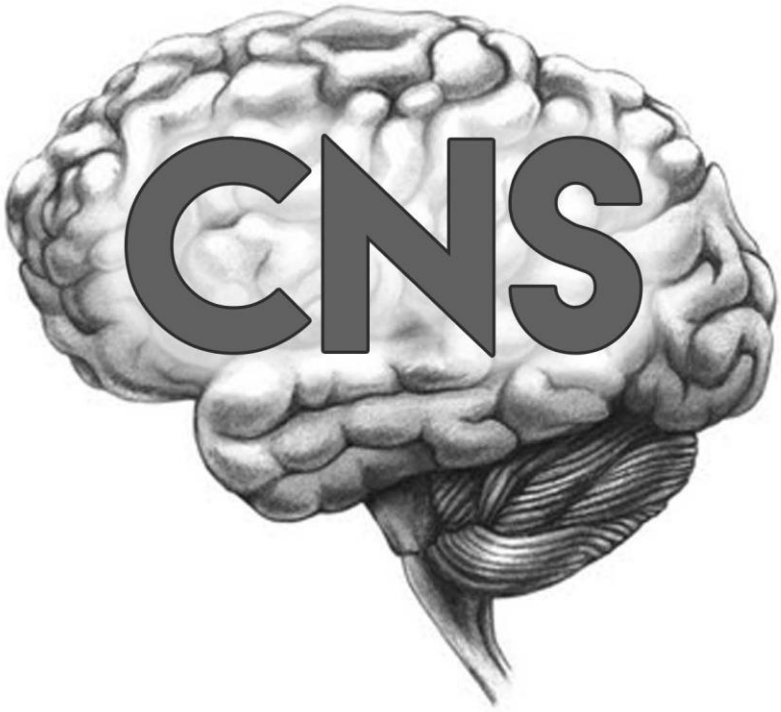

ANATOMY

☑ Sheet ○ Slide ○ Handout

Number

13

Subject

Sensory pathways & hemi-section lesion

Done By

Abdallah S. S.

Corrected by

Doctor

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Date:

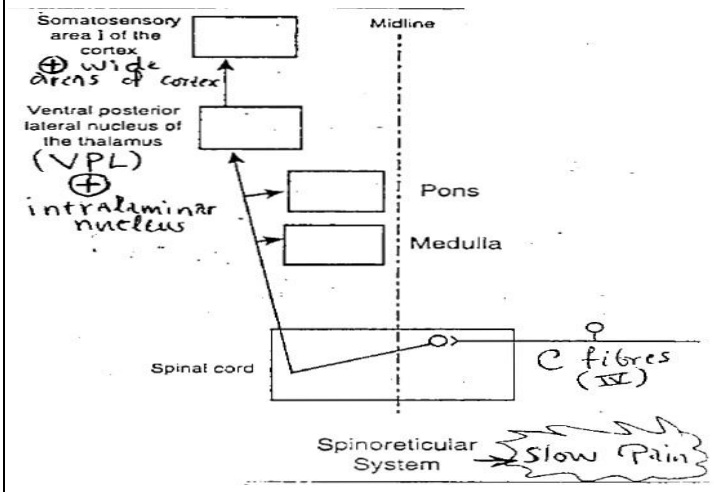
Price:

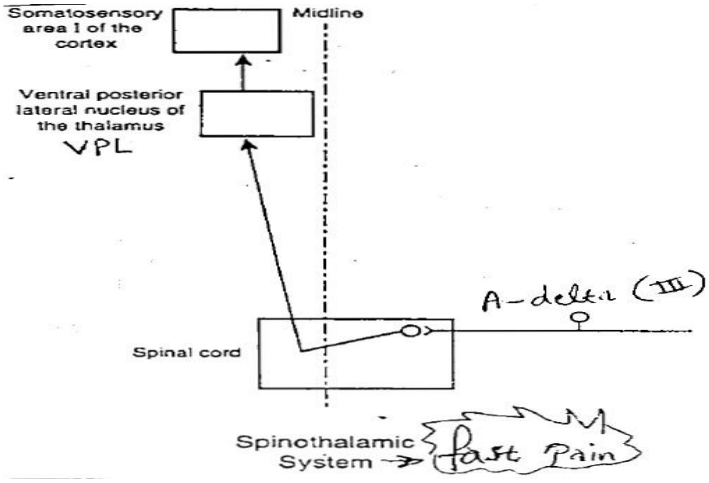
بسم الله الرحمن الرحيم

This sheet was written according to section 2 recordings (13 & 14). Hope you enjoy it.

