CENTRAL NERVOUS SYSTEM

- [x] Handout
- [ ] Sheet
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- [x] Anatomy
- [ ] Physiology
- [ ] Pathology
- [ ] Biochemistry
- [ ] Microbiology
- [ ] Pharmacology
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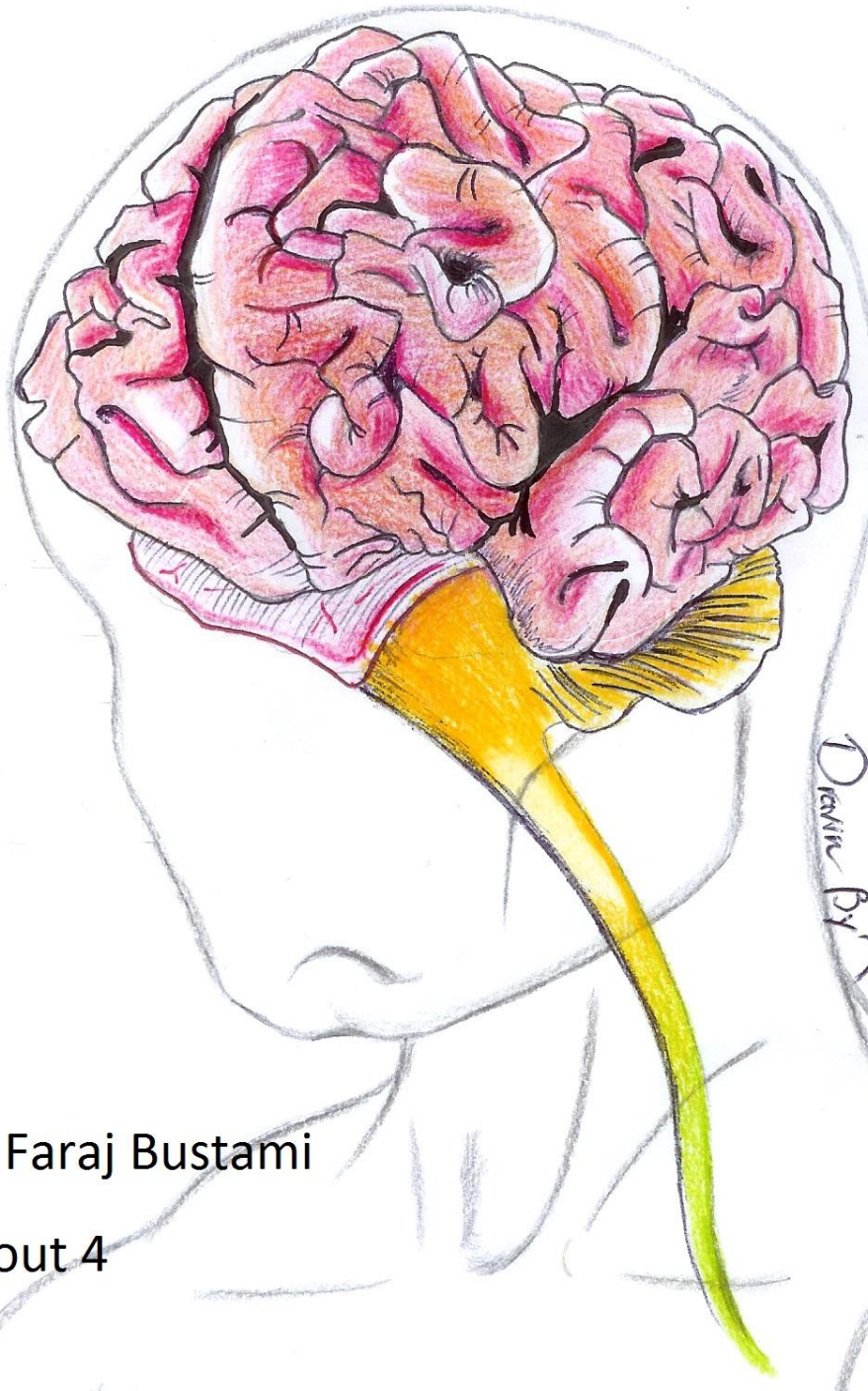

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Lec #: Handout 4

All encapsulated nerve endings are mechanoreceptors

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Table 5-1 Morphological types of cutaneous nerve endings, related to function

Morphological type	*Hairy skin*	*Glabrous skin*	*Sensory modality*
Free	+	+	Pain Touch Heat Cold
Applied			
follicular	+	–	Touch
Merkel's discs	+	+	Touch
Encapsulated			
Meissner	–	+	Touch
Ruffini	+	+	Stretch
Pacinian	–	+	Vibration

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⊕ L.f. vibration
⊖ H.f. »

SKIN RECEPTORS
Touch, Temperature, Pain, & Pressure

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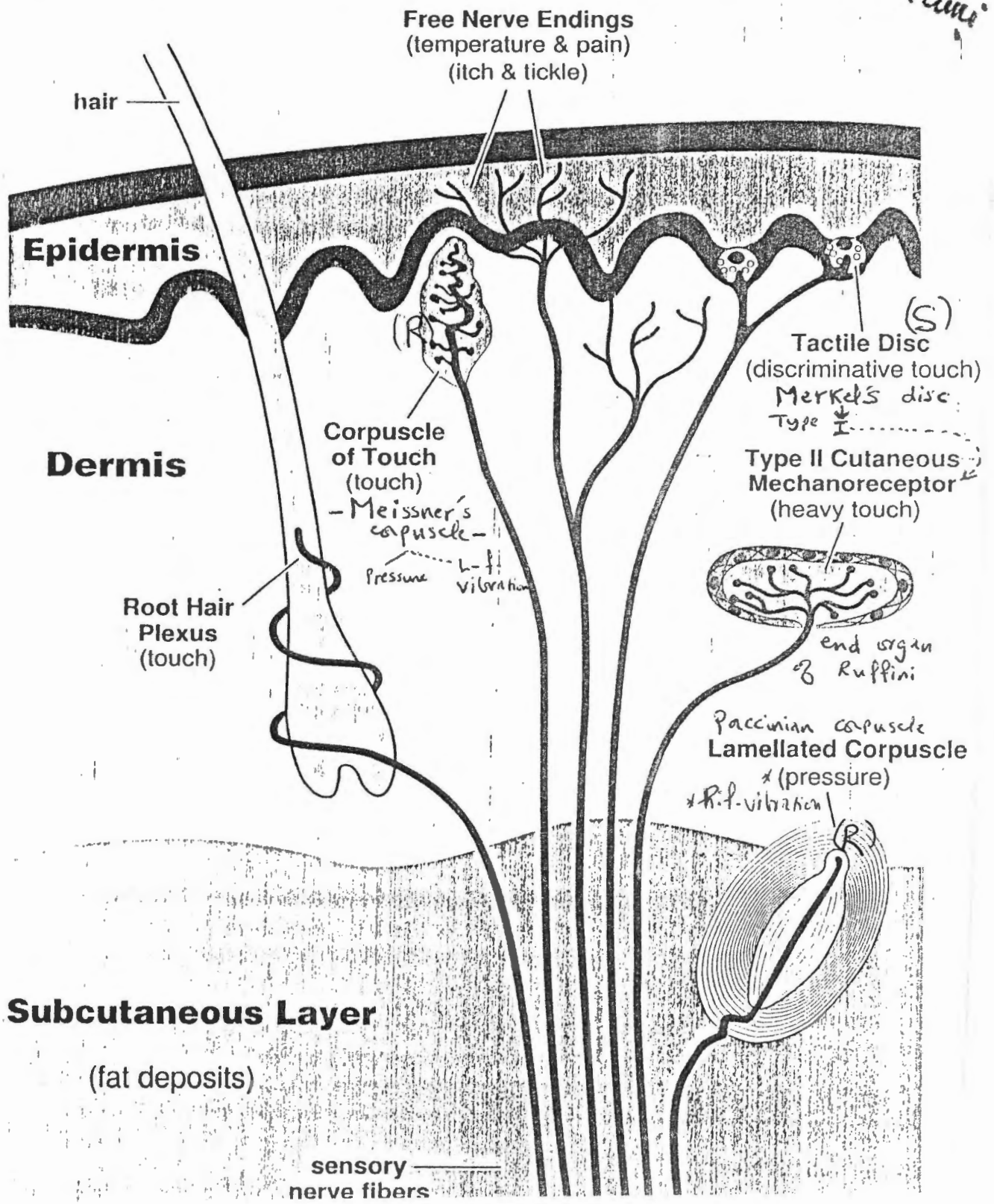


SENSORY SYSTEM / Skin Receptors

Receptors in the skin monitor 3 basic types of cutaneous sensations: tactile, thermal, and pain.

