CENTRAL NERVOUS SYSTEM

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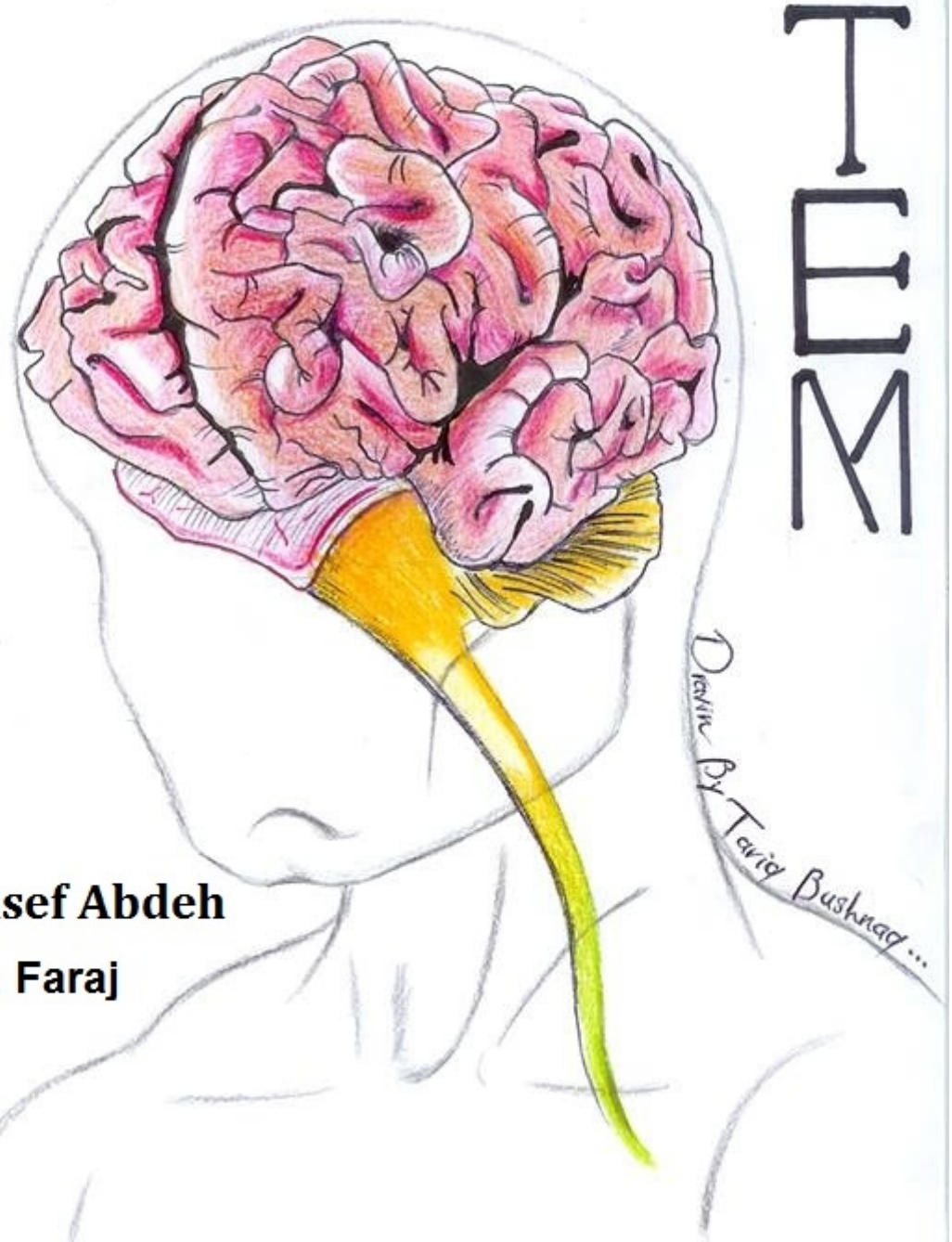

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Sensory Pathway (cont'd)

Hello, a great deal of information has been rearranged in the making of this sheet to ensure that it goes as easy as a glove in the hand "hopefully".