ꕥ we'll talk about two pain pathways : SLOW (spinoreticular-SR) & FAST (spinothalamic-ST)

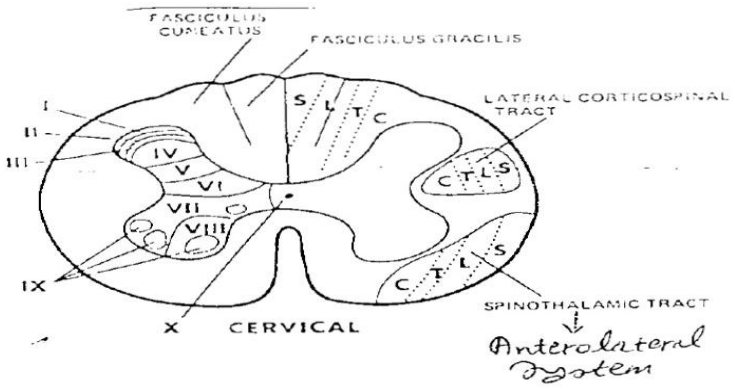
- For both pathways the 1st order neuron is in the dorsal root ganglion, and the peripheral process is at the receptor where the central process is at the 2nd order neuron.
- The 2nd order neuron for ST is located at lamina I & V
- The 2nd order neuron for SR is located at lamina I & II
- The 2nd order neuron sends out fibers that cross the midline toward the 3rd order neuron in the thalamus ; * VPL nucleus (in case of ST) which is part of the ventro-basal complex
  * VPL &<u>intraluminar nucleus (it sends awakening impulses to the cortex to treat the pain)</u>
- **<u>Fast</u>** pain is transmitted via **<u>A-delta</u>** fibers .It is very well localized & mostly of **<u>superficial</u>** structures. (i.e. pinprick is felt within 0.1s)
- **<u>Slow</u>** pain is difficult to endure and transmitted via **<u>C</u>** fibers. It's in both **<u>superficial & deep</u>** structures. (i.e. intestinal colic, toothache, burn, etc…)





- <u>Fibers arrangement</u>;

Here there is a sensory pathway called antero-lateral system for pain of both temperature and touch and dorsal column system. Fibers are **<u>inversely arranged</u>**, where cervical and thoracic regions (UL) are represented most medially & lumber and sacral regions (LL) are represented laterally.



- <u>According to the site of stimulation</u> : three types of pain can be described ;
  cutaneous→ from the skin , it is very well localized as skin is rich in receptors ,
  deep somatic→ its receptors are in muscles , bones , joints & ligaments , dull diffuse ,&
  visceral→poorly localized & transmitted via C fibers , and as deep somatic , both are associated with **<u>autonomic manifestations</u>** as : tachycardia, HTN, hypotension ,sweating ,vomiting , etc.. .

- <u>Adequate stimulus</u> : the lowest threshold needed for the receptor to be stimulated ;
  - ✓ For deep somatic pain:*mechanical (muscletraction, pressure on a bone, etc..)
    *chemical (venoms)
    *ischemia (cardiac muscle→angina pectoris , LL →intermittent claudication)

☞ Intermittent claudication : is a bad sign especially for diabetic patient with atherosclerosis , he starts walking without pain but after a while as his blood supply is not enough to remove the metabolites (esp. lactic acid) he feels pain , pain also can be felt during rest . Gangrene is a predictable complication.

- ✓ Visceral pain is not felt unless there is a considerable inflammation due to few receptors there;
  *distention of bladder & intestines
  *spasm: leads to blood vessels compressions and accumulation of metabolites.
  *chemical irritants:HCl from perforated duodenal ulcer (most severe pain)

- Visceral pain is often referred (i.e. acute appendicitis is felt around the umbilicus) , and can be accompanied with <u>rigidity</u> in the overlying muscles (i.e. for the appendicitis if the inflammation causes peritonitis the abdominal wall is a board-like)

- **<u>Referred pain mechanism</u>**: (*<u>convergence theory</u>* / misinterpretation) ;

E.g. Viscus→ Appendicitis is inflamed then sends pain impulses via sensory fibers with sympathetic or parasympathetic through spinal cord to the 2nd order neuron at T10, which also represents the skin around the umbilicus. The 2nd order neuron sends out a spinothalamic tract to the cortex that in turn misinterprets and thinks that the pain is from the umbilical region as it has much more receptors.
→The sensory fibers that come out from an affected organ goes to a cell that receives other sensory fibers from the body wall.
The pain is then localized if the inflammation reaches the peritoneum.

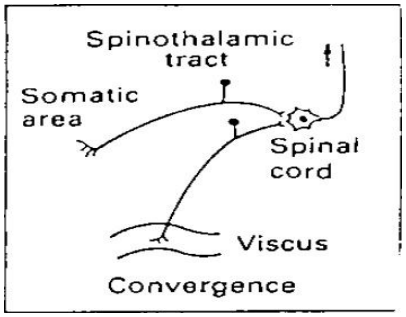


Table 17.4 Important examples of referred pain

Organ	Site of referral
Heart	Precordium; inner aspect of left arm; epigastrium
Appendix	Umbilicus
Small intestine	Umbilicus
Central part of diaphragm	Tip of shoulder
Pleura	Abdomen
Kidney	Costovertebral angle (loin)
Ureter	Testicle
Trigone of bladder	Tip of penis
Tongue	Ear
Teeth	Head
Hip	Knee
Uterus	Low back radiating to lower abdomen

C٣, ٤&٥

C٤

Kidney stone

Acute tonsillitis; CN IX

CN IX

CN V

Illustration to the table:

- Precordium: ant. to the heart , sternum region

- Pain is felt at the inner aspect of Lt. arm , as it's supplied by intercostobrachial nerve (T٢), where the heart sensation is supplied by (T٢,٣&٥).