TACTILE SENSATIONS

Touch Receptors

Corpuscles of Touch (Meissner's corpuscles) : encapsulated nerve endings; rapidly adapting touch receptors that recognize exactly what point of the body is touched.

Root Hair Plexuses : dendrites arranged in a network around hair follicles; rapidly adapting touch receptors that detect movement when hairs are disturbed.

Tactile Discs (Merkel's discs / Type I Cutaneous Mechanoreceptor) : expanded nerve endings (flattened dendrites); slowly adapting touch receptors for discriminative touch.

Type II Cutaneous Mechanoreceptors (end organ of Ruffini) : expanded nerve endings embedded in the dermis; slowly adapting receptors that detect heavy and continuous touch.

Pressure & Vibration Receptors

Lamellated Corpuscles (Pacinian corpuscles) : oval structures composed of a connective tissue capsule, layered like an onion, that enclose a dendrite; rapidly adapting receptors that respond to pressure and high frequency vibrations.

Corpuscles of Touch (Meissner's corpuscles) : rapidly adapting receptors that respond to low frequency vibrations, as well as to pressure and touch stimuli.

Itch & Tickle Receptors

Free Nerve Endings Free nerve endings are the receptors for both tickle and itch sensations.

Adaptation *Rapidly adapting* receptors respond at the onset and removal of a stimulus with a burst of action potentials. *Slowly adapting* receptors respond throughout the duration of a stimulus with a sustained discharge.

Receptive Fields The receptive field is the region of the skin that is monitored by a given sensory receptor. If a receptor has a small receptive field it provides precise information about the shape and texture of the object indenting the skin. These receptors are highly concentrated at the finger tips. A large receptive field can cover a whole finger or part of the palm. These receptors respond to vibrations, stretching of the skin, and movement of joints.

THERMAL SENSATIONS (Thermoreceptors)

Free Nerve Endings The sense receptors for cold and warm are called thermoreceptors. They are free (naked) nerve endings.

Warm receptors are most sensitive to temperatures above 25 C (77 F); above 45 C pain receptors are stimulated (burning sensation). Cold receptors are most responsive to temperatures between 10 C & 20 C (50 — 68 F); below 10 C pain receptors are stimulated (freezing sensation). Both warm and cold receptors adapt rapidly; sensations disappear within minutes.

PAIN SENSATIONS (Nociceptors)

Free Nerve Endings The sense receptors for pain are called nociceptors. They are free (naked) nerve endings located between cells of the epidermis. Nociceptors respond to all types of high intensity stimuli and stimuli that cause tissue damage.

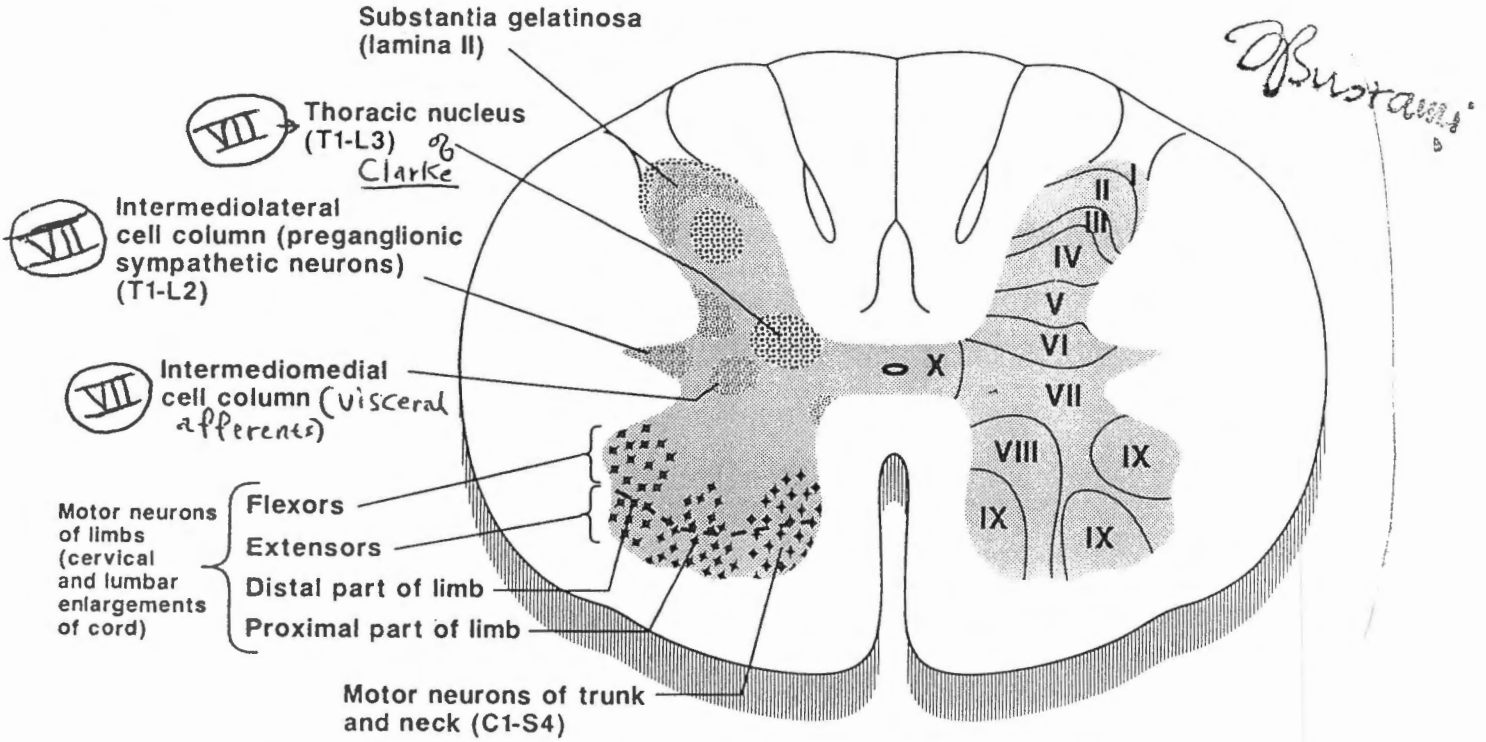
Architecture of the Spinal Cord Gray Matter

Neurons of the spinal cord gray matter are arranged in longitudinal columns according to similarity in appearance and function. In transverse section these cell columns appear as layers or laminae. This laminar scheme, as described by Rexed, is more useful in functionally organizing the gray matter than the older method of giving separate names to each of the cell columns (or nuclei). However, a few of the latter are worthy of continued use, and these will be mentioned in association with the laminae in which they reside. The laminae are numbered by Roman numerals, beginning at the tip of the posterior horn and moving anteriorly into the anterior horn (Figure 2).

Lamina I is a thin layer of neurons capping the posterior horn. It receives some pain and temperature afferent fibers from the dorsal roots and contributes some fibers to the contralateral *spinothalamic tract*.

