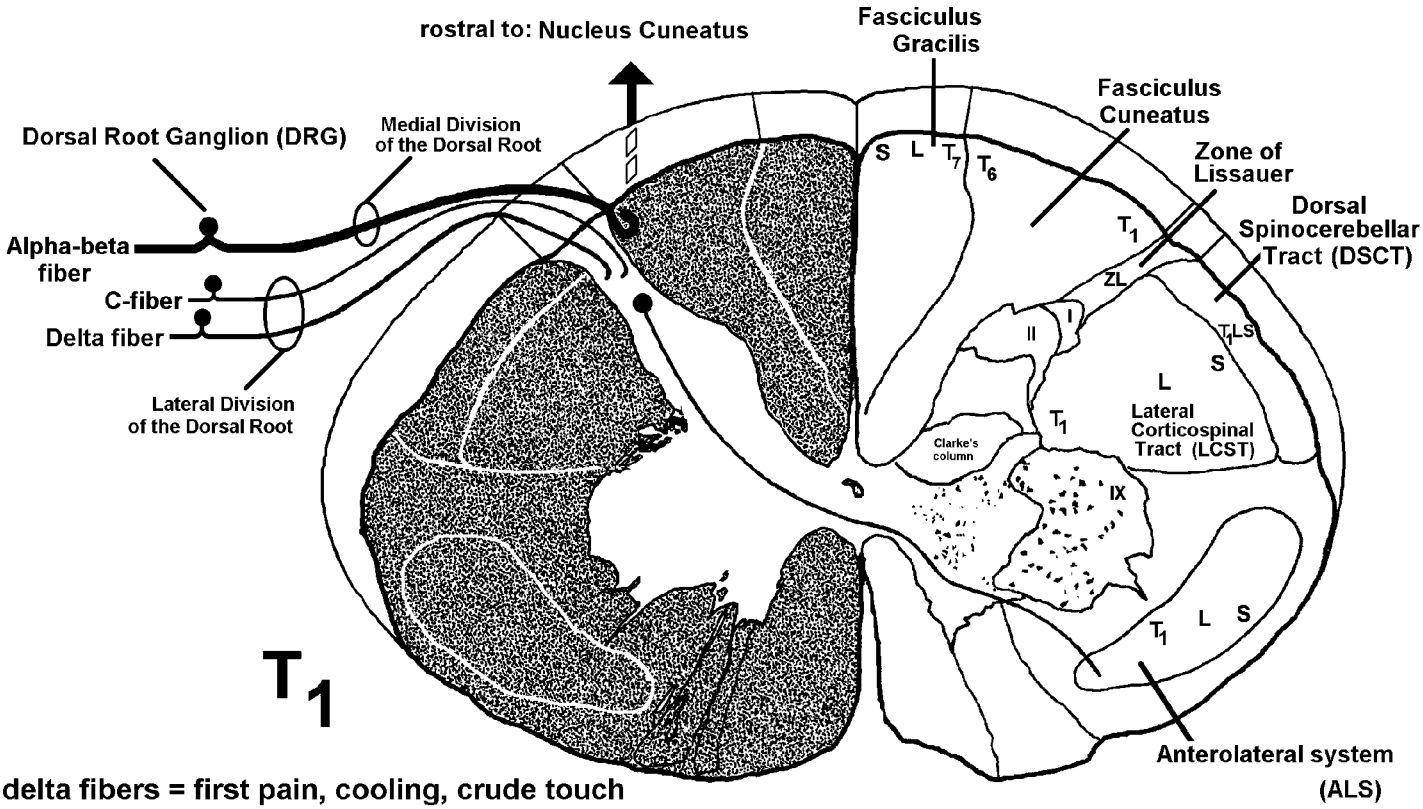
The second pathway that I want to talk about is very important since it carries pain and temperature. Dorsal root ganglion cells pick up the information and bring it into the spinal cord. Cells in the dorsal horn of the spinal cord then send the information to the **contralateral** thalamus. Cells in the thalamus then project to the cerebral cortex.



In the discussion of the dorsal columns, I mentioned that the entering alpha-beta fibers course within the **medial** division of the dorsal root. As I hope you recall, alpha-betas are relatively large, myelinated and fast-conducting. In contrast, the fibers in the **lateral** portion of the dorsal root are thinner and consist of both myelinated and unmyelinated axons.

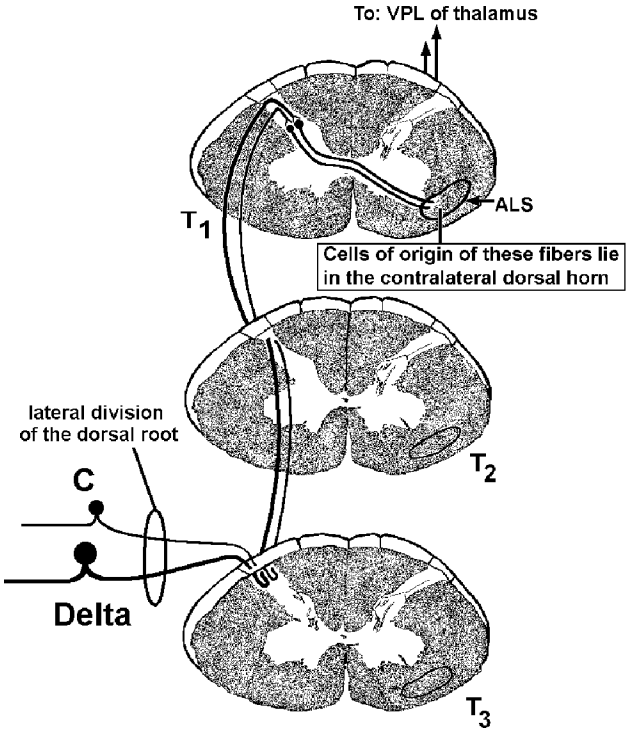
There are two types of dorsal root processes in the **lateral** division. One is called a **delta** fiber. This axon measures 1-5μm in diameter (compare with 6-12μm for alpha-betas) and conveys information regarding the sense of **cooling** and what is referred to as **pricking** or **first pain**. Delta fibers also convey what is referred to as **crude touch**. This contrasts with discriminative touch carried in the dorsal columns and is simply the sense of **contact**. You know that you are being contacted by something but have difficulty localizing the stimulus.

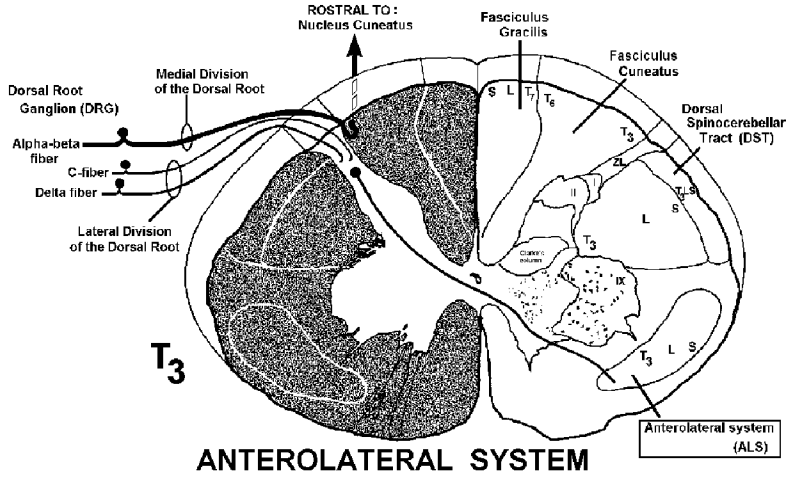
The thinner of the two fibers in the lateral division of the dorsal root is called a **C fiber.** This fiber is unmyelinated (slower conducting than a delta) and measures 0.2-1.5μm in diameter. The C fibers carry information regarding the sense of **warming** and **slow** or **burning pain.** The difference between first and second pain can be felt when you touch a hot pan. At first (no pun intended!!) there is a shooting pain (delta fibers which conduct faster than Cs) and then a slow, agonizing, burning, lasting pain (C fibers). Most of the receptors for pain, temperature and crude touch are **naked nerve endings** (compared with the elaborate receptors associated with alpha-betas).



delta fibers = first pain, cooling, crude touch
C-fibers = second pain, warming

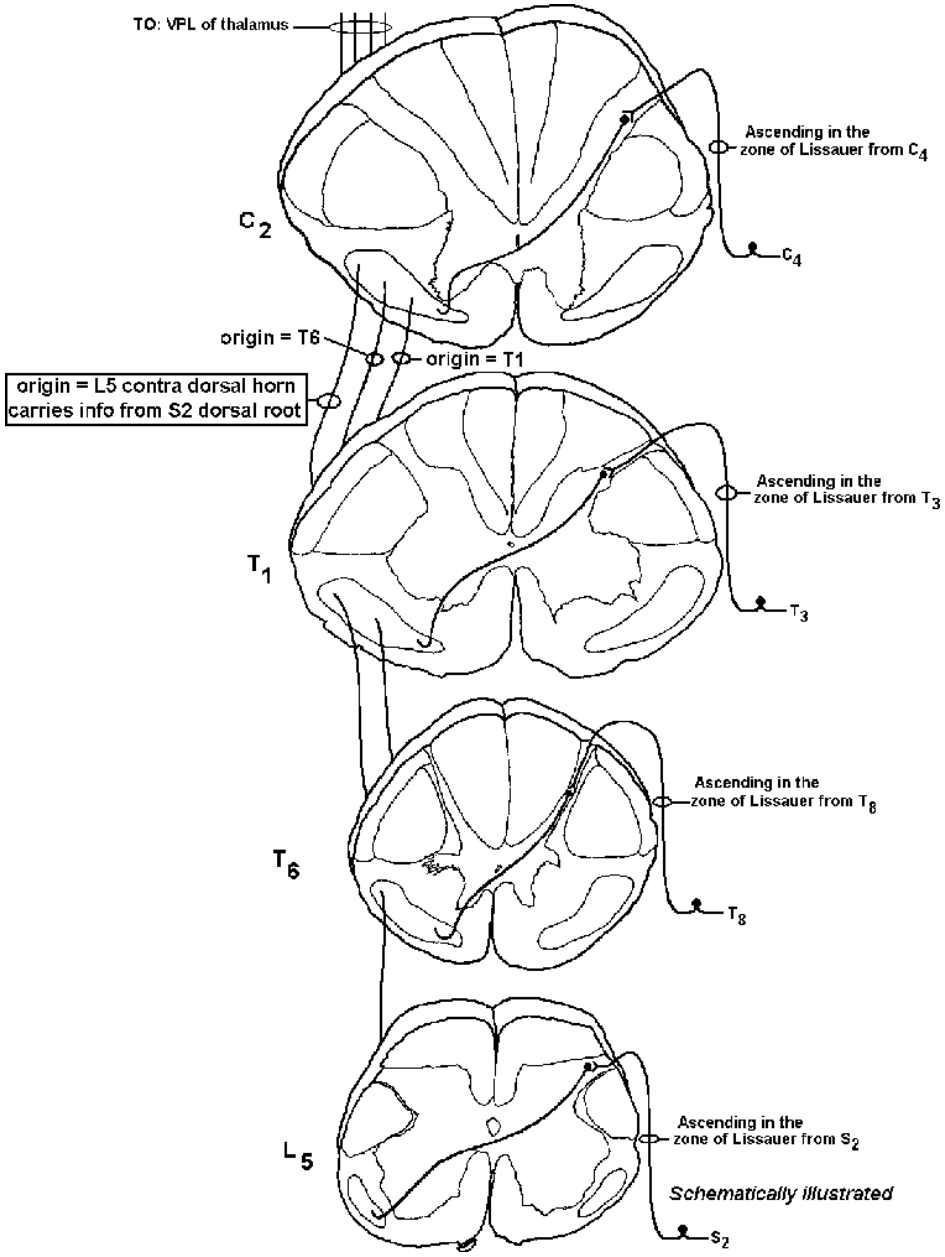
The central processes of delta and C fibers in the **lateral division** of the dorsal root do something quite different than the alpha-betas. These axons enter a zone at the top of the dorsal horn called the zone of Lissauer (ZL) and then course **ROSTRALLY** for approximately **2 spinal segments** within this zone before they dive into the dorsal horn, **where they synapse**. **THERE HAS BEEN NO CROSSING YET!!!!!** Cells in the **DORSAL HORN** that receive this pain and temperature information then send axons which **CROSS** and enter the **anterolateral** portion of the lateral funiculus, where they ascend to the thalamus (the great gateway to the cortex). In particular, they terminate in the **ventral posterolateral nucleus (VPL)**. The VPL then relays the information to the somatosensory cortex (Areas 3, 1, and 2). Information carried over the pain, temperature and crude touch pathway begins in the processes of dorsal root ganglion cells, but the **ANTEROLATERAL SYSTEM ([ALS] axons in the anterolateral part of the white matter) TAKES ORIGIN FROM CELLS IN THE CONTRALATERAL DORSAL HORN. THIS SYSTEM IS ALSO CALLED THE *SPINOTHALAMIC PATHWAY* (ORIGIN IN SPINAL CORD, TERMINATION IN THALAMUS).**



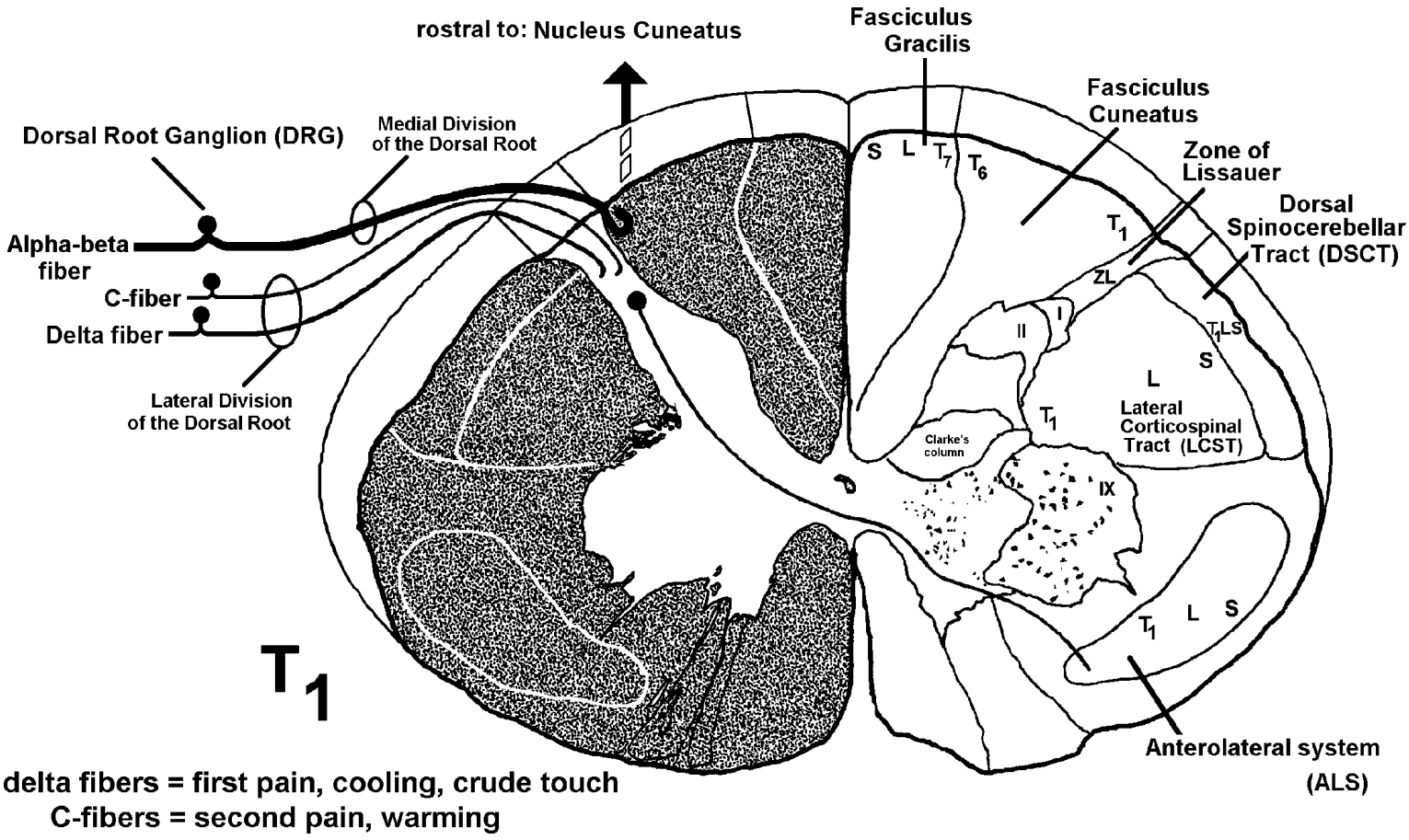


Fibers in the ALS are also somatotopically organized. However because the fibers cross the midline to reach the ascending tract (ALS) they are always added on to the medial edge of the tract as we ascend in the spinal cord. For example, you can see in the drawing below that at spinal level C2 axons from cells in the contralateral dorsal horn at L5 lie lateral to axons which arise from T6. These T6 axons lie lateral to axons that arise from T1. You should remember that this somatotopy is opposite to that in the fasciculus cuneatus and gracilis.

In **gross** terms a lesion of the **anterolateral system (spinothalamics)** will result in a deficit in **pain, temperature and crude touch on the contralateral** side of the body **below** the level of the lesion. That is, a lesion of the ALS in the upper cervical region results in a pain and temperature deficit of the entire **contralateral** side of the body. A lesion of the ALS in the upper thoracic cord will result in a deficit in pain and temperature from the thorax and lower extremities on the **contralateral** side because the fibers which are interrupted at this level convey pain and temperature information from these areas. The upper extremity is **safe** because the fibers conveying information regarding pain and temperature from this region are still reaching the thalamus (VPL) and in turn the cortex. These fibers lie in the anterolateral system **ROSTRAL** to the lesion and are not interrupted.



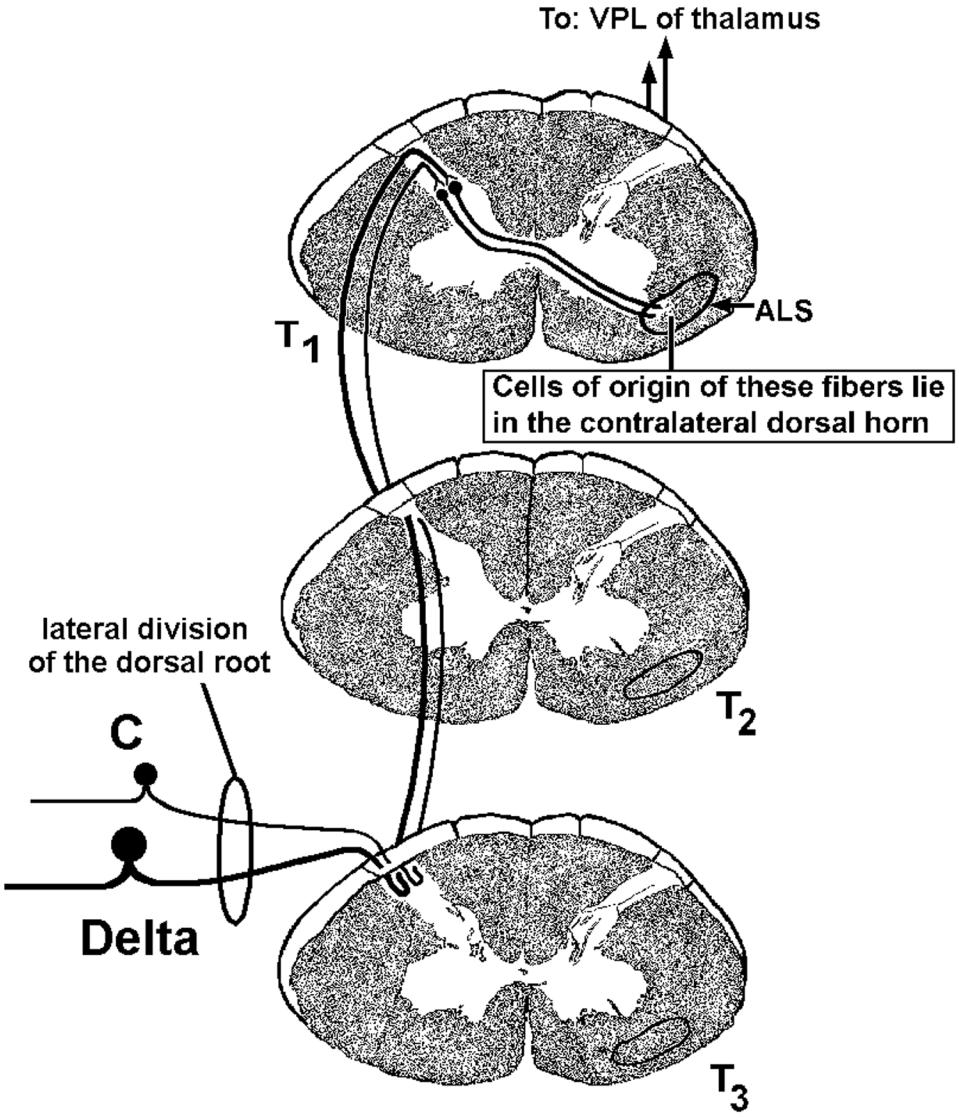
The **contralateral** deficits in pain and temperature following a lesion of the **ALS** are more detectable than the deficit in crude touch. Why?? Because the dorsal columns are intact and it "covers" for the loss during the neurological exam. For example, following a lesion of the **left** ALS at C2 there is a deficit in crude touch on the **right** (contra.) side but the dorsal columns are OK on the **right. Of course,** if you have a lesion of the dorsal columns only, you still have the crude touch being conveyed by the ALS. For example, following a lesion of the **right** fasciculus gracilis, you still have crude touch from the right lower extremity because the **left** ALS is fine and dandy!!!



delta fibers = first pain, cooling, crude touch
C-fibers = second pain, warming

We know that the central processes of delta and C fibers **ASCEND** approximately 2 levels before they synapse in the dorsal horn. In other words, **dorsal horn cells** which send their axons across into the anterolateral system receive their pain and temperature information from **TWO** spinal segments below. Thus, instead of the gross approximation of deficits of pain and temperature below the level of the lesion on the contralateral side, it is best to say the deficits start two levels below the lesion of the ALS and includes everything below this level. Of course, the deficits are contralateral.

For example, a lesion of the ALS (spinothalamics) at T1 will result in deficits in pain, temperature, and crude touch from T3 (2 segments below) and below on the contralateral side of the body.



"SPEED PLAY"

If there is reduced pain/temperature sensation in one limb and reduced position/vibration sensation in the contralateral limb, the lesion must be somewhere in the spinal cord (on the side of the position/vibration deficit.)

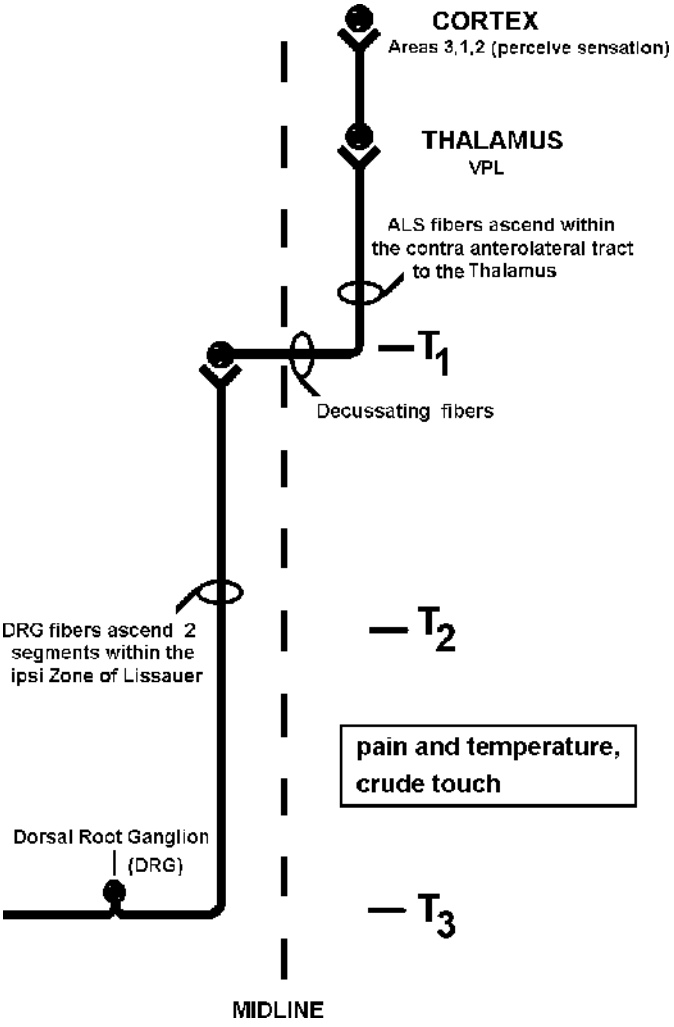
LET'S REVIEW THE ANTEROLATERAL (SPINOTHALAMIC) SYSTEM

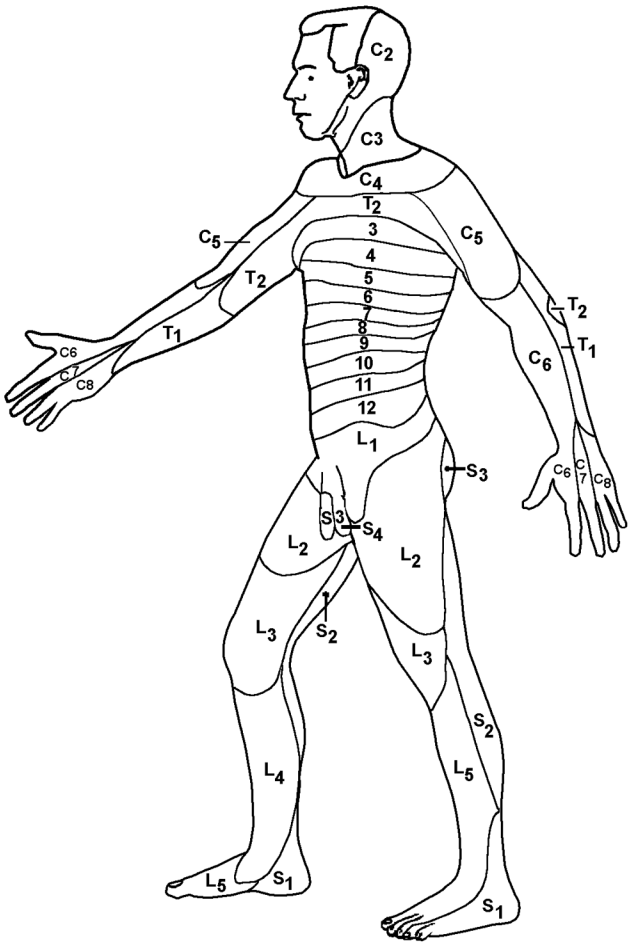
1. CELLS OF ORIGIN = contralateral dorsal horn (delta fiber; 1-5μ -1st pain and cooling)
(c-fiber; 0.2-1.5μ warm, *burning*)

2. LOCATION = anterolateral quadrant

3. TERMINATION = ipsilateral ventral posterolateral nucleus (VPL) of thalamus (the ALS is the ALS only after the axons are in the anterolateral quadrant of the spinal cord. Not before!!)