- The pain sensation at the lower jaw due to MI is not explained yet.

- fever with cough and abdominal pain → thinks of pleurisy

Adequate Stimulus

Pain receptors are specific, and pain is not produced by overstimulation of other receptors. On the other hand, the adequate stimulus for pain receptors is not as specific as that for others, because they can be stimulated by a variety of strong stimuli. For example, pain receptors respond to warmth, but it has been calculated that the threshold for thermal energy is over 100 times that of the warmth receptors. Pain receptors also respond to electrical, mechanical, and, especially, chemical energy(Polymodal receptors)

It has been suggested that pain is chemically mediated and that stimuli which provoke it have in common the ability to liberate a chemical agent that stimulates the nerve endings. The chemical agent might be histamine, which causes pain on local injection.

Illustration;

-Cold receptors can be stimulated by overheating, and you'll sense cold, while the pain can't be sensed by overstimulation of other receptors.

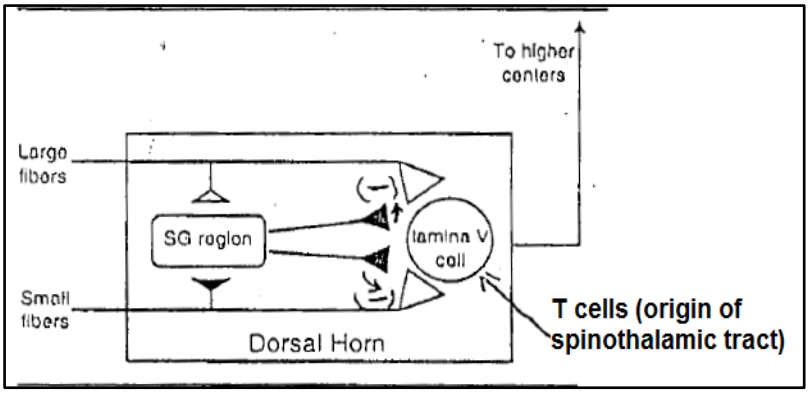
-Adequate stimulus for pain is not specific: Pain receptors can respond to electrical, mechanical & chemical stimuli as they cause tissue damage.

ꕥ Endogenous pain pathway: (substantia gelatinosa, lamina II) to modify pain.

It can reduce pain impulses to high brain centers.

Lamina I, II & V cells are the origin cells of spinothalamic tract that sends out pain pathways (spinothalamic & spinoreticular).

- ✓ If stimulated → action potential reaches the cortex → opened-gate
- ✓ If inhibited → closed-gate
  →This is called Gate controlled theory.



- Large fast fibers transmit touch.
- Small slow fibers transmit pain.
- Both these synapse at lamina I , II & V cells.
- Substantia gelatinosa sends inhibitory fibers for both large and small fibers near there ends (axo-axonic synapse)
- Touch (large) fibers before reaching the lamina cells they excite SG, so increasing its inhibitory effect, and the gate is closed.
- On the other hand, Pain (small) fibers inhibit SG, removing its inhibitory effect, so the gate is opened.
- This is applied as follows: when you feel pain, you scratch your hands so activating touch fibers to close the gate and no more feeling pain. However, in case the pain is severe this is not applied as pain impulses from small fibers are much stronger than touch impulses. (Modify pain)

ꕥ **Endogenous pain control system : (see the figure at the next page)**

1. Ascending pathway

–spinoreticular pain impulses project to the periaqueductal gray of the midbrain. PAG

2. Descending raphe–spinal pathway

–excitatory neurons of the periaqueductal gray project to the nucleus raphae magnus of the pons.
–excitatory neurons of the nucleus raphae magnus project serotonergic fibers to enkephalinergic inhibitory neurons of the substantia gelatinosa.
–enkephalinergic neurons of the substantia gelatinosa inhibit afferent pain fibers (substance P) and tract neurons that give rise to the spinoreticular and spinothalamic tracts.

3. Descending ceruleospinal pathway

– projects from the locus ceruleus to the spinal cord.
–is thought to directly inhibit tract neurons that give rise to the ascending pain pathways.

Notes:

-the excitatory neurotransmitter at PAG & NRM (nucleus raphae magnus) is **serotonin**.

-the inhibitory neurotransmitter at SG is **enkephaline**.

- We inhibit Lamina I, II & V either;
 *directly via locus cerleus (from pons)
 *or indirectly from the midbrain(PAG) & pons (NRM)

-these pathways partly inhibit pain transmission, so we give analgesics for severe pain.

