NEUROSCIENCE UNIT 1
DETAILED ANSWERS FOR SPINAL CORD PRACTICE QUESTIONS

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SPINAL CORD—DORSAL COLUMNS

DORSAL COLUMNS #1
The shaded area is the right fasciculus gracilis at spinal level C2.
A. FALSE The right fasciculus gracilis terminates in the right nucleus gracilis, not cuneatus.
B. FALSE The right fasciculus gracilis arises from cells in the right dorsal root ganglia (DRG; alpha-betas) from T7 and below (caudal). Cells in the dorsal horn differ from those in the dorsal root ganglia.
C. FALSE The right fasciculus gracilis arises from cells in the right dorsal root ganglia (alpha-betas) from T7 and below (caudal). Fasciculus cuneatus arises from DRG T6 and above (rostral).
D. FALSE The right fasciculus gracilis arises from cells in the right DRG (alpha-betas) from T7 and below (caudal). The pathway travels on the same side as the cells of origin (DRG T7 and below on the right).
E. TRUE

DORSAL COLUMNS #2
The shaded area is the right fasciculus gracilis at spinal level C2.
A. FALSE The right fasciculus gracilis carries two pt. etc from T7 (index finger is C6-C7) and below on the right/ipsilateral side.
B. FALSE The right fasciculus gracilis carries two pt. etc from T7 (index finger is C6-C7) and below on the right.
C. FALSE The right fasciculus gracilis carries two pt. etc from T7 and below on the right/ipsilateral side.
D. FALSE The right fasciculus gracilis carries two pt. etc from T7 and below on the right.
E. TRUE The right fasciculus gracilis carries two pt. etc from T7 and below on the right and the big toe is L5.

DORSAL COLUMNS #3
The shaded area is the fasciculus cuneatus at T1.
A. FALSE the axons at this level arise form DRG on the right from T6-T1.
B. FALSE cells in the dorsal horn do not give rise to axons in the fasciculus cuneatus; such axons arise from DRG.
C. TRUE the right fasciculus cuneatus terminates in the right nucleus cuneatus.
D. FALSE the right fasciculus cuneatus arises from cells in the ipsilateral (right) DRG T6 and above.
E. FALSE with T7 and below, think *gracilis*, not cuneatus.

DORSAL COLUMNS #4
Shaded area is fasciculus cuneatus at T1.
A. FALSE lesions of the fasciculus cuneatus at T1 result in ipsilateral loss of 2 pt. etc from T1-T6.
B. FALSE the shoulder is innervated by dermatomes C4-C5 alpha-beta fibers from these levels have not yet entered the fasciculus cuneatus at level T1.
C. FALSE fasciculus cuneatus does not carry information from the toe, much less the opposite toe!
D. FALSE the index finger is innervated by dermatome C6. The lesion involves fibers from T1-T6 on the right/ipsilateral side.
E. TRUE the upper chest is innervated by T2-T4 and the lesion involves fibers from T1-T6.

DORSAL COLUMNS MATCHING

A. left fasciculus gracilis at L2 (spinal levels L3-S1=dermatomes of the leg).
B/1.
C. the right fasciculus cuneatus
D. the right fasciculus cuneatus at C5 and rostral (C6-C8 are dermatomes of hand)
E/2. (T1/T2 is the dermatome of the elbow)

DORSAL COLUMNS #5

Explanation: Since this is a drawing at C2, fasciculus gracilis contains the central processes of T7 most laterally and coccygeal 1 most medially. Dermatomes of the foot are L4, L5 and S1. Your shading should not include the most medial fibers in fasciculus gracilis nor should it go too far laterally in the fasciculus. The shading should only involve the part of the fasciculus gracilis containing the central processes of L4, L5 and S1. Think about where they lie relative to T7 (most lateral) and Coc1 (most medial).

DORSAL COLUMNS #6

Explanation: The neck is the province of dermatomes C2 and C3. The most lateral fibers in fasciculus cuneatus at C2 should be shaded.

SPINAL CORD—ANTEROLATERAL SYSTEM (ALS)

ALS #1

Shaded areas are right fasciculus gracilis and right ALS at C2.

A. FALSE alpha beta axons in the fasciculus gracilis arise from cells in the ipsilateral DRGs.
B. FALSE the ALS arises from cells in the contra lateral dorsal horn
C. FALSE the ALS is the ALS only after it is in the anterolateral quadrant of the spinal cord. It terminates in the ipsilateral VPL of the thalamus. The cells in the dorsal horn and their axons before crossing are NOT the ALS!
D. FALSE C and D fibers terminate in the dorsal horn on the same side as that which they enter the spinal cord. They do not comprise the ALS or fasciculus gracilis.
E. TRUE

ALS #2

The two shaded pathways are the right ALS and right fasciculus gracilis at spinal level C2.

A. FALSE fasciculus gracilis terminates in the ipsilateral nucleus gracilis and the ALS terminates in the ipsilateral VPL
B. FALSE alpha-beta axons do not cross when entering the spinal cord
C. FALSE fasciculus gracilis is associated with DRG T7 and below
D. FALSE dorsal horn cells project into the opposite ALS
E. TRUE cells in the left dorsal horn project into the right (contra lateral) ALS

ALS #3

The two shaded pathways are the right ALS and right fasciculus gracilis at spinal level C2.

A. FALSE the 2 pt discrimination carried in fasciculus gracilis at C2 comes from spinal levels T7 and below on the ipsilateral side of the body. The index finger is not in the province of fasciculus gracilis. Nothing in the dorsal columns is related to contra lateral side of the body!

B. FALSE the index finger (C6/C7) is not in the province of fasciculus gracilis (T7 and below).

C. TRUE the right ALS at C2 carries pricking (first, delta) information from C4 down on the contra lateral side; this would include the left (contra lateral) big toe.

D. FALSE the ALS pathway in the spinal cord carries burning pain information from the opposite/ contra lateral side of the body.

E. FALSE the ALS in the spinal cord carries cooling (delta) information from the opposite side of the body.

ALS #4

The two shaded pathways are the right ALS and right fasciculus gracilis at spinal level C2.

A. FALSE the right fasciculus gracilis at C2 carries information from the **right** (ipsilateral) hip

B. FALSE the right ALS at spinal level C2 carries first pain from the left (contra lateral) index finger

C. TRUE a lesion of the right ALS at C2 interrupts warming sensation from C4 on down on the left (contra lateral) side; this would include below the knee.

D. FALSE spinal cord pathways carrying conscious proprioception (fasciculi gracilis and cuneatus) are uncrossed.

E. FALSE. fasciculus gracilis carries information from T7 and below; the thumb is innervated by the C6 spinal nerves and lies in the province of fasciculus cuneatus.

ALS #5 (Matching)

A. right ALS at C3 and rostral

B. right fasciculus cuneatus

C. is the right fasciculus cuneatus at C6 and rostral

D/2 the left ALS at C2 contains cooling information from spinal levels C4 and below on the right.

E/1 the right fasciculus gracilis at C2 contains information regarding conscious proprioception from spinal levels T7 and below on the right.

ALS #6

Explanation: A lesion of the right fasciculus cuneatus at spinal level C2 will account for the deficit in conscious proprioception, vibration, and two-point discrimination from spinal segments C2-T6 on the right/ipsilateral side. A lesion of the right ALS at spinal level C2 will account for the deficit in pain and temperature from C4 and below.

SPINAL CORD—DORSAL SPINOCEREBELLAR TRACT (DSCT)

DSCT #1

The shaded pathways are the right fasciculus gracilis, ALS and DSCT at C2.

A. FALSE GTOs are associated with 1as, Ibs, and IIs, not with alpha-betas.
B. FALSE deltas are associated with cooling and first pain, not muscle spindles
C FALSE the right DSCT terminates in the right (ipsi) cerebellum
D. FALSE delta and C fibers arise from cells in the DRGs and terminate in the dorsal horn. The ALS is comprised of the processes of cells in the contra lateral dorsal horn, and these cells are not deltas and Cs.
E. TRUE the right DSCT at C2 arises from cells along the entire length of the ipsilateral/right Clarke's column

DSCT #2

The shaded pathways are the right fasciculus gracilis, ALS and DSCT at C2.