4. LESION DEFICITS = CONTRA. pain, temp. and crude touch





You will find this dermatome chart useful in the problem solvings. We will provide more details later in the course.

PROBLEM SOLVING

Match the best choice in the right hand column with the pathway or cell group in the left hand column. **There might be deficits that are not included in the responses.**

_____1. right fasciculus gracilis at C2

_____2. left anterolateral system at C1

A. lesion results in deficit in pain from the left arm

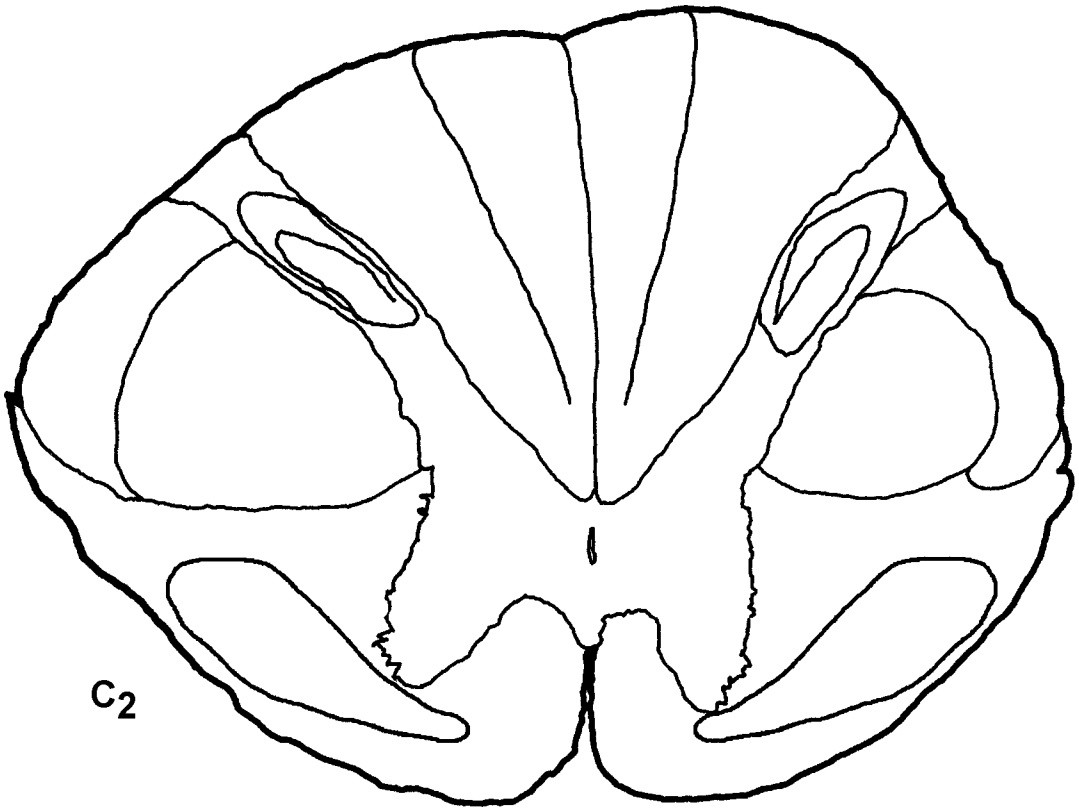
B. axons arise from dorsal roots T6 and above on the right

C. axons carry info. about vibration from the right thumb

D. lesion results in deficit in sense of cooling from the right foot

E. lesion results in deficit in conscious proprioception from the right knee

PROBLEM SOLVING



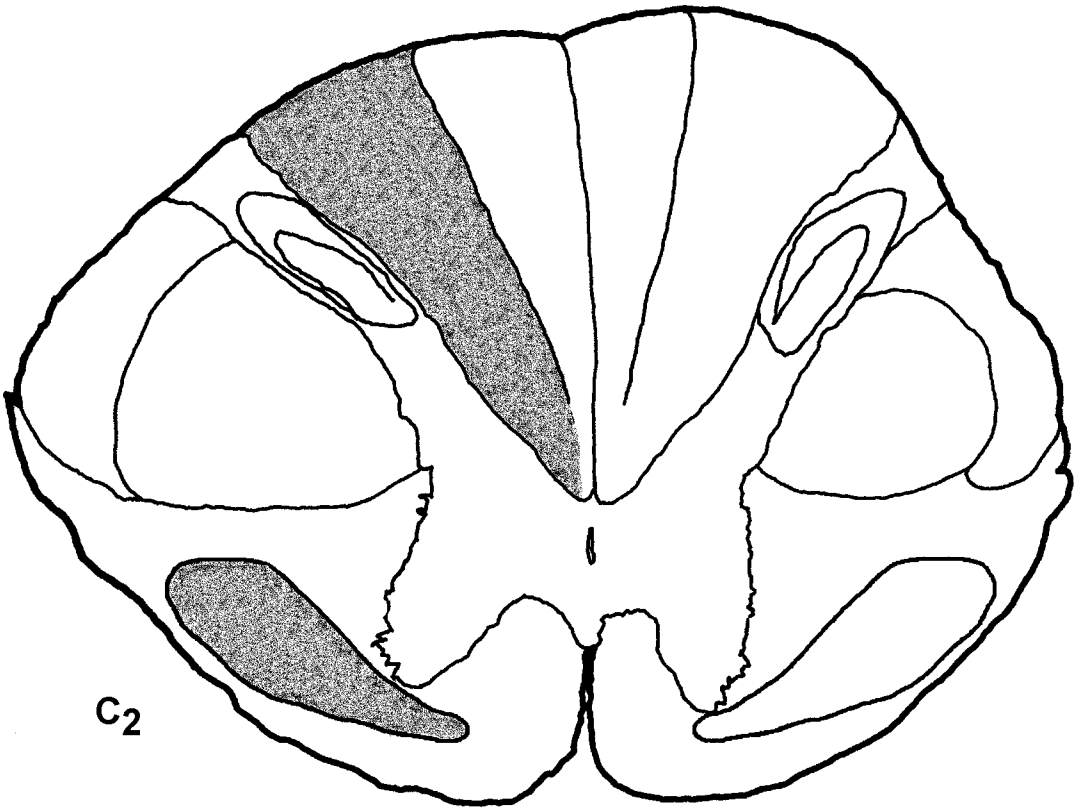
RIGHT LEFT

Shade in the location of lesions in the above drawing that will account for only the following neurological deficits:

a deficit in conscious proprioception, vibration, and two point discrimination from spinal segments C2-T6 on the right and a deficit in pain and temperature from the left side of the body below C4

Spinal cord
Anterolateral system (ALS)
Problem solving

PROBLEM SOLVING ANSWER

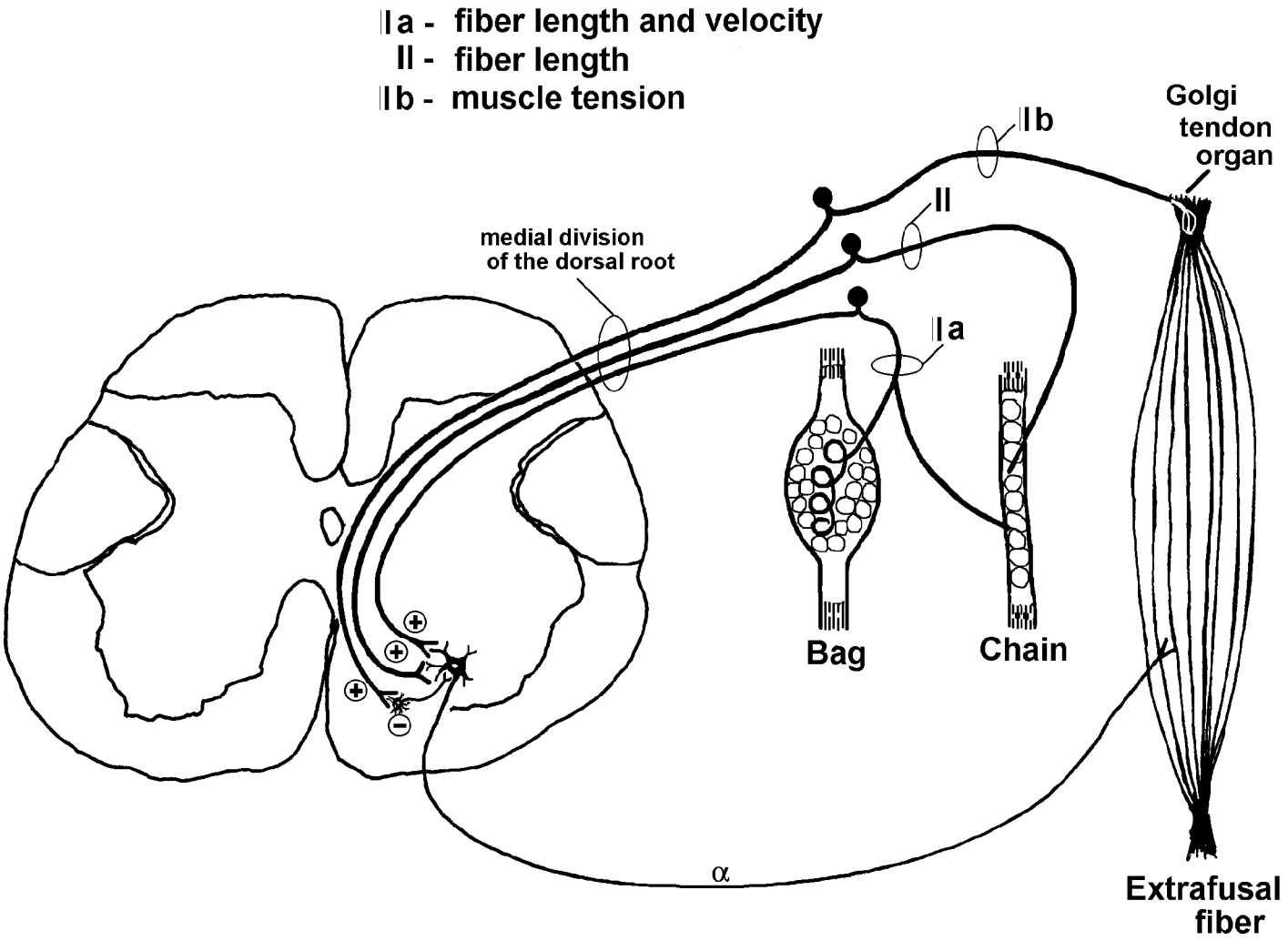


RIGHT LEFT

3 DORSAL SPINOCEREBELLAR TRACT (DSCT)

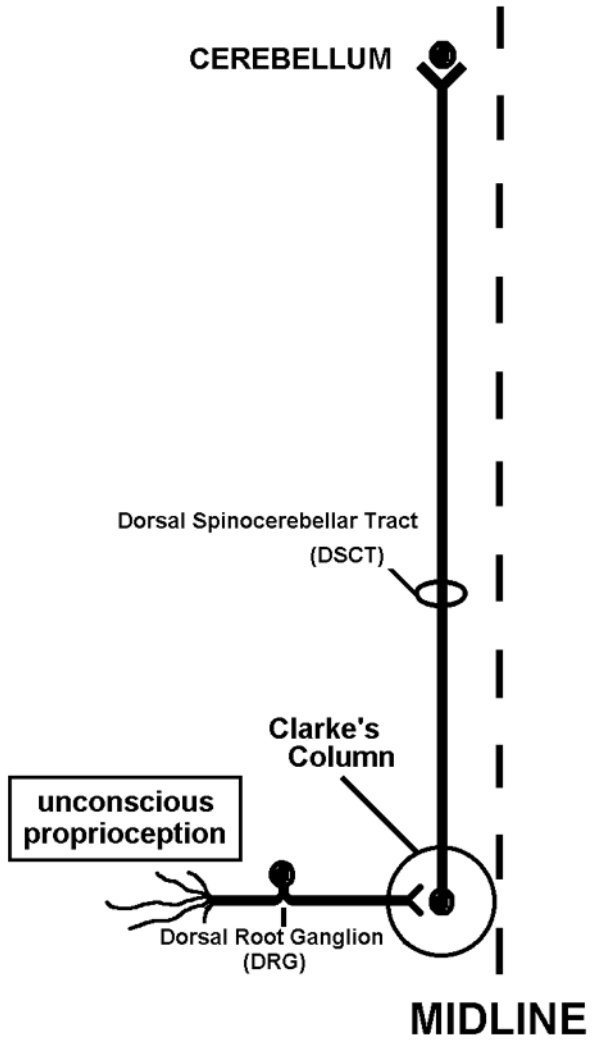
So far we have discussed **3 types** of fibers that comprise the dorsal root. The **alpha-betas** are associated with the dorsal columns (fasc. gracilis and fasc. cuneatus) while the **deltas** and **Cs** are associated with the anterolateral system (ALS). The alpha-betas are bigger than the **deltas** and **Cs**, but there are fibers in the dorsal root that are even bigger (12-20μm) in diameter. These are called **Ia, Ib and II** fibers. Since these fibers (whose cell bodies lie in the dorsal root ganglia) are big, guess which division of the dorsal root they use when entering the spinal cord???? Of course, the **MEDIAL**, along with the **alpha-betas**. Remember that the **skinny** ones lie **laterally** (ahh, that hurts!!) and the more **rotund** ones **medially**.

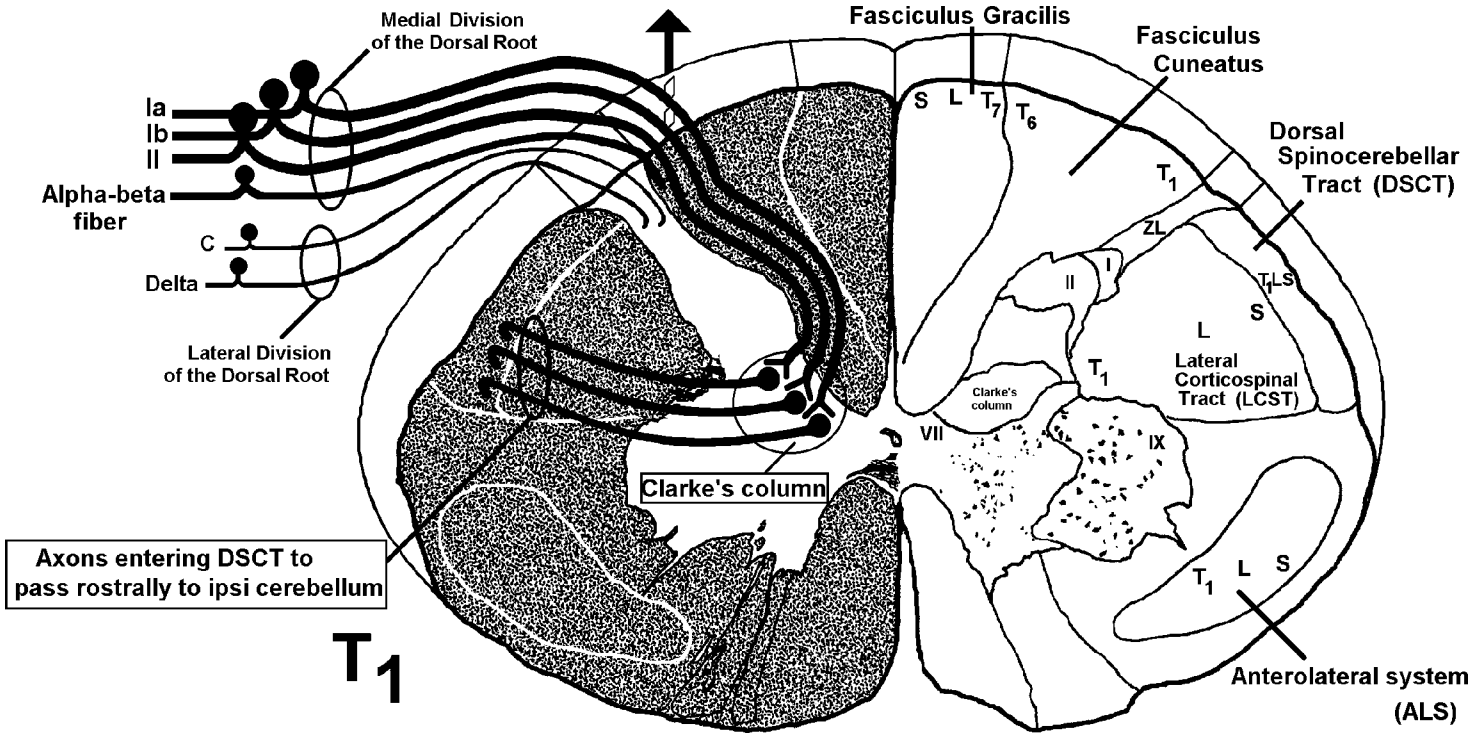
You have heard in Physiology that **Ia** and **II** fibers convey information from muscle spindles, while **Ib** fibers carry information from Golgi tendon organs. As Dr. Moss mentioned, this information is utilized for reflexes. However, all of this information also ascends to the **cerebellum** (L., little brain) in order to participate in motor coordination. How does this information reach the cerebellum?



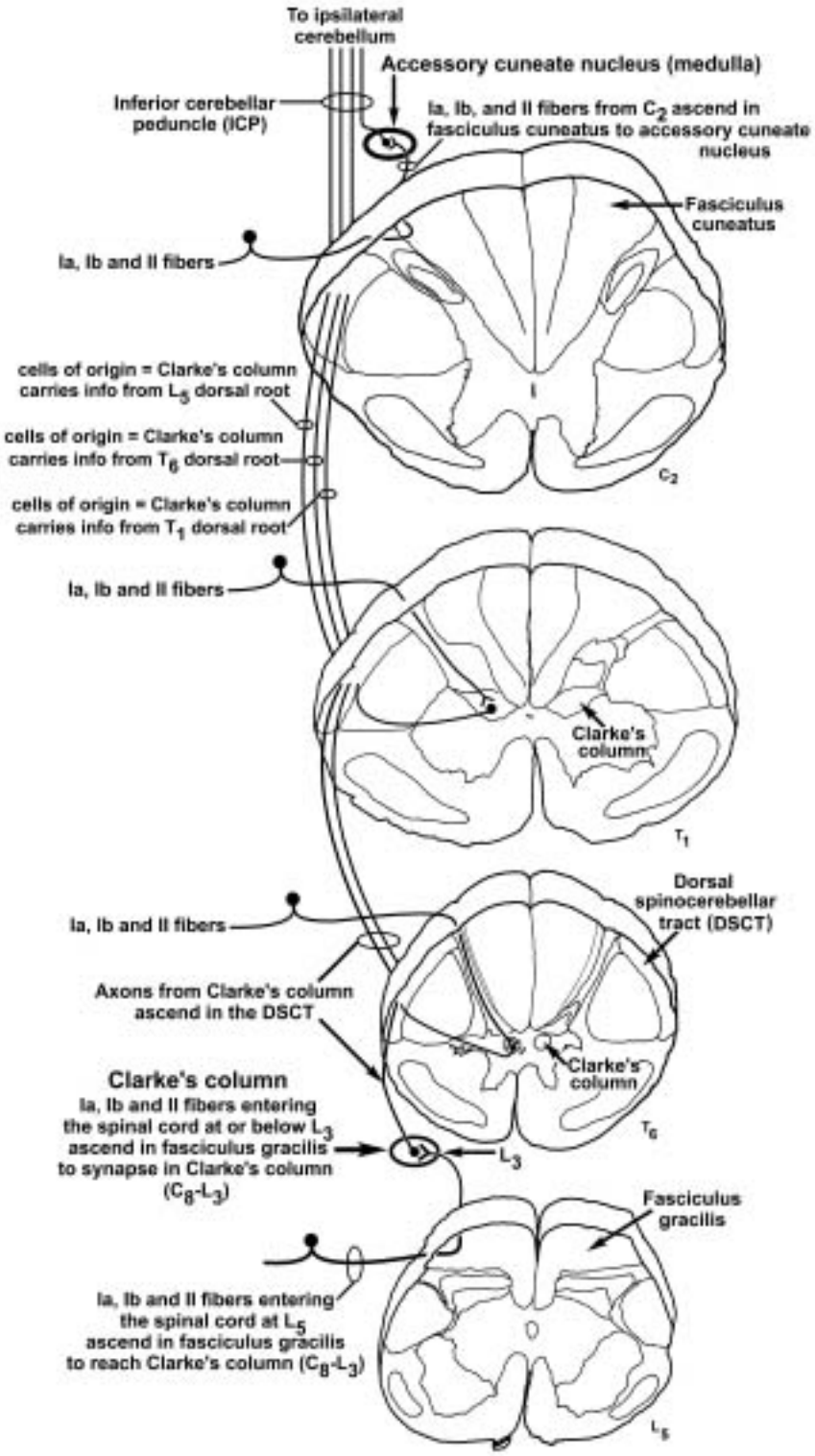
When the Ia, Ib, and II axons enter the spinal cord they dive into the grey matter of the dorsal horn until they reach its base. An investigator by the name of Rexed has divided the grey matter of the cord into layers or laminae, and the base of the dorsal horn is called lamina **VII**. Within this lamina **at spinal segments C8-L3, AND ONLY AT THESE LEVELS,** there is a very distinctive cell group called **CLARKE'S NUCLEUS OR COLUMN**. **The Ia, Ib and II fibers terminate on cells in Clarke's nucleus**. From there, cells in Clarke's nucleus send axons into the **IPSILATERAL** lateral funiculus where they are located dorsal and laterally. These axons comprise the **DORSAL SPINOCEREBELLAR TRACT (DSCT)**. The cells of origin of this tract lie in the **IPSILATERAL** Clarke's nucleus. The pathway passes rostrally in the lateral funiculus and eventually terminates within the **IPSILATERAL CEREBELLUM.**

As fibers from cells in Clarke's column enter the **DSCT** and ascend, they are organized such that the most **caudal** fibers lie **laterally** within the DSCT, while the most **rostral** (C8) lie **medially** in the DSCT. Compare this with the dorsal columns and ALS.





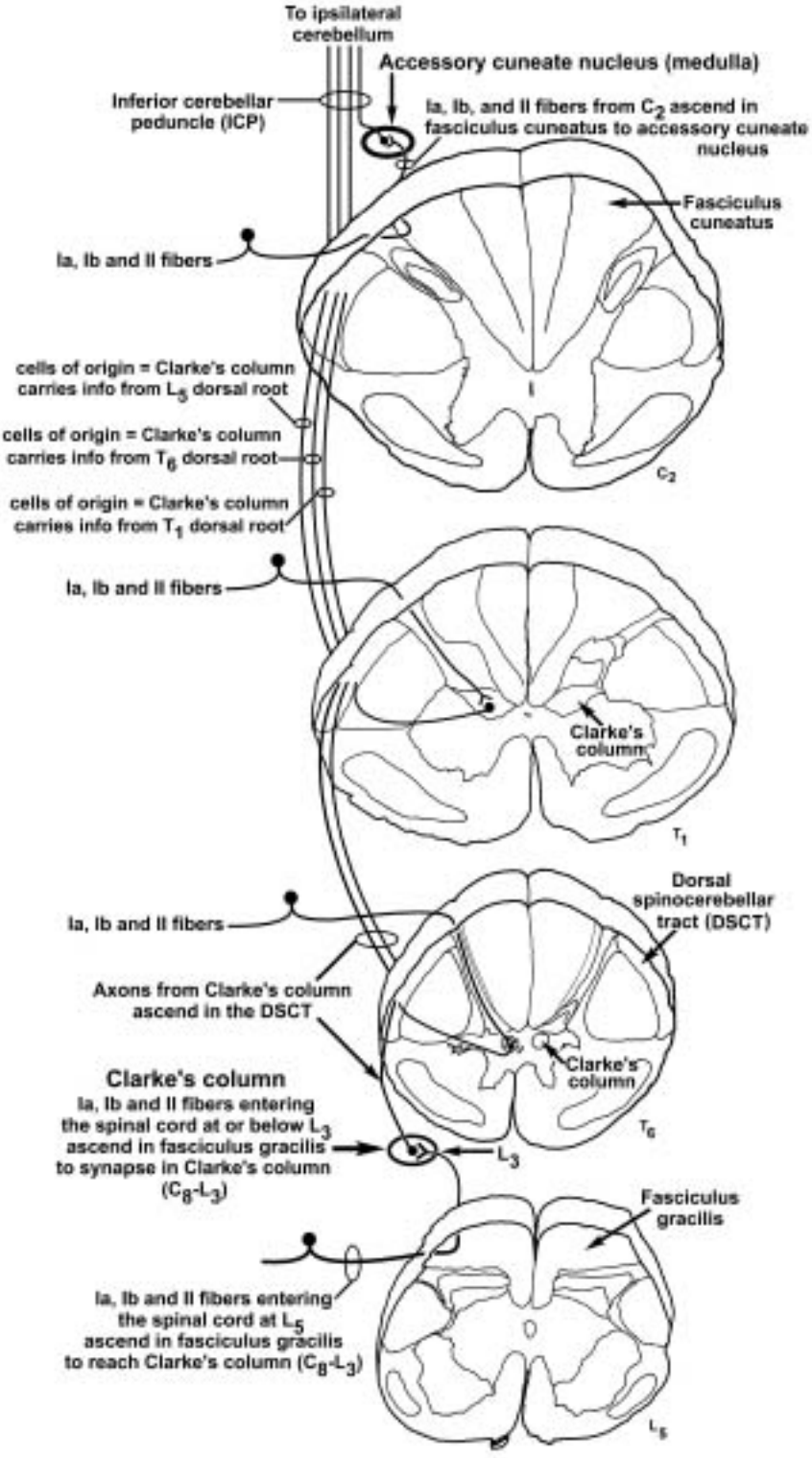
To get into the cerebellum, the DSCT courses within (is a component of) the **inferior cerebellar peduncle** (L., a little foot) or restiform body. Think of a cerebellar peduncle as a bundle of axons connecting the spinal cord/brain stem and the overlying cerebellum. There are three of these peduncles. More on this later in the course!!