Referred Pain:

As for today, we'll start our talk with examples of referred pain & that you should know the table below like anything.

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

Notes:

1) **Heartache:** like in cases of, angina pectoris, ischemia or MI will refer to the chest wall in the precordium (through segments T2/T3/T4/T5), might also refer to the inner aspect of the arm (through segment T2, specifically through the intercostobrachial nerve) and finally to the epigastrium.
2) **Appendix:** will refer to the umbilicus; innervation of both the appendix & the skin around umbilicus is the same (through segment T10).
3) **Small intestines:** will refer to the umbilicus and the periumbilical region in specific.
4) **Central part of diaphragm:** will refer to the tip of shoulders; innervation of both the central part of diaphragm & the tip of the shoulder is the same (through segment C4 mainly).
5) **Pleura:** will refer to the upper abdominal region; innervation of both the pleura & the upper abdominal region is the same (through the intercostal nerves).
6) **Kidneys:** will refer to the loin i.e. costovertebral angle.
7) **Ureters:** will refer to the testicles in males.
8) **Trigone of bladder:** post. surface of bladder will refer to the tip of penis in males.
9) **Tongue:** will refer to the ear; innervation of both the tongue & the middle ear is the same (through the glossopharyngeal nerve "CN IX").
10) **Teeth:** will refer to the head and face; innervation of the head, face & teeth is the same (through branches of the trigeminal nerve "CN V").

11) **Hip:** will refer to the knee; innervation of both the hip & knee is the same (through the femoral, obturator & sciatic nerves).
12) **Uterus:** during labor or due to menstrual periods, pain will refer to the lower back & abdomen.

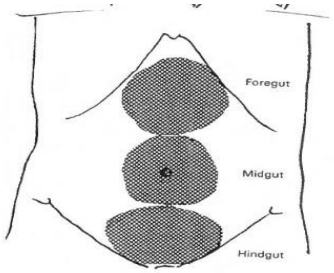
Examples:

1) A patient comes to you complaining of pain in his shoulder & an x-ray, CT & MRI of the shoulder revealed nothing so the patient is having a lesion either in the liver or the gallbladder which irritates the diaphragm or peritoneum.
2) A child with severe pain in the knee, you examined the knee both clinically and radiologically & you found nothing so the patient is having a problem in the hip.
3) A child is presented to you with fever, upper abdominal pain & a productive cough so pneumonia might be affecting the pleura which in turn refers to the upper abdominal area.
4) A child with acute tonsillitis might come crying from pain in the ear, a patient with carcinoma in the post. third of the tongue might also come complaining of pain in the ear too.

Visceral Pain:

Visceral pain can be classified as follows:

I. **Pure visceral pain:** felt at the site of the affected organ. Example: the pain experienced as the initial intestinal, biliary or ureteric obstruction. A case of appendicitis where pain is felt in the right iliac fossa (at Mcburney's point).
II. **Viscerosomatic pain:** caused by the spread of visceral disease to somatic structures. Example: another case of appendicitis in which the inflammation has spread to the parietal peritoneum of ant. abdominal wall where a reflex contraction of muscles there would result in severe rigidity in that region.
III. Now regarding the last type:
Referred visceral pain: refers to the body's wall (chest/ abdomen) having the same nerve supply as mentioned previously.



Visceral referred pain from the gastrointestinal tract.

Notes on the above figure:

Pain in the gut might refer to these midline areas as follows:

1) Pain from the Foregut (stomach, gallbladder, liver & pancreas) → Patient complains of pain in the epigastrium.
2) Pain from the Midgut (small intestines, part of large intestines & appendix) → Patient complains of pain in the periumbilical region.
3) Pain from the Hindgut (left colic flexure, descending & sigmoid colon, rectum & anus) → Patient complains of pain in the hypogastria.
    - A question is why do these pains refer to the midline? Recall back from GI's embryology, the gut at the 5th week was a midline structure "before rotation" & it received its innervation during that time so the brain stored this information as it is i.e. that the gut is a midline structure.