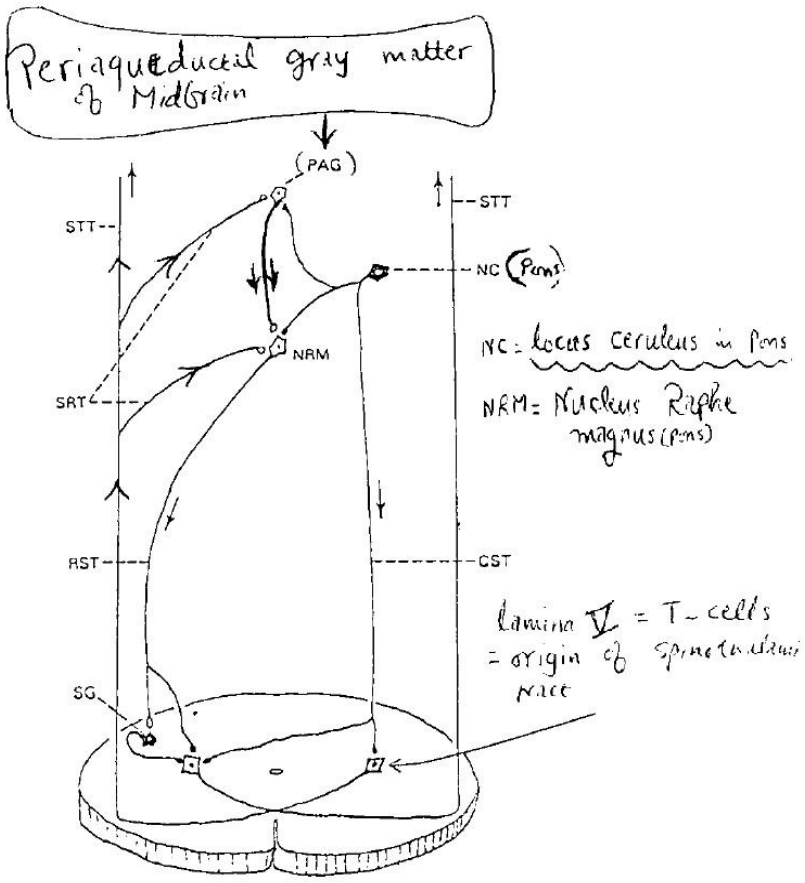


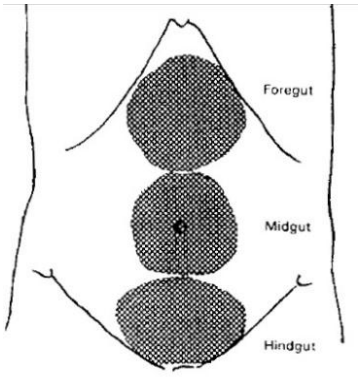
Fig. 15-7 Two supraspinal gate controls: the raphespinal tract (RST) from the nucleus raphe magnus (NRM), and the ceruleospinal tract (CST) from the nucleus ceruleus (NC). PAG, periaqueductal gray matter; SG, substantia gelatinosa; SRT, spinoreticular tract; STT, spinothalamic tract.

ꕥ **<u>Visceral pain</u>** : of three types

- ✓ Pure visceral pain; felt in the region of the affected organ. (E.g. at the beginning of intestinal & bile duct obstructions, solid organ's capsule stretch due to inflammation ...)
- ✓ Visceral referred pain; explained previously, visceral pain is referred to somatic structures innervated from the same segmental levels of spinal cord.

  →From the GI tract, pain is referred to anterior midline area:

  - From foregut to epigastrium.
  - From midget to the periumbilical region.
  - From hindgut to hypogastrium
  - This is because during the early embryonic life the gut is innervated while it was a mid-line structure, then the gut-rotation occurs.
  - E.g. a patient complains of an epigastric pain, which organs you suspect can be affected? Foregut (stomach, liver, gall bladder & pancreas...)



Visceral referred pain from the gastrointestinal tract.

- ✓ Viscerosomatic pain ; caused by the spread of visceral disease to somatic structure , (e.g. ant. abdominal wall muscles rigidity in case of peritonitis)

After talking about the spinothalamic tract, we will start talking about **dorsal (post.) column system**:Regarding this system we have two theories concerning its structure and components, an old one and a new one.

- ✓ The old theory: it says that the first order neuron is found in the ganglion, the peripheral process of this ganglion comes from the receptor (in skin for example), and the central process reaches the spinal cord. The central process continues as **gracile and cuneate tracts** (so they are the axons of first order neurons).
- **Gracile receives impulses from the lower limb, while cuneate receives impulses from the upper limb.**
  Gracile and cuneate tracts continue to gracile nucleus and cuneate nucleus respectively in the medulla ***the second order neuron***.
  The most important thing in second order neurons is that the tracts should cross to the opposite side when reaching them, and that's what happens here, the two tracts cross (in the medulla) and this decussation is called **sensory decussation.**
- This sensory decussation forms the **medial lemniscus** tract, so we can call the whole tract as
  dorsal column - medial lemniscus tract.
- ☞ You should note the following: injury to this tract **before** the decussation results in symptoms to the same side; injury to the **medial lemniscus** results in symptoms in the opposite side in both UL & LL.