A. FALSE pathway terminates in the contralateral (left) nucleus gracilis in the medulla
B. FALSE pathway arises from cells in the contralateral (left) Clarke's column
C. FALSE pathway terminates in the contralateral (left) VPL
D. **TRUE** the DSCT is not present below L3. There are unconscious proprioceptive (1a, 1b and II) fibers ascending in fasc. gracilis on their way to the most caudal part of Clarke's column (L3), but these fibers are NOT called the DSCT.
E. FALSE pathway arises from cells in the ipsilateral (right) dorsal horn

DSCT #3

The shaded pathways are the right fasciculus gracilis, ALS and DSCT at C2.

A. FALSE unconscious proprioception from the left arm is carried in the left/ipsilateral DSCT
B. FALSE conscious proprioception from the left index finger (C6) is carried in the left/ipsilateral fasciculus cuneatus
C. FALSE pricking pain from the right big toe (L5) is carried in the left/contra lateral ALS
D. **TRUE** burning pain from the left ankle is carried by the right/contra lateral ALS
E. FALSE unconscious proprioception from the left big toe is carried by the left/ipsilateral DSCT

DSCT #4

Explanation: Deficit in conscious proprioception, vibration, and two-point discrimination from spinal segments C2-T6 on the right: Lesion in right fasciculus cuneatus at spinal level C2. Deficit in pain and temperature from the left side of the entire body (below the neck): Lesion of right ALS at C2. Deficit in unconscious proprioception from the entire right side of the body: Lesion of right DSCT at C2 results in right sided loss of unconscious proprioception C8 and below. Lesion of right fasciculus cuneatus at C2 interrupts ascending 1a, 1b and II fibers associated with C2-C7 that are destined for the accessory cuneate nucleus in the medulla. These two lesions together result in loss of unconscious proprioception from C2 and below.

DSCT MATCHING

A/3. lesion of left DSCT at T6=ipsi loss of unconscious proprioception T6 and below on left
B/1. right fasciculus gracilis arises from DRGs T7 and below on right
C. involves left fasciculus cuneatus
D. involves right ALS
E. has to involve right fasciculus cuneatus (elbow is T1/T2 dermatome)
F. no such pathway exists; the zone of Lissauer contains central processes of C and delta fibers, but the ALS is comprised axons of dorsal horn cells from the opposite side.
G/2. true even though the lesion in the ALS would result in loss of other parts of the body too!

SPINAL CORD—LATERAL CORTICOSPINAL TRACT (LCST)

LCST #1

The shaded pathways are the right fasciculus cuneatus, right ALS, and right LCST at spinal level T6.

A. FALSE the stippled/shaded pathways are the right fasciculus gracilis, (and a little fasciculus cuneatus), ALS, and LCST at spinal level T6. The right LCST arises from cells in the left/contra lateral motor cortex.
B. **TRUE** the right LCST terminates on ventral horn cells on the right/ipsilateral side of the spinal cord.
C. FALSE the right ALS terminates in the right (ipsilateral to ALS) VPL
D. FALSE the DSCT is not shaded.
E. FALSE

LCST #2

The shaded pathways are the right fasciculus cuneatus, fasciculus gracilis, right ALS, and right LCST at spinal level T6.

A. FALSE the right LCST lesion occurs at T6 and thus there would be no hemiplegia. In addition, the lesion would result in a **right**/ipsilateral problem.
B. FALSE there would be a **right**/ipsilateral Babinski.
C. FALSE the right ALS lesion would result in left/contra lateral problems.
D. the right LCST lesion at T6 results in spasticity on the right/ipsilateral side below the chest (i.e., would not include the arms)
E. TRUE an extensor plantar response (Babinski) of the right big toe would result from the lesion of the right LCST at T6.

LCST #3

Explanation: The shading shown involves the axons that would normally innervate the spinal levels related to the right arm and leg. The most medial fibers in the LCST are not shaded as they exit to innervate spinal levels rostral to the arm innervation. The deficit is fast pain from the left foot is accounted for by the lesion of the right ALS at C2 that damages the more lateral fibers.

LCST MATCHING

A/3. lesion of left DSCT at T6 would result in loss of uncon. proprio from left leg
B. defines left fasciculus gracilis
C/1. right fasciculus gracilis at C1 would carry vibration from right big toe
D/2. lesion of left ALS at C1 will result in loss of cooling from right foot
E. is the left fasciculus cuneatus rostral/above spinal level T2 (dermatome of elbow=T2)
F. is the right fasciculus cuneatus rostral to C5 (dermatomes of arm=C5 and T2)
G. is the right ALS above/rostral to C6 (dermatomes of hand=C6-C8)
H. is the right LCST above L1
I/4. left LCST begins in right motor cortex

SPINAL CORD—VENTRAL HORN

VENTRAL HORN #1

The shaded pathways are at spinal level T1 and include the right fasciculus gracilis and ALS and the shaded cell group is the ventral horn.

A. FALSE the ventral horn cells project to the ipsilateral (right) muscles.
B. FALSE the right fasciculus gracilis terminates in the right (ipsilateral) nucleus gracilis.
C. FALSE the right fasciculus gracilis arises from cells in the ipsilateral/right DRG from T7 and below.
D. FALSE the DSCT is not shaded
E. TRUE

VENTRAL HORN #2

The shaded pathways are at spinal level T1 and include the right fasciculus cuneatus, right ALS, and right DSCT, and the shaded cell group is the ventral horn.

A. TRUE Cells in the ventral horn at T1 project to ipsilateral (right) intrinsic hand muscles.
B. FALSE the right LCST is not shaded. Lesion of ventral horn cells=flaccidity and atrophy.
C. FALSE a lesion of the right ALS results in loss of pricking (first, delta) pain from the left/contra lateral big toe
D. FALSE the LCST is not shaded on either side.
E. FALSE a lesion of the right DSCT at T1 will not influence unconscious proprioception entering the spinal cord further rostrally via 1as, 1bs and IIs in the DRG at C4

VENTRAL HORN #3

Explanation: The spinal level shown is T1. The problem in pricking pain from the right foot can be accounted for by a lesion of peripherally located fibers in the left ALS at T1. The “foot” lies within the province of dermatomes L4, L5 and S1. Remember, in the ALS the peripheral fibers carry information from more caudal segments than more centrally coursing fibers in the ALS. So, the lesion should be peripherally placed but should not include the most peripheral fibers (carrying information from levels caudal to S1). The atrophy of the left intrinsic hand muscles can be accounted for by a lesion in the left ventral horn at T1.

VENTRAL HORN MATCHING
A/3. is the left DSCT above/rostral to approximately L3
B/1. left fasciculus gracilis
C. is the right fasciculus gracilis
D/2. left ALS at and above/rostral approximately spinal level L2 (“foot”=L4-S1).
E/5. left fasciculus cuneatus at and rostral to T2
F. is the right DSCT
G. is the right ALS at and rostral to C6 (hand=C6-C8
H/4. left ventral horn at T1
I. left LCST

SPINAL CORD—LATERAL HORN

LATERAL HORN #1
The shaded areas are the right fasciculus gracilis, ALS, ventral horn and lateral horn.
A. FALSE preganglionic sympathetic neurons in the lateral horn at T1 terminate in the ipsilateral/ right SCG (superior cervical ganglion)
B. FALSE preganglionic sympathetic neurons in the lateral horn at T1 terminate in the ipsilateral/ right SCG; they do NOT go directly to the dilator. This muscle is innervated by cells in the SCG, not lateral horn.
C. TRUE preganglionic sympathetic neurons in the lateral horn at T1 are targeted by axons arising from the ipsilateral/right hypothalamus
D. FALSE the axons in the right fasciculus gracilis arise from DRGs on the right/ipsilateral side of the spinal cord.
E. FALSE the axons comprising the left ALS arise from cells in the left/contralateral dorsal horn.

LATERAL HORN #2
The right fasciculus gracilis, ALS, ventral horn and lateral horn are shaded.
A. FALSE the ventral horn on the right is shot and thus there is atrophy of the intrinsic hand muscles. They can’t be spastic if they are dead!!!!
B. FALSE the lateral horn on the right is shot. The right/ipsilateral pupil would be dilated.
C. FALSE the lateral horn on the right is shot and thus the right/ipsilateral side of the face is flushed. The loss of sympathetic innervation to cutaneous blood vessels of the face causes vasodilation. The face also would be dry as the sympathetic innervation stimulates sweat glands.
D. FALSE lesion of the right ALS at T1=loss of sense of warming on left from T3 and below.
E. TRUE

LATERAL HORN #3
Explanation: The spinal level shown is T1. A lesion of the right ventral horn will account for the atrophy of the intrinsic hand muscles. Damage to the right lateral horn will result in the miosis (constriction) and ptosis. A lesion of the more peripheral fibers in the right ALS will result in a deficit in burning pain (C fiber) in the left/contra lateral leg.