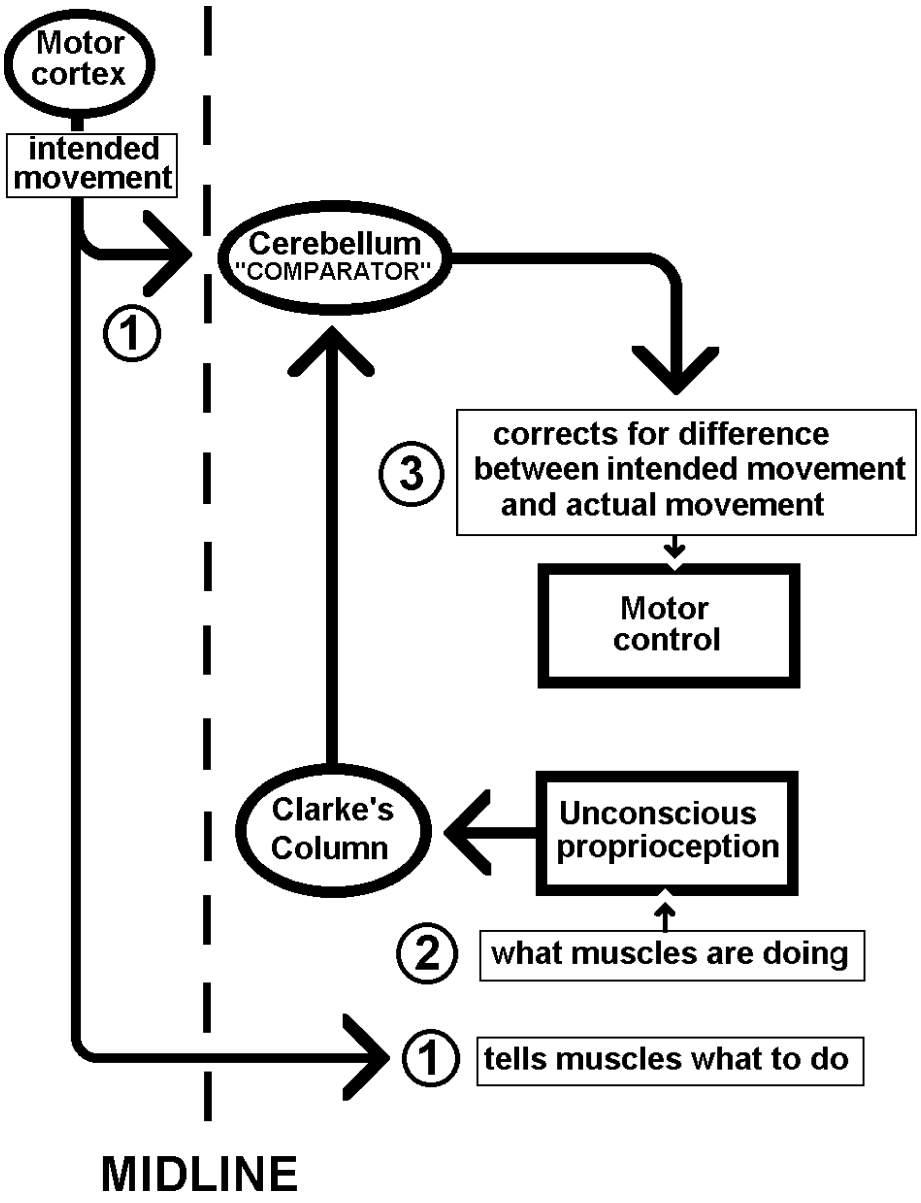


It's a crying shame that Clarke's nucleus is not present at every spinal cord level. As I mentioned earlier, it is only present at spinal cord segments **C8-L3**. So, if a Ia, Ib, or II axon comes into the spinal cord between C8-L3, fine!!! There is a Clarke's nucleus waiting for it and bingo, the fiber dives into the nucleus and the information that it is conveying is relayed to the cerebellum (via the DSCT). However, think about a Ia, Ib or II fiber coming in at spinal level L5. It looks around and there is no Clarke's nucleus to hitch a ride on. What would you do if you were a fiber who wanted to get your information to the cerebellum?? Personally, I would pass rostrally in the fasciculus gracilis (remember, no fasciculus cuneatus is present here!!) until I got to L3, where there is a Clarke's column, and dive into the nucleus. This is exactly what happens! Ia, Ib, and II fibers that enter the cord at L4 or below travel in the fasciculus gracilis with the ascending alpha-beta fibers to get to Clarke's nucleus.

What about Ia, Ib and II fibers associated with dorsal roots above C8? Well, they enter the cord, and find that like L4 and below they don't have a Clarke's nucleus, so they enter the fasciculus of the upper extremity (fasciculus cuneatus) until they reach the caudal medulla, where they synapse in the **ACCESSORY CUNEATE NUCLEUS**. Cells in the accessory cuneate nucleus project to the **IPSI** cerebellum via the inferior cerebellar peduncle (just like cells in Clarke's column do).

I realize that this is a **tremendous** amount of information about the DSCT. Some instructors are fearless enough to talk about a ventral spinocerebellar tract. I am not fearless at this point! But you should know that you might hear the term SPINOCEREBELLAR **PATHWAYS** sometime in your career!

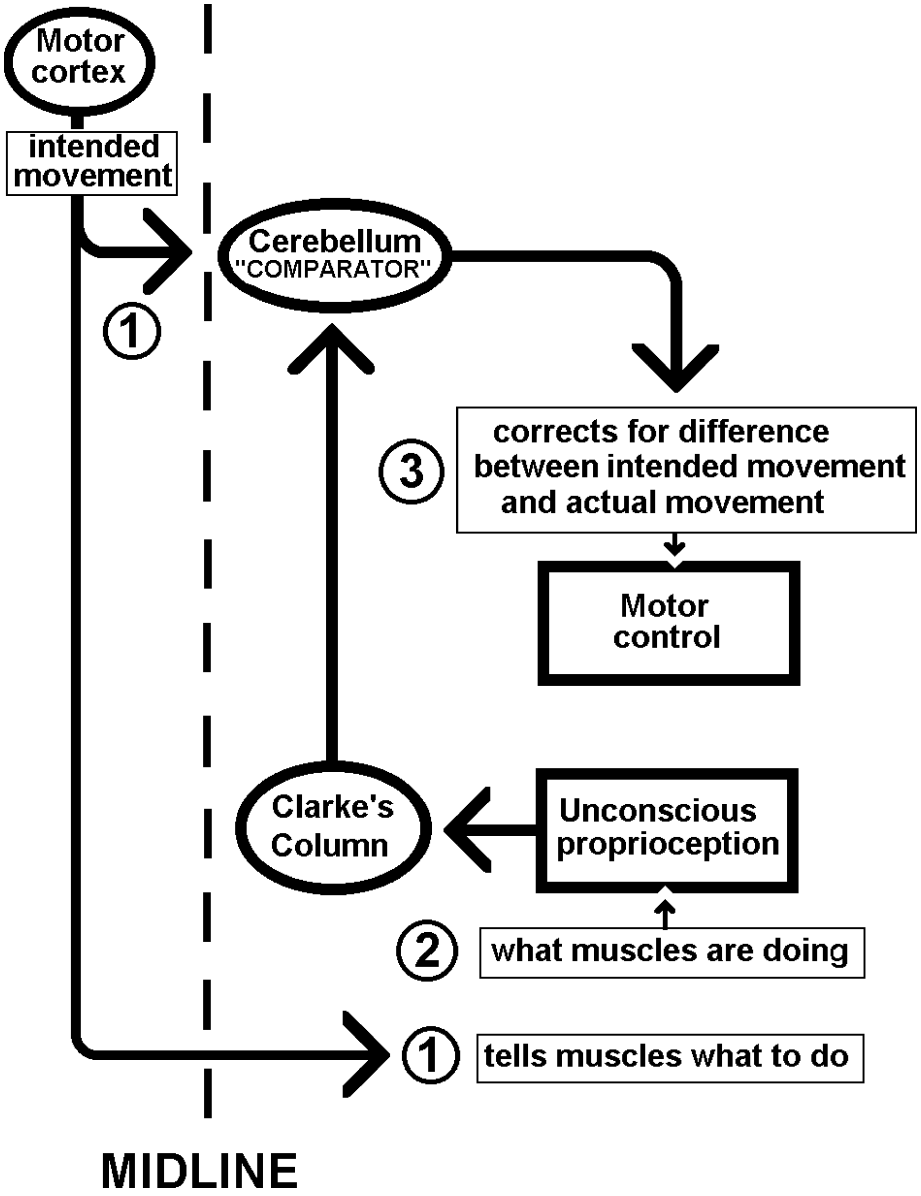




Clinicians will rarely talk about the deficits resulting from a lesion of the DSCT. Lesions in the spinal cord usually damage other tracts that mask such deficits. (One of these pathways lies right next to the DSCT and we will talk about it next!). But if we think about the information this tract is carrying we can see that such a lesion would result in a loss of information regarding the constant and changing lengths of muscle and tension on muscles. This information is going to the cerebellum and we are not really aware of it as we fish, ice skate, shoot buckets, bike through the arboretum, or start our backswing at the Ridge. This information tells the cerebellum about how long each muscle is, how fast each muscle is moving and how much tension is on each muscle **(#2 above)**. The cerebellum then can **compare** this ascending information regarding what **the muscles are doing** with other information (the sources of which we will learn later) regarding what higher motor centers want the muscles to do **(#1 above).** Then a correction can occur via pathways that leave the cerebellum to influence motor performance (#**3 above).** Whew!!

For our problem solving, let's equate a lesion of the **DSCT** with loss of **unconscious proprioception** and **incoordination or ataxia**. This incoordination deficit will be **IPSILATERAL** to the lesion because **there is no crossing of information in the spinal cord**. The **DSCT** is **IPSI** to the receptors. **Also, the cerebellum influences the same or ipsilateral side of the body (via several output pathways)**. Think about the dorsal columns. Is there crossing from the receptors to the fasc. gracilis and fasc. cuneatus in the spinal cord?? How about the pain and temperature pathways?

A lesion of the DSCT means there is a loss of input to the cerebellum. Anytime the cerebellum or its input/output pathways are damaged there is a "**cerebellar ataxia**" (contrast with a sensory ataxia associated with dorsal column disease). In these instances the patient cannot stand with his/her feet together and eyes **open** so there can not be a Romberg sign (swaying when eyes are closed) as the patient is unstable even before closing the eyes.



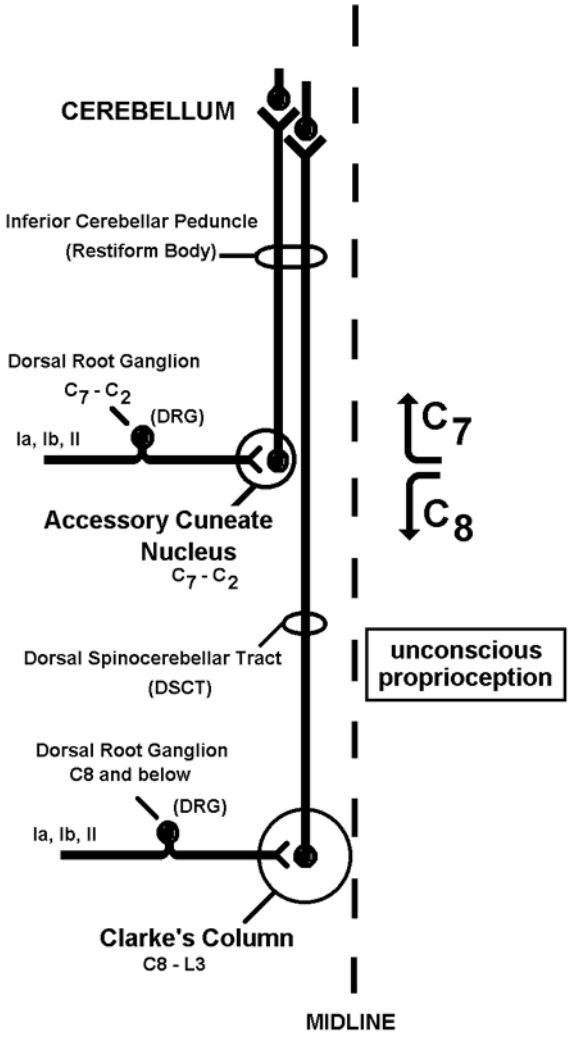
LET'S REVIEW THE DORSAL SPINOCEREBELLAR TRACT

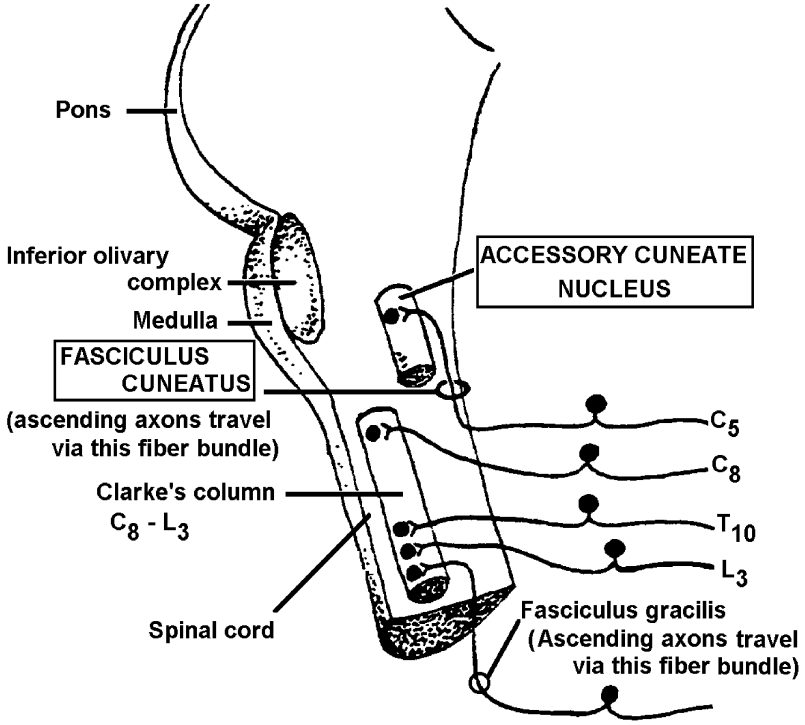
1. CELLS OF ORIGIN = ipsilateral Clarke's column (1a, II, 1b; 12-20μ)

2. **LOCATION** = dorsolateral part of lateral funiculus

3. TERMINATION = ipsilateral cerebellum

4. LESION DEFICIT(S) = **ipsilateral** muscle incoordination/**ataxia**





PROBLEM SOLVING MATCHING

Match the best choice in the right hand column with the pathway or cell group in the left hand column. **There might be deficits that are not included in the responses.**

_____1. right fasciculus gracilis at C2

_____2. left anterolateral system at C2

_____3. left dorsal spinocerebellar tract at T6

A. lesion results in deficit in unconscious proprioception from the left leg

B. axons arise from dorsal roots T7 and below on the right

C. axons carry information about vibration from the left thumb

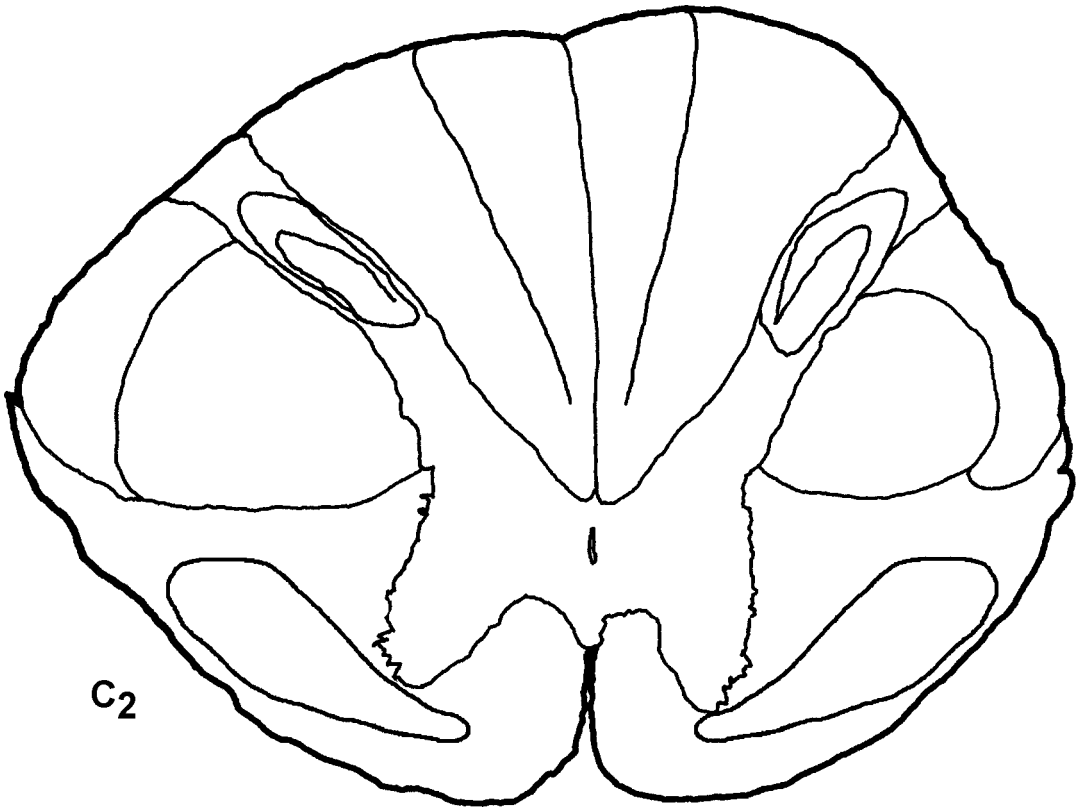
D. lesion results in deficit in sense of cooling from the left foot

E. lesion results in deficit in conscious proprioception from the right elbow

F. pathway comprised of central processes of delta and C fibers of contra. dorsal roots

G. lesion results in deficit in the sense of warming in right hand

PROBLEM SOLVING



RIGHT LEFT

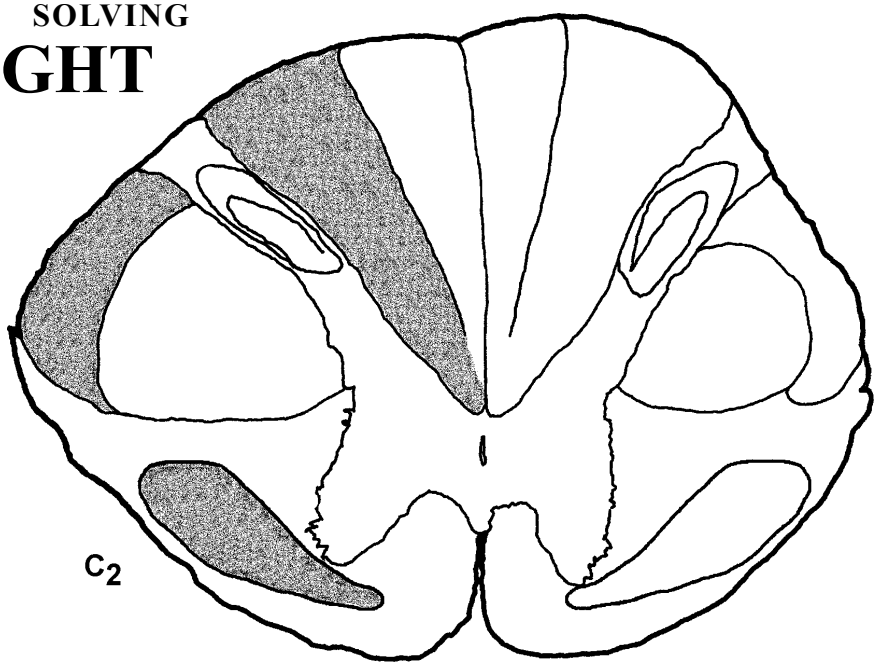
Shade in the location of unilateral lesions in the above drawing that will account for the following neurological deficits:

deficit in conscious proprioception, vibration, and two point discrimination from spinal segments C2-T6 on the right, deficit in pain and temperature from the left side of the entire body (below the neck) and deficit in unconscious proprioception from the entire right side of the body (Think about how fibers get to the accessory cuneate nucleus!!!)

Spinal cord
Dorsal spinocerebellar tract (DSCT)
Problem solving

PROBLEM SOLVING

RIGHT



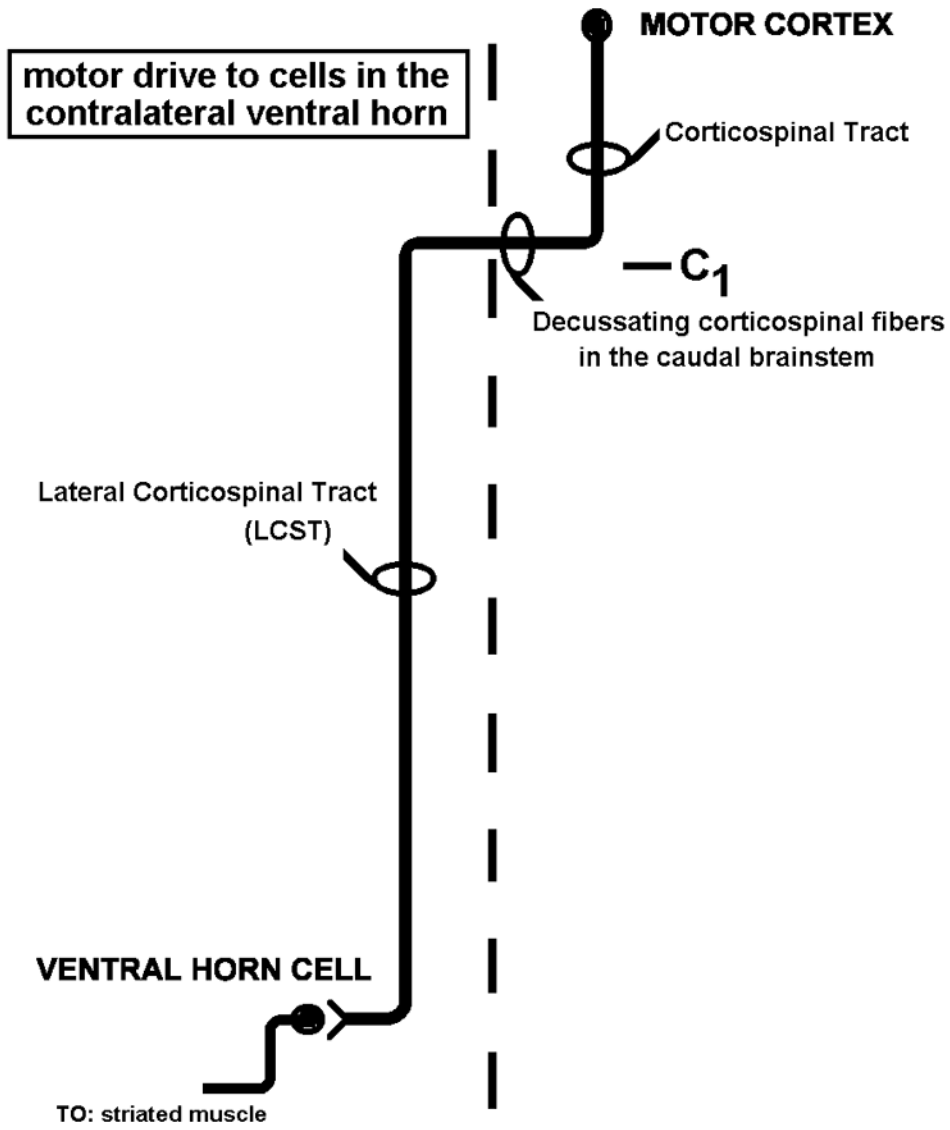
ANSWER

LEFT

4 LATERAL CORTICOSPINAL TRACT (LCST)

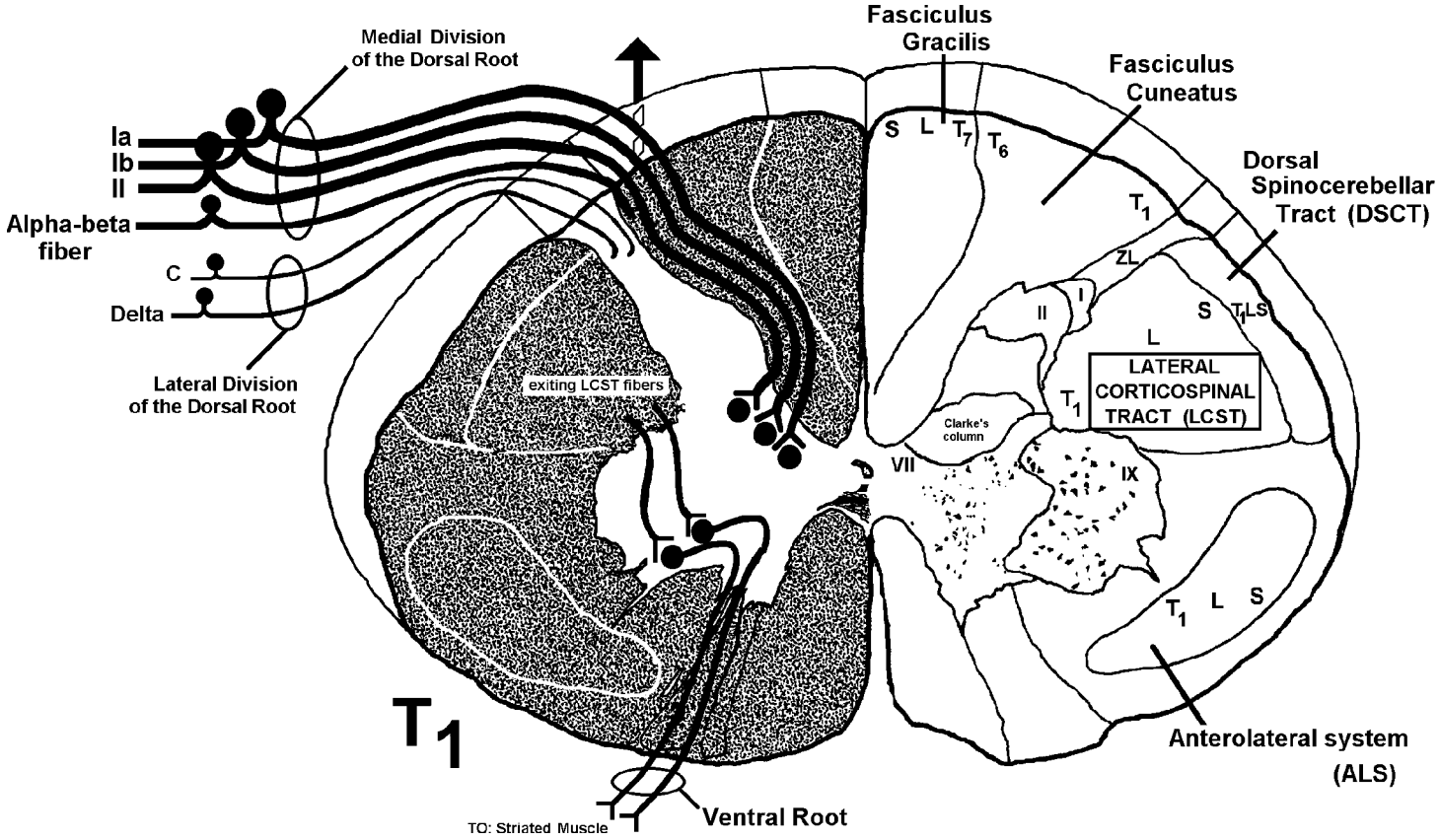
The lateral corticospinal tract (LCST) is the most important pathway we have for making voluntarily movements and is one of, if not THE, most important pathways in clinical neurology.