Lamina II corresponds to the Substantia gelatinosa. It receives considerable input relating to pain both from the dorsal root afferents and higher centres (brainstem). Its neurons DO NOT contribute to the ascending (1st) Pain Pathway (spinothalamic tract) however, they can modify the transmission of Pain sensation.

Figure 2 **Composite spinal cord section with nuclei on the right and laminae on the left**



Laminae III & IV → (Nucleus Proprius) : receive large number of dorsal root Pain, temperature and touch afferents The dendrites of large neurons in lamina V reach these layers

Lamina V → receives dorsal root afferent fibres and interneurons from laminae II, III & IV.

→ Axons of its neurons CROSS TO THE OPPOSITE SIDE FORMING THE ↑ SPINOTHALAMIC TRACT (major part of) [Pain, temperature & light touch Pathway)

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Lamina VI is present mainly in the cervical and lumbosacral enlargements. It receives proprioceptive input from muscles.

Lamina VII contains several important nuclei as well as many interneurons. The *intermediolateral cell column* occupies and forms the *lateral horn* of the gray matter from T_1 to L_2 and consists of the cell bodies of preganglionic sympathetic neurons. The *intermediomedial cell column* is present throughout the spinal cord and receives visceral afferent fibers. The *thoracic nucleus* (formerly known as the nucleus dorsalis or Clarke's column) is present from T_1 to L_3 and receives proprioceptive afferent fibers from neuromuscular and neurotendinous spindles. Axons of these cells form the ipsilateral *posterior spinocerebellar tract*. The *sacral parasympathetic nucleus* is present from S_2 to S_4 and consists of preganglionic parasympathetic neurons.

Lamina VIII receives descending fibers from the vestibulospinal and reticulospinal tracts involved with muscle tone, postural adjustments, and reflexes. These cells project, both ipsilaterally and contralaterally, to laminae [illegible] IX.

Lamina IX consists of groups (nuclei) of somatic efferent neurons whose axons leave the spinal cord in the ventral roots to supply skeletal muscles. The more medial nuclei supply the muscles of the trunk and are present at all spinal cord levels. The lateral nuclei supply the limb muscles and are present only in the cervical and lumbosacral enlargements. Both alpha and gamma motor neurons are located here.

Lamina X surrounds the central canal and is composed of decussating axons, neuroglia, and interneurons.

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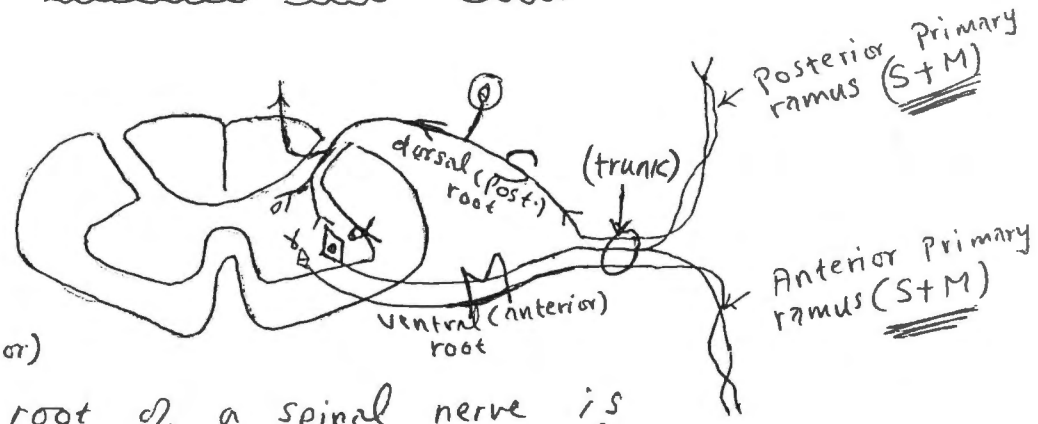


Remember : (Posterior)

① the dorsal root of a spinal nerve is formed of sensory (afferent) nerve fibres and their cell bodies are within the dorsal root (spinal) ganglion

② the ventral (anterior) root is formed of the motor axons of α & γ motoneurons

③ the trunk of a spinal nerve → mixture of sensory & motor fibres

④ the anterior and posterior primary rami Each is formed of ← Sensory nerve fibres, Motor " ", Sympathetic " "

⑤ Sensory fibres (afferents) bring sensation from skin, muscles & joints

⑥ motor fibres (efferent) supply skeletal muscles

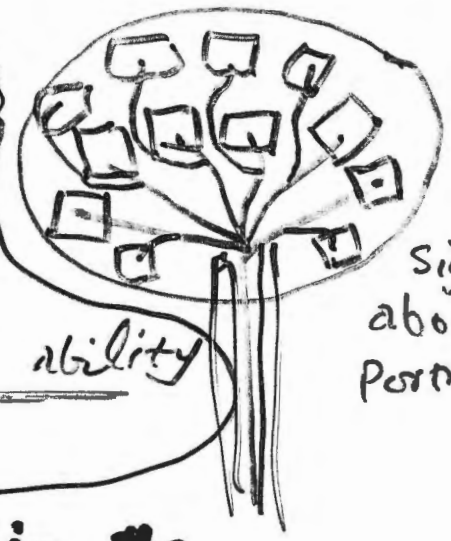
Receptive fields at the tips of fingers are small

↓

Each neuron signals information about small discrete portions of the fingers

1

The smaller the receptive field → the greater its acuity or discriminative ability



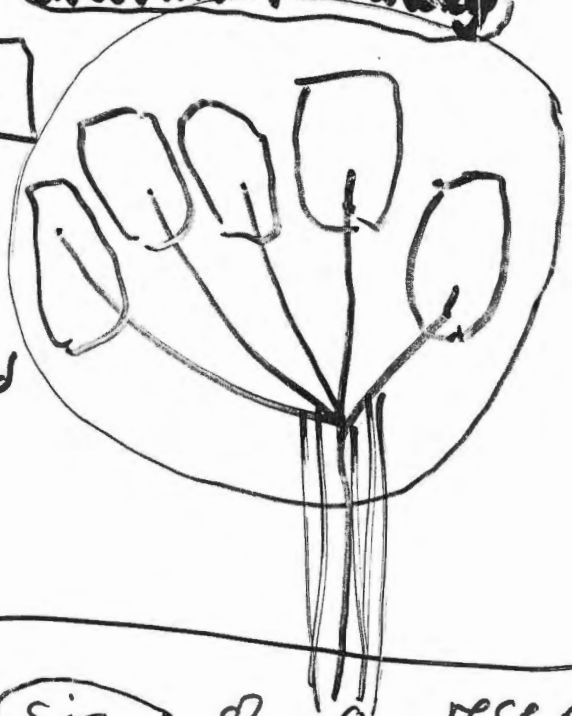
Each Receptor in the skin interacts with the CNS through a distinct pathway

↓

Labelled Line

↓

Activation of sensory receptor produces the same sensation independent of the stimulus that activated the receptors



larger receptive field at elbow

2

The size of a receptive field varies inversely with the density of receptors in the region

* 3 **Cortical representation** corresponds to innervation density

↓

more cortical space from areas with smaller receptive field

Classification of Somatic Senses

3 physiological types

1. **Mechanoreceptive** somatic senses
   - tactile sense
     - touch
     - pressure
     - vibration
     - tickle
   - Position Sense (frequently called proprioceptive sense)
     - Static Position
       - Conscious perception of the orientation of the different parts of the body with respect to one another
     - dynamic → Kinesthesia or movement sense
2. **Thermoreceptive** → heat / cold
3. **Nociceptive** (pain) sense
   - activated by any factor that damages tissues

- Anatomically the dorsal horn neurons can be classified as PROJECTING NEURONS & INTERNEURONS