LATERAL HORN MATCHING

A. is the right DSCT at or above L3
B/1. left fasciculus gracilis at and above T7
C. is the right fasciculus gracilis at and above L5
D. is the left ALS at and above L4 (foot=L4-S1)
E/5. left fasciculus cuneatus at and above T2 (elbow)
F/3. left Clarke's nucleus (C8-L3)=cells origin of left DSCT (present from L3 on up).
G. is the right ALS at and above C6 (hand=C6-C8)
H/4. left ventral horn at T1 innervates ipsi intrinsic hand muscles
I/2. left LCST
J/6. none are related to poor lateral horn!

SPINAL CORD—BRINGING IT ALL TOGETHER

BRINGING IT ALL TOGETHER #1

The pathways involved are the fasciculus gracilis, fasciculus cuneatus, zone of Lissauer, DSCT, ALS, LCST and descending autonomics; the ventral and lateral horns are the cell groups involved. All lesions are on the right.

A. FALSE the DSCT arises from the ipsilateral/right Clarke's column
B. FALSE the LCST fibers on the right arise from the left/left motor cortex.
C. FALSE nothing from dorsal roots on the left are ascending on the right. Axons in the zone of Lissauer arise from C and delta fibers in ipsilateral DRGs.
D. FALSE come on!!! Only preganglionic sympathetics arise from the lateral horn at T1. The postganglionic sympathetics coming from the SCG are the fibers that end on/in the vessels.
E. TRUE the descending autonomics from the hypothalamus that are headed for T1-L2 travel medial to the LCST and are uncrossed.

BRINGING IT ALL TOGETHER #2

The pathways involved are the fasciculus gracilis, fasciculus cuneatus, zone of Lissauer, DSCT, ALS, LCST and descending autonomics; the ventral and lateral horns are the cell groups involved. All lesions are on the right.

A. TRUE anesthesia means **no** sensation. The interruption of fibers in the zone of Lissauer on the right means no pain and temp from T1-T3. Remember, the C and delta fibers from T1 are entering here at T1 and are cut. The ascending C and delta fibers from T2 are ascending to get to C8 and are kaput!!! Same thing for the ascending fibers from T3 that are headed for the dorsal horn at T1. When you add this pain and temp loss at T1-T3 to the loss of 2 pt etc at the same levels from interruption of fasciculus cuneatus, you have no sensation at these three levels.
B. FALSE analgesia means absence of pain. There would be no pain from T1-T3 on the right due to the lesion of the zone of Lissauer. In addition, there is a loss of pain and temperature from T3 and below on the left/contra lateral side.
C. FALSE interruption of the descending autonomics just medial to the LCST at T1 and/or the cells in the lateral horn at T1 means that the preganglionic sympathetics have lost an important "drive" from the hypothalamus. This results in the parasympathetics to the pupil dominating and thus a constricted pupil.
D. FALSE-interruption of the descending autonomics at T1 means that not only is sweating from the face lost, but also the preganglionic sympathetic neurons below T1 have lost their drive. This means that there would be a lack of sweating below the head, too.
E. FALSE see A.

BRINGING IT ALL TOGETHER #3

1. spasticity of the **left**: **TRUE**—left LCST
2. flushed on the **left** side of the face: **TRUE**—damage to cells in left lateral horn or left descending autonomics
3. lack of sweating on **left** side of face: **TRUE**—same as #2
4. lack of sweating on **left** side of body: **TRUE**—damage to desc. autonomics
5. miosis of **left** pupil: **TRUE**—same as #2
6. ptosis of **left** eyelid: **TRUE**—same as #2
7. spasticity of **left** intrinsic muscles of the hand: FALSE—can't have spastic muscles if they are dead or dying (are flaccid, atonic)
8. spasticity of **left** biceps: **FALSE**—left LCST at T1 is below level of lower motor neurons that innervate biceps (C5).
9. atrophy of **left** toe muscles: **FALSE**—ventral horn is damaged only at T1. UMN lesions=no atrophy
10. loss of pain and temp from dermatome innervated by **left** dorsal root T3 (forget about dermatome overlap): **TRUE**—the c and d fibers of T3 are ascending in the zone of Lissauer (or have entered the dorsal horn) at T1.
11. bilateral loss of crude touch at T3: **TRUE—**crude touch is carried by c and d fibers and the ALS. Such information from spinal level T3 *on the left* is lost due to interruption of the ascending fibers from T3 traveling in the zone of Lissauer or as they synapse in the left dorsal horn. Of course, the cells in the left dorsal horn receiving this input are also dead. The lesion of the left ALS results in loss of crude touch from T3 on down *on the right.*
12. loss of tone in **left** intrinsic muscles of the hand muscles: **TRUE**—if the muscles are dead or dying, there is no tone via the stretch reflex.
13. spasticity of **left** shoulder muscles: **FALSE**—the damaged LCST lesion at T1 is below the spinal level(s) containing the lower motor neurons that innervate the shoulder (C4-C5).
14. loss of vibration sense from the **left** toe: **TRUE**—left fasciculus gracilis.
15. right Babinski: **FALSE**—it would be a left Babinski because LCST problems are ipsi.
16. loss of **unconscious** proprioception from the region of the body innervated by the ipsilateral dorsal root L4 (remember, Clarke's column ends at L3): **TRUE**—the left DSCT at T1 contains unconscious proprio. from T1 on down. Clarke's column at L3 "serves" L3 and below.
17. **increased** muscle stretch reflex in the **left** quadriceps upon tapping the patellar tendon: **TRUE**—hyper-reflexia is a cardinal UMN sign. The LMNs in the ventral horn that innervate the quadriceps (L3-L4) lie below the damaged LCST at T1.
18. some of Clarke's column on the **left** is involved: **TRUE**—Clarke's column runs from C8-L3.

BRINGING IT ALL TOGETHER #4

Explanation: A lesion of the zone of Lissauer on the right will interrupt ascending c and d fibers from T3 and T2. The lesion will also interrupt the incoming fiber of T1. There is **NO WAY** to account for these deficits on the right via a lesion of the left ALS. The best you can do with an ALS lesion is to have T3 on the right involved!

BRINGING IT ALL TOGETHER #5

Explanation: Interruption of the descending autonomics on the right at C8, (which lie just medial to the LCST), will mean that preganglionic the sympathetic neurons in the lateral horn (T1-L2) will not receive the important drive from the ipsilateral hypothalamus.

BRINGING IT ALL TOGETHER #6
Explanation: This is easy. The lesion is in the right ALS (at T1).

BRINGING IT ALL TOGETHER #7
Explanation: This is easy too! The lesion is in the right DSCT at T1.

SPINAL CORD—SOMATOTOPIC ORGANIZATION

SOMATOTOPIC ORGANIZATION #1
The pathways involved at C2 are the more medial fibers in fasciculus gracilis, the most lateral fibers in the DSCT, fibers in the lateral one-half of the LCST and fibers in the ventrolateral periphery of the ALS.
A. FALSE the axons carrying pain and temp from the left arm are more medially located than those shaded/stippled
B. FALSE 2 pt etc from the right arm travels in the right fasciculus cuneatus, not gracilis.
C. FALSE LCST fibers destined for the ventral horn at C8 would not be so laterally placed in the right LCST
D. FALSE unconscious proprioception from T1 on the right travels more medially in the right DSCT than those that in the shaded/stippled area.
E. TRUE

SOMATOTOPIC ORGANIZATION #2
The shaded axons involved at T1 on the right are in the lateral most part of fasciculus gracilis, the most medial part of the DSCT and LCST and the entire ALS.
A. FALSE the right ALS at T1 contains axons that have arisen from dorsal horn cells in the left dorsal horn from T1 on down. Think about that cell in the left dorsal horn at T1 and how its axon crosses and travels medially in the right ALS. That axon carries the most rostral information about pain and temp that is in the ALS at T1. The axon from the left dorsal horn cell at C7 is not in the right ALS at spinal level T1.
B. FALSE fibers carrying vibration from the right foot travel more medially in the fasciculus gracilis than those stippled/shaded.
C. TRUE the medially shaded LCST fibers would be exiting soon and would innervated ventral horn cells at T2.
D. FALSE deficit always starts **TWO LEVELS BELOW THE ALS** lesion if the zone of Lissauer is not involved
E. FALSE the shaded LCST fibers do not include the more laterally placed fibers destined for the ventral horn at lumbosacral levels of the spinal cord.