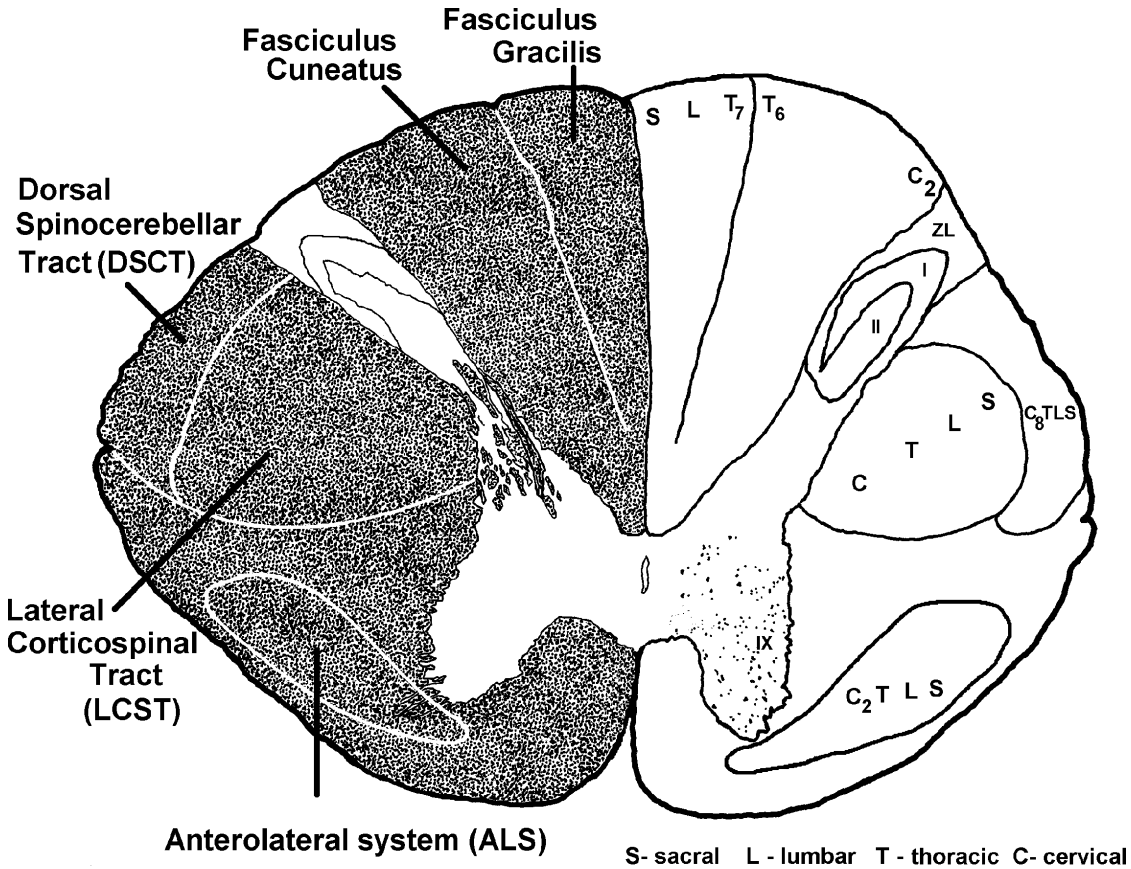
Cells in the cerebral cortex, especially the motor cortex (area 4; precentral gyrus) possess very long axons that **descend** through an extensive region of the brain to eventually reach the spinal cord. Right before entering the cord these corticospinal fibers cross or **decussate** (L., to make an X) and enter the **LATERAL FUNICULUS** where they travel **medial** to the **DSCT**. These fibers, which are now called the **lateral** (they are in the lateral funiculus) **corticospinal tract** (**LCST**), innervate neurons in the spinal cord along its entire length. Once in the grey matter (where the cells are) LCST axons synapse upon cells in the ventral horn. **This is the first synapse in a pathway over which the cerebral cortex informs cells in the CONTRALATERAL spinal cord about a voluntary movement that it wishes to perform**. Once the cells in the ventral horn receive this cortical information, they **directly** drive the muscles via axons that pass out the ventral root. The fastest way you can move a body muscle voluntarily is by utilizing **2 neurons.** The first one is an upper motor neuron and lies in the **cerebral cortex**. The second one lies in the **contralateral** or opposite **ventral horn** and is a lower motor neuron.



Descending fibers in the LCST are somatotopically organized such that the most **medially** located fibers in the tract terminate before (**rostral** to) the more the **laterally** placed fibers.

Lateral corticospinal tract (LCST)





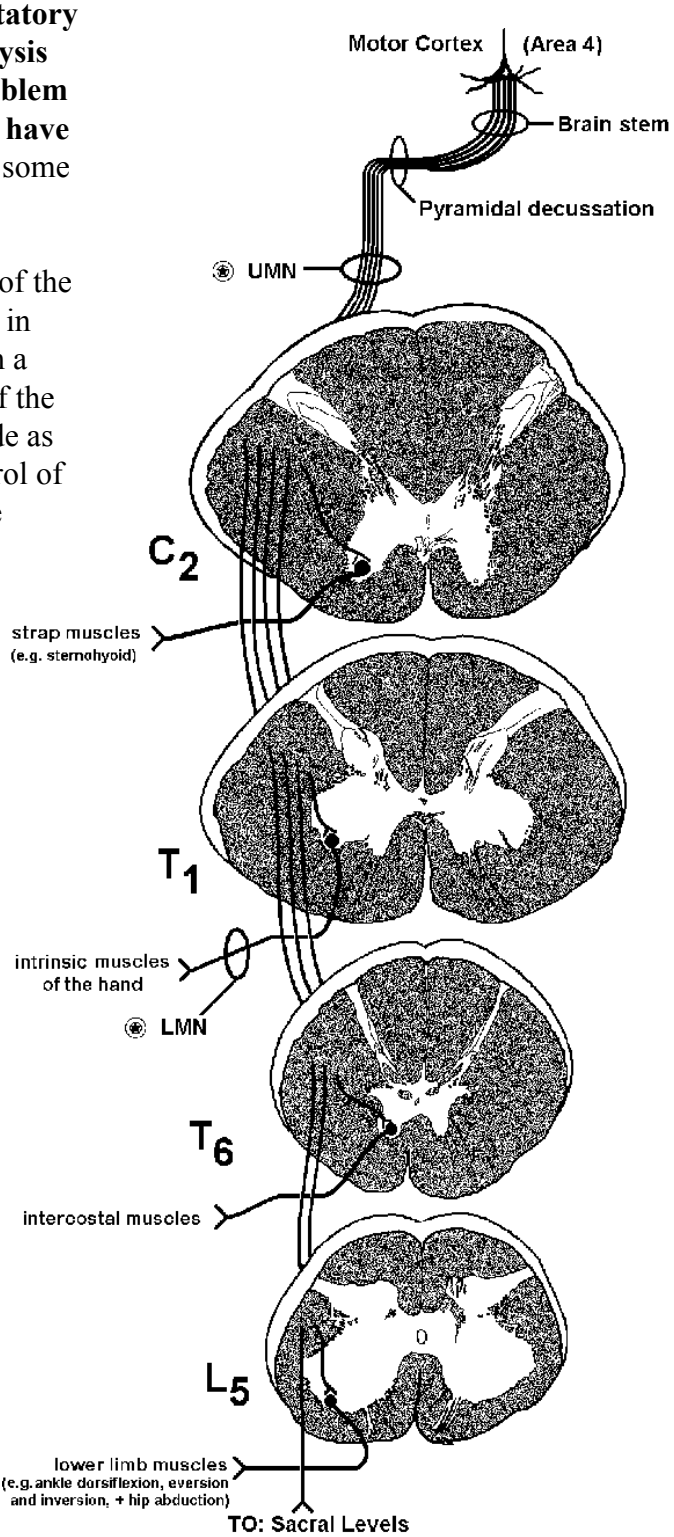
S- sacral L - lumbar T - thoracic C- cervical

ZL - Zone of Lissauer
I - Marginal zone of the dorsal horn
II - Substantia Gelatinosa
IX - Motor Nuclei (includes neurons of spinal accessory C.N. XI)

C2

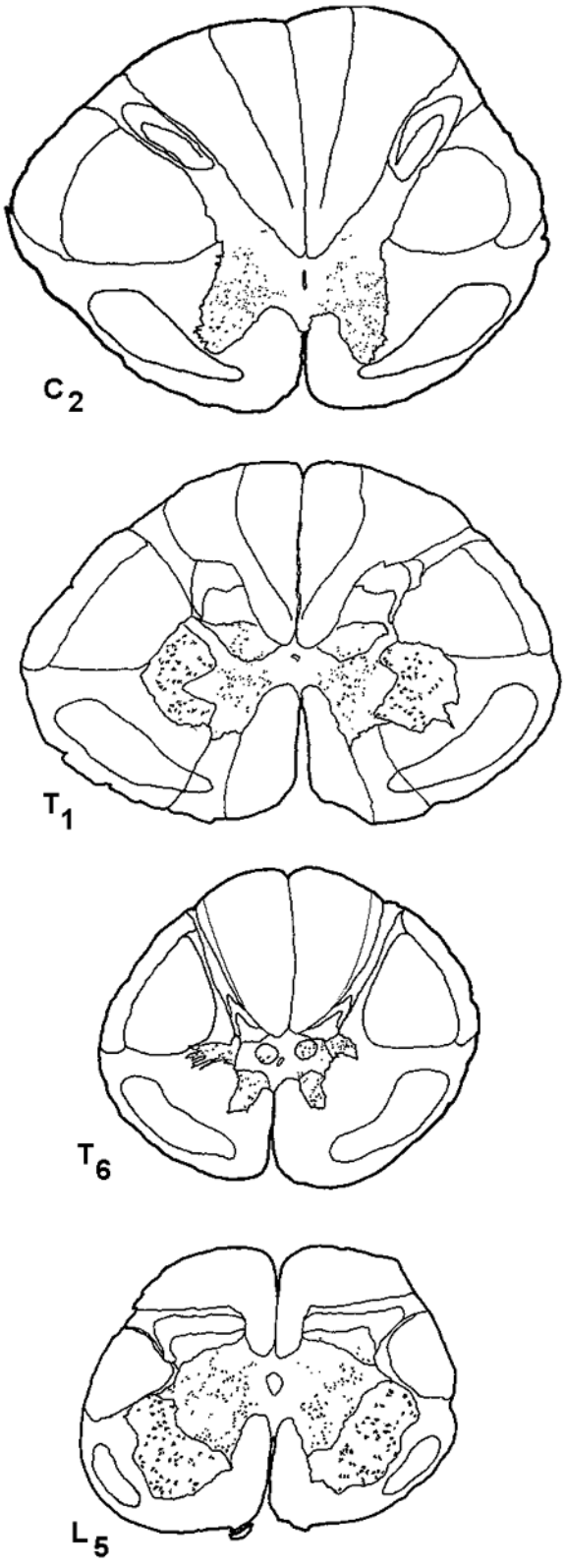
Interruption of the LCST means that neurons in the spinal cord that innervate or drive muscles have lost a tremendously important input. These muscles are still innervated by the spinal cord neurons in the ventral horn, but these cells have lost a large part of their drive. This results in **weakness in those muscles that are innervated by spinal neurons that have lost their LCST excitatory drive. Such a lesion does NOT result in paralysis because the muscles are still ALIVE. The problem is that the neurons that innervate the muscles have lost a large part of their drive.** (There are still some other inputs to these cells).

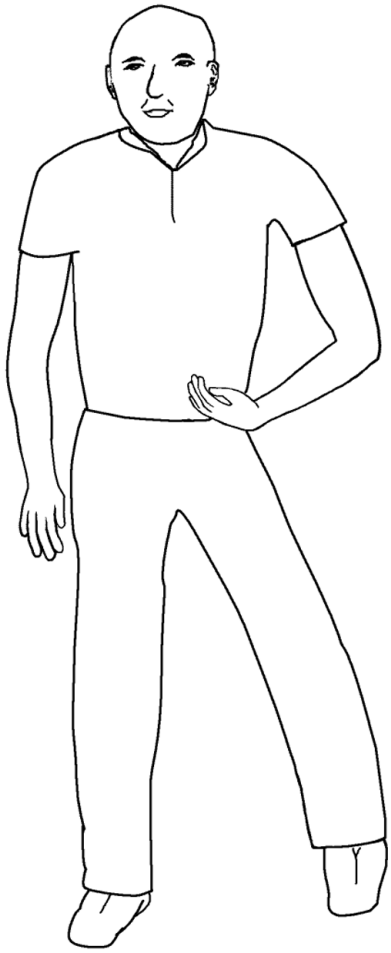
It is important to understand that lesions of the LCST at different levels of the spinal cord result in different muscles being affected. Let's start with a lesion at C1. Such a lesion will interrupt **ALL** of the **LCST** fibers to the spinal cord on the **SAME** side as the lesion. The result is a loss of voluntary control of all of the muscles on the **IPSILATERAL** (to the lesion) side of the body. This is called **HEMIPLEGIA** (plegia = stroke or paralysis). Notice that the muscles are **not** paralyzed, only weak.



LATERAL CORTICOSPINAL TRACT (LCST)

What about a lesion of the LCST at T3. Such a lesion spares the voluntary control to the upper extremity since the LCST fibers to the spinal neurons innervating the cervical enlargement have "already gotten off" and are doing their job. Only the **IPSILATERAL lower** extremity is affected. Play around and sketch some lesions at different rostrocaudal locations in the LCST. This is a good way to learn!!!!

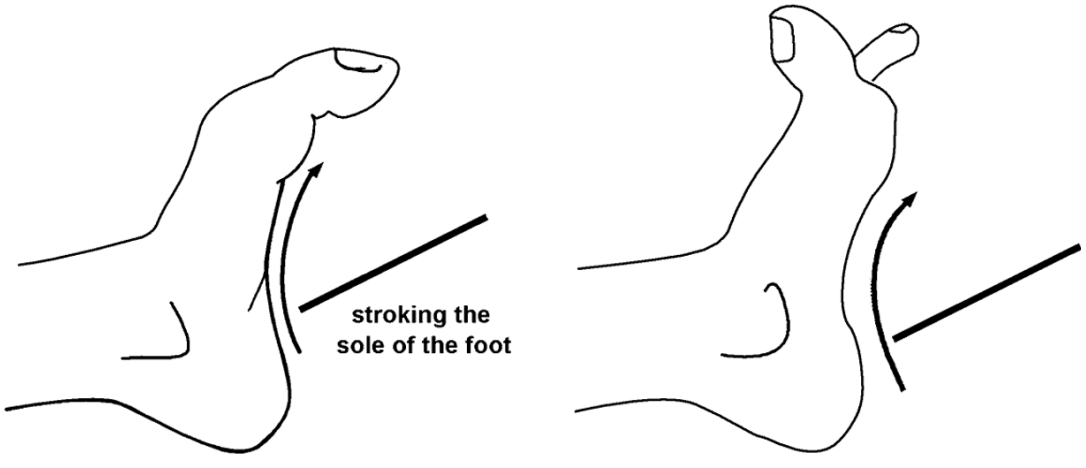




Stroke patients in which the corticospinal or lateral corticospinal tract is damaged exhibit a **flexed arm** and an **extended leg**. That is, the resting length of these muscles is **shortened**. When you passively **lengthen** the patient's flexed arm or extended leg, you feel **more** resistance or tone than in a normal person. This is due to an **increase** in the muscle stretch reflexes that serve to maintain the length of muscles. The **hypertonia/hyperreflexia** in a stroke patient is especially apparent in the **flexor muscles of the arm** and the **extensor muscles of the leg** (ipsilateral to the spinal cord lesion or **contralateral** to the cortical lesion). These increased muscle stretch reflexes are velocity dependent. That is, the faster you try to extend the flexed arm and flex the extended leg the more resistance you will feel. The **increase in muscle tone (hypertonia/hyperreflexia) seen when passively moving a limb is called SPASTICITY. This is a very important term in clinical neurology, one that you should NEVER FORGET!! PLEASE!!!!!!!** The essential feature of spasticity is a velocity-dependent increase in the resistance of muscle to passive stretch.

Keep in mind that if you test for reflexes via your hammer (knee jerk in contrast to passively moving the limb) the reflex is labelled on the chart as Increased. What I'm saying is that you passively move a limb to look for spasticity and use your reflex hammer to test for hyperreflexia (or reflexive increase).

The hyperreflexic state that characterizes spasticity often takes the form of **clonus**, a series of rhythmic involuntary muscular contractions occurring at a frequency of 5 to 7 Hz in response to an abruptly applied and sustained stretch stimulus. It is usually designated in terms of the part of the limb to which the stimulus is applied (e.g, patella, ankle). The cutaneomuscular abdominal and cremasteric reflexes are usually abolished in these circumstances, and a **Babinski sign** is usually present. That is, stroking the ventral surface of the foot causes the big tow to go "**UP**", which is plantar extension, instead of the normal down movement, which is plantar flexion. Other less famous pathological reflexes that reflect LCST damage include: the **Chaddock** sign, (toe goes up upon stimulation of lateral aspect of the foot), the **Bing** sign (toe goes up upon jabbing dorsal surface of the big toe), and the **Hoffman** sign (flicking the middle finger causes the index finger and thumb to reflexively flex).



Normal plantar reflex **Dorsiflexion = Babinski sign**

Lesions of the LCST also result in the loss of the cremasteric reflex. That is, the cremaster muscle fails to contract when the skin on the medial side of the thigh is stroked—reflex arc involves L1. The abdominal reflex, which is also lost, is demonstration by scratching each of the four quadrants of the abdomen. The response is considered normal if the umbilicus moves slightly toward the direction scratched; reflex arc involves T6-T12. The abdominal and cremeasteric reflexes are lost ipsilateral to the LSCT lesion.

LET'S SUMMARIZE SOME DIFFICULT (AND CONFUSING??) MATERIAL INTO A "SIMPLE RULE":

A LESION OF THE LCST RESULTS IN IPSILATERAL WEAKNESS AND SPASTICITY OF MUSCLES THAT ARE INNERVATED BY VENTRAL HORN CELLS *AT AND BELOW* THE SPINAL LEVEL OF THE LESION. THERE ALSO WILL BE IPSILATERAL CLONUS AND A BABINSKI SIGN.

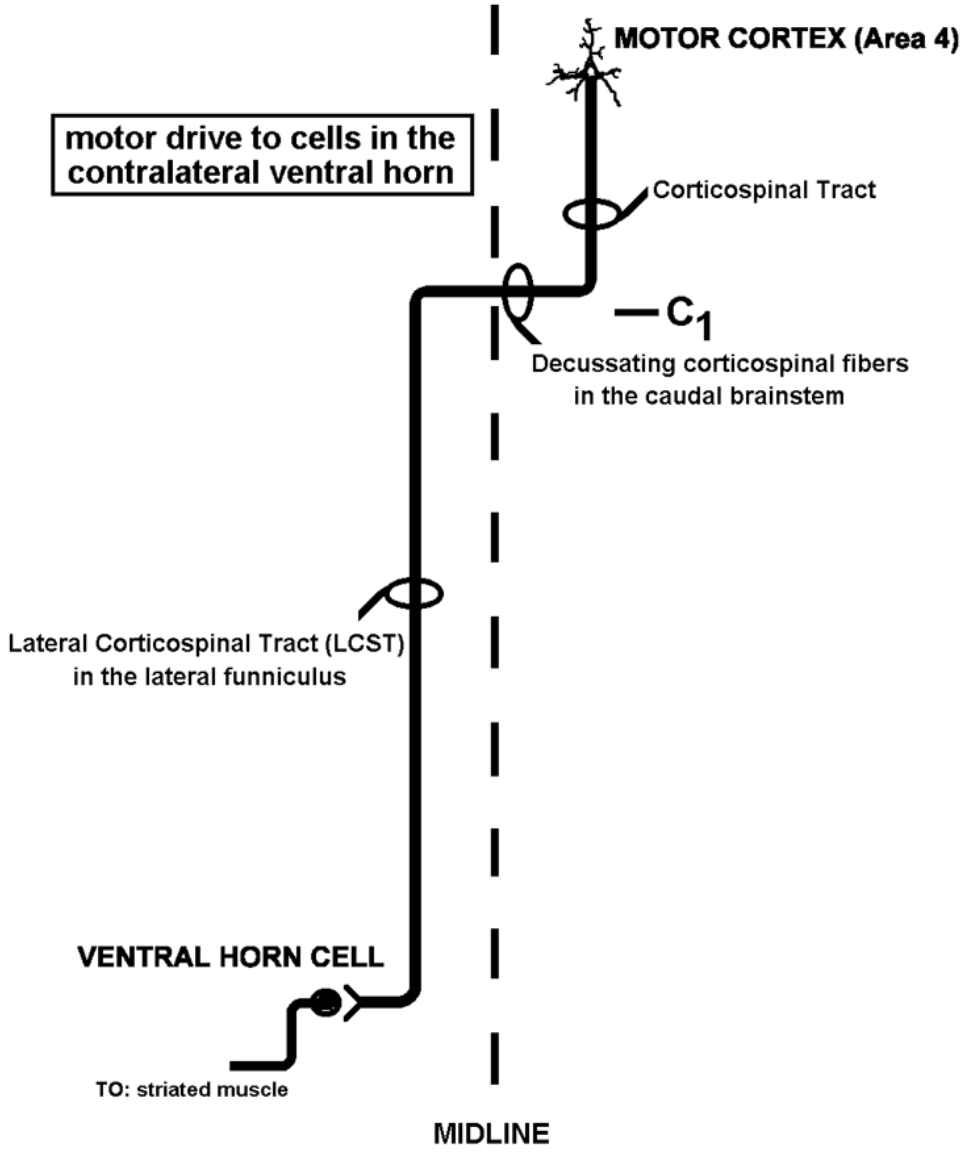
LET'S REVIEW THE LATERAL CORTICOSPINAL TRACT

1. CELLS OF ORIGIN = contralateral motor cortex

2. LOCATION = lateral funiculus

3. TERMINATION = ipsilateral ventral horn

4. LESION DEFICITS = ipsilateral muscle weakness (loss of excitatory drive) and increased tone and deep tendon reflexes in muscles innervated by spinal segments at and below the level of the lesion. Also, a Babinski sign.