Applied Anatomy:

1) Myocardial ischemia (infarction), in addition to all the previously mentioned referral sites we also add the dorsal roots of spinal nerves entering into the inter-scapular region & the lower jaw as well. Sometimes pain starts as a pure visceral one in the epigastrium.

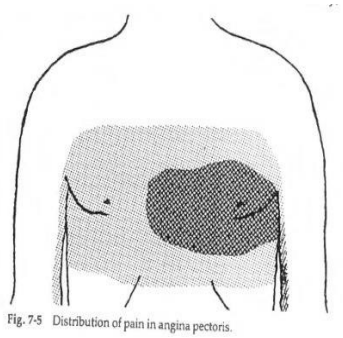


Fig. 7-5 Distribution of pain in angina pectoris.

2) Ureteric colic, pain will refer to the lower abdomen or external genitalia; all are supplied by nerves of L1 & L2 (iliohypogastric & ilioinguinal nerves). Once the patient has a stone & it starts descending, he will feel pain that goes down which is really a good sign; it indicates that the stone is getting towards the urinary tract.

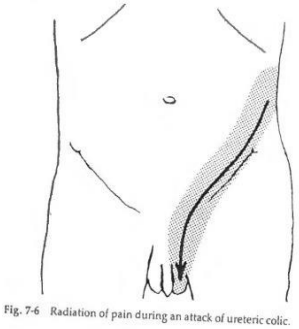
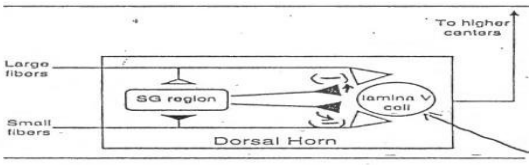


Fig. 7-6 Radiation of pain during an attack of ureteric colic.

Endogenous Pain Relieving System & Gate Control Theory:



This system aids in overcoming pain in our bodies through :

1. **Gate control theory**, which states that " At the level of spinal cord we can block pain & prevent it from ascending to higher levels."

   Now regarding the way it works:

   ➔ We knew that the mother cells from which the spinothalamic tract originates from are present within laminae 1 & 5 for the spinoreticular tract are from laminae 1 & 2. We consider these neurons here as a "gate", if you stimulated these neurons, the gate would open & nerve impulses would be transmitted along the spinothalamic tract to the brain ultimately. While if you inhibited these neurons, the gate would close & no impulses would ascend to the brain. Now the question is, how do we open/close these gates? i.e. how do we stimulate/inhibit neurons within this laminae? In the dorsal root of the spinal nerve we have 2 kinds of fibers: Large ones conducting touch & Small ones conducting pain. Both types of fibers will enter the dorsal horn of gray matter & synapse there exciting neurons within laminae 1,2 & 5.

   Touch fibers, which are large ones, stimulate "Substania Gelatinosa" but before that occurs S.gelatinosa, neurons in lamina 2 and might be also found in 3 too, would inhibit large & small fibers (HOW?) → Through "Pre-synaptic inhibition" i.e. axons of S.gelatinosa will be at the terminal parts of large & small fibers banning the release of transmitters.

   <u>Bottom line:</u>

   i. large fibers excite S.gelatinosa → excited S.gelatinosa inhibition on large fibers will be increased so the effect on laminae 1, 2 & 5 is decreased, the gate is now closed & no impulses will reach higher centers so you don't feel pain anymore.
   ii. Small fibers inhibit S.gelatinosa → inhibited S.gelatinosa, which inhibits laminae 1 & 5, will stimulate neurons in lamina 5, the gate is opened & impulses will reach higher centers and pain is now felt.