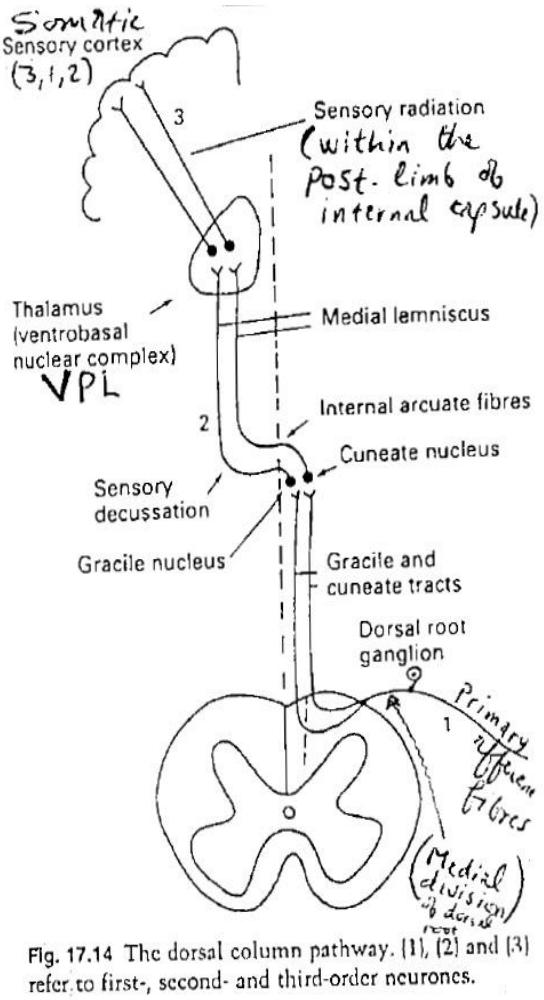


Fig. 17.14 The dorsal column pathway. (1), (2) and (3) refer to first-, second- and third-order neurones.

- From the medial lemniscus we should reach the third order neuron in the thalamus (VPC ;VPL in this case or VPN from the face). From the thalamus the tract reaches area 3,1,2 - somatic sensory cortex via sensory radiation tract (through the **posterior limb of internal capsule**), and then to area 5,7 (to perceive the type of sensation).
  Note: lesion to the sensory radiation → temporary hemi-anesthesia, crude sensation returns at the level of the thalamus.

- ✓ The new theory:In addition to what we have mentioned previously, the new theory states that the **dorsal spinocerebellar tract** has a role in this system.
- According to what we have learned, the stimulus from the lower limb reaches the nucleus of Clarke, this stimulus continues with the dorsal spinocerebellar tract to reach the cerebellum transmitting **unconscious** proprioception, but now, new studies have shown that part of the dorsal spinocerebellar tract has the ability to transmit **conscious** proprioception. (see the fig.at the next p.)

The fibers of this part reach **nucleus Z** in the medulla, then these fibers unite with the gracile and cuneate tracts forming the medial lemniscus.

- **So the new theory says that the medial lemniscus consists of axons of gracile, cuneate and nucleus Z.**
- This has important implications clinically.

Function of this system:

1-This tract transmits **fine** sensation → so its receptor fields are small.

- Fine sensation means discriminative touch:

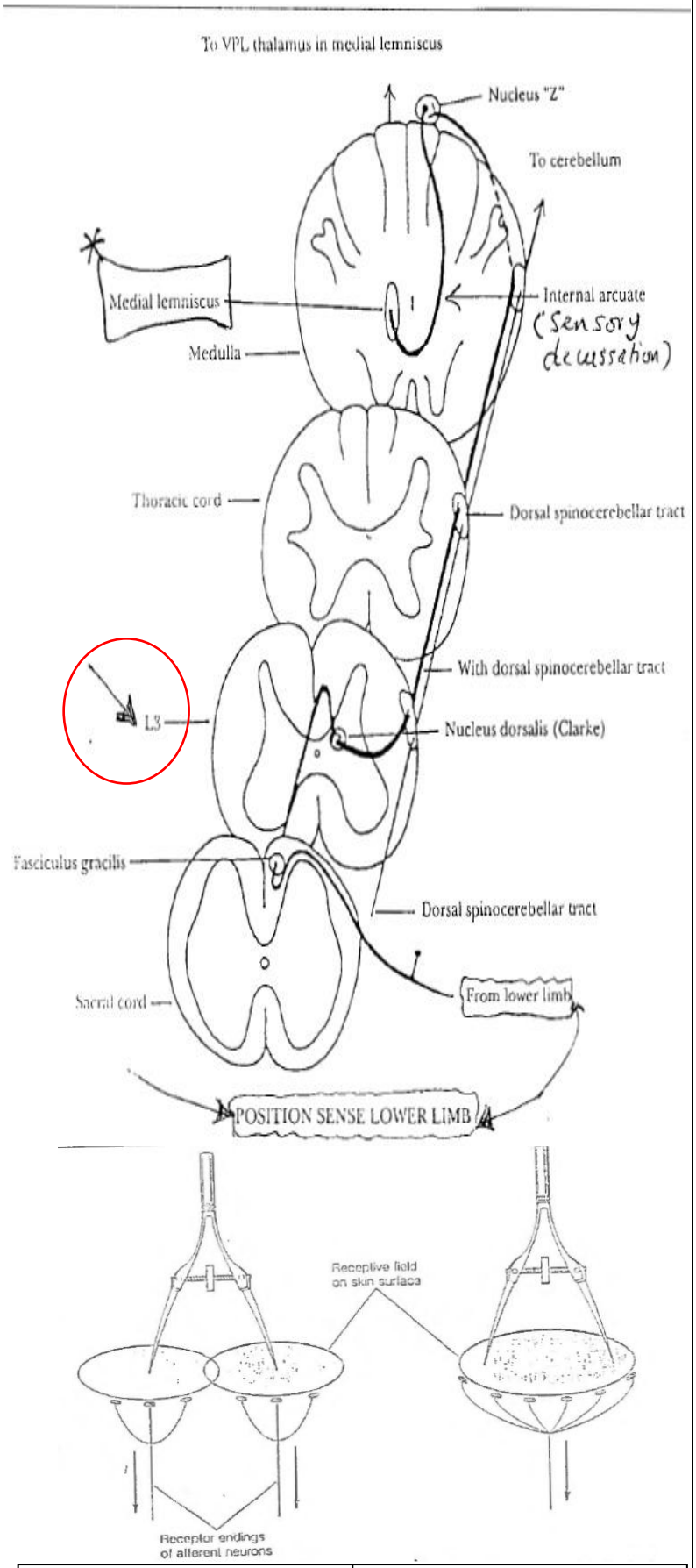
  A- If we have two pins on the hand, and the distance between them is small (stimulating two nearby receptor fields), we should recognize them as two stimuli and not as one. This is called **2 point discrimination**.