THE MOST ASKED ABOUT OF THEM ALL!!!

SOMATOTOPIC ORGANIZATION #3
The shaded areas are the medial part of fasciculus gracilis, the lateral part of the LCST and the ALS all on the right at spinal level L5. The DSCT is not present at this level because we are below Clark's column (C8-L3).

A. TRUE There are axons in the left ALS at L5 that have their cell bodies in the right dorsal horn at S1. Do this simple exercise: Draw a cell in the left dorsal horn at L5. Now, draw its axon as it crosses and enters the right ALS to lie in its medial part. Now, answer this simple question. "Is there an axon in the right ALS at L5 whose cell body lies in the contra lateral/left dorsal horn at L5." Please say YES!!! Now, do the same thing at S1 (yes, draw the cell in the left dorsal horn and then its crossing axon) Now, answer the question, "is there an axon in the right ALS at S1 whose cell body lies in the left/left dorsal horn at S1. Please say YES. If you said YES, where is this axon at L5? Please say it is in the right ALS!!!
B. FALSE the shaded fibers in the LCST at L5 are destined for more caudal levels and thus their interruption would not affect S1 LMNs
C. FALSE same as B
D. Ia, II or Ib fibers carrying unconscious proprio. from L5 would enter fasciculus gracilis and be positioned at the lateral most part of the fasciculus (last one in is most lateral as you ascend). The shaded axons in fasciculus gracilis are from levels more caudal than L5.
E. FALSE

SOMATOTOPIC ORGANIZATION #4 (Matching)
A. lesion results in muscle atrophy (**ventral horn or root**)
B. primary source of information about how fast a muscle is changing length (**1a fiber**)
C. pathway carries pain and temperature and terminates within the ipsilateral thalamus (**ALS**)
D. axons carry primarily information regarding the constant length of muscle (**II fiber**)
E. pathway arises from Clarke's column (**DSCT**)
F. axons convey information about fast pain and cooling (**d/delta fiber**)
G. smallest and slowest conducting axon in the dorsal root (conveys info. about warming) (**c fiber**)
H. axon conveys information about vibration (**alpha-beta fiber**)
I. axon conveys info. about muscle tension (**Ib**)
J. axon carries info. about pain and temperature from regions of the body innervated by contra lateral dorsal root at T3 **(The axon labeled T1 in the right ALS means that it arose from the contra lateral dorsal horn at T1. Such an axon is carrying information from contra lateral T3 dermatome. This has caused confusion in the past, so don't confuse the dorsal horn origin of a fiber and the information it carries. Separate the dorsal *horn* and the dorsal *root* in your mind. Think about the fact that there is NO axon in the ALS at T1 that carries pain and temp. information from the part of the body innervated by contra lateral dorsal root of T1).**

SOMATOTOPIC ORGANIZATION #5
A=DRG of c/d fiber(s); B=ALS axon; C=VPL neuron; D=cell in somatosensory cortex; E=dorsal horn cell
1. FALSE the cells of origin of the ALS (**B**) lie in the contra lateral **dorsal horn**, not the DRG. The ALS is in the ANTEROLATERAL quadrant of the spinal cord white matter. The axon of the DRG or dorsal horn cell before entering the ALS is **NOT** the ALS.
2. FALSE the peripheral process of **A** supplies the dermatome of that spinal level.
3. FALSE the ALS terminates in VPL nucleus of the thalamus
4. FALSE VPL receives input from the contra lateral dorsal horn
5. FALSE **D** is in the cortex
6. FALSE **E** lies in the dorsal horn
7. FALSE **E** receives information about pain and temperature
8. FALSE **B**, the ALS, lies in the anterolateral quadrant of the spinal cord
9. TRUE
10. FALSE the ALS does not go straight to the cortex; it has to first go through the gateway to the cortex (the thalamus) via its VPL nucleus

SOMATOTOPIC ORGANIZATION #6

A=corticospinal neuron in the motor cortex; B=LCST; C=ventral horn cell; D=cortical axon before (rostral to) pyramidal decussation.

1. FALSE this is an UMN lesion. You need a LMN problem for atrophy.
2. FALSE any lesion of an UMN rostral to the decussation leads to contra lateral spasticity
3. FALSE a dead muscle can't be spastic. It's dead!! How can there be "**a velocity-dependent increase in the resistance of muscles to a passive stretch stimulus**"?
4. FALSE a Babinski sign is indicative of UMN problems. The descending signals from the LCST are lost and the normal flexion of the big toe becomes extension. If there is a LMN lesion, the screwed up reflex circuitry is meaningless; the muscle won't move and would atrophy!
5. FALSE the LCST travels in the lateral funiculus!
6. FALSE remember, the muscle has to be alive to be spastic!
7. FALSE UMN lesion=Babinski=up-going toe.
8. **TRUE** injured axon is contra lateral to the muscle
9. FALSE injured axon is ipsilateral to muscle
10. FALSE axon is not in the lateral funiculus of the spinal cord yet.

SOMATOTOPIC ORGANIZATION #7

Illustrates the dorsal column/medial lemniscal pathway A: alpha-beta DRG; B: its central process in the dorsal column (gracilis *or* cuneatus); C: is the nucleus in the medulla (nucleus gracilis or cuneatus); D: VPL of the thalamus; E: somatosensory cortex.

1. FALSE **A** is a DRG.
2. FALSE **E** receives information about 2 pt. etc.
3. FALSE **C** is either nucleus gracilis or cuneatus, both of which are in the caudal medulla.
4. FALSE **B** is traveling in the dorsal funiculus/column.
5. FALSE **D** is in the thalamus.
6. FALSE **C** lies in the caudal medulla.
7. FALSE **A** carries information about 2 pt. etc (including *conscious* proprioception).
8. FALSE **B** carries information about 2 pt. etc.
9. FALSE **C** carries information about 2 pt. etc.
10. **TRUE B** is the central process of an alpha-beta DRG.

SOMATOTOPIC ORGANIZATION #8

This schematic illustrates the DSCT. D represents Ia, Ib and II fibers of a DRG (unconscious proprio.); C is a cell in Clarke's column (C8-L3); B the DSCT and A a cell in the cerebellum.

1. FALSE **B** travels in the lateral funiculus, lateral to the LCST.
2. FALSE lesion of **A** results in ipsilateral motor incoordination.
3. FALSE D carries information about unconscious proprioception.
4. FALSE D represents Ia, Ib, and II fibers.
5. FALSE Clark's column is present only from C8-L3.
6. FALSE via inferior cerebellar peduncle (also called restiform body!)
7. FALSE Clarke's, not Royce's.
8. **TRUE** compare with preganglionic sympathetics, which are located from T1-L2 ("sandwiched" by the levels containing a Clark's nucleus).
9. FALSE nope, it is the good ol' DSCT.
10. FALSE nope, it is the good ol' DSCT.

SPINAL CORD—WEAKNESS

WEAKNESS #1.

A. FALSE the SNAP is the sum of all the action potentials generated in sensory nerve fibers by an electrical shock. For instance, you can stimulate the medial nerve by placing a stimulating electrode on the skin over the antecubital fossa. You then record the SNAP at the index finger. The nerve fibers in the median nerve at the elbow (where they were stimulated!) that run out along the index finger are purely sensory. Remember, the cell bodies of these sensory fibers lie in DRGs, and when you stimulate near the antecubital fossa the impulses can go either toward or away from the DRG cell bodies. The SNAP recorded at the finger is in micro volts (μV; CMAPS are in millivolts; mV) and of course the latency can be recorded (in ms). The SNAP is about 1000 times smaller than the CMAP, making sensory nerve conduction technically more difficult to measure. A lesion of the LMNs in the ventral horn of the spinal cord causes no change in sensory axons, whose cell bodies lie in DRGs.