- Projecting neurons → have direct projections to thalamus &/or medulla & Pons
  - Mostly located in lamina I
  - a small number = = V
- Neurons in laminae II & III (substantia gelatinosa) have few direct projection to the brain

Sensory Pathways

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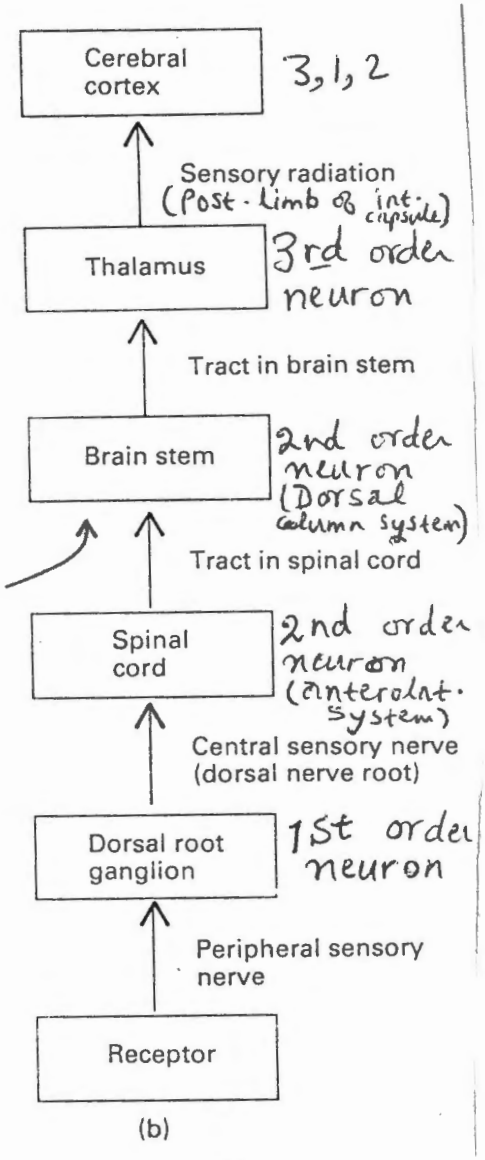
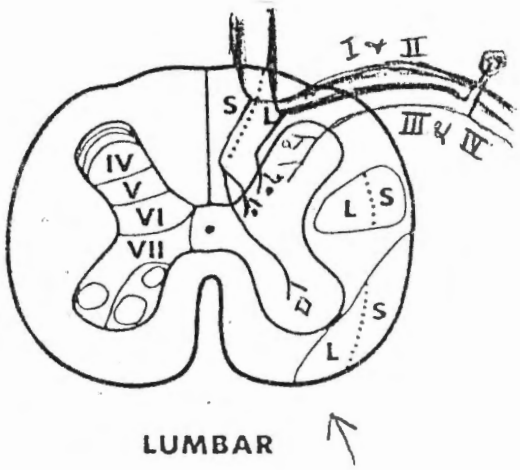




DORSAL Root entry Zone (between the end of the dorsal root & the apex of the dorsal horn)

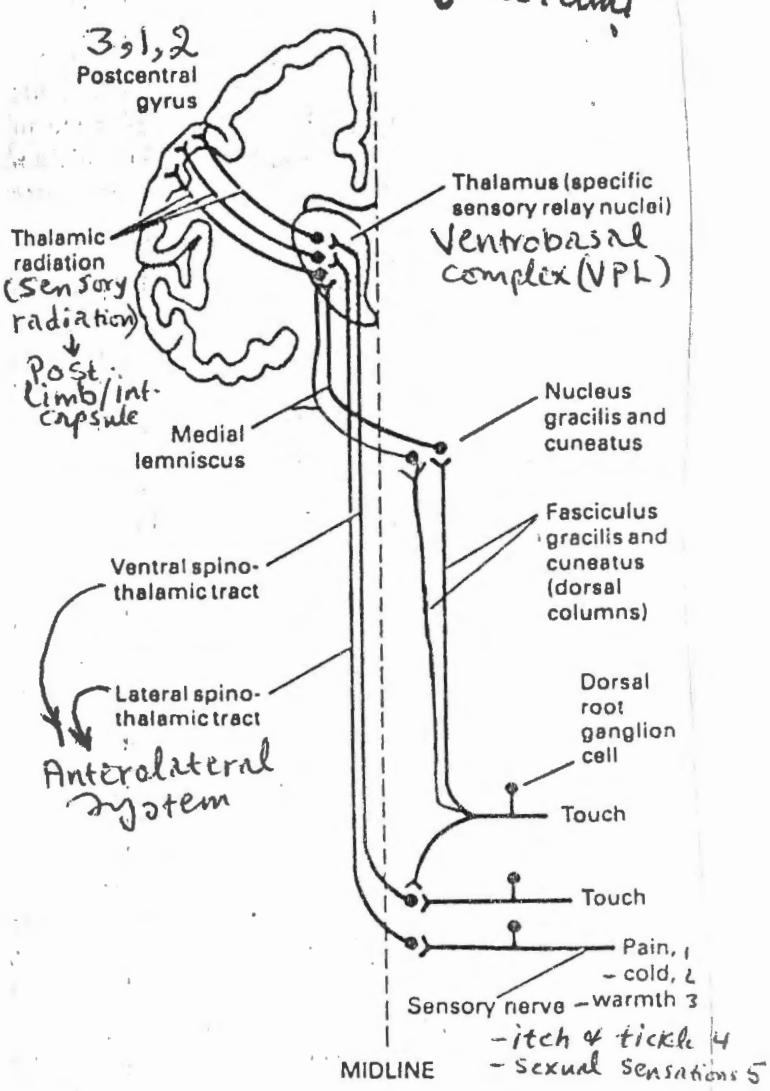
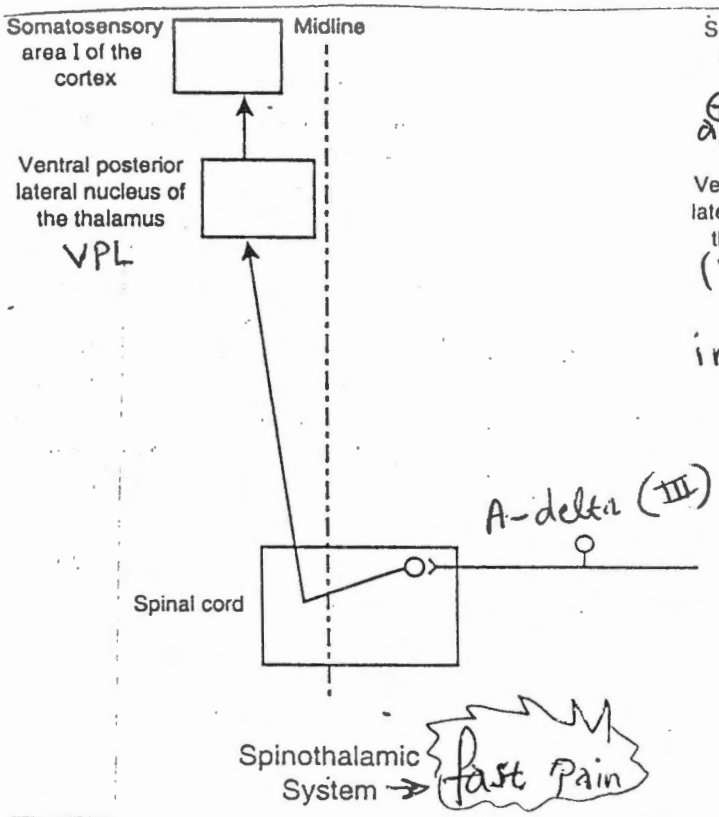
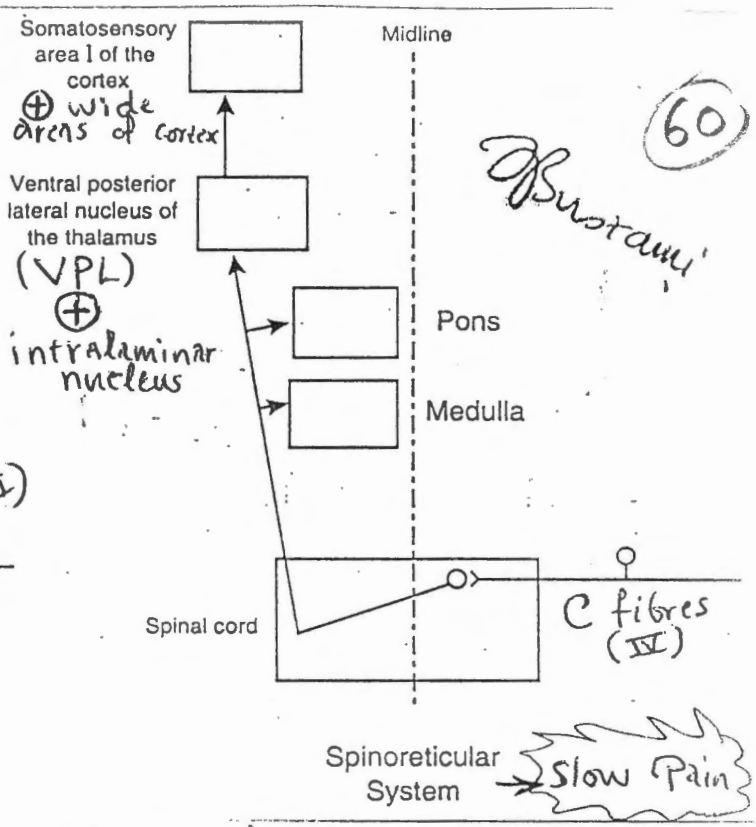