PROBLEM SOLVING MATCHING

Match the best choice in the right hand column with the pathway or cell group in the left hand column. **There might be deficits that are not included in the responses.**

_____1. right fasciculus gracilis at C1

_____2. left anterolateral system at C1

_____3. left dorsal spinocerebellar tract at T6

_____4. left lateral corticospinal tract

A. lesion results in deficit in unconscious proprioception from the left leg

B. axons arise from dorsal roots T7 and below on the left

C. axons carry information about vibration from the right big toe

D. lesion results in deficit in sense of cooling from the right foot

E. lesion results in deficit in conscious proprioception from the left elbow

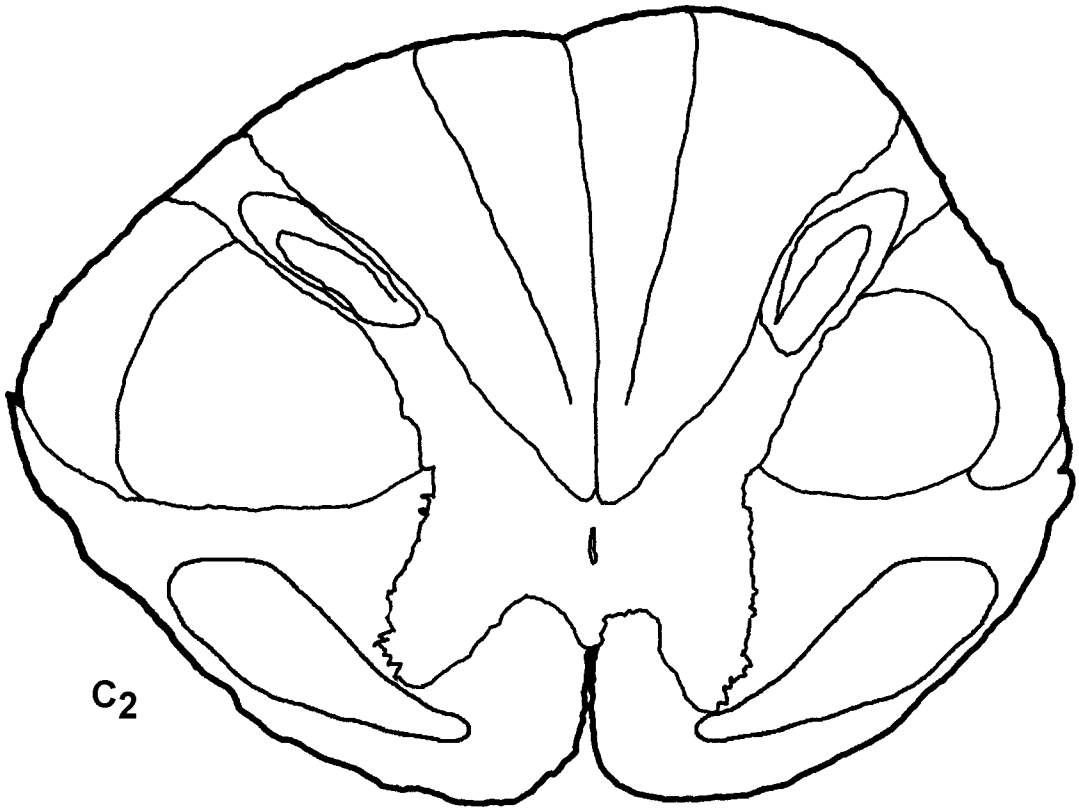
F. lesion results in deficit in distinguishing position of the right arm in space with eyes closed

G. lesion results in deficit in sense of warming in left hand

H. lesion results in right Babinski

I. cells arise in right motor cortex

PROBLEM SOLVING



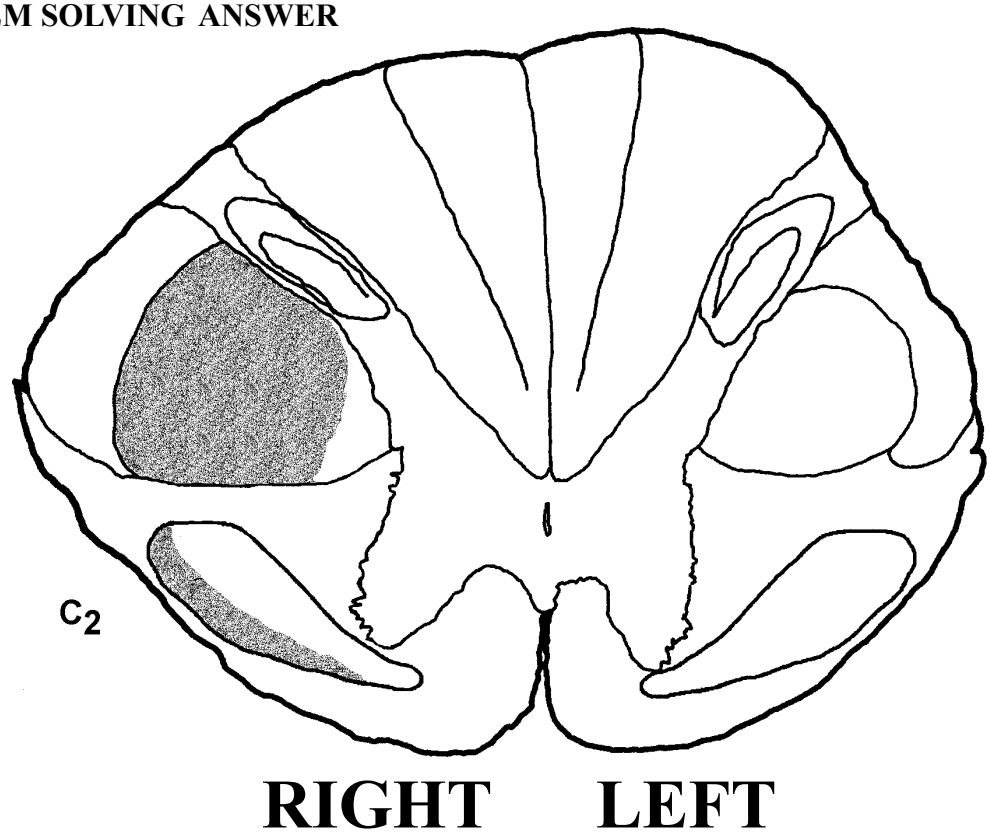
RIGHT LEFT

Shade in the location of unilateral lesions in the above drawing that will account for the following neurological deficits:

stroking the bottom of the right foot results in extension (up) of the right big toe, spastic right leg and arm, and deficit in fast pain from the left foot

PROBLEM SOLVING ANSWERS
POINTS 1-4

PROBLEM SOLVING ANSWER



ANSWERS TO PROBLEM SOLVING QUESTIONS RELATED TO ORIENTATION AND POINTS 1-4

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

Point #1 Dorsal Columns

Matching B,E

Point #2 Anterolateral System

Matching E,D

Point #3 Dorsal Spinocerebellar Tract

Matching B,G,A

Point #4 Lateral Corticospinal Tract

Matching C,D,A,I

5 VENTRAL HORN

The ventral horn contains some of the largest cells in the central nervous system. The large neurons directly innervate muscles. We need to think about what happens when there is a lesion that damages these large ventral horn motor neurons. When such damage occurs the motor neurons die, their axons in the ventral root die, and the muscles that they innervate eventually die. The muscle dies because it has lost its source of nourishment or trophic source (trophic = nourishment). When the muscle dies, it **ATROPHIES** or shrinks up. (atrophy = wasting). Atrophy occurs only when the cells that **DIRECTLY** innervate the muscle die. Also, as the lower motor neurons die, the muscles they innervate sometimes twitch. These twitches, as seen by the naked eye, are called fasciculations.

CELLS THAT DIRECTLY INNERVATE MUSCLES ARE CALLED

LOWER MOTOR NEURONS (LMNs)

Let's do some problem solving using this **extremely important** concept. What will happen if there is a lesion in the ventral horn at T1??? Well, it seems to me that the logical answer is that motor neurons in the ventral horn at this level, **and only at this level,** die. Their axons also die, and then the muscles innervated by these axons die, and atrophy. It's as simple as that!!!! T1 motor neurons innervate the intrinsic hand muscles.

Since the muscles innervated by ventral motor neurons at spinal level T1 are dead, they atrophy and are flaccid (flaccidus L = flabby). They will not respond to stretch and therefore there is **atonia** and **areflexia.**

Now, another important concept. As you already know, the lateral corticospinal tract (LCST) fibers synapse upon lower motor neurons in the spinal cord. This pathway is therefore called an **UPPER MOTOR NEURON (UMN)** pathway. Clinically, it is "***THEEEEEE" UMN pathway.*** Star, underline in many colors, and remember for the rest of your life:

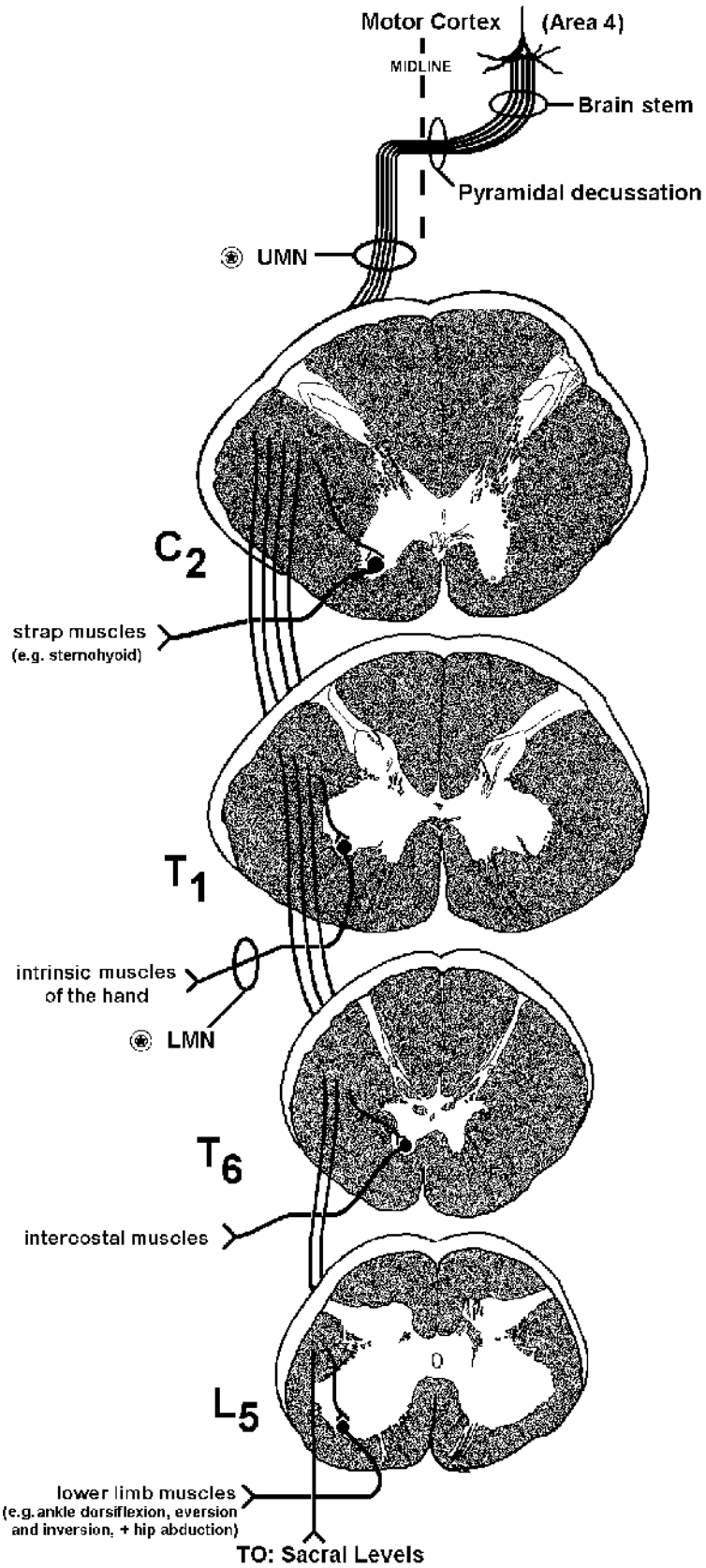
LCST = UMNs while VENTRAL HORN CELLS = LMNs.

UPPERS TELL LOWERS WHAT TO DO—BUT ONLY LOWERS TELL MUSCLES WHAT TO DO!!!!!!

LOWER MOTOR NEURON LESION = ATROPHY, FLACCIDITY, ATONICITY AND AREFLEXIA

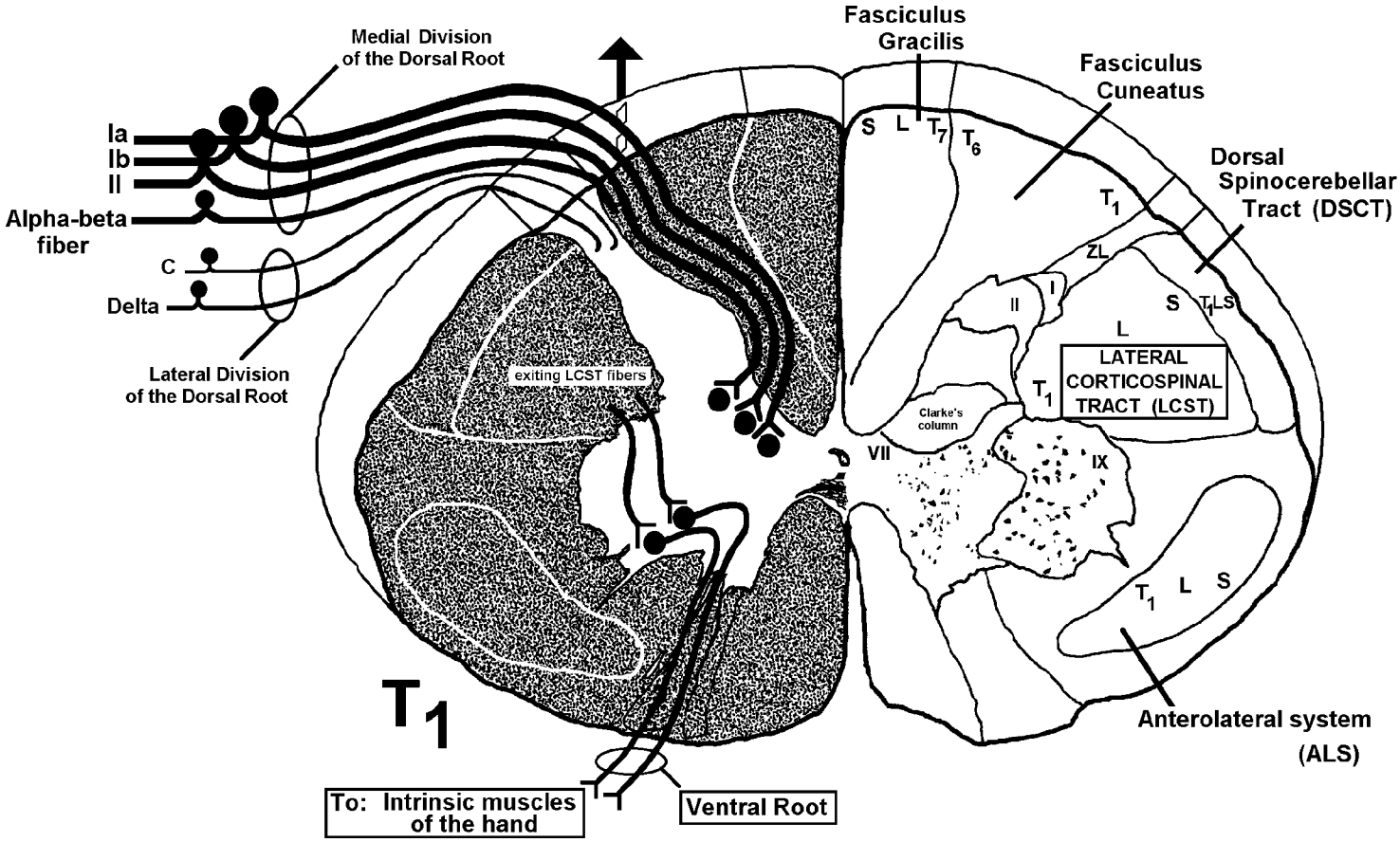
UPPER MOTOR NEURON LESION = SPASTICITY, BUT NO ATROPHY

Spinal cord
Ventral horn



LATERAL CORTICOSPINAL TRACT (LCST)

Now, for an interesting caveat. **Immediately** following a lesion of the LCST, **SPINAL SHOCK** occurs. During spinal shock there is atonia and flaccidity below the level of the lesion. This state lasts for varying periods of time, after which it is replaced by the classic clinical signs of UMN disease: **weakness and spasticity**.



PROBLEM SOLVING MATCHING

Match the best choice in the right hand column with the pathway or cell group in the left hand column. **There might be deficits that are not included in the responses.**

_____1. left fasciculus gracilis at C2

_____2. left anterolateral system at C2

_____3. left dorsal spinocerebellar tract at T6

_____4. cells in left ventral horn at T1

_____5. left fasciculus cuneatus at C2

A. lesion results in deficit in unconscious proprioception from the left leg

B. axons arise from dorsal roots T7 and below on the left

C. axons carry info. about vibration from the right big toe

D. lesion results in deficit in sense of cooling from the right foot

E. lesion results in deficit in conscious proprioception from the left elbow

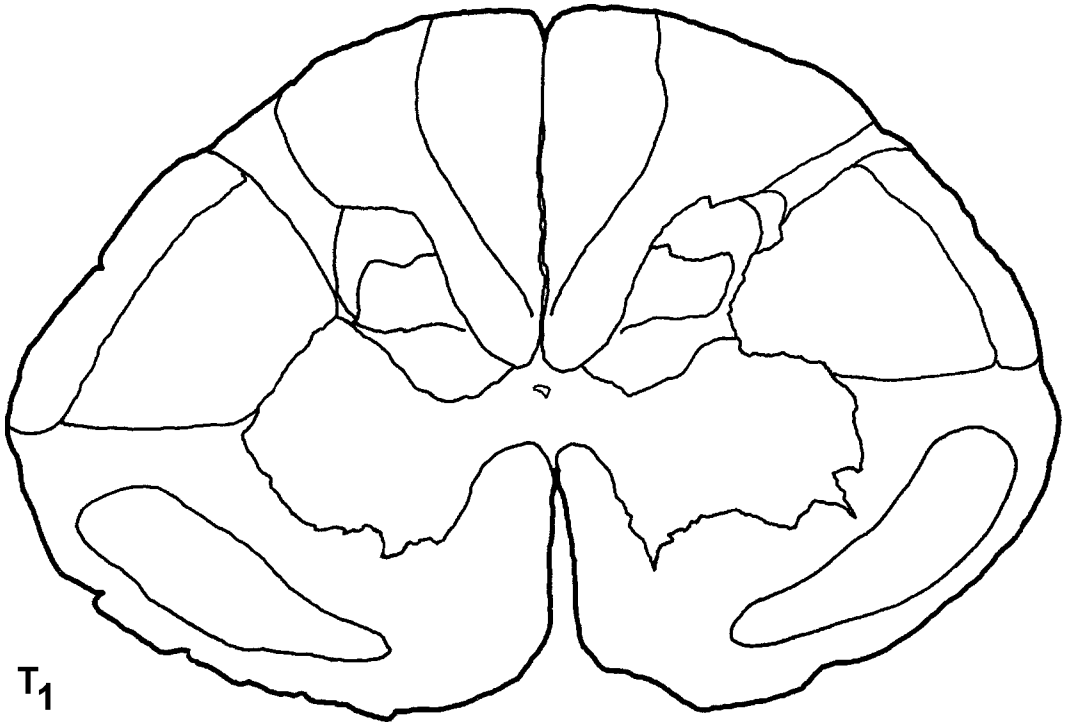
F. cell bodies lie in Clarke's nucleus on the right side

G. lesion results in deficit in the sense of warming in the left hand

H. lesion results in atrophy and fasciculations of the intrinsic muscles of the left hand

I. cells arise in right motor cortex

PROBLEM SOLVING



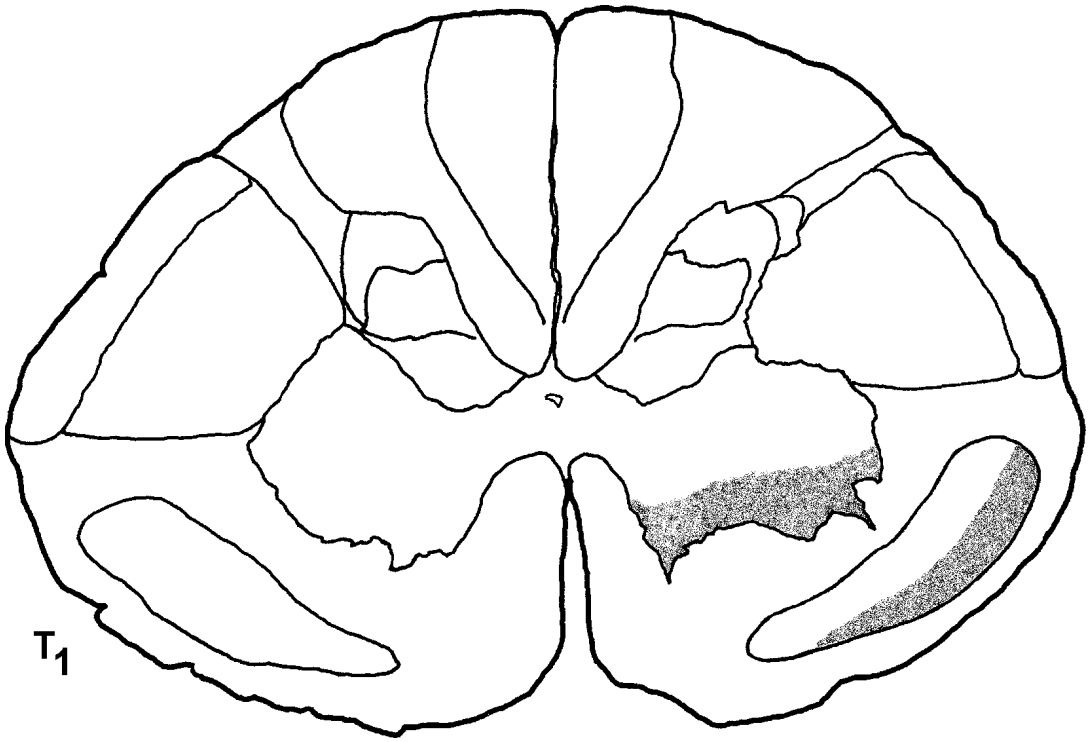
RIGHT LEFT

Shade in the location of unilateral lesions in the above drawing that will account for the following neurological deficits:

deficit in pricking pain from the right foot only, and atrophy of intrinsic muscles of the left hand

Spinal cord
Ventral horn
Problem solving

PROBLEM SOLVING ANSWER

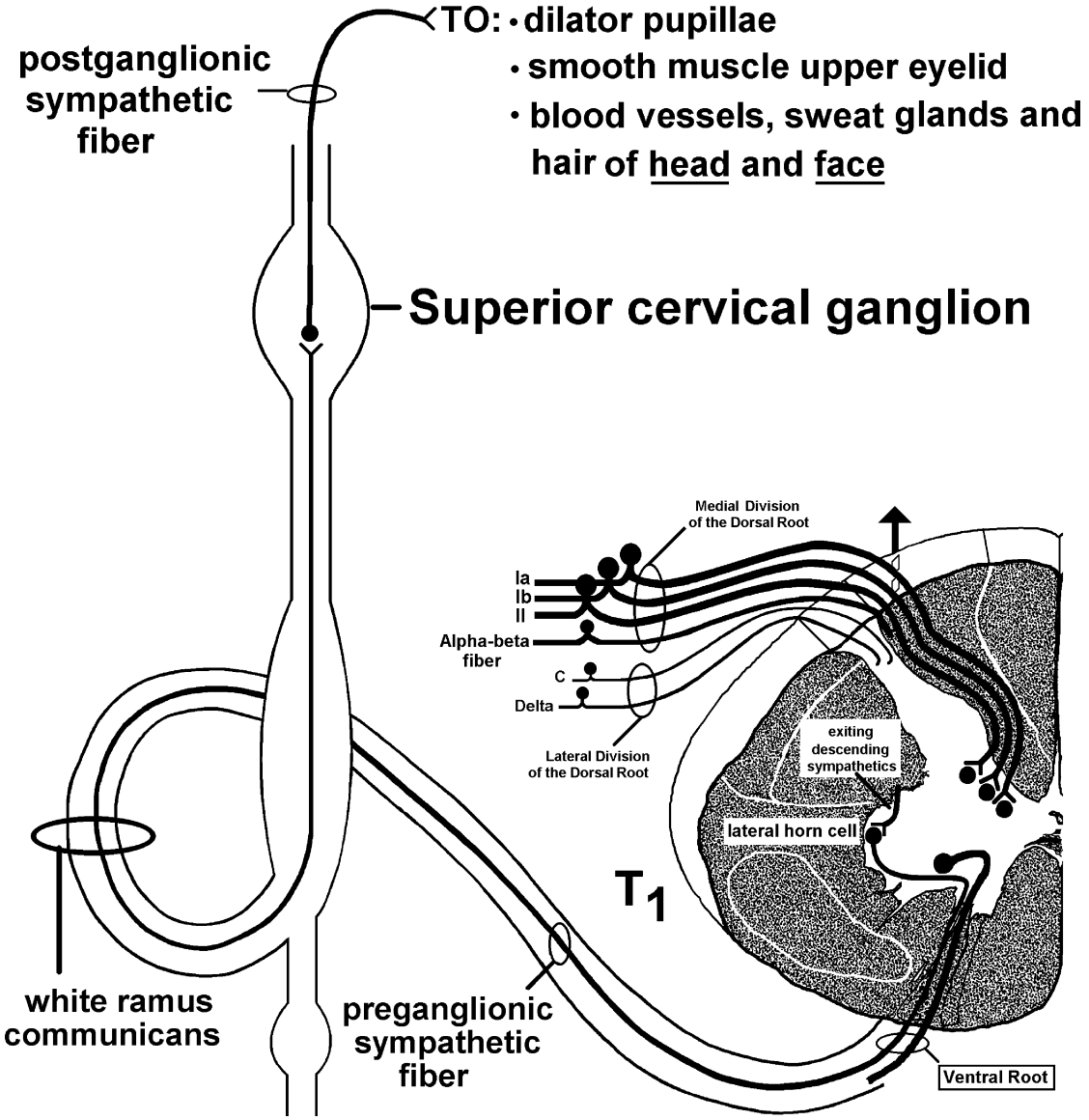


FYI and enjoyment

The brain of the great physicist Albert Einstein weighed 1,230 grams. This is far below the average brain weight of 1,400 grams. (Reference: *Neuroscience Letters*, 210:161-164, 1996.)
The adult human spinal cord weighs about 35 grams (0.1 lb).
The average length of the adult spinal cord is 45 cm for men and 43 cm for women.
The brain of an elephant weighs about 6 kg (13 lb)