B. FALSE the electrical activity of the action potentials does not depend upon myelin but rather on the nerve axon. The **potential difference** across the neuron cell membrane (not myelin) is the basis for generating electrical signals.

C FALSE

D. TRUE in a myopathy (muscle disease) the sensory nerves are OK, and thus the SNAPs are normal.

E. FALSE

WEAKNESS #2.

A. TRUE the muscle fibers (MF) innervated by a ventral horn cell and its axon are termed the motor unit (MU). The MU is the basic functional element of a skeletal muscle. Conversely, a muscle may be considered a grouping of MUs. While one MF is supplied or innervated by one MN (motor neuron), the reverse is obviously not true. One MN may supply from 6-10 MFs in extraocular muscles to hundreds of MFs in a large proximal limb muscle such as the biceps brachii. The result is a remarkable expansion from controlling unit (MN) to the endpoint of its apparatus (i.e., MFs), in both anatomic and physiologic terms. In addition to the function of the MU in motor control, the MN also has a trophic effect in maintaining the integrity of the MFs at its endplate. The signature electrical signal generated by a MU is termed the motor unit action potential (MUP). That is, when the MN discharges, all of its MFs respond by generating an action potential (AP) and collectively, these single MF APs summate to form the MUP.

In a low-power histological cross-section of muscle containing approximately 100 MFs, approximately 20-25 MUs are represented, with approximately 1-5 MFs per MU. You know from your histology lectures that muscle fibers can be stained and that type 1 and 2 differ in their appearance (dark versus light) depending upon the stain and pH. Thus, if a muscle contains both types of fibers (type 1 slow twitch; type 2 fast twitch), the histological section of muscle looks like a checkerboard, with dark fibers mixed with light (the dark can be type 1 or 2, depending on the stain and pH). While an MU is homogeneous for the histochemical type of MF it contains, those MFs belonging to individual MUs still cannot be discerned in histological sections. For example, if we say that the dark MFs are type 1s, then the dark ones belong to more than one MU.

Soooooo, back to type grouping/group atrophy and anterior horn disease. In a ventral horn disease like amyotropic lateral sclerosis (ALS), the process is characterized by ongoing MU loss with compensatory re-innervation (a neurogenic disorder). In this chronic process, compensatory re-innervation, type grouping, can occur in step with MU loss. Therefore, patients do not appreciate

any early change in strength. This compensatory re-innervation process is so successful that as many as half of the MUs may be lost before the re-innervation mechanism begins to fail, i.e., group atrophy, and clinical weakness becomes manifest. EMG studies in such processes can show MUPs that may reach sizes approximately 20 times normal, implying an extraordinary ability to re-innervate and add MFs to a MU. An interesting fact (not in the course book) is that as a "normal" MU reinnervates additional MFs, its new collateral and their end plates are not 100% normal. Thus, it will take longer to turn on all of the MFs in the new/enlarged MUPs and the duration of the neurogenic MUP is increased.
B. FALSE-in NMJ disease the LMNs and their axons are OK and you don't get type grouping and group atrophy
C. FALSE in a myopathy (disease of muscle!!) the MFs die, so there is no reinnervation of MFs and thus no type grouping/group atrophy as in a neuropathy
D. FALSE myasthenia gravis involves damage to the NMJ, which does not damage LMNs or axons.
E. FALSE myelin can die with the axon (axis cylinder) being OK

WEAKNESS #3.
A. FALSE no muscle is dying and therefore there is no increase in CK
B. FALSE there are no LMNs or axons dying
C. TRUE demyelinating neuropathies affect multiple focal areas of every nerve and you have normal segments in between. Thus, you do not find "normal" and "abnormal fibers;" all the fibers are affected to some degree. In axonal neuropathies, some fibers are affected, others not. The normal fibers conduct normally and thus you do not see significant slowing of nerve conduction velocities, but you see a drop in the SNAP and CMAP amplitudes.
D. FALSE no LMNs or axons are dying
E. FALSE CMAP amplitude decreases when motor neuron axons die or when there is a myopathy present.

WEAKNESS #4.
A. FALSE if the axons are dying, their action potentials (APs) are affected. Thus, the amplitude of the combination of all the APs in the nerve, the SNAPs and CMAPs, decrease. The dying axons in the nerve no longer turn on the MFs innervated by that axon. This, of course, assumes group atrophy.
B. TRUE the normal axons will camouflage the dying ones
C. FALSE the MUPs will be larger (neurogenic) due to group typing (larger motor units). MUPs are smaller in muscle diseases (myopathies).
D. FALSE decremental CMAPs are seen only in NMJ problems such as myasthenia gravis
E. FALSE type grouping and group atrophy are classic signs of axonal loss and subsequent reinnervation by living axons due to either ventral horn or peripheral nerve problems.

WEAKNESS #5.
A. FALSE myasthenia gravis (MG) is an acquired autoimmune disorder characterized clinically by weakness of skeletal muscles and fatigability on exertion. Thomas Willis reported the first clinical description in 1672. As you know, the presynaptic terminal contains vesicles filled with acetylcholine (ACh). Upon arrival of a nerve action potential, the contents of these vesicles are released into the synaptic cleft in a calcium-dependent manner. The released ACh molecules diffuse across the synapse and bind to the Ach receptors (AchRs) on the postsynaptic membrane of the muscle cell. Such binding of transmitter with receptor results in the opening of ion channels, the influx of so-

dium, and a tiny depolarization of the muscle membrane. The change in end plate potential (EPP) of the muscle fiber membrane resulting from the release of a single vesicle of Ach is about 1 mV, and is called a miniature end plate potential (MEPP). The EPP resulting from the summation of MEPPs generated from the release of a quanta from a single nerve terminal depolarization is about 40 mV, or 25 mV above the threshold EPP required for the generation of a propagated MF AP. This overshoot is called the **safety factor** of NMJ transmission. At the slow rates of 2-5 Hz repetitive nerve stimulation, the safety margin can be reduced to 5 to 10 mV, due to the depletion of the immediately releasable pool of Ach vesicles at the nerve terminal. So, how is this diatribe related to myasthenia gravis and a decremental CMAP? Well, remember, there are fewer healthy AchRs on the MFs and, in fact, some MFs won't be turned on at all when testing the CMAP (too far gone!). So what happens is that the CMAP resulting from the first stimulation will be relatively normal. Then, the second stimulation/CMAP will be smaller. This is the result of MFs that fired on the first stimulus not firing on the second. Why? Well, consider a muscle fiber that has lost some of its AchRs. It fired an AP during the first stimulus because there was plenty of Ach around to bind to a reduced number of receptors and the MF fired was just able to fire an AP. However, on the second stimulation of the nerve, there is less Ach around and this combined with fewer receptors means no MF AP. That is, a point is reached during slow repetitive stimulation where insufficient numbers if AchR ion channels are opened to produce a threshold EPP.
B. FALSE MUPs are usually normal as there has been no neurogenic reinnervation (increase in MUPs) or death of MFs (decrease in MUPs, myopathies).
C. FALSE SNAP amplitudes are normal since the sensory axons are normal
D. TRUE see A
E. FALSE see B

WEAKNESS #6.
A. FALSE fatigue is a key finding. MG is characterized by fluctuating weakness increased by exertion. Weakness increases during the day and improves with rest.
B. FALSE large calves are seen because there is replacement of muscle by fat and connective tissue (pseudohypertrophy). However, the weakness is in **proximal** muscles.
C. FALSE there is a reduction in MFs, so how could the CMAP be normal? They are decreased!
D. FALSE there is a reduction in MFs, so how could the number of MFs in a motor unit increase? They are decreased!
E. TRUE myotonia is a neuromuscular disorder characterized by the slow relaxation of the muscles. Symptoms may include muscle stiffness and hypertrophy (enlargement). The disorder is caused by a genetic mutation involving the chloride channel of the muscles. The muscle stiffness, which particularly occurs in the leg muscles, may be enhanced by cold and inactivity, and is often relieved by exercise

WEAKNESS #7.
A. FALSE there is usually not a breakdown of the muscle cell membranes in NMJ disease. Because most of the CK in the body normally exists in muscle, a rise in the amount of CK in the blood indicates that muscle damage has occurred or is occurring.
B. FALSE decremental CMAPS is a classical finding in myasthenia gravis (NMJ disease)
C. TRUE and type II are fast conducting.
D. FALSE type grouping is associated with LMN and axonal death and reinnervation.
E. FALSE inflammation is a hallmark of myopathy.