Figure 7–2. Touch, pain, and temperature pathways from the trunk and limbs. The anterolateral system (ventral and lateral spinothalamic and related ascending tracts) also projects to the mesencephalic reticular formation and the nonspecific thalamic nuclei.

Medial division → large myelinated fibres (I (Aα), II (Aβ))
↓
Carry Precise sensory informations (sense of Position (Proprioception) discriminative touch, Stereognosis...

lateral division small myelinated (Aδ = III) & Unmyelinated (C = IV)
↓
carry information about Simple touch, thermal sense, Pain & visceral sensation





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*Spinoreticular tract : C (IV) afferent fibres → cells of origin are located in laminae I & II → ascend to terminate in both reticular formation & thalamus (VPL & intralaminar nuclei). The intralaminar nuclei are part of the Reticular activating system (RAS) which Projects to wide areas of cerebral cortex → Thus they AROUSE one from sleep, create a sense of urgency and Promote defence reactions to rid the person of Pain.

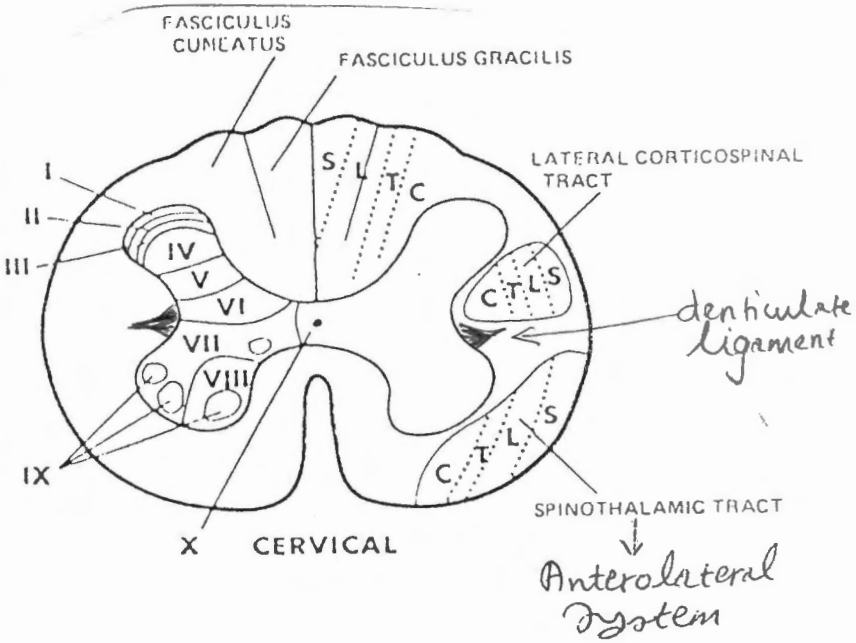
QUALITIES OF PAIN

Classically, the sensation of pain is subdivided into two components: ← fast (Pricking P.) slow (aching P.)

1 *Pricking pain*. This is often referred to as first pain. It is a fast acute sensation, which occurs within 0.1 s after application of a painful stimulus. It is usually well localized, the kind of sensation felt when a pin is stuck into the skin or the skin is cut with a knife. Pricking pain is usually superficial and is not felt in most of the deeper tissues. It is transmitted via type Aδ fibres.

2 *Burning or aching pain*. This is often referred to as second pain. It is a slow pain, which increases slowly over a period of many seconds or minutes. This component is the type that is difficult to endure and can occur both in the skin and in the deeper tissues. A good example is intestinal colic, toothache or a burn. Slow pain is transmitted by unmyelinated type C fibres.

The two qualities reflect not only the dual nature of the input (i.e. Aδ and C fibres), but also the two sets of connections within the nervous system.

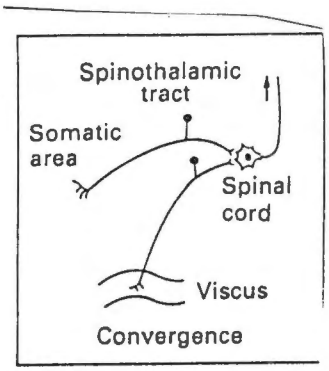


Pain and temperature (and light touch)
Spinothalamic system
(Anterolateral system)

Nerve fibres that carry { Pain (Nociception), temperature, light touch } form the lateral division of dorsal root and are formed of group III (A delta) fibres → ASCEND within Lissauer's tract for 1-2 segments → synapse with neurons in laminae I & V (These are considered as T or Transmission tract neurons) → Axons of these neurons CROSS at the White commissure of spinal cord → ASCEND through the Anterolateral white matter in medulla → Pons → midbrain → Terminate in VPL (Ventral Posterolateral) part of Ventrobasal complex of thalamus → 3,1,2

Mechanism of referred pain

Convergence and facilitation both play a part in the production of referred pain.



Convergence theory According to this theory, pain fibres from an area of skin and a diseased viscus supplied by the same spinal segment converge on the same second-order neurone in the dorsal horn (Fig. 17.22). The skin has a much richer nerve supply than any viscus, and it is more exposed to stimulation than any viscus. As a result, the somatosensory area of the cerebral cortex is more used to receiving impulses from skin than from a viscus. Thus, the brain misinterprets impulses coming along the common pathway as coming from the skin (i.e. the sensation is projected to the skin area) and not from the viscus.

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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

{Visceral pain is conducted along C fibres → those from thoracic viscera & lower pelvic viscera reach the CNS along parasympathetic nerves (Vagi & pelvic nerves) while those from the rest of the viscera reach the CNS through sympathetic nerves}

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.