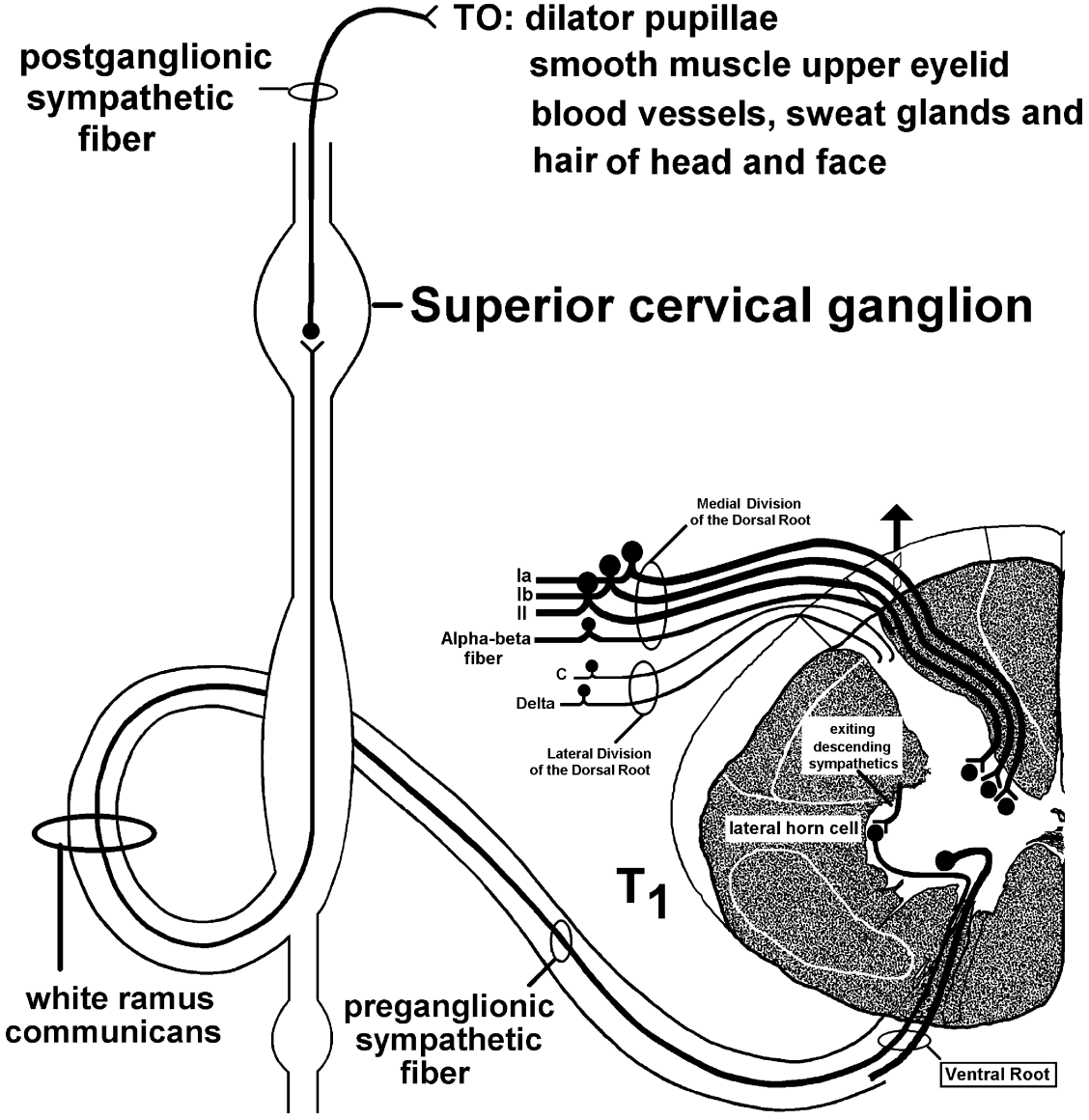
6 LATERAL (INTERMEDIOLATERAL) HORN (preganglionic autonomics)

Hopefully, you will remember from Gross Anatomy some of the important details regarding the organization of the **autonomic nervous system** within the spinal cord. What I want to emphasize at this time is the **sympathetic** outflow. There are **PREGANGLIONIC SYMPATHETIC** cells present in the spinal cord at spinal levels T1-L2. This clump of cells comprises the **LATERAL CELL COLUMN (also called the intermediolateral cell column by some investigators)**. Axons of these cells pass ventrally and comprise part of the ventral root (from T1-L2). These **preganglionic sympathetics** can then do a number of things.



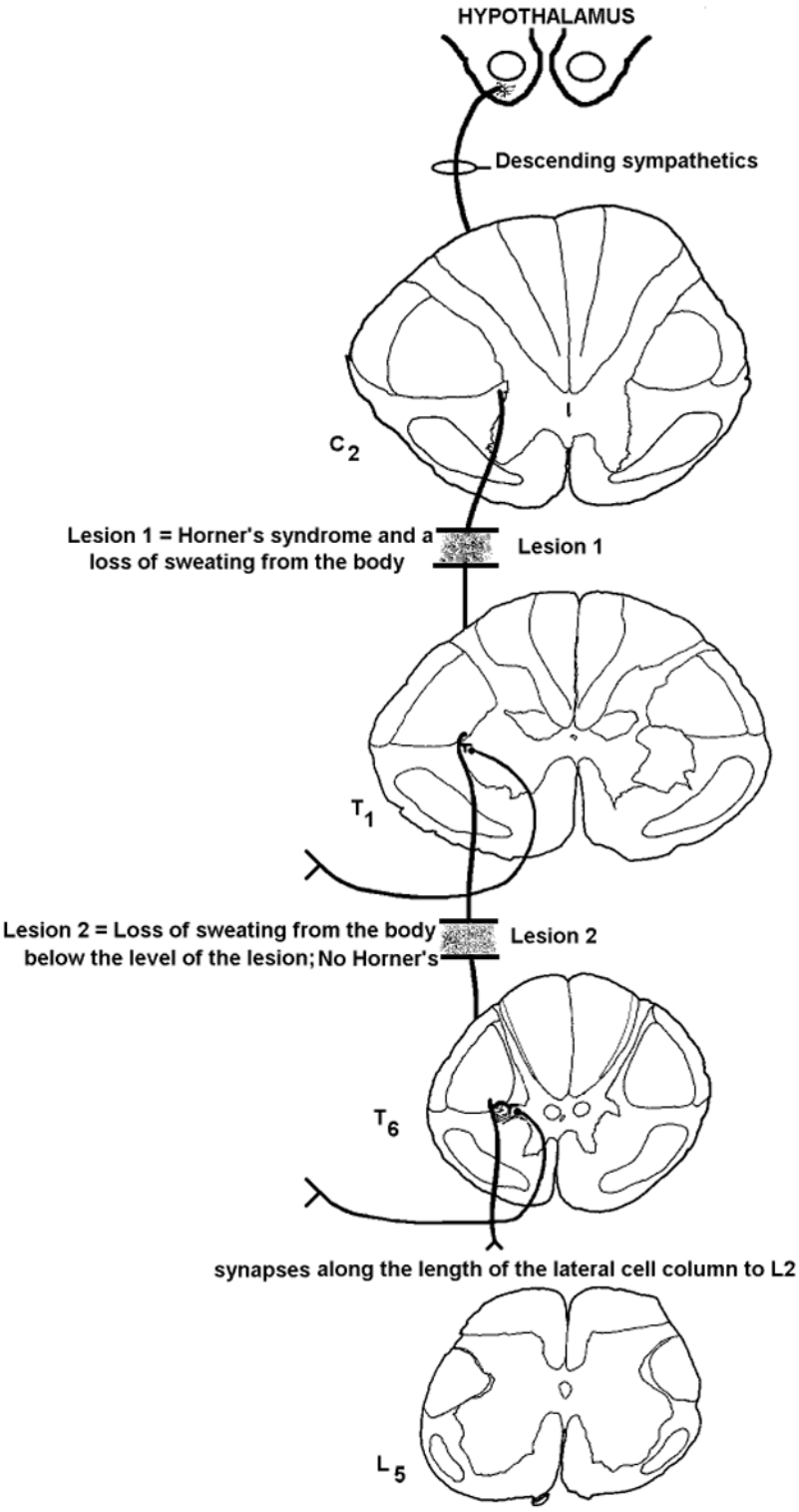
Spinal cord
Lateral horn

The **preganglionic sympathetics** pass to the sympathetic trunk via the white communicating rami. They can synapse in the autonomic trunk (paravertebral ganglia), go up or down and synapse, or go through to comprise the splanchnics. Preganglionic sympathetics from spinal levels T1 to T5 ascend to the superior cervical ganglion, but most of these fibers arise from **T1**. Cells in the superior cervical ganglion (which receive their main drive from cells in the lateral horn at spinal level T1) then innervate, via **postganglionic sympathetics**, the smooth muscle of the dilator pupillae, the smooth muscle of the upper eyelid, the blood vessels, sweat glands, and hair of the head and face. A **LESION at spinal level T1**, either in the spinal cord or the ventral root, interrupts the sympathetic drive to these structures. This results in what is called **HORNER'S SYNDROME**. On the side **IPSILATERAL** to the spinal T1 lesion there is a drooping eyelid (**PTOSIS**), a constricted pupil (**MIOSIS**; remember, the boring parasympathetics are "in charge"), and a flushed (vasodilation, since sympathetics to the skin vasoconstrict) and dry face.



If lesions interrupt the lateral horn or the ventral roots as far caudal as L2, the loss of sympathetic drive to the thorax, abdomen and pelvis **is most noticeable** in the lack of sweating in the area innervated by the particular nerve (heart rate and visceral control are relatively normal at rest).

NOW FOR A DIFFICULT CONCEPT. There are sympathetic-related fibers that arise from cells in the ***hypothalamus*** and descend through the brain stem to reach the spinal cord. In the spinal cord they travel in the lateral funiculus in the **most medial part of the LCST**. If these fibers are interrupted anywhere ***above*** T1, (in the brain stem or between spinal levels C1 and T1) cells in the lateral horn at spinal level T1 have lost their major drive. The result is similar to a lesion in the lateral horn or ventral root at T1. That is, the sympathetic outflow to the head is interrupted, resulting in a Horner's syndrome. ALSO SWEATING OVER THE REST OF THE BODY IS AFFECTED because cells in the lateral horn below T1, have lost their drive. So, a lesion of the descending sympathetics above T1 = loss of sweating over the **entire body** ipsi to the lesion. Lesion of descending sympathetics below T1 spares the head and involves loss of sweating below the level of the lesion.



PROBLEM SOLVING MATCHING

Match the best choice in the right hand column with the pathway or cell group in the left hand column. **There might be deficits that are not included in the responses.**

_____1. left fasciculus gracilis at C1

_____2. left lateral corticospinal tract at C1

_____3. left dorsal spinocerebellar tract at C1

_____4. cells in left ventral horn at T1

_____5. left fasciculus cuneatus at C1

_____6. left lateral horn at C6

A. lesion results in deficit in unconscious proprioception from the right leg

B. axons arise from the dorsal roots T7 and below on the left

C. axons carry info. about vibration from the right big toe

D. lesion results in deficit in sense of cooling from the right foot

E. lesion results in deficit in conscious proprioception from the left elbow

F. cell bodies lie in the left Clarke's nucleus

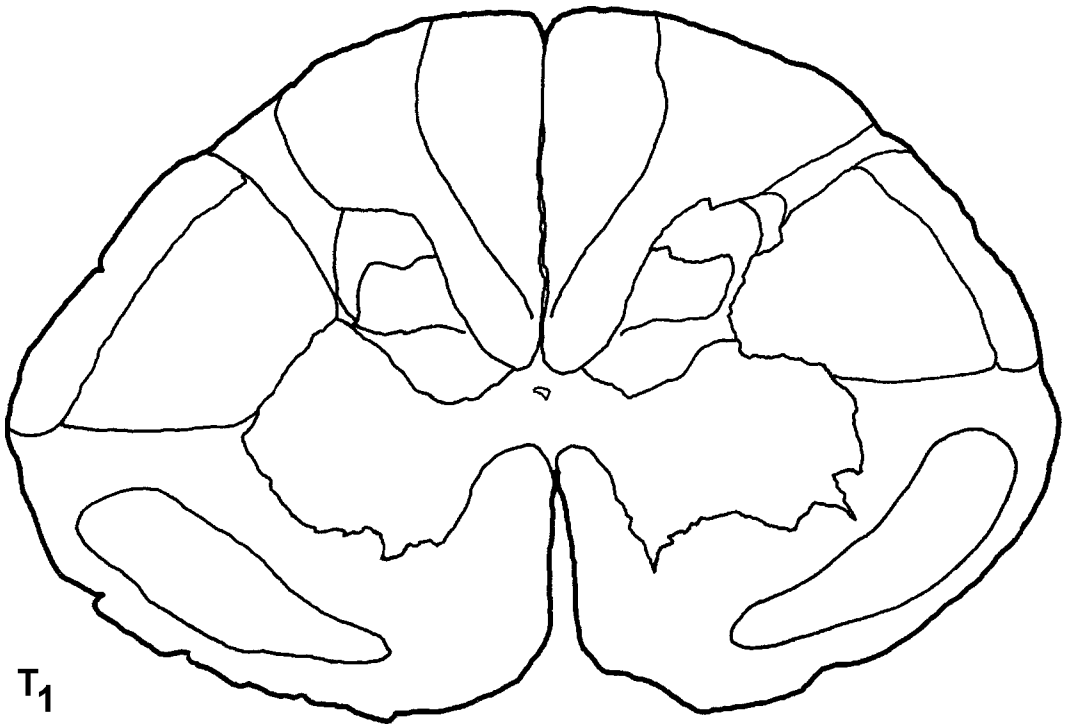
G. lesion results in deficit in sense of warming from the left hand

H. lesion results in atrophy ofthe intrinsic muscles of the left hand

I. axons arise from cells in right motor cortex

J. NO appropriate response

PROBLEM SOLVING

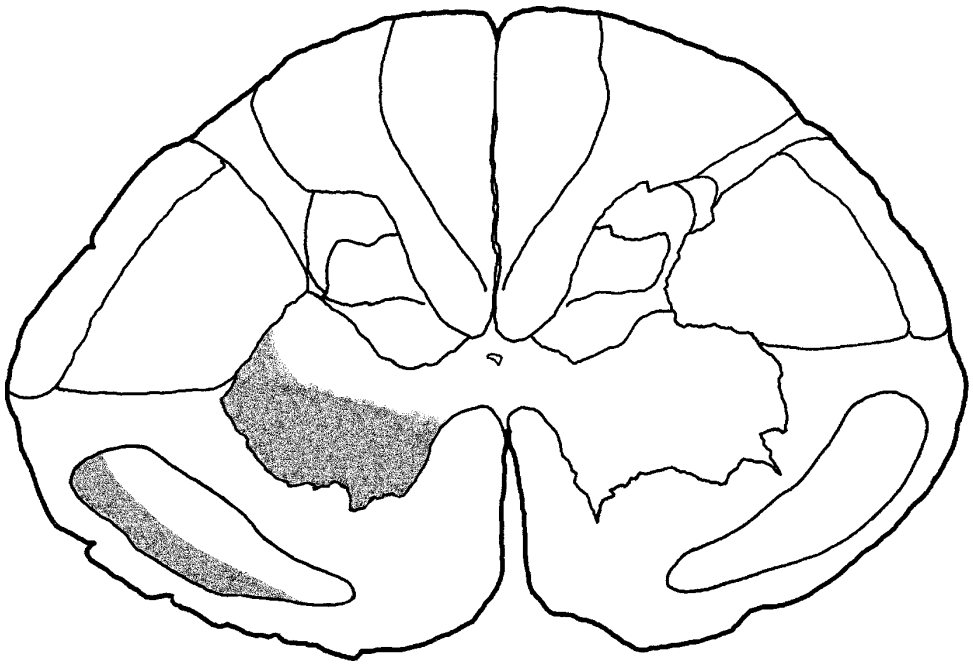


Shade in the location of two, unilateral lesions in the above drawing that will account for the following neurological deficits:

atrophy of the intrinsic muscles of the right hand, deficit in burning pain from the left leg, **ptosis** of the right eyelid and **miosis** of the right pupil

Spinal cord
Lateral horn
Problem solving

PROBLEM SOLVING ANSWER

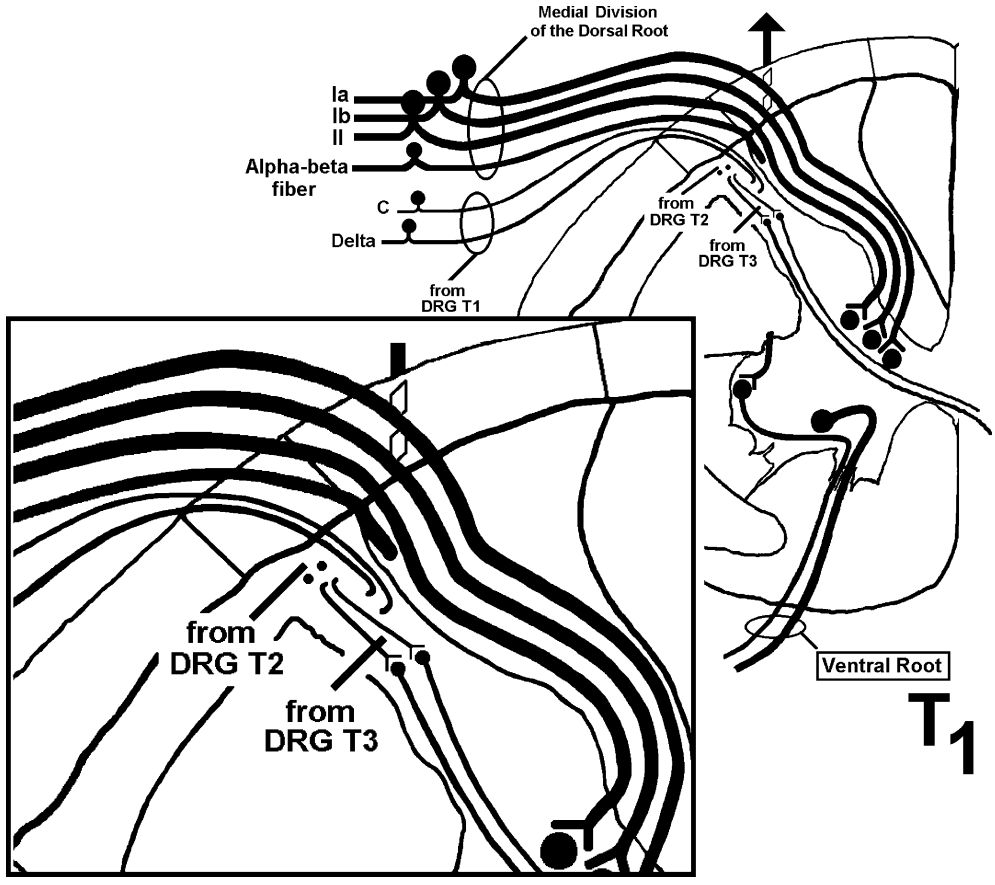


BRINGING IT ALL TOGETHER

Let's go through this by making a lesion of each of the pathways or cell groups discussed so far. We will make these lesions at spinal level T1. You will soon discover how much you know!! Remember in this point all pathways and cell groups of one half of the spinal cord are dead.

1. Dorsal root entry zone and zone of Lissauer at spinal level T1.

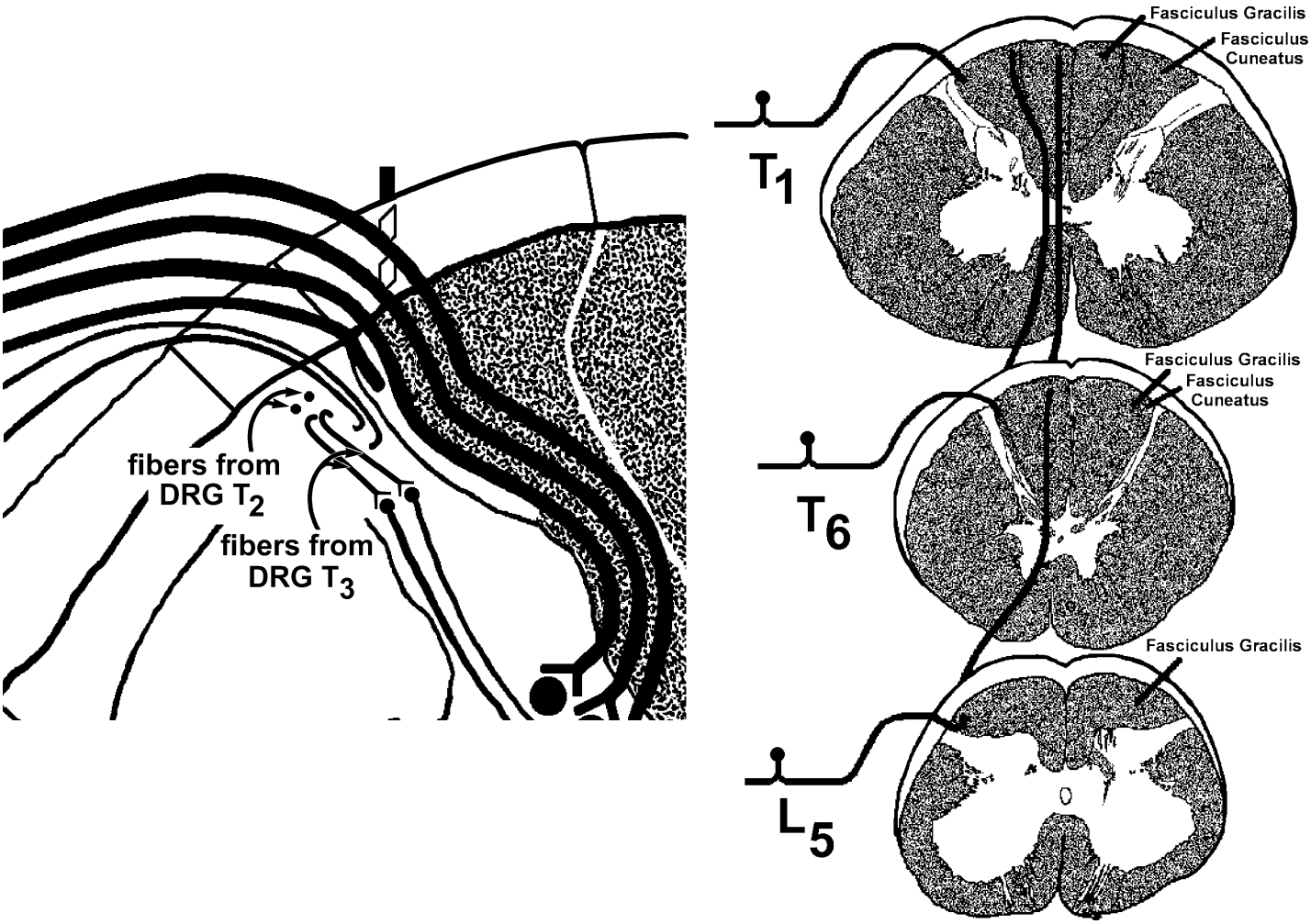
A lesion in this area interrupts the lateral and medial divisions of the dorsal root and the ZL. Interruption of the dorsal root means that NO sensory information gets into the brain from the area of the **ipsilateral** body innervated by the T1 dorsal root. This includes **2 pt. discrimination, vibration and conscious proprioception (alpha-betas), unconscious proprioception (1a, 1b and II) and pain and temperature and crude touch (C's and deltas)**. In addition, remember that pain and temperature fibers **ASCEND IPSILATERALLY** in the zone of Lissauer for two segments before they enter the dorsal horn and synapse at T1. A lesion interrupts these fibers too. The ascending delta and C fibers from T3 are headed for the dorsal horn at T1—they are dead. The ascending fibers from T2 are headed for the dorsal horn at C8—they are dead!! And of course, the delta and C fibers coming in at T1 via the dorsal root are dead!!! Thus, on the **IPSI** side the pain and temp. loss is from **T1-T3**. The loss of all sensory information from T1 means that no reflexes (e.g. stretch, pain) can occur via this dorsal root.



2. Dorsal columns at T1.

Now let's add the loss of the dorsal columns at T1.