WEAKNESS #8.

A. FALSE increased tendon reflexes are seen only in lesions of the CNS, especially UMNs. Lesions of nerves result in decreased tendon reflexes either because the sensory input is lost (think of the stretch reflex) or the motor output to the muscle is interrupted.

Read on if interested—A neuropathy is a condition in which the peripheral nerves are damaged or not working correctly. There are hundreds of different types of neuropathies and many different ways to categorize them. A polyneuropathy is a neuropathy pattern, whereby the nerve damage initially starts in both feet and may progress to involve the feet, calves, and fingers/hands. Another word for this pattern is a "Glove and Stocking Neuropathy." Many patients with polyneuropathy may not even have any symptoms; in this case, the diagnosis is made by a physical examination or EMG and nerve conduction velocity test (NCV). Some patients with polyneuropathy have only numbness, "tingling," and/or "pins and needles"(paraesthesia). Less often, some unlucky patients with polyneuropathy experience pain. There are many causes of painful polyneuropathy. The most common cause is diabetes, both Type 1 and Type 2. Other causes include old age, certain drugs (such as some chemotherapy drugs), alcohol abuse, AIDS, environmental toxins, and inherited neurological neuropathies. However, in up to one-third of patients with painful polyneuropathy, no underlying cause can be found. Importantly, the chance of obtaining pain relief with proper treatment is the same for patients with or without a known etiology. The actual injury to the nerves may result from several different problems. Possible injuries include: 1) not enough blood supply to the nerves, resulting in loss of oxygen and other needed nutrients to the nerve and thus damage to the nerve, and 2) abnormal function of the nerve itself, such that the nutrients within the nerve are not properly metabolized. In any individual patient, it is not possible to find out which type of problem exists.

B. FALSE some polyneuropathies affect sensory fibers, some affect motor and some affect both (sensorimotor). Moreover, some affect axon cylinders, some myelin and some both. For example, alcohol affects both sensory and motor fibers, as does diabetes (which also affects autonomic fibers). In **Guillain Barre syndrome**, there usually is a deficit in the myelin so that nerve conduction velocities are affected, resulting in eventual conduction block and flaccid paralysis. Motor fibers are affected more than sensory.

C. TRUE glove and stock is classic polyneuropathy!!! Remember this for life!!!! Read on if interested!! The symptoms of polyneuropathy start in the toes and feet (right and left). In some patients the symptoms gradually rise up the calves and into the knees. This is called a "stocking pattern." Then, in some the symptoms may also begin in the fingers and hands — causing a "**glove and stocking**" pattern." It cannot be predicted how any one patient's symptoms will spread. In some patients, the pain does not spread beyond the toes or feet; in others, the progression to calves and hands occurs in months; and yet in others the spread is very gradual, over many years.

Patients who develop pain with polyneuropathy describe the pain using a variety of words, including "burning," "raw skin," "skin sensitivity," "sharp," "electric-like," "deep ache," "freezing cold," "like walking on ground glass," "itchy," and others. Some patients say they don't have pain but have unpleasant and irritating sensations, which may include "buzzing," "like bugs crawling," and "aching." Some patients have constant pains, day and night, whereas others only have noticeable pain at bedtime. Often, patients may complain that the pain interferes with their sleep. Some patients with polyneuropathy may have difficulty feeling things with their feet or hands. Therefore, it is very important that these patients examine their affected skin areas regularly to make sure they haven't injured themselves (cuts, burns, infections, etc.). Also, some patients with neuropathy have trouble with their balance when walking; these patients should keep a nightlight on in their bedrooms and bathrooms, so they do not fall when they get

up at night. As with all chronic pain, patients with painful polyneuropathy may develop depression and sleep problems.

D. FALSE the distal regions are more affected.
E. FALSE diabetes is the number one cause of damage to the peripheral nerves, and peripheral polyneuropathy is the most frequent complication of diabetes. The most common early dysfunction is abnormal nerve conduction studies due to demyelination. Sensory fibers are usually more affected than motor. The first clinical sign that usually develops with abnormal nerve conduction is decrease or loss of ankle jerks or decrease or loss of vibratory sensation over the great toes.

WEAKNESS #9.
A. FALSE a SNAP represents the APs of all axons in the nerve being tested.
B. FALSE a CMAP represents all of the APs of all MFs innervated by the nerve being tested/stimulated
C. TRUE
D. FALSE a MUP represents the APs of all MFs belonging to a LMN (a motor unit). You might be interested in how one motor unit can be turned on so as to be tested. Well, say you want to examine the biceps brachii. Individual MUP measurement can be made by having the patient slowly and minimally activate the muscle. The EMG signal should contain only the discharges of a few MUPs with adequate baseline between discharges to enable full identification of their size and shape.
E. FALSE it is a postsynaptic defect. If you are interested, Lambert Eaton myasthenic syndrome (LEMS) involves a presynaptic problem. Recent work demonstrates that LEMS results from an autoimmune attack against voltage-gated calcium channels on the presynaptic motor nerve terminal. Cancer is present when the weakness begins or is later found in 40% of patients with LEMS. This is usually a small cell lung cancer.

WEAKNESS #10.
A. FALSE a myopathy would result in lower CMAPs
B. FALSE dead muscle has no relationship to how fast normal axons conduct
C. FALSE reserved for myasthenia gravis (postsynaptic problem at NMJ)
D. FALSE dying muscle=smaller MUP
E. TRUE when muscle membranes break and CK gets into the blood stream

WEAKNESS #11.
Shaded area is ventral root
A. TRUE dying motor axons=reinnervation=type grouping
B. TRUE dying motor axons=reinnervation=type grouping and group atrophy
C. TRUE dying motor axons=reinnervation=type grouping, group atrophy and fewer MFs=lower CMAP
D. FALSE dying motor axons=reinnervation=type grouping=increase in size of MUPs (followed by group atrophy)
E. TRUE it is relatively normal as there is some slight increase due to the group atrophy

WEAKNESS #12.
Shades structure is spinal nerve.
A. FALSE the axon is fine, therefore no reinnervation etcetera!
B. FALSE see A
C. FALSE the messages to fire get to the muscle, only slower.
D. FALSE see A
E. TRUE classic sign

WEAKNESS #13.
Shaded area is spinal nerve.
A. TRUE dying motor axons=reinnervation=type grouping
B. TRUE dying motor axons=reinnervation=type grouping=group atrophy
C. FALSE assuming that this spinal nerve entered a larger nerve bundle with normal axons, the normal axons will camouflage the bad ones and give a normal NCV
D. TRUE dying motor axons=reinnervation=type grouping=increase in size of MUPs
E. TRUE you need a myopathy for breakdown of MF membranes and the release of CK into the bloodstream.

WEAKNESS #14.
Shows group atrophy.
A. TRUE fewer normal MFs in a MU
B. TRUE that is what the drawing shows
C. FALSE the normal nerves camouflage the dying ones
D. TRUE assuming that other MUs are reinnervating MUs that are dying
E. TRUE only myopathies display increased CK

WEAKNESS #15.
Drawing shows nerve fiber with shortened internodes that are hypomyelinated.
A. TRUE that is what we are trying to show in this drawing!
B. TRUE less myelin=slower NCV
C. TRUE axon/axis cylinder is fine but loss of myelin reduces efficiency of saltatory conduction
D. FALSE reinnervation occurs only when axons to muscles die
E. TRUE loss of myelin alone does not lead to reinnervation

WEAKNESS #16.
Shows a normal and a decremental CMAP.
A. FALSE referring to the decremental CMAP, it is only seen with NMJ problems
B. FALSE see A
C. TRUE this is seen in myasthenia gravis
D. FALSE a lesion of the LCST will **NOT** result in any problems from the LMN out to the muscle
E. FALSE this is a NMJ problem; there is no breakdown of MF membranes

WEAKNESS #17. Fill in the blanks in the chart below.
AHC—**Rep Stim is normal (no decremental CMAPs)**
PN/axonal—**MUPs increased; fibrillations present (yes)**
PN/demyelin—**Conduction velocity (NCV)decreased**; **histology normal**
Myast. Gravis—**Blood CK is normal**
Muscle disease (myopathy)—**SNAPs are normal**
UMN lesion-**CMAPs are normal; no fasciculations**