According to the Site of Stimulation Pain can be classified into:
- Cutaneous
- deep somatic
- Visceral

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(1) **Cutaneous Pain** – Produced by stimulation of Pain receptors of the skin → Pain occurs in 2 phases: fast Pricking, slow burning
→ can be accurately localized (large number of receptors in the skin)

(2) **Deep Somatic Pain**
- Receptors are in deep structures: muscles, bones, joints, ligaments
- dull, diffuse & prolonged
- usually associated with autonomic Stimulation
  e.g change of heart rate & blood Pressure
  sweating; vomiting
- adequate stimulus for deep somatic pain
  - Mechanical → e.g severe Pressure on a bone, or a muscle traction
  - chemical → venoms
  - Ischaemia → angina pectoris in cardiac muscle
    → intermittent claudication in calf muscle

(3) **Visceral Pain**
- adequate stimulus
  - distension of hollow organs (the pain we experience when the urinary bladder is full)
  - Spasm of a viscus → ischaemia
  - Chemical irritants → HCl from a perforated gastric ulcer → severe pain
- Pain receptors in the Viscera → very few ??
  Pain is felt when there is extensive injury to the Viscera
- visceral pain is conducted along type C (IV) fibres
- Visceral Pain → poorly localized
  → often referred to other site
  Often associated with autonomic disturbances e.g vomiting, sweating, tachycardia
  may be associated with rigidity of nearby skeletal muscles

N.B Visceral Pain → referred to the dermatome (area of skin) supplied by the dorsal roots through which impulses from diseased structure reach CNS

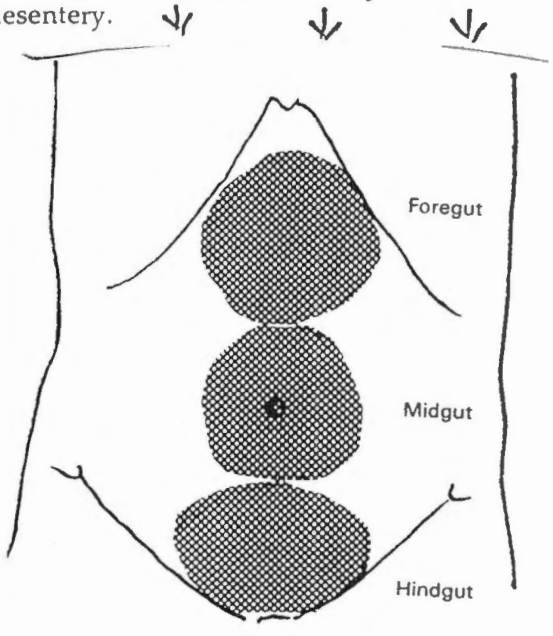
There are three fundamental types of visceral pain:
1 pure visceral pain, felt in the region of the affected organ,
2 visceral referred pain, projected into the somatic territory of the corresponding spinal nerves,
3 viscerosomatic pain, caused by the spread of visceral disease to somatic structures. e.g Parietal Peritoneum ← Reflex contraction of Ant. Abdominal wall muscles → Rigidity

Pure visceral pain

This is characteristically vague and deep-seated and often accompanied by autonomic responses (sweating or nausea). It is experienced as the initial pain in intestinal, biliary or ureteric obstruction, or when the capsule of a solid organ (liver, kidney or pancreas) is stretched by underlying disease.

Visceral referred pain

As its severity increases, visceral pain is 'referred' to somatic structures innervated from the same segmental levels of the spinal cord. For example, the pain of myocardial ischaemia is usually referred to the chest wall, biliary and intestinal colics are referred to the anterior abdominal wall, and labor pains are referred to the sacral area of the back. From the gastrointestinal tract, pain is referred to the anterior midline area: from foregut to epigastrium, from midgut to the periumbilical region, and from hindgut to hypogastrium (Fig. 7-3). This is because the visceral afferents invade the alimentary tract during the fifth week of embryonic life, at which time the primitive gut is attached to the posterior abdominal wall by a midline dorsal mesentery.



Visceral referred pain from the gastrointestinal tract.

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Myocardial ischemia

In acute myocardial ischemia the patient may initially experience pure visceral pain, expressed as a sense of acute epigastric discomfort. Referred pain usually supervenes or may be present from the beginning. Most commonly,

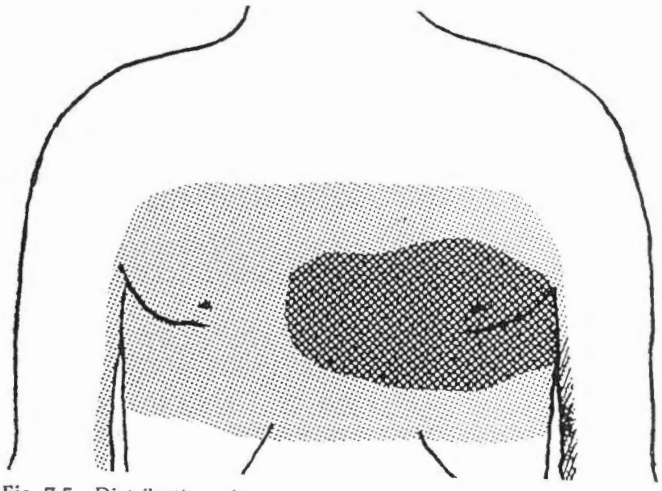

Fig. 7-5 Distribution of pain in angina pectoris.

spinothalamic neurons at T2–T5 levels are excited by cardiac C fibers traversing the sympathetic route. The stimulus is projected to the somatic territory of spinal afferents entering the cord at the same levels. The territory includes that of the intercostobrachial nerve (lateral branch of the second intercostal) which supplies the skin of the medial arm (Fig. 7-5). Reference along posterior rami may produce interscapular pain. Severe autonomic responses (sweating and pallor) are characteristic, and there may be a sense of impending death.

For no clear reason, pain of myocardial origin may be felt mainly or even entirely outside the thorax – notably in the epigastrium (where it may be interpreted as 'indigestion') or in the neck and lower jaw.

Ureteric colic

The passage of a stone down the ureter elicits intense peristaltic activity. The ureter is innervated by afferent fibers entering L1 and L2 segments of the spinal cord. The pain is excruciating – the patient rolls around the floor – and is referred to the territory of the iliohypogastric and ilioinguinal nerves: the loin, the groin and the scrotum or labium majus (Fig. 7-6). In males, the pain may be felt exclusively in the testis.

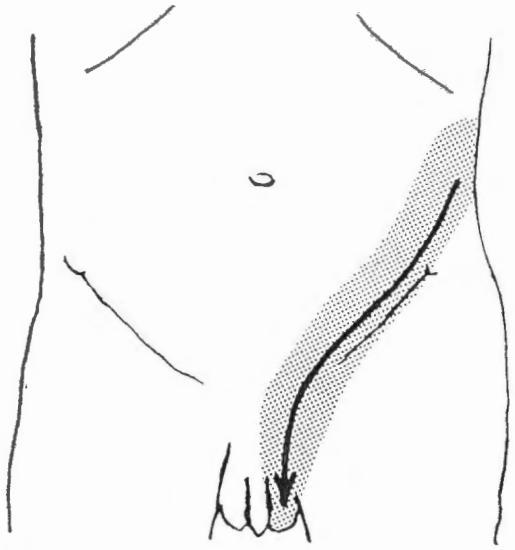

Fig. 7-6 Radiation of pain during an attack of ureteric colic.