One should ask himself, how are these mechanisms kept under balance? Let's take an example where you feel cold & your hands are freezing and you start rubbing them. What you are doing is that you are stimulating touch fibers which in turn made you feel less pain & that if pain is of

small magnitude while if it was of higher magnitude previously mentioned gates would open and you'd feel pain.

2. **Tracts from higher levels,** Coming up contralaterally from both sides of laminae 1,2 & 5 are the spinothalamic & spinoreticular pathways.

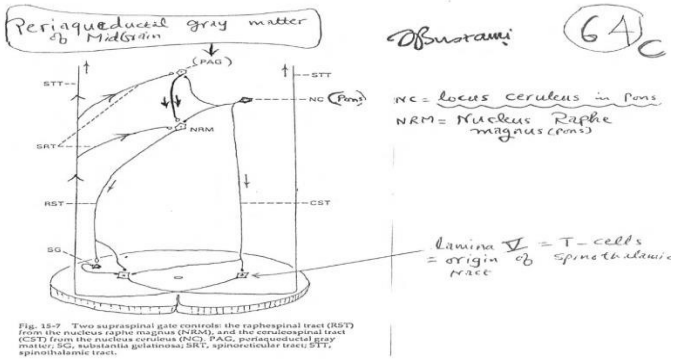


Fig. 15-7 Two supraspinal gate controls: the raphespinal tract (RST) from the nucleus raphe magnus (NRM), and the ceruleospinal tract (CST) from the nucleus ceruleus (NC). PAG, periaqueductal gray matter; SG, substantia gelatinosa; SRT, spinoreticular tract; STT, spinothalamic tract.

If we trace these two pathways along the figure above, we'll see that they will reach & stimulate the "Periaqueductal gray matter" found around the cerebral aqueduct of midbrain which sends impulses projecting to "Nucleus Raphe Magnus, NRM". Descending from this nucleus impulse going down to S.gelatinosa & from S.gelatinosa to laminae 1,2 & 5 inhibiting these neurons. So the beginning is by stimulating the periaqueductal gray matter through the ascending pathways.

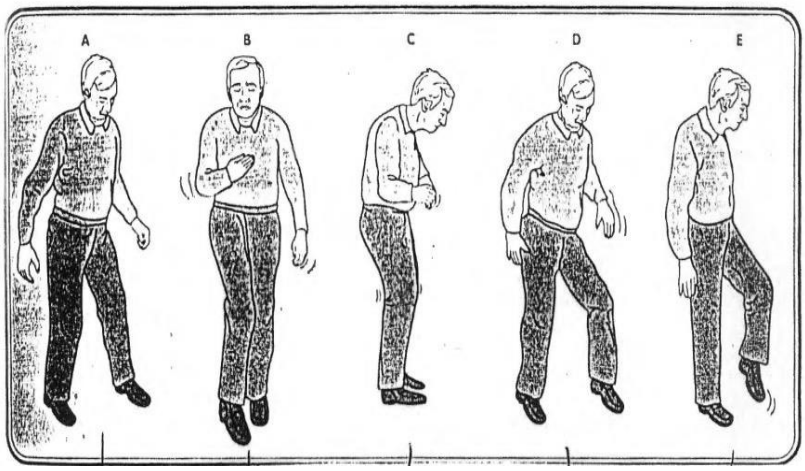
We do also have a tract of unknown function which is called the "Spinomesencephalic", some says that it goes along the spinoreticular one, reaching the mesencephalon stimulating cells there. Impulses descending from the periaqueductal gray matter to NRM then to S.gelatinosa if stimulated, will inhibit neurons of laminae 1,2 & 5 blocking pain from ascending to higher centers.

Another station is in the pons called "Locus Cerulleus", it's been said that a tract descends directly from it to laminae 1,2 & 5 inhibiting them.

"A comparison between both descending tracts"

	the Raphe-spinal tract	the Ceruleo-spinal tract
Origin	Originates from NRM.	Originates from Locus Cerulleus.
Site	Present in the midbrain.	Present in the pons.
Course	NRM → S.gelatinosa → Laminae 1,2 & 5.	Locus Ceruleus → directly to laminae 1,2 & 5.