  B- Also we should know the precise area that has been stimulated (like we should know if the pin has been stimulating the thumb or index, from the tip or the base), and this is called **tactile localization**.
- Note: in spinothalamic tract (at the level of thalamus) we have **no** discriminative touch (large receptive fields).

2-Stereognosis: identifying an object without seeing it. E.g.: putting keys in your hand and knowing that you have keys in your hand without using your sense of sight.
To test the LL→ draw shapes

3- This tracts also transmits **pressure** sensation.
4- Vibration.
5- Proprioception: sense of position

☞ All these sensations can be referred to as **mechanoreceptive sensations**.



2 receptive fields stimulated → 2 points felt	1 receptive fields stimulated → 1 point felt

(Recall: spinothalamic tract transmits wide spectrum of sensations like temperature, simple touch, itch and tickle, sexual sensation and fast & slow pain, so we can't put them in one category).

- Note: spinothalamic tract transmit signals from rapidly adapting receptors (e.g. touch) and slowly adapting receptors (e.g. temperature).

VERY IMPORTANT: (lesions in this pathway)
Old theory: if gracile tract is damaged we lose stereognosis and proprioception of the LL.
if the lesion in the dorsal column pathway is above L3 (thoracic or cervical regions) we lose stereognosis & proprioception from both UL and LL

New theory: if gracile tract is damaged, we lose only stereognosis **as the proprioception of the LL is transmitted via nucleus Z in the medulla**.
if the lesion in the dorsal column pathway is above L3 (thoracic or cervical regions)
we lose stereognosis from both UL and LL , while the sense of position is lost only in UL.

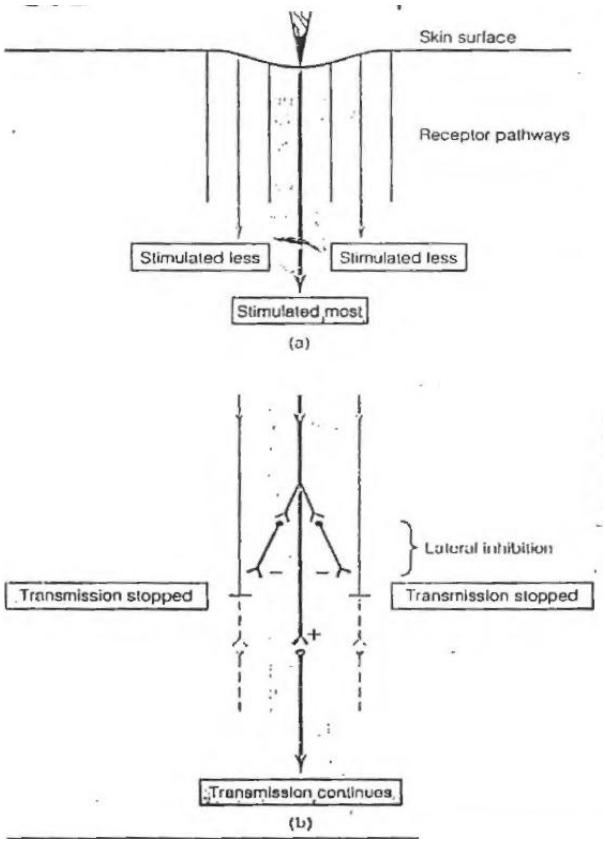
Explanation: as these impulses travel via gracile tract, the tract synapse at the level of L3 at nucleus of Clarke that sends out dorsal spinocerebellar tract partially to the cerebellum to transmit un**conscious** proprioception, where the rest of the tract travels to nucleus Z to transmits **conscious** proprioception.
(So gracile nucleus is now replaced by nucleus Z for the proprioception of the LL)

Q: a patient has lost his sense of position in his foot, the lesion could be in all of the following except?