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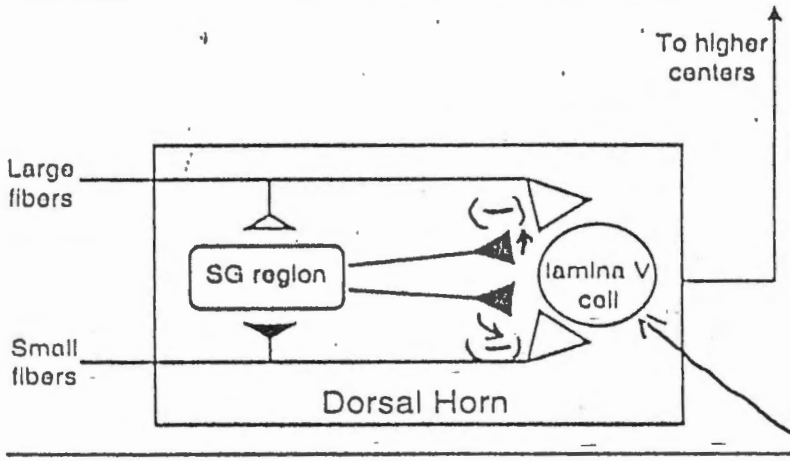
Fig. 7-12. The gate-control theory. Large and small fibers excite a lamina V neuron that projects to the brain. Neurons in the substantia gelatinosa (SG) inhibit transmission between large fibers and lamina V cells and those from small fibers and lamina V cells. Activation of large fibers excites SG cells and therefore increases their inhibitory effect on the lamina V cells and closes the gate. Activation small fibers inhibits SG neurons and removes their inhibitory effect on the lamina V neurons and therefore opens the gate. (Modified from: Melzak, R., and Wall, P. D. Pain mechanisms: A new theory. *Science* 150:971, 1965.)



T cells (origin of spinothalamic tract)

GATE CONTROL THEORY

In the dorsal horn of the spinal cord onward transmission of nerve impulses from nociceptive afferent fibres via **T or transmission cells** depends on the activity of large sensory afferent neurons from peripheral touch receptors. Impulses in these touch sensory afferents can block the pain pathway by stimulating an interneuron in the **substantia gelatinosa** which will presynaptically inhibit all input to the T-cell. If impulse traffic in the nociceptor afferents is greater than in the touch receptor afferents then afferent impulses will pass on and pain will be appreciated. This theory explains why rubbing your skin in a painful area can help to lessen the pain.

Periaqueductal gray matter of Midbrain

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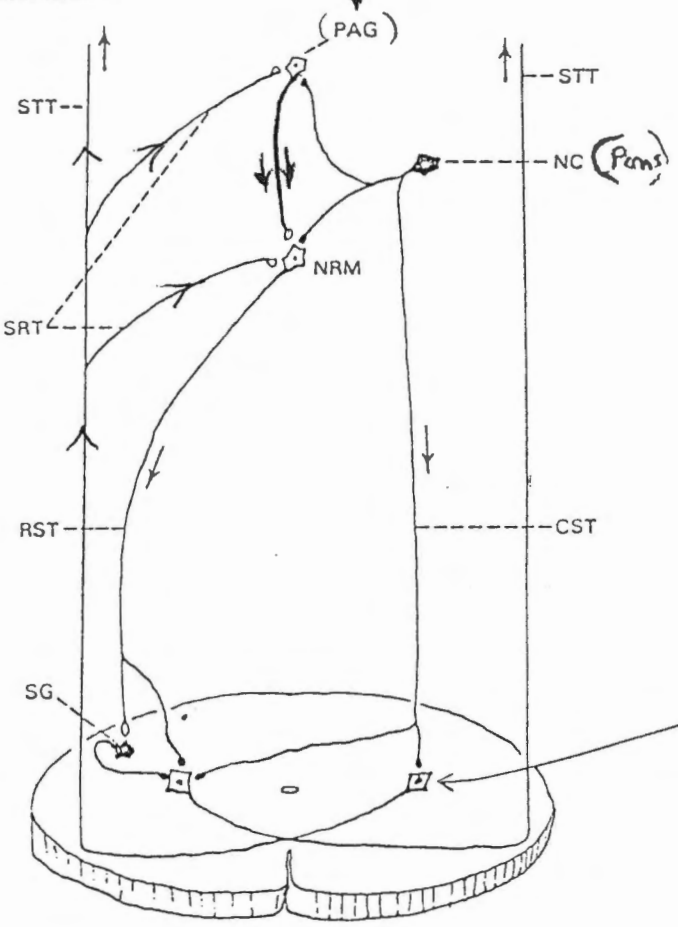
64 C



NC = locus ceruleus in pons

NRM = Nucleus Raphe magnus (pons)

lamina V = T-cells = origin of spinothalamic tract

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.

Ascending pathway

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

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.

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.

Endogenous Pain Control System

N.B Substantia gelatinosa = laminae II + III

Posterior Column Pathway

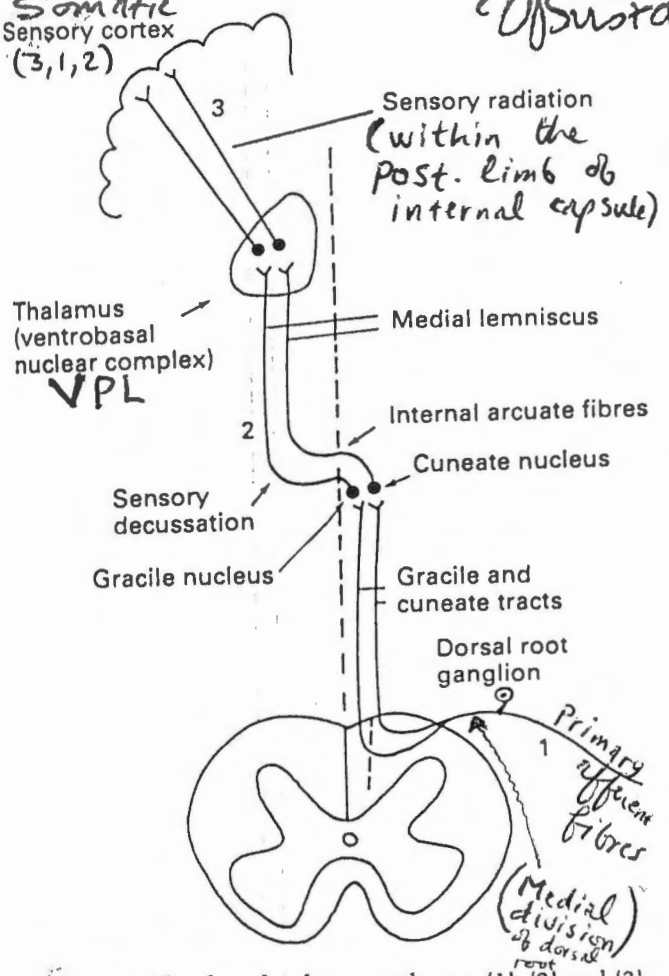


Fig. 17.14 The dorsal column pathway. (1), (2) and (3) refer to first-, second- and third-order neurones.

The paths of the three orders of neurones are as follows:

1 *First-order neurones.* Afferent fibres enter the spinal cord via the medial portion of the dorsal root. They enter the ipsilateral dorsal column and ascend upwards as the gracile and cuneate tracts to end, respectively, in the gracile and cuneate nuclei of the medulla.

2 *Second-order neurones.* These are located in the gracile and cuneate nuclei and give rise to axons, which cross to the opposite side, forming the sensory decussation. They then pass upwards as the medial lemniscus, which traverses the brainstem to end in the ventrobasal nuclei of the thalamus (VPL nucleus)

In its pathway through the brain stem, the medial lemniscus is joined by additional fibres from nuclei of the trigeminal nerve. These fibres subserve the same sensory functions for the head that the dorsal column fibres subserve for the body.