Disturbances of gait:



A. cerebellar ataxia,
   Cause: A lesion affecting the flocculonodular lobe of cerebellum causing disturbance to spine muscles maintaining posture.
   Clinical picture: Patient stands & walks on a wide-based gait to maintain his balance & posture.
B. Hemiparetic (Hemiplegic),
   Cause: Stroke.
   Clinical picture: increased tone in flexors of upper limb and extensors of lower limb. In addition to that the patient can't bend his leg on knee joint.
C. Parkinsonism gait,
   Clinical picture: Stooped posture, loss of arm swing, shuffling or festinating steps in addition to difficulty in starting & terminating a move & in rotating his body to either sides.
D. Sensory ataxia (Stamping gate),
   Cause: Loss of conscious pro-prioception due to degeneration of peripheral nerves or in the dorsal root & it's central extension "gracile & cuneate"
   Clinical picture: Stamping gait.
E. Unilateral foot drop (Steppage gait),
   Cause: A lesion in the common peroneal nerve leading to paralysis of the ant. & lateral compartments of the leg.
   Clinical picture: Unable to perform dorsi flexion and eversion so presents with planter flexion and inversion.

F. Bilateral foot drop,
Cause: Disc prolapsed, vertebral disc compressing on L5 root.

***Note on point (D):** Tabes Dorsalis, which is a complication of an old STD "syphilis", the problem arises in the dorsal root and it's central process which continues as gracile & cuneate. At a point in time, patient would have sensory ataxia; degeneration occurs to the central process of the dorsal root so no gracile & cuneate.

Steps for examining the nervous system of a patient:

1. Examine muscles of both upper & lower limbs & check if there is wasting or not.
2. Examine the power of muscles "actively", like if you suspect a patient is having weakness in his right hand, you ask him to shake hands with you & then you compare it with the opposite hand.
3. Examine the muscle tone "passively", like if you suspect a patient with hypotonia (flaccidity) or hypertonia (spasticity), you start to move the limbs passively & compare it with the other side, you either find resistance or not.
4. Examine reflexes (jerks), it's either (+) indicating normalcy or (++ or more) indicating hyper-reflexia.
5. Examine the planter reflex "Babinski's sign".
6. Examine movement co-ordination, you check for paralysis, mono/hemi/para/quadriplegia.
7. Examine Sensations as below,

Sense	Method
Touch	Moving a piece of cotton anywhere in the body.
Pain	Pin prick testing.
2 point discrimination	Putting 2 pins within a certain distance between them.
Temperature	Applying hot/cold test tubes on any part of the body.
Vibration	Putting a vibrating tuning fork on any bony prominence.
Sterognosis (Upper limbs)	Holding an object with the eyes being closed.
Graphesthesia (Lower limbs)	Drawing certain figures on thigh or foot with the eyes being closed.

Spinal Cord Injuries & lesions:

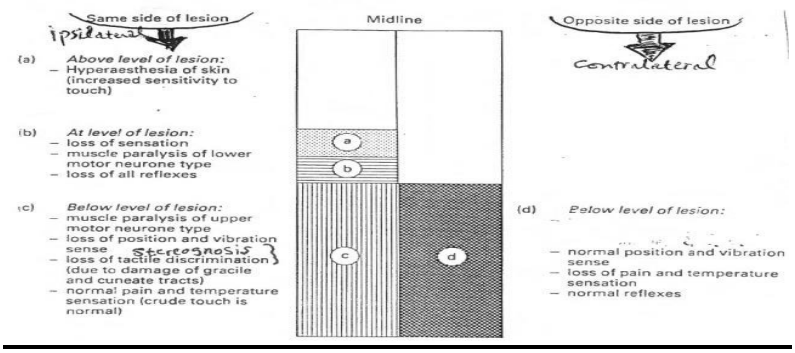
Spinal cord injuries show a combination of signs from both sensory & motor pathways injuries; from the white matter there are the descending motor pathways "pyramidal & extra-pyramidal" & going up is the spinothalamic tract crossing at the spinal cord & the dorsal column tract crossing at the medulla.