SPINAL CORD—RADICULOPATHY

RADICULOPATHY #1.
A. FALSE most common cause is herniation of the intervertebral disc. They may also be caused by spinal stenosis, where the vertebral canal narrows and squeezes the spinal column and nerve roots. Moreover, as we age, the water content in our body cells diminishes and other chemical changes occur that can cause the disk to shrink. Without sufficient cushioning, the vertebrae may begin to press against each other, pinching the nerve or causing the formation of bony spurs. Cervical strains may also cause cervical radiculopathy. Cervical strains are usually caused by injuries such as being struck on the head by a heavy object or automobile accidents. When these injuries occur, the muscles, tendons, and ligaments may become inflamed and therefore irritate nerves in the neck region. Osteoarthritis is another condition that can create radiculopathy. As the arthritis progresses, bony spurs become more and more prevalent and larger. Eventually, these spurs can begin to irritate the nerve roots. There may be many other causes for radiculopathies, such as muscle spasms, cancer, infections, as well as many other disease processes. Remember, radiculopathy means disease affecting one of the nerve roots that arise from the spinal cord, travel for a short distance in the spinal canal, and then exit through one of the neural foramina alongside the spinal column. Radicular symptoms can include weakness, pain, or loss of feeling in the area of the body to which the affected nerve goes.
B. FALSE dorsal roots can be affected, but so can ventral
C. FALSE ventral roots can be affected, but so can dorsal
D. FALSE see A
E. TRUE remember this for life!!!

RADICULOPATHY #2.
A. FALSE anesthesia mean absence of sensation
B. FALSE analgesia means absence of pain, and the hallmark of acute or chronic nerve root compression is pain
C. TRUE remember this for life!!
D. FALSE you only see spasticity following a lesion inside the central nervous system; radiculopathies involve the peripheral nervous system.
E. FALSE see C

RADICULOPATHY #3.
A. FALSE lumbar roots emerge from **below** their respective vertebrae
B. FALSE the cauda equina or “horse’s tail” is formed by lumbar and sacral nerve roots lying in subarachnoid space below the termination of the spinal cord (conus medullaris) which is at approximately L1-L2 vertebral body.
C. TRUE a classic!! Remember it for life
D. TRUE a classic!! Remember it for life
E. TRUE see C and D

RADICULOPATHY #4.
A. FALSE C7 exits above the C7 vertebra. Because in the cervical region there are only 7 vertebrae despite 8 cervical roots, the root number exiting between two vertebrae is always the number of the lower vertebra. For example, the C5 root exits between the C4-C5 vertebrae and would be effected by a C4/5 disc herniation; the C8 root exits between C7-T1 vertebrae and would be compressed by a C7/T1 disc.
B. FALSE lumbar roots emerge from **below** their respective vertebrae.
C. FALSE a C7 radiculopathy, which is the most common in the cervical region, radiates into the dorsum of the middle finger.
D. FALSE see C above and remember C6=six shooter!!
E. TRUE yes, another classic, and yes, remember it for life!

RADICULOPATHY #5.
A. FALSE brachialgia is pain in the arm and transient paresthesia occurring only at night
B. FALSE root pain can be accompanied by paraesthesias and sensory loss (motor loss, too!)
C. FALSE brachialgia is usually accompanied by neck pain
D. TRUE remember, radiculopathies compress/involve roots whose sensory fibers are associated with dermatomes.
E. FALSE radiculopathy/pain is often the result of mechanical causes like a herniated disc, bone spur, or stretching event.

RADICULOPATHY #6.
A. FALSE dull=proximal
B. FALSE at this level of the vertebral column only the spinal nerves are present. It is below the spinal cord and the associated ascending and descending pathways. The only thing that a lesion will affect is the spinal nerves and this results in lower motor neuron deficits only; there are no UMN pathways (LCST) present!!!!
C. FALSE wow, so false!! LMN all the way!!
D. FALSE C7 is the most common root affected by herniated discs in the cervical region. L4-L5 disc/L5 root is the champ!!
E. TRUE so true, so very, very true!!!

RADICULOPATHY #7. SELF EXPLANATORY—Remember, dorsum of foot and big toe=L5, and lateral aspect of foot=S1!

RADICULOPATHY #8. SELF EXPLANATORY—Your thumb and index finger make a little pistol, your very own six shooter. How convenient for your C6 dermatome!!

RADICULOPATHY #9.
Shows herniation of the intervertebral disc rostral (above) the vertebral body of L5—i.e., the L4-L5 disc. This will result in a L5 radiculopathy and A is labeled L5.
A. FALSE it is the L5 root
B. FALSE S1=little toe, L5=big toe
C. FALSE it is the L5 root
D. FALSE you must be thinking L3
E. TRUE so true!!

RADICULOPATHY #10.
A. FALSE **A** is the S1 root. The motor component is involved with hip extension and ankle plantar flexion (stand on toes)
B. FALSE **B** is the L5 root. The motor component is involved with the hip abductors, and ankle dorsiflexion, (stand on heels!), eversion and inversion
C. FALSE **C** is the L4 spinal root. The motor component is involved with leg extension at the knee joint
D. FALSE **D** is the L3 spinal root. The motor component is involved with leg extension at the knee joint and hip flexion.
E. TRUE B is the L5 spinal root. The motor component is involved with the hip abductors, as well as ankle dorsiflexion, (stand on heels!), eversion and inversion

RADICULOPATHY #11.
A. TRUE B is the C6 spinal root. The motor component (along with C5) does supply the deltoid as well as the biceps, brachioradialis, infraspinatus, and supraspinatus.
B. FALSE **D** is the C8 spinal root. The motor component supplies the extensors and flexors of the wrist and the intrinsic hand muscles
C. FALSE **B** is the C6 spinal root. See response A
D. FALSE **B** is the C6 spinal root. See response A and C. Interosseous muscles are intrinsic muscles of the hand (C8/T1).
E. FALSE **C** is the C7 spinal root. The motor component supplies the triceps and extensors and flexors of the wrist.

RADICULOPATHY #12.
A. TRUE The lumbar and sacral roots are damaged. The perineum is innervated by roots S2-S4
B. TRUE the ankle reflex involves S1
C. TRUE the knee jerk involves L4
D. FALSE this is an UMN sign. We are only talking LMNs with cauda equina lesions!!
E. TRUE see responses A, B, and C

SPINAL CORD—AUTONOMIC DYSFUNCTION

AUTONOMIC DYSFUNCTION #1.
A. TRUE right after the lesion=spinal shock=flaccid; later on spastic
B. TRUE in a flaccid bladder (during spinal shock) a large amount of urine remains in the bladder. In a spastic bladder, a small residual amount remains in the bladder.
C. TRUE urinary incontinence indicates absence of normal control of the bladder/urination.
D. TRUE there is retention because the bladder is hypotonic (during spinal shock). It fills and spills.
E. TRUE see all responses above!

AUTONOMIC DYSFUNCTION #2.
A. TRUE all of the descending higher cortical pathways present at C2 are lost. This means that the cells of origin of the phrenic nerve (LMNs at C3-C5) and LMNs innervating the intercostals, accessory muscles in the neck and shoulders, and abdominal muscles have lost this descending control/ input
B. TRUE the muscles referred to in response A are still receiving input from their LMNs in the spinal cord and the LMNs are still receiving descending brain stem inputs
C. TRUE descending control over the bladder is lost due to the lesion at L5. The bladder could be flaccid or spastic. If flaccid, there will be urinary retention
D. TRUE the diaphragm is innervated by LMNs located at C3-C5
E. TRUE see responses A-D

AUTONOMIC DYSFUNCTION #3.
Lesion involves crossing fibers of the ALS at spinal level T3.
A. FALSE the cell bodies of the crossing fibers lie in the dorsal horn at the same level, T3
B. FALSE the cell bodies of the crossing fibers lie in the dorsal horn at the same level, T3
C. FALSE the crossing fibers at T3 enter the most medial part of the ALS. They would lie **medial** to an axon that arose from a cell in the contra lateral dorsal horn cell in **T11** (which receives information from spinal nerve S1, which innervates the lateral part of the foot!)
D. TRUE the crossing axons arise from dorsal horn cells at T3. These cells receive pain and temp information from T5 spinal nerves (two levels down!).
E. FALSE the only way for this to be true is to have bilateral lesions of the **ALS!!! THIS DISTINCTION IS VERY IMPORTANT!**

AUTONOMIC DYSFUNCTION #4.
A. FALSE the only way for the diaphragm to atrophy is to have a lesion that affects the LMNs that innervate it or via a lesion of the phrenic nerve(s). A lesion at C1 would not damage any of the LMNs, which are at spinal levels C3-C5
B. FALSE see A
C. FALSE the LMNs innervating the diaphragm and the intercostals have lost their descending drive
D. FALSE the LMNs innervating the diaphragm and the intercostals have lost their descending drive
E. TRUE

AUTONOMIC DYSFUNCTION #5.
A. FALSE the only way for the diaphragm to atrophy is to have a lesion that affects the LMNs that innervate it or via a lesion of the phrenic nerve(s). A unilateral lesion at C1 would not damage any of the LMNs, which are at spinal levels C3-C5
B. FALSE see A
C. FALSE the unilateral lesion would result in the diaphragm still functioning
D. FALSE the unilateral lesion would result in the diaphragm still functioning
E. TRUE lesion needs to be bilateral!!