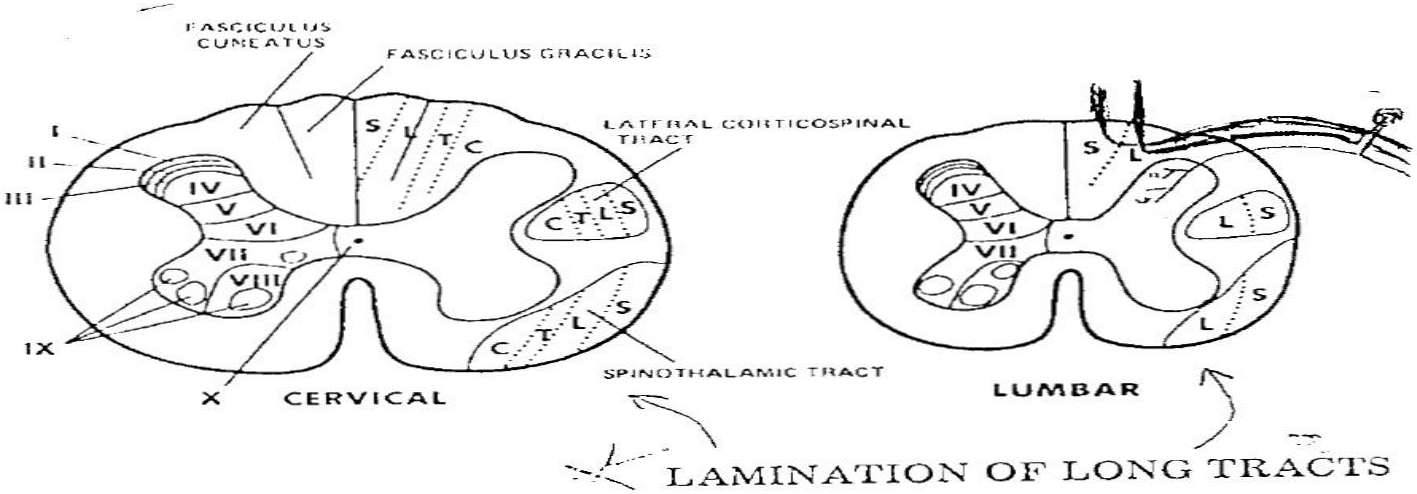
a)gracile tract b)nucleus Z c) dorsal spinocerebellar tract **d)gracile nucleus**

Lateral inhibition:

- If we have a pen pushing on your hand, we should have signals reaching the brain from the area found directly under the pen (strong stimulus) and from the area surrounding the pen tip (weak stimulus). If these two stimuli have reached the brain together, this will cause a disturbed signal interpretation (misleading perception for the site and intensity of the pen stimulus) so the brain needs to sharp the signal, and that is done by sending inhibitory signal through interneurons to the receptors surrounding the pen tip in your hand from the CNS, inhibiting the weak stimuli from reaching the brain, and that leads to more precise sensation; this is called lateral inhibition.
- **The inhibitory signals are sent from the CNS and not from the receptors themselves.**



- This phenomenon has a critical role in vision: it sharps the image and makes it more intense.



These section in both cervical & lumber regions.
In the cervical region: we have both gracile (medially)& cuneate (laterally) tracts, while the spinothalamic tract is inversely arranged.
In the lumbar region: we have only the gracile tract

· comparison	· spinothalamic tract	· dorsal column tract
· Types of fibers	· A delta & C → slow	· A beta (ii) → fast
· Blocking the tract	by : spinal cord endogenous analgesic system or brain analgesic system	· We cannot
· Receptive fields	· large	· small
· accuracy	· low	· high

- Finally : lesions of the spinal cord & peripheral nerves ;
- Any peripheral nerve (sensory, motor or sympathetic) lesion .E.g.if sensory fibers are affected all types of sensation are lost, also if motor fibers are damaged , it causes flaccid paralysis (LMNL). If sympathetic fibers are damaged, you'll find that the affected limb is dry and red (vasodilation). Peripheral nerve injury may be due to many diseases, especially diabetic neuropathy.
- Dorsal root injury; dorsal root is a sensory expansion → loss of all types of sensation in the dermatome supplied by the dorsal root.
- Spinal cord injuries: whatever is partially or completely damaged, the effect appears below the injury level, neither the sensory goes up, nor the motor goes down.
  At the level of injury we have two motor pathways (pyramidal & extra-) and two sensory pathways (spinothalamic & dorsal column).

℘ **Hemi-section injuryof the spinal cord**(As for bullet injury).

Also called →**Brown-Sequard syndrome**

- ☞ If we cut the spinal cord at the right side as shown in the figure (T10 level), it affects the dorsal column ipsilaterally below the level of damage, where the spinothalamic tract is affected contralaterally as it's decussates.

  According to the motor pathways, paralysis & paresis are ipsilaterally below injury level.
- ☞ Hyperaesthesia is above level of lesion .

  (↑ Sensitivity to touch). (i.e. simple touch is felt as pain)
- ☞ At the levelof lesion → alpha & gamma fibers of more than one segment (ventral horn) & ventral and dorsal roots are damaged→ loss of all types of sensation and **flaccid** paralysis (LMNL).
- ☞ **Spastic** paralysis (hypertonia) with hyper-reflexia ipsilaterally **below**the level of injury (UMNL), as both motor pathways are affected. (Babinski +)

  Why? Medullary reticulospinal tract is not active.