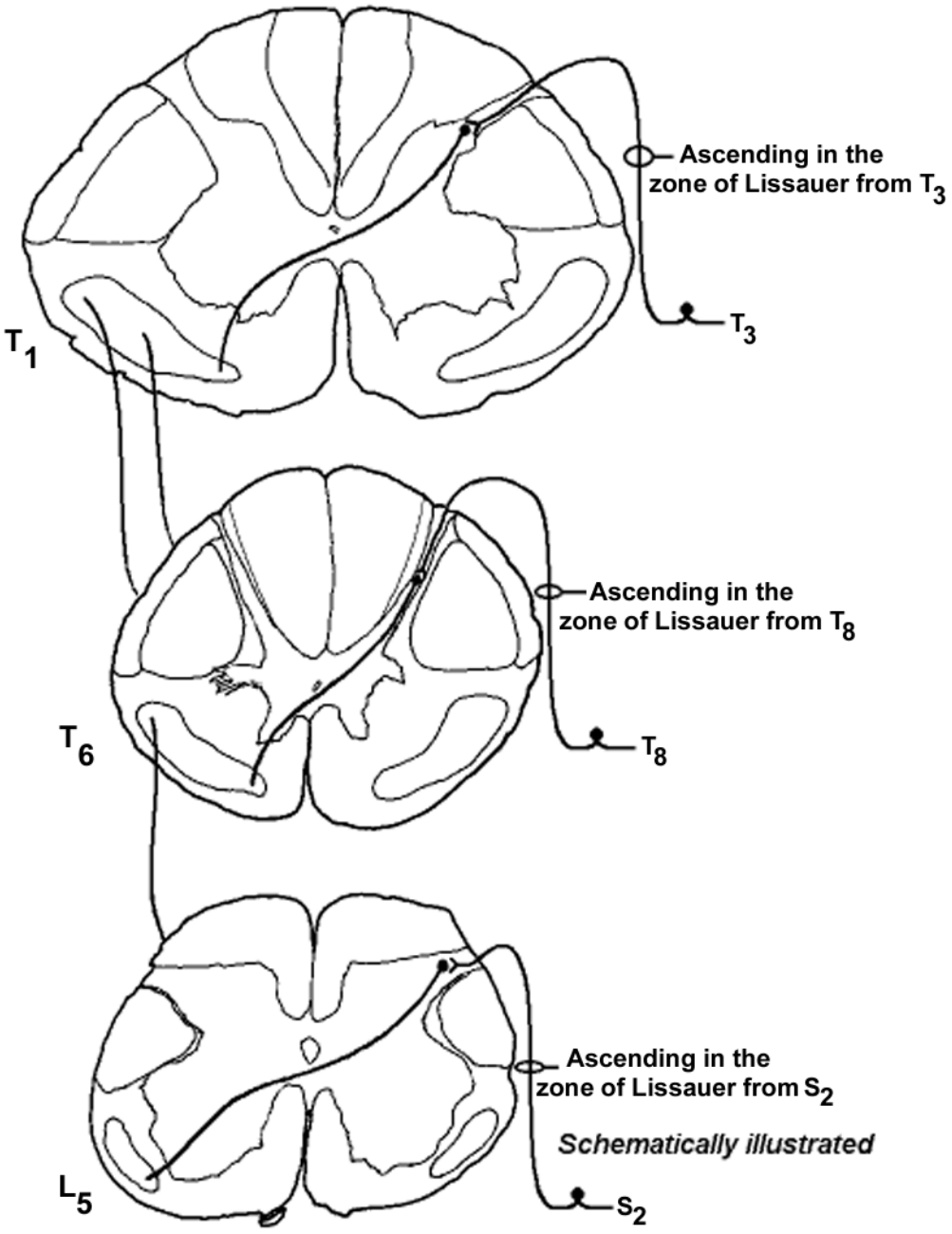
The deficits are **IPSI** and involve T1 and below. The deficits include two point discrimination, vibration sense, conscious proprioception, astereognosis and agraphesthesia. If you add these deficits to the loss of pain, temperature, and crude touch from T2 and T3 (by interupting the ascending C and delta fibers discussed above in #1), you can see that spinal levels T2 and T3 are, like T1, **ANESTHETIC** ("add" the dorsal column "loss" and zone of Lissauer "loss" for segments T1, T2 and T3). Any problems in coordination from a lesion of the dorsal columns are overshadowed by other deficits (spasticity and muscle atrophy) to be discussed.



3. Anterolateral system at T1.

Now let's add the loss of the ALS at T1.

There is a **CONTRA** deficit in pain and temperature from T3 and below (alllll the way down!!). This is called **ANALGESIA** (Gr., no pain). There also is a deficit in crude touch in the same area.

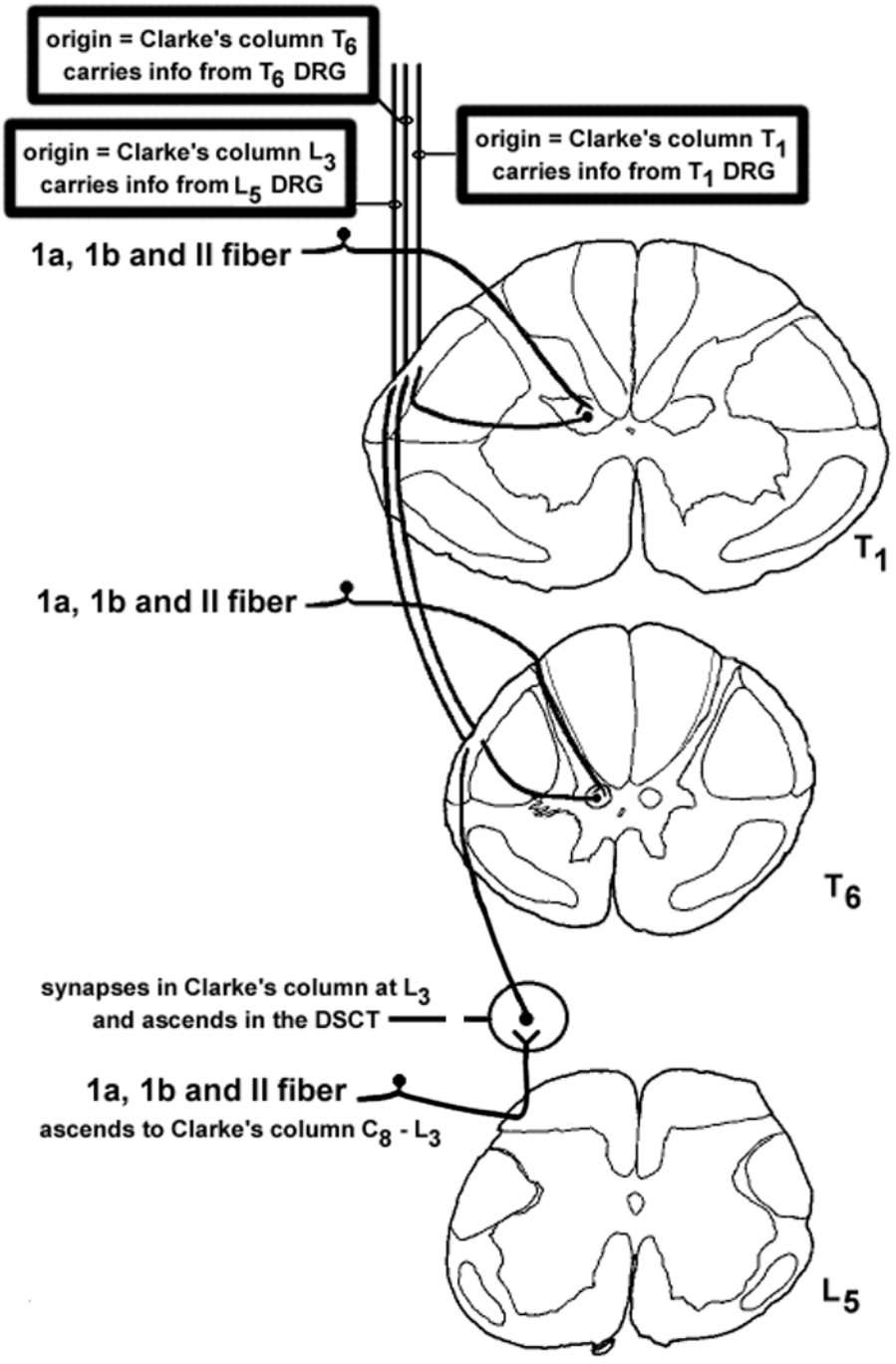


This deficit is **"covered"** by the ***intact dorsal columns*** on that side.

4. Dorsal spinocerebellar tract at T1.

Now let's add the loss of the DSCT at T1.

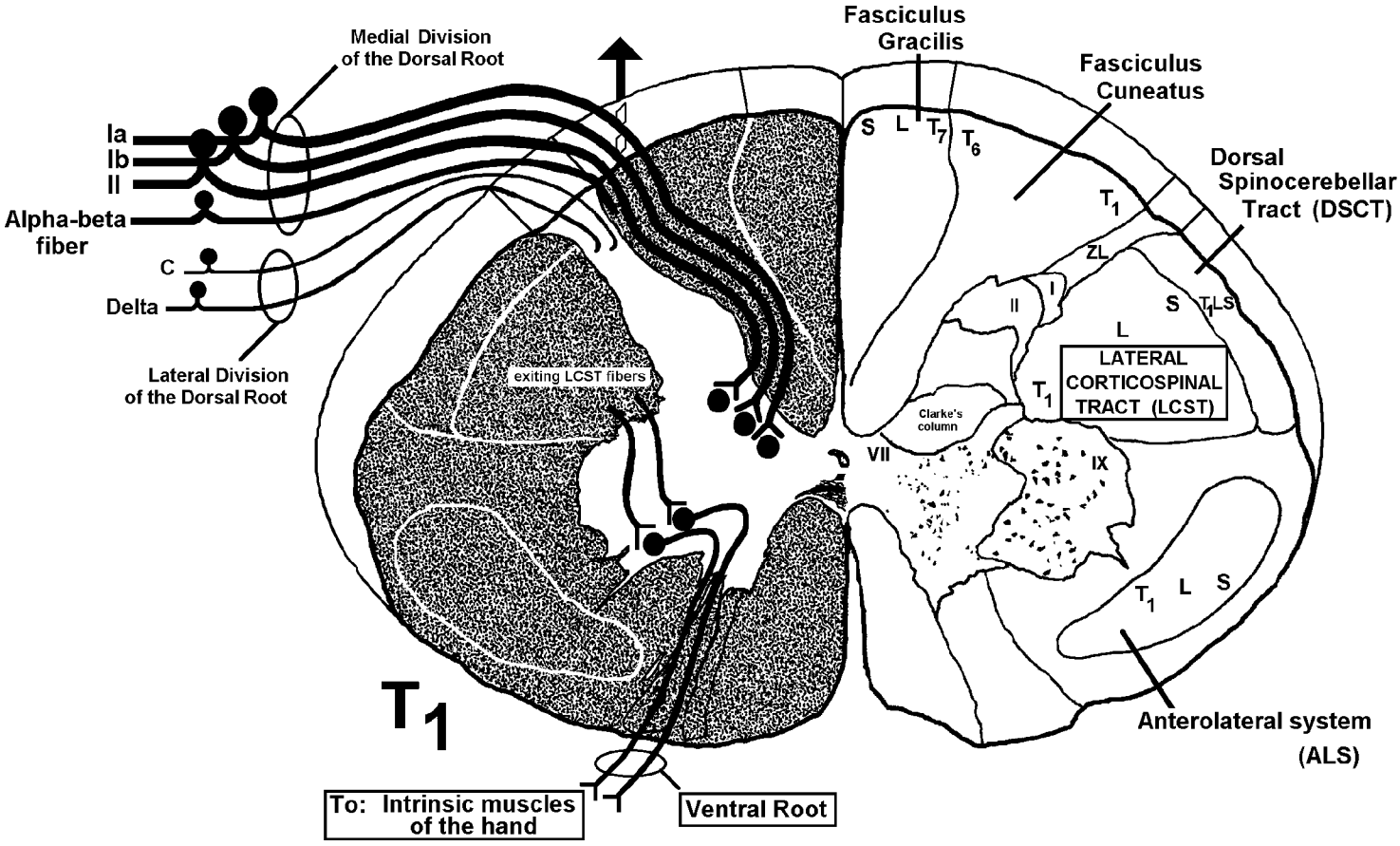
There is a loss of unconscious proprioception **IPSILATERAL** to the lesion from T1 and below. The deficit involves incoordination of the lower extremity (and the forearm). This deficit will be ***camouflaged*** by problems related to interruption of the **LCST**.



5. Cells in the ventral horn at T1 are dead.

Now let's add the loss of the ventral horn at T1.

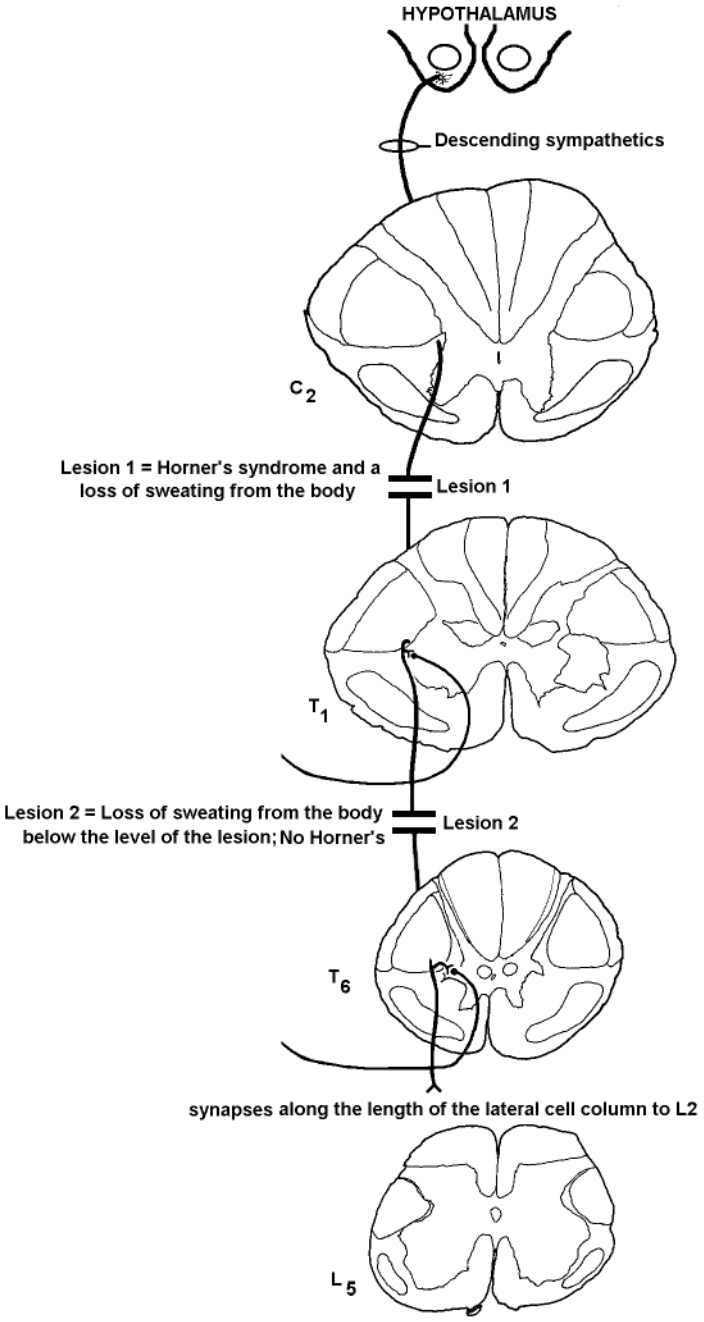
There is atrophy of the ipsilateral intrinsic muscles of the hand that are innervated by the lower motor neurons in the ventral horn at T1.



6. Lateral horn at T1.

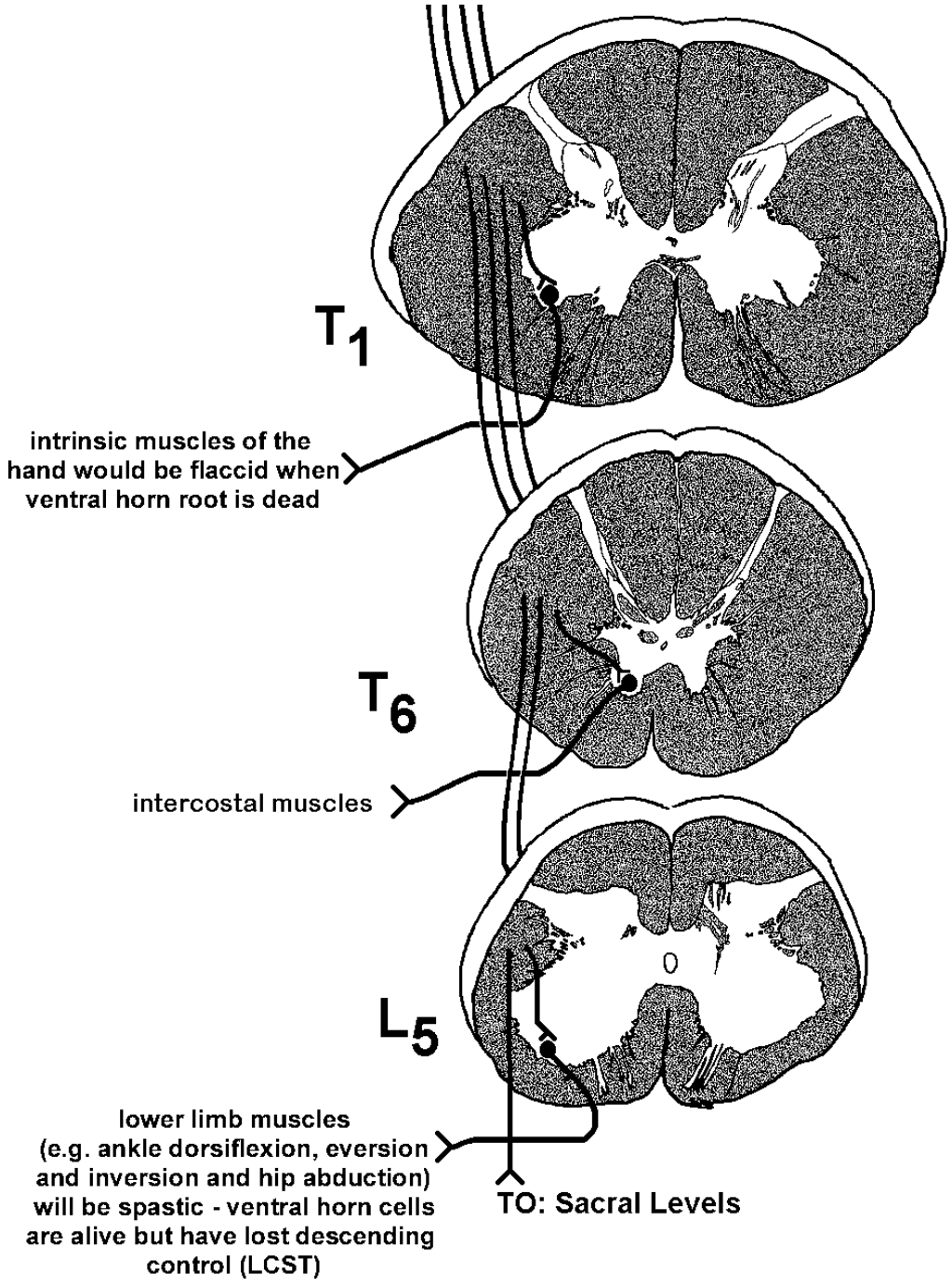
Now let's add the loss of the lateral horn at T1.

There is an **IPSILATERAL** Horner's Syndrome (ptosis, miosis, dry face, etc.). The interruption of the **descending sympathetics** (that no longer pass to regions of the spinal cord below T1) means that there is no sweating on the entire IPSI side of the body.

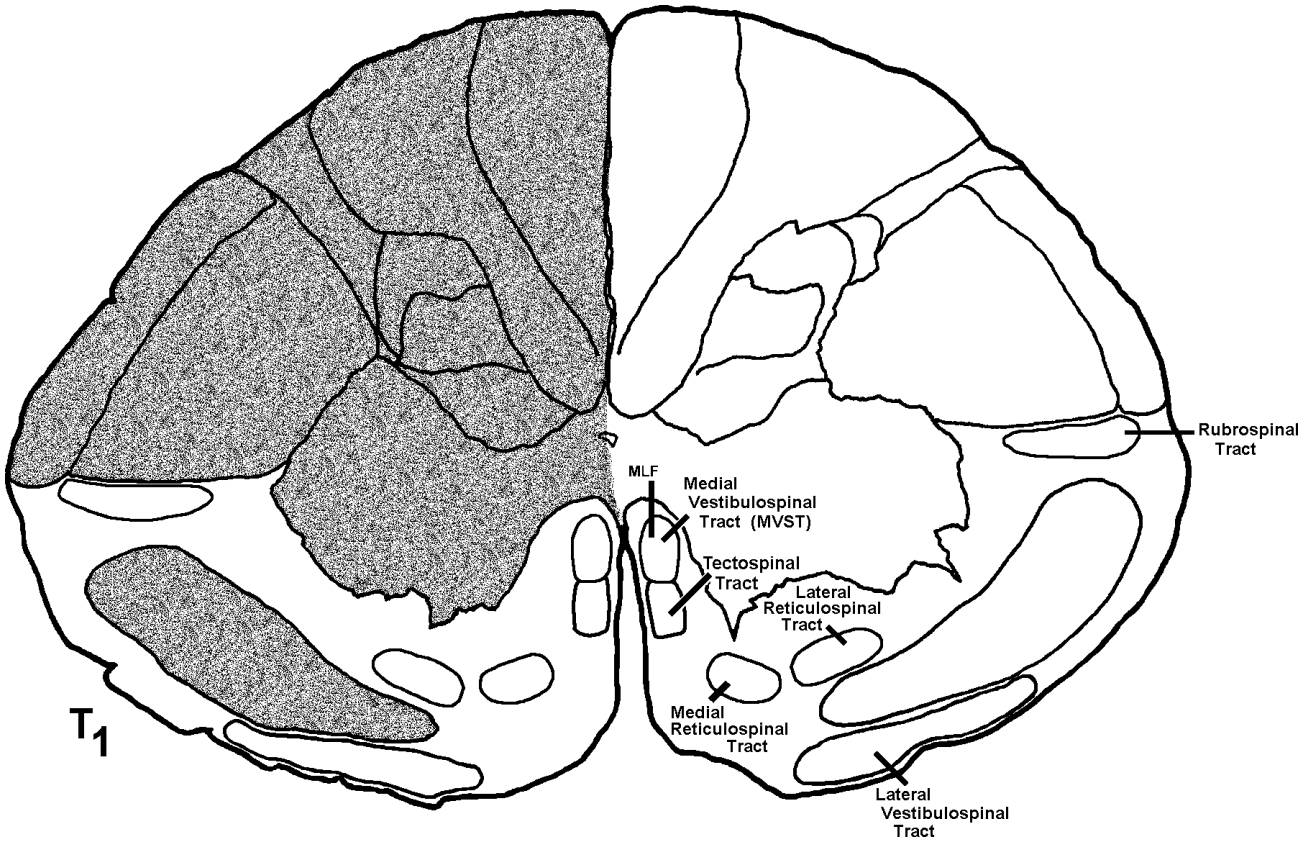


7. Lateral corticospinal tract at T1.

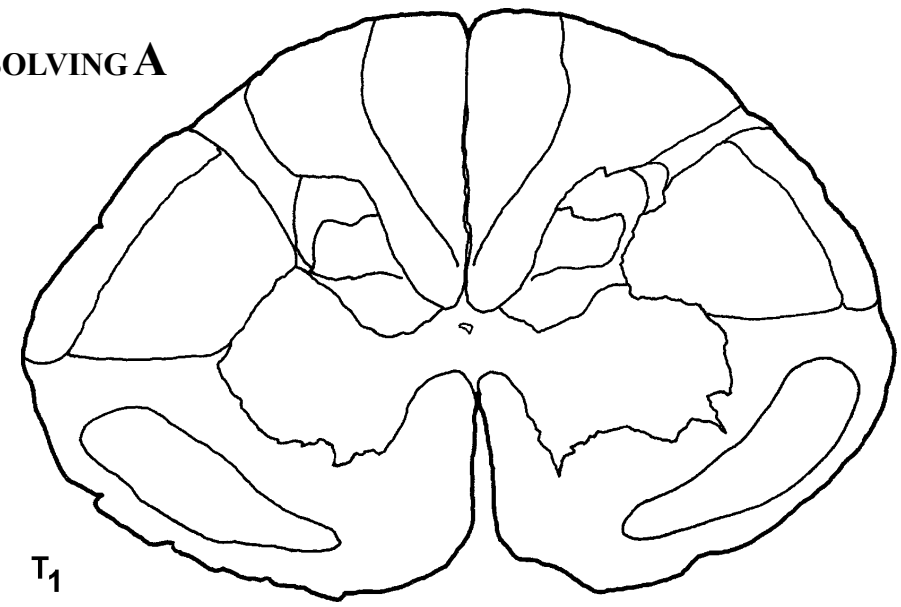
Now let's add the loss of the LCST at T1. Remember, all of the pathways we have thus far discussed are dead! A lesion of the LCST at T1 will result in weakness and spasticity of all muscle innervated by spinal segments **T2** and below. These muscles are spastic. There is a Babinski sign. The reason that the muscles innervated by **T1 are not spastic** is that cells in the ventral horn at T1 are ***dead*** . You need an intact stretch reflex to have spasticity. Remember, if you quickly stretch a spastic limb it will contract and then release, contract and release. This could not happen without a dorsal root bringing in the muscle spindle information and a ventral root causing the contraction.



As you can see from the figure below, there are several spinal cord pathways that we have not discussed. These pathways will be covered in the next few weeks. Right now, realize that a lesion of the LCST, following a stroke, will **not** disrupt **all** descending motor control. You may have seen such patients and it is clear that there is some motor control that is conveyed via several of the pathways labeled below.