AUTONOMIC DYSFUNCTION #6.
The lesion involves the entire right side of the spinal cord at L5.
A. FALSE lesion needs to be bilateral
B. FALSE lesion needs to be bilateral
C. FALSE lesion needs to be bilateral
D. FALSE there is no DSCT at L5. Clarke's column does not start until L3.
E. TRUE

AUTONOMIC DYSFUNCTION #7.
Lesion involves S2-S5 bilaterally in the caudal equina.
A. TRUE the bladder is flaccid, atonic, hypotonic, autonomous, or LMN (Whew!!).
B. TRUE see A
C. TRUE the bag fills and fills and spills.
D. FALSE frequency and urgency occur following an **UMN** lesions involving the bladder. In the spastic bladder, small degrees of filling results in frequent (automatic) emptying. If any sensory information gets up the cord to the cortex, there also is urgency.
E. TRUE when the preganglionic and postganglionic input to the bladder is damaged it is called a LMN bladder. This does not fit exactly with the traditional concept of LMN input (directly to skeletal muscle), but such lesions do result in bladder weakness, atrophy, and hyporeflexia.

AUTONOMIC DYSFUNCTION #8.
A. FALSE spontaneous, radicular, pain is a very prominent symptom
B. TRUE preganglionic parasympathetics cell bodies are at spinal levels S2, S3 and S4. Without these cells working, there are major autonomic problems with the bladder
C. FALSE huh? Glove and stocking=polyneuropathy!
D. FALSE the knee jerk reflex needs LMN and sensory afferents associated with spinal levels L3/L4 to be intact; only sacral levels are present in the conus medullaris
E. FALSE huh? The cauda equina=LMNs=atrophy

AUTONOMIC DYSFUNCTION #9.
A. FALSE nipple=T4
B. FALSE belly button=T10
C. FALSE big toe=L5
D. TRUE C6 innervates the thumb and index finger (can conform to shape of a gun!!)
E. FALSE little toe=S1

AUTONOMIC DYSFUNCTION #10.
A. FALSE no atrophy in UMN lesions
B. FALSE loss of LMNs means fewer axons in a nerve and smaller CMAPs from a particular muscle when you stimulate the nerve.
C. FALSE automatic=spastic and autonomous=flaccid
D. TRUE keeps it alive!!!
E. FALSE L5 helps you stand on your heels, while S1 helps you stand on your toes

SPINAL CORD ANATOMICAL LOCATION

ANATOMICAL LOCATION #1.
A. TRUE think of Duchenne muscular dystrophy
B. FALSE neuropathic (nerve) disorders exhibit several different patterns. However, proximal weakness and the absence of sensory abnormalities does not occur.
C. FALSE syringomyelia is first associated with the bilateral loss of pain and temperature at one level (two levels down from the lesion). So there is a sensory disturbance. You should note, however, that a syrinx (lesion) can move into the ventral horn and cause weakness that could be proximal, depending on the specific site of the lesion
D. FALSE I guess this could be true if just the LMNs innervating proximal muscles are affected. However, it would be pretty selective not to include a single sensory pathway
E. FALSE hey, the "**distal"** says it all!

ANATOMICAL LOCATION #2.
A. FALSE both are part of the peripheral nervous system. The only way you get spasticity is to have a lesion of the CNS and in particular the CST/LCST.
B. FALSE the motor part of L5 is involved with eversion and inversion of the ankle, dorsiflexion of the foot and hip abductors
C. FALSE sensory from the big toe is classic L5 radiculopathy. But remember, the L5 root travels in the peroneal nerve on its way to the big toe! (peroneal=L4-S2 roots). The peroneal nerve is one of the two major divisions of the sciatic, the other one being the tibial.
D. TRUE the peroneal nerve is in the leg!!! How could a lesion affect the back?
E. FALSE only one (D) response is TRUE

ANATOMICAL LOCATION #3.
Bilateral weakness of deltoid and hips areas.
A. FALSE this would be pretty unlikely. You would need some pretty selective lesions in both LCSTs.
B. FALSE "distal" is the key
C. TRUE CK goes up in muscle disease.
D. FALSE dying muscle=smaller CMAPs
E. FALSE what does dying muscle have to do with axon conduction velocity?

ANATOMICAL LOCATION #4.
Depicts "glove and stocking" sensorimotor loss.
A. FALSE the key word is easily.
B. FALSE it is a distal polyneuropathy. In the case of the stocking, both the tibial and peroneal branches of the sciatic are affected. Think about losing both roots L5 (dorsiflexion of foot) and S1 (plantar flexion of foot)
C. FALSE polyneuropathy=peripheral nervous system damage. Only get SPASTICITY AND BABINSKI IN CNS LESIONS!
D. FALSE here we assume that some of the axons are OK and there is reinnervation=**increase** in MUP amplitude
E. TRUE classic symptom of polyneuropathy!

ANATOMICAL LOCATION #5.
Sensorimotor loss in the distribution of L5 root.
A. FALSE think about the peroneal nerve. Its distribution is much larger/extensive than the shaded area
B. FALSE think about the distribution of the tibial nerve
C. FALSE "poly" is the key—many or several **NERVES**! This looks like one root!
D. FALSE it is a problem with the L5 root. To get this you need herniation of the L4-L5 disc.
E. TRUE see D

ANATOMICAL LOCATION #6.
Sensorimotor deficit in a cape and gown distribution. Assume that only the pain and temperature is affected under "sensory"!
A. FALSE "best be accounted" is the key here.
B. FALSE mononeuropathy multiplex refers to the situation in which two or more individual nerves or branches are involved. Thus the symptoms and signs are restricted to the territories of these damaged nerves. (In a polyneuropathy, the peripheral nerves are affected symmetrically, and usually the longest nerve fibers are damaged first and maximally. Thus the symptoms and signs involve both feet first, and as the disorder progresses, the hands are also both involved).
C. FALSE such a lesion would result in pain and temperature being lost from sensorimotor deficits from C6 on down!
D. FALSE syringomyelia is a central cord (and can go into the brain stem too!) lesion but it can also extend laterally into the ventral horns.
E. TRUE Explanation: Think of a lesion that affects the crossing fibers from C2-C8. This accounts for the pain and temperature loss. In addition, the lesion also extends bilaterally into the ventral horns from C4-T2.

ANATOMICAL LOCATION #7.
Loss of all sensation and movement from T10 and below.
A. FALSE the "down two levels" rule only relates to lesions of the ALS. A complete lesion of the spinal cord at T10 does damage the ALS on both sides and this would mean a loss of pain and temp from T12 on down bilaterally. However, you need to add the bilateral loss of the dorsal horn cells and zones of Lissaur at T10. When this addition is done, you can see that the pain and temp loss starts at T10.
B. TRUE neurogenic means "nerve related." There would be a loss of all descending neural control over the bladder.
C. FALSE the lesion is at T10. The diaphragm is doing fine and so are accessory muscles and many intercostals.
D. FALSE the lesion is at T10 and the T10 LMNs are dying. Dead muscles cannot be spastic or hyper-reflexive.
E. FALSE **HYPER**-reflexive after UMN lesions. Knee jerk reflex depends on L3/L4.