I. **Peripheral nerve injury** If there is a lesion in a peripheral nerve keeping in mind that every peripheral nerve carries motor, sensory and sympathetic fibers. How would a peripheral nerve be damaged?
   - ➔ Interruption of a peripheral nerve through cutting like in case of a wound passing by the ulnar or median nerves.
   - ➔ Degeneration due to demyelination disease like MS.
   - ➔ Degeneration to the nerve's axon.

A case of diabetes mellitus which causes peripheral neuropathy where we have degeneration of the axon . For unknown reasons, the patient will lose all types of sensations in the distal parts of both upper (gloves) & lower (stockings) limbs producing the clinical picture of glove & stocking anesthesia or hypoesthesia which includes: LMNs, atrophy & paralysis to muscles supplied by that nerve, the limb will turn red & it will be dry.

***Recall** that each peripheral nerve contains motor, sensory & sympathetic fibers so a damage to the motor part would cause LMNs while an injury to the sympathetic fibers would increase blood flow and decrease sweating in that limb.

II. **Brown-Sequard Syndrome** aka hemi-section of spinal cord. So a hemi-section of spinal cord on the right half would show results on both the ipsilateral & contralateral sides below the site of lesion as follows:



Notes:

-**Below the level of lesion ipsilaterally:** patient shows spastic paralysis, +ve Babinski's sign & hyper-reflexia due to damage occurred to both pyramidal & extra-pyramidal tracts. Patient also loses pro-prioception, sterognosis & 2 points discrimination due to

damage occurred to the dorsal column pathway. **While on the contralateral side** patient loses pain & temperature sensations due to damage occurred to the spinothalamic pathway.
-**Above the level of lesion ipsilaterally:** patient shows hyperalgesia for unknown reason.
-**At the level of lesion ipsilaterally:** patient shows signs of LMNLs i.e. paralysis & atrophy due to destroying alpha & gamma neurons.

III. **Syringomyelia,** A disease affecting females mostly in which there is an abnormal cavitation in the spinal cord not in the central canal it's either above it or below it. A cavity near the white commissure would affect the spinothalamic tract as it crosses at that part of the spinal cord. This cavitation would affect the decussating fibers of spinothalamic tract i.e. if this lesion is in the cervical part of spinal cord "where it occurs mostly" it would affect the upper limbs leading to loss of pain, touch, temperature from upper limb's skin while having a spared dorsal column a condition referred to as "Dissociated sensory loss".

Example: A middle-aged female is presented to you, complaining of burning her fingers while smoking. What you should be doing in the very first place is ordering a CT or MRI for the spinal cord & then you'd observe a cavity & it's a case of syringomyelia not a peripheral neuropathy due to DM. In addition to that she might show signs of LMNLs if it the cavity was big enough to compress the ventral horn.

IV. **Dorsal Root lesion,** the dorsal root which contains thick fibers for touch & thin ones for pain, so if the patient had a lesion in it he would lose all these sensations from the area of skin supplied by the dorsal root & he would also lose the reflexes as they are part of the dorsal root.

V. **Complete section of the spinal cord:**

a. **Thoracic cord lesion,** the effect is below the level of lesion, so affected lower limbs with normal upper limbs i.e. normal reflexes or in other words UMNLs; no pyramidal & extra-pyramidal reaching alpha & gamma & spasticity due to destruction of medullary reticulospianl tract, which decreases tone. We also lose all kinds of sensations & signs of sensory ataxia in lower limbs & also urinary retention.
b. **Lumbar & sacral lesions,** like in cases of crash injury where we destroy alpha & gamma neurons leading to LMNLs in lower limbs i.e. paralysis with atrophy & loss of sensation, areflexia & a sort of sensory ataxia.

Yours sincerely,

Yousef Abdeh.