  (Normally it reduces the tone)
- ➢ **See the figure below (very important)**

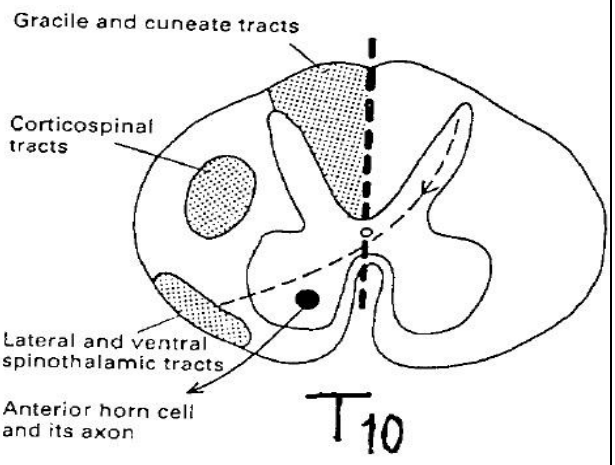
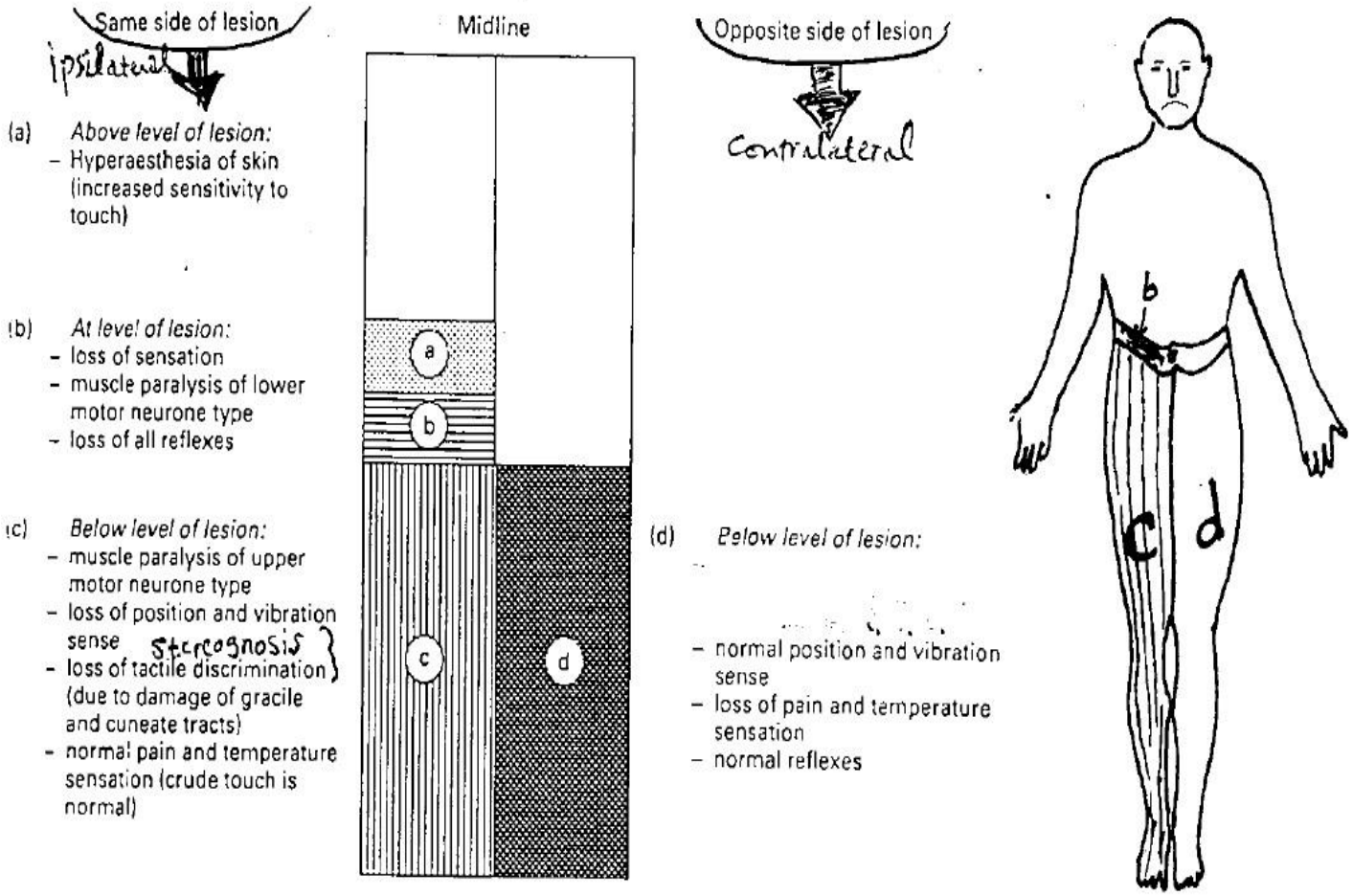


Fig. 17.18 Brown-Séquard syndrome. The pathways (tracts) damaged in Brown-Séquard syndrome are shown by the shaded areas. The thick broken line indicates the limits of the lesion.



A schematic diagram showing the manifestations of Brown-Séquard syndrome.