PROBLEM SOLVING A

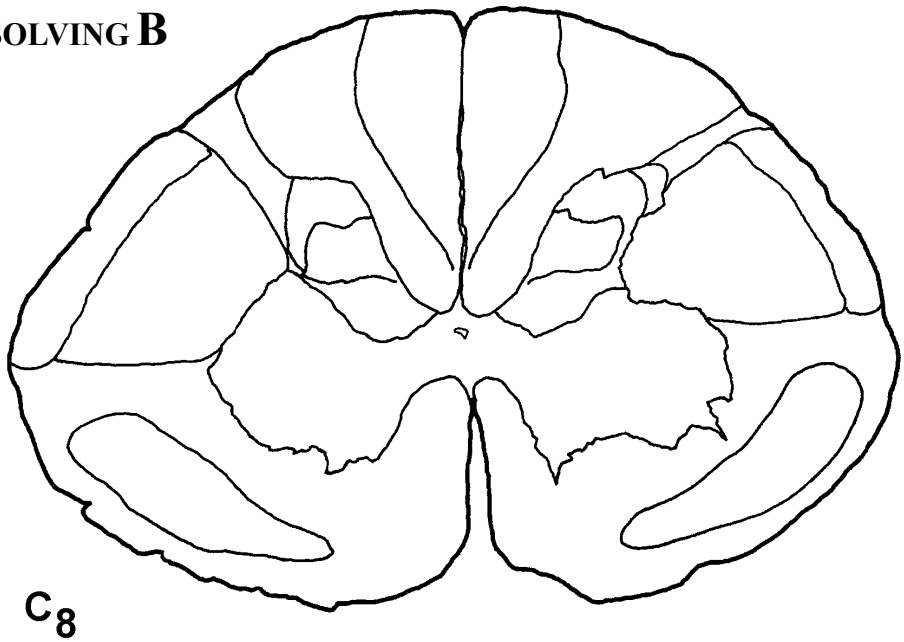


RIGHT LEFT

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

loss of pain and temperature from **only** the region of the body innervated by T1-T3 on the **right**

PROBLEM SOLVING B

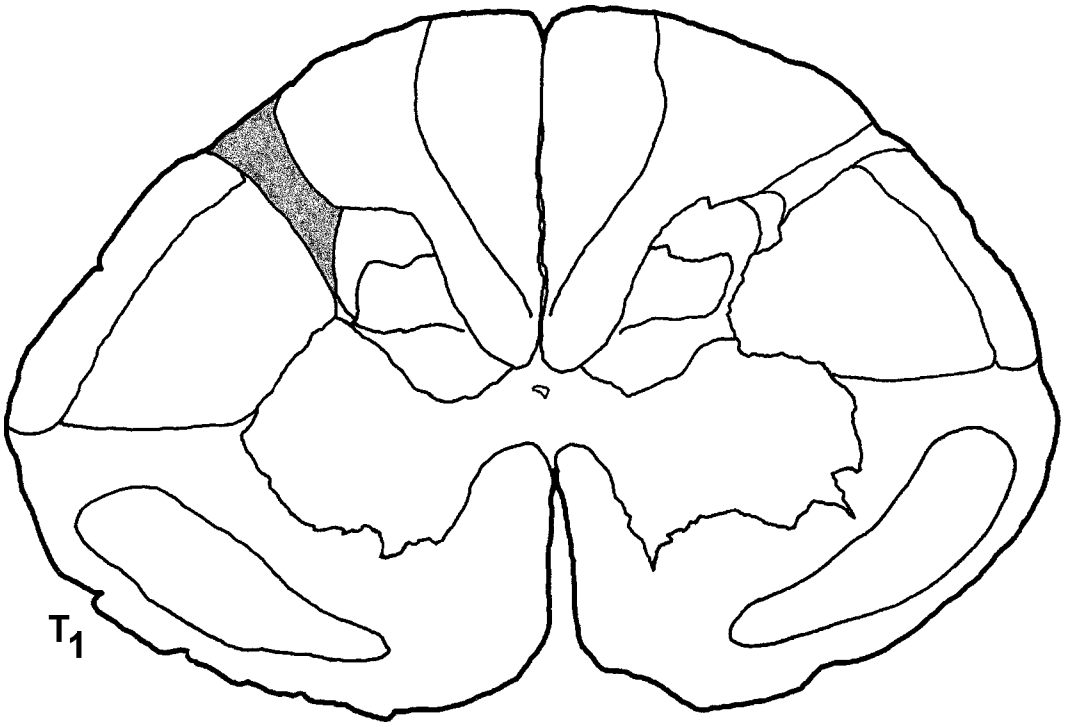


RIGHT LEFT

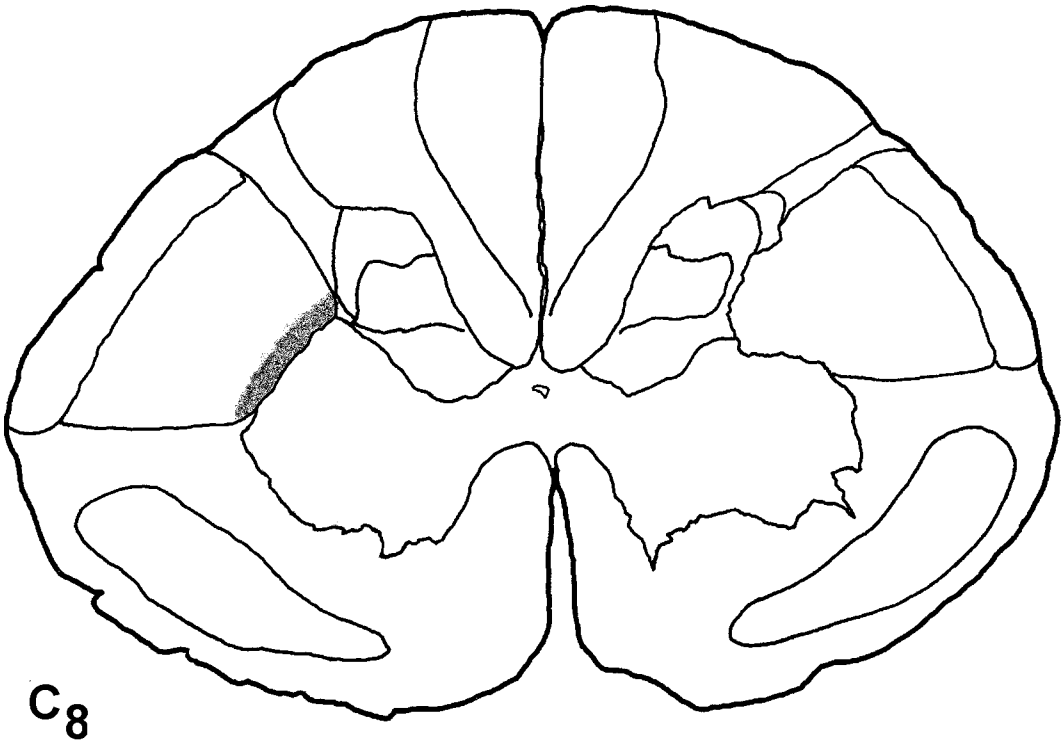
Shade in the location of a single, continuous, unilateral lesion in the above drawing that will account for the following neurological deficits:

loss of sweating from the entire **right** side of the body and head

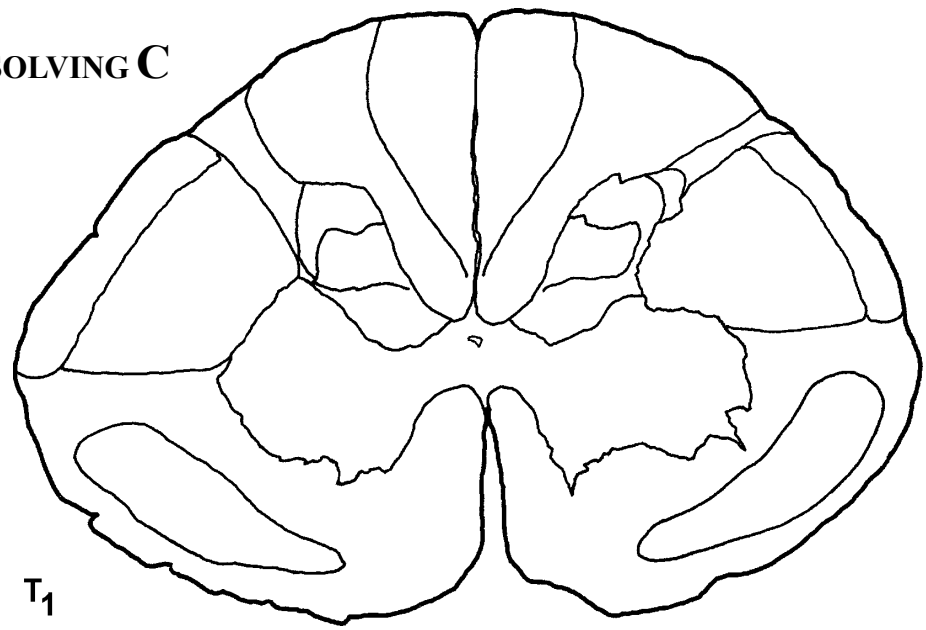
PROBLEM SOLVING ANSWER A



PROBLEM SOLVING ANSWER B



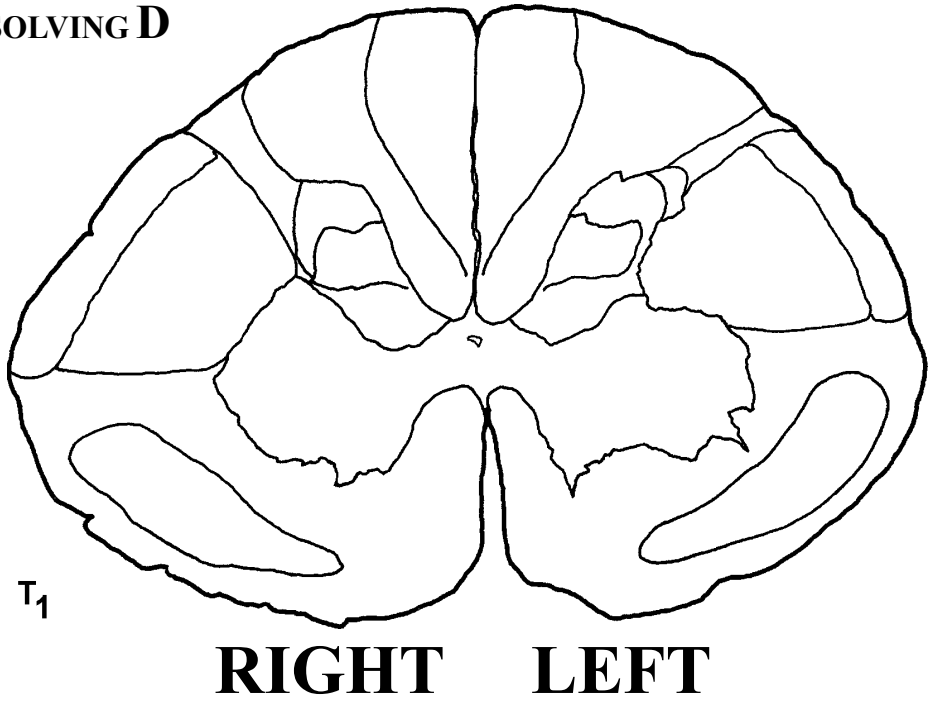
PROBLEM SOLVING C



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

loss of pain and temperature from the region of the body innervated by spinal segments **T3 and below** on the **left**

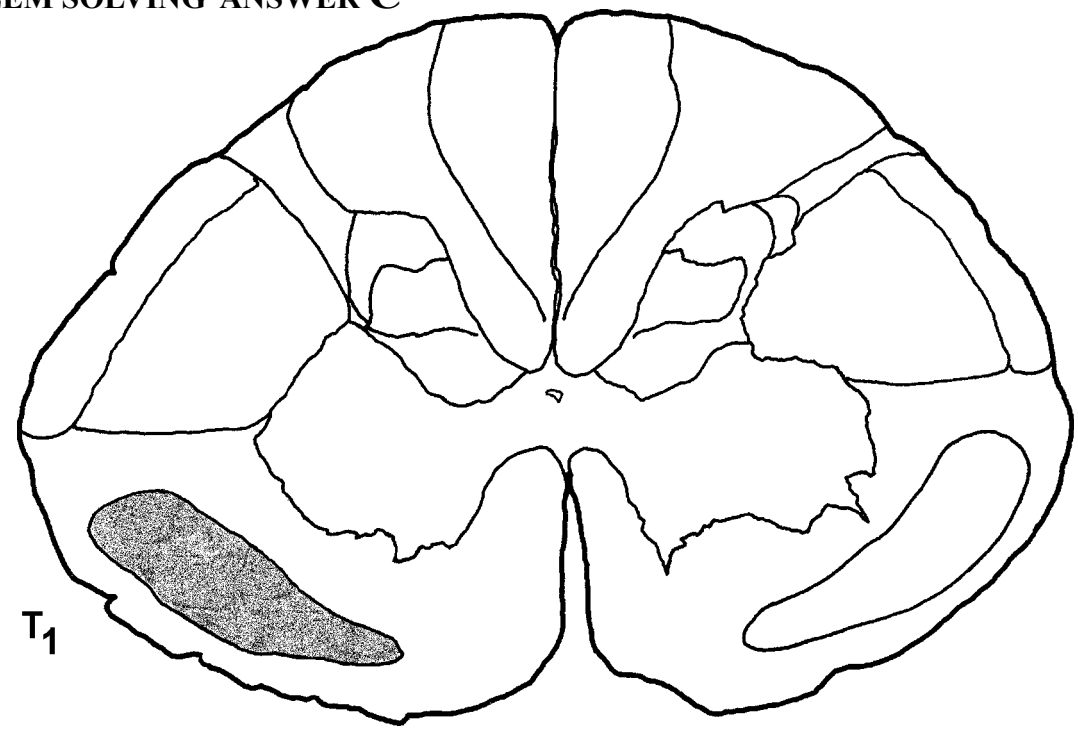
PROBLEM SOLVING D



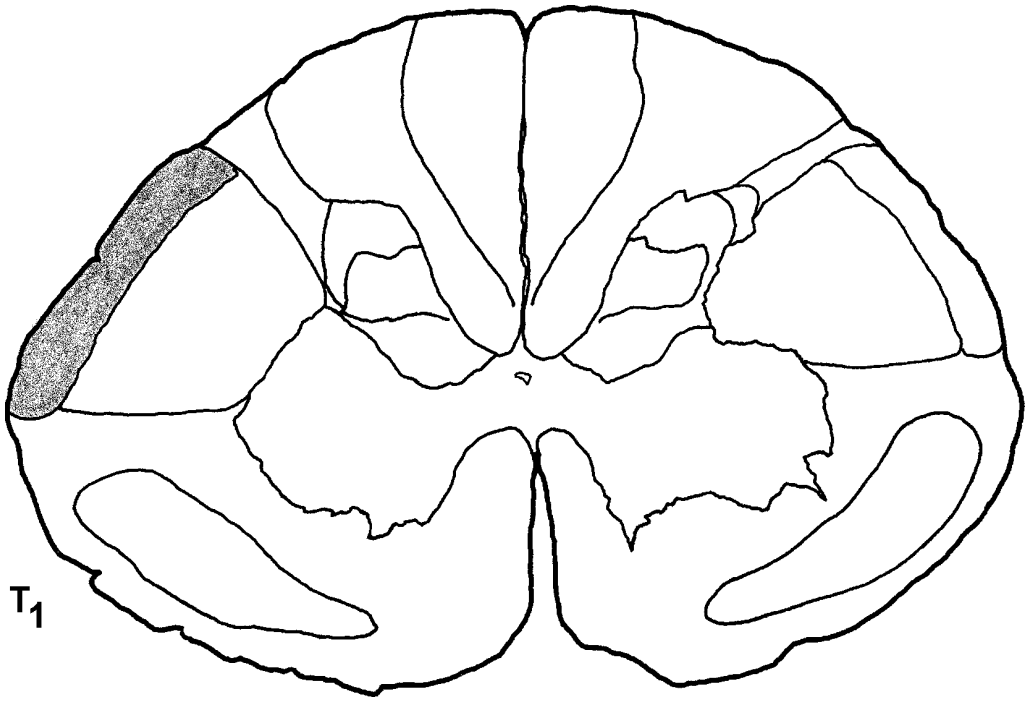
Shade in the location of a single, continuous, unilateral lesion in the above drawing that will account for the following neurological deficits:

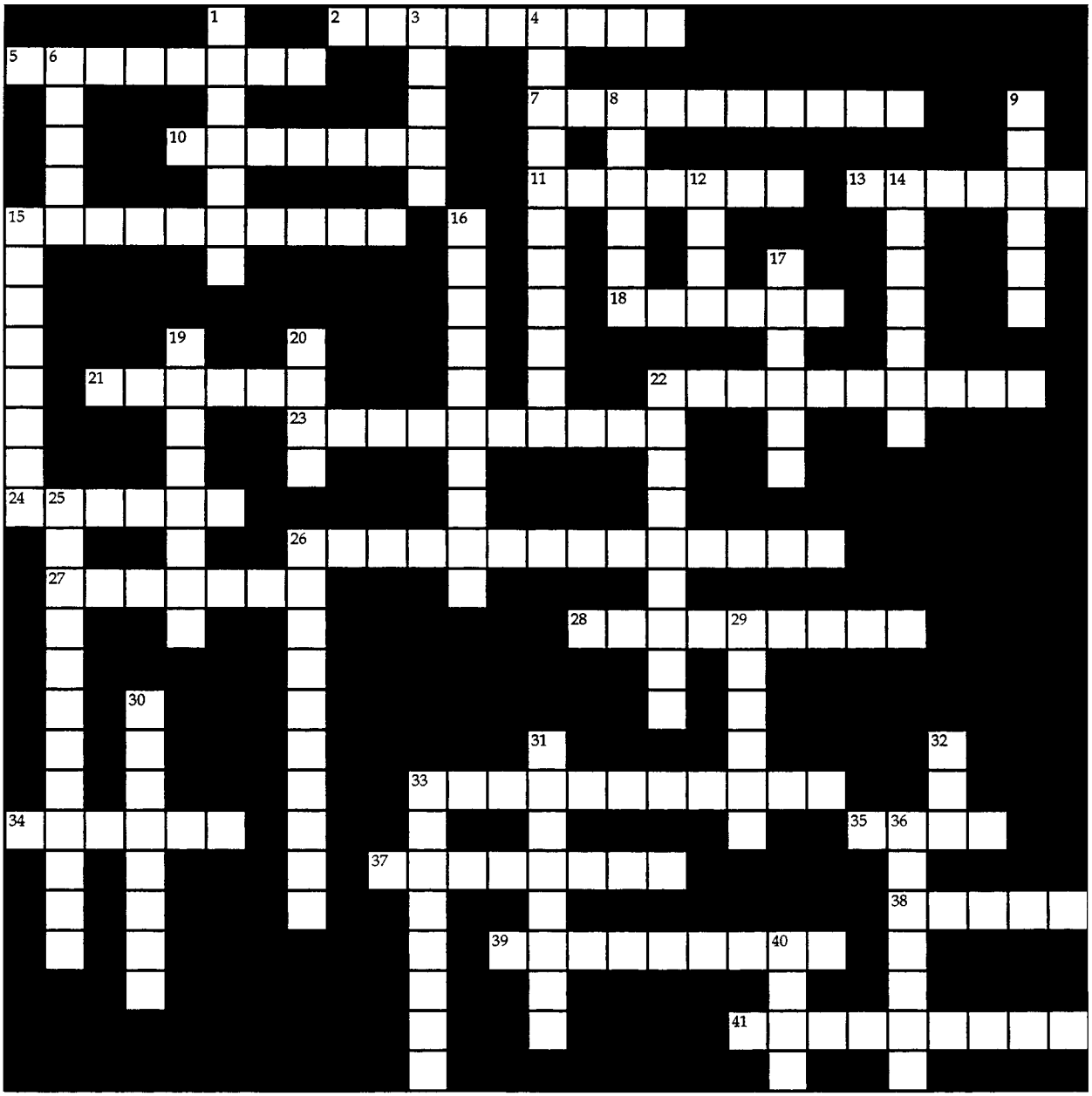
loss of **unconscious** proprioception from regions of the body innervated by spinal segments **T1** and below on the **right**

PROBLEM SOLVING ANSWER C



PROBLEM SOLVING ANSWER D



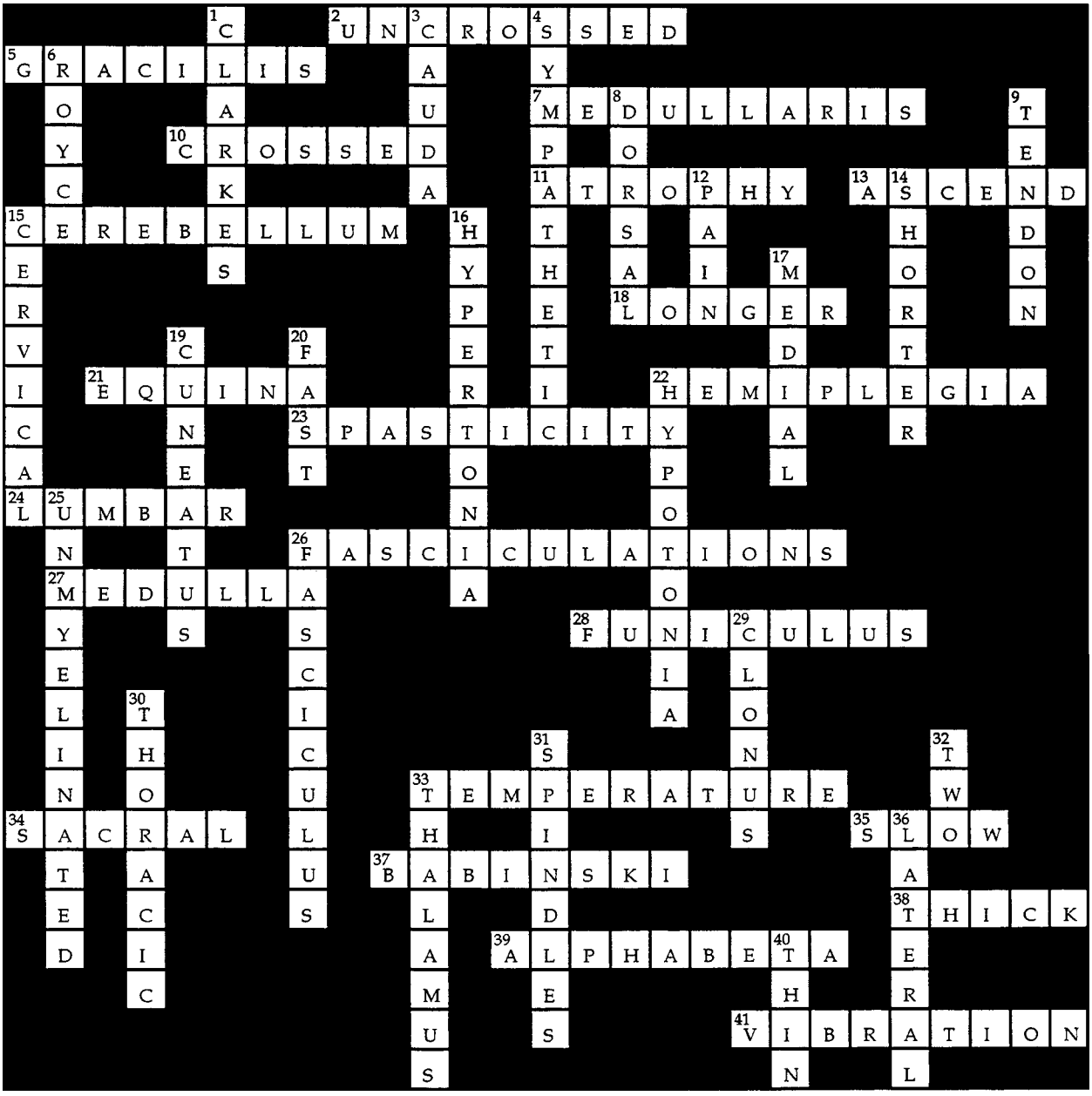


ACROSS

2. laterality(crossed/uncrossed) of dorsal spinocerebellar tract
5. arises from dorsal root ganglia T7 and below
7. conus _____
10. decussate
11. follows lesion of ventral horn
13. what c and delta fibers do before entering the dorsal horn
15. target of cells in Clarke's column
18. relative relationship of vertebral column to spinal cord
21. cauda _____
22. follows interuption of lateral corticospinal tract at C1
23. loss of descending control of gamma efferents
24. fibers lie lateral to thoracics in anterolateral system
26. tiny muscle twitches
27. area of brain targeted by fasc. gracilis and fasc. cuneatus
28. contains fasciculi
33. one type of information carried by anterolateral system
34. fibers lie medial to lumbars in fasciculus gracilis
35. conduction speed of c-fibers
37. follows lesion of the lateral corticospinal tract
38. size of fast conducting fiber
39. fibers associated with conscious proprioception
41. type of information carried in the dorsal column

DOWN

1. cell group that receives Ia, Ib and II information
3. _____equina
4. lateral cell column from approx. T1-L2
6. taught nerve module in Histo.
8. funiculus containing fasciculus gracilis
9. golgi _____ organ
12. lesion at C2 results in contralateral loss
14. relative relationship of spinal cord to vertebral column
15. fibers lie medial to thoracics in lateral corticospinal tract at C2
16. follows lesion of lateral corticospinal tract
17. relative location of lumbar "information" to sacral in the DSCT
19. T6 and above
20. type of pain carried by delta fibers
22. follows a lesion of either the dorsal or ventral root
25. c-fibers
26. part of a funiculus
29. follows a lesion of the lateral corticospinal tract
30. cord region containing lateral horn and small ventral horn
31. muscle _____
32. number of levels pain and temp. fibers ascend in the zone of Lissauer
33. contains nuclei that project to the cerebral cortex (ALS, and nuc. grac. and nuc. cuneatus project here!!) GATEWAY TO THE CORTEX
36. division of dorsal root containing smallest fibers
40. size of fibers that comprise the lateral division of the dorsal root



ANSWERS TO PROBLEM SOLVING QUESTIONS RELATED TO POINTS 5-6

NOTE: The answers to ALL #3s (#4s for points 5 and 6) illustrated on the back side of the question.

Point #5 Ventral Horn

Matching B,D,A,H,E

Point #6 Lateral (Intermediolateral) Horn

Matching B,I,F,H,E,J