3 *Third-order neurones.* The ventrobasal nuclear complex of the thalamus gives rise to axons, which project in the sensory radiation to the somatosensory cortex (area 3,1,2)

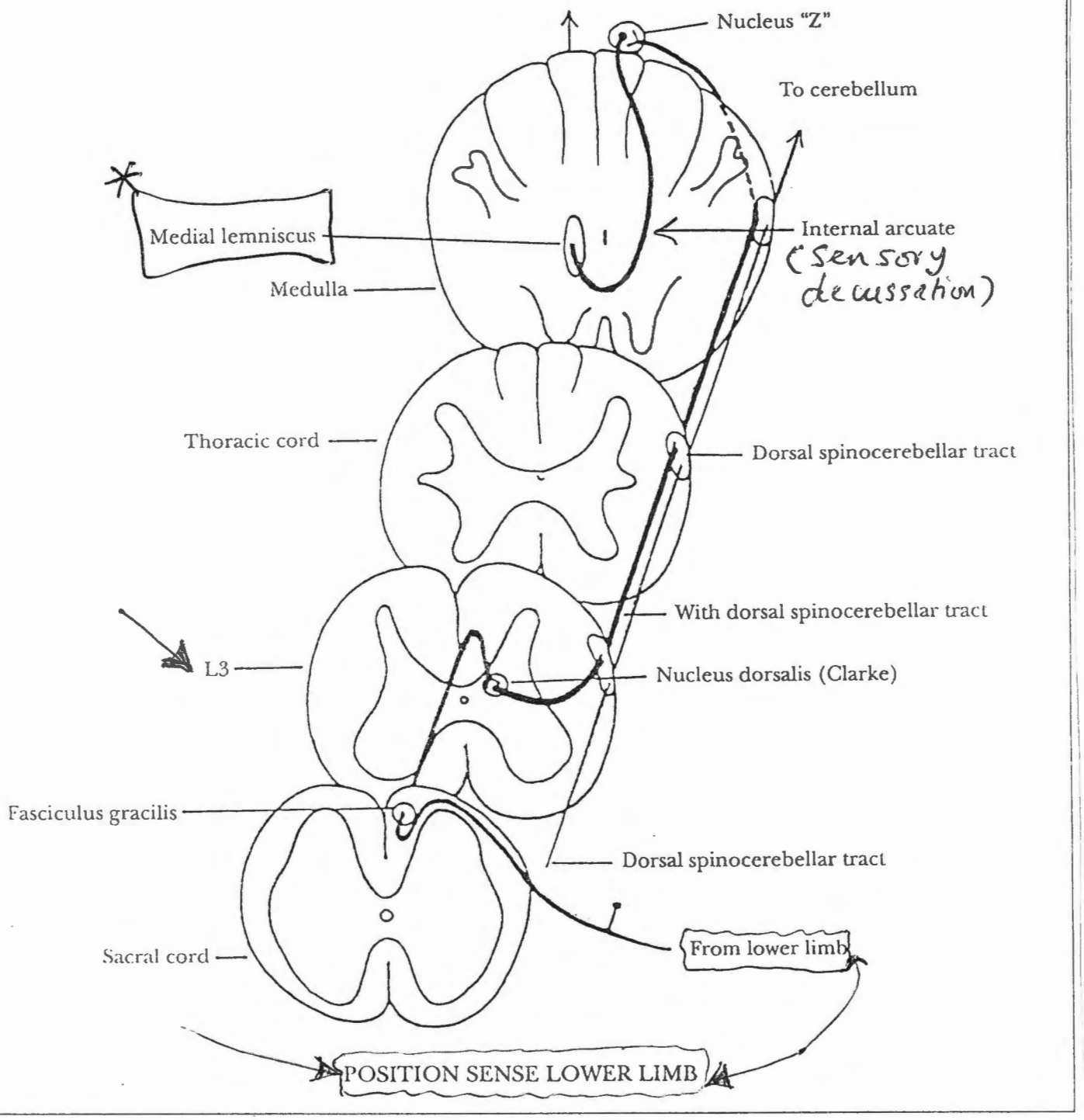
Dorsal Column - Medial Lemniscal Pathway : Concerned with

1. Discriminative touch → Precise localization of touch including two-point discrimination
2. Vibration → (Phasic sensation)
3. Position Sensations — Static, dynamic (Kinesthesia or movement sense)
4. Stereognosis
5. Pressure

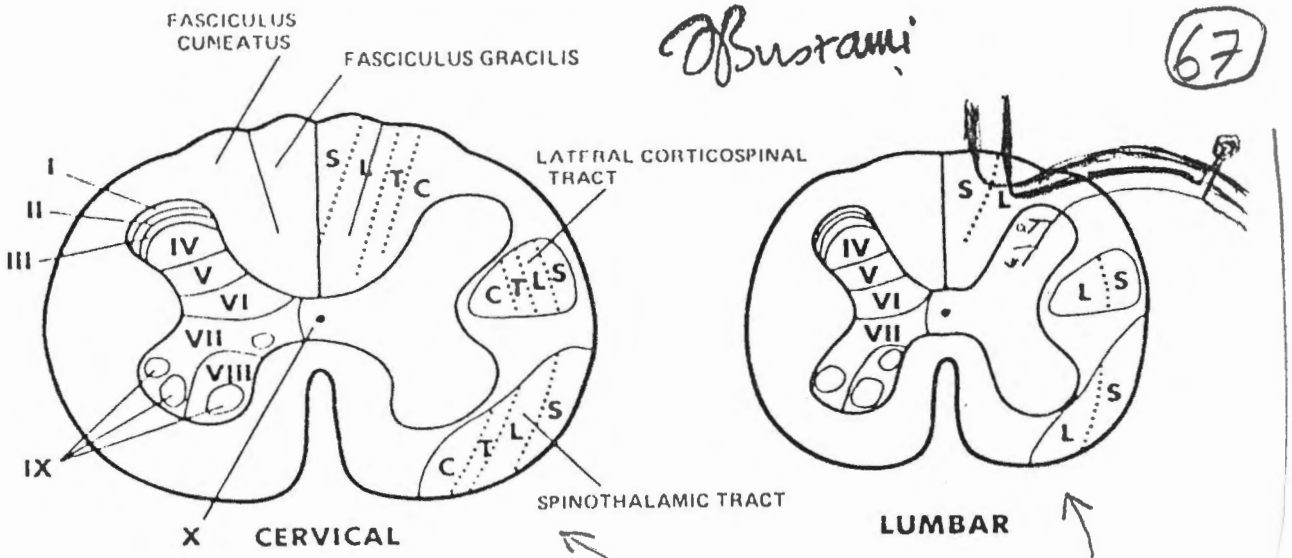
* Lesion of Post. Column - Medial Lemniscal System → Loss or diminution of the following Sensations:
- Vibration
- Position sense
- 2 Point discrimination (including Stereognosis)
- Touch

1. Vibration → tested by placing a vibrating tuning fork over a bony prominence
2. Position sense → tested by moving the tip of the patient's finger or toe dorsally or ventrally and asking the patient (whose eyes are closed) to identify the position of the part moved
3. Two-point discrimination → tested by simultaneously pricking the patient in 2 adjacent areas of skin. Under normal conditions a person is able to recognize these 2 stimuli as separate stimuli if the distance between them is not less than 5 mm on the fingertips and not less than 10 cm on the shin (front of leg)
4. Stereognosis the ability to recognize objects by touch without the aid of vision

Touch → tested by placing a cotton ball gently over the skin



The significance of all this is that this anatomy explains the persistence of lower limb proprioception when lesions affect the spinal cord dorsal columns above L3. A high posterior column lesion will result in loss of *stereognosis in both upper and lower extremities, but loss of position sense only in the upper extremities.* The gracilis nucleus is only concerned with stereognosis, the cuneate nucleus with both stereognosis and proprioception. Above nucleus gracilis and nucleus "Z", the path for *all* stereognostic-proprioceptive information is the same.



LAMINATION OF LONG TRACTS

Dorsal column lemniscal pathway	Anterolateral (Spinothalamic) pathway
(1) Is concerned with fine type of transmission	Is concerned with a cruder type of transmission
e.g. There is a high degree of spatial orientation of nerve fibres with respect to their origin on the surface of the body	e.g. Poor degree of spatial orientation
(2) Responsiveness is not greatly altered by stimuli from other areas of the nervous system	Responsiveness can be greatly altered by stimuli from other areas of the nervous system (i.e. brain and spinal cord analgesic system)
(3) Fibres from the lower parts of the body lie towards the centre, while fibres from higher levels form successive layers laterally	Fibres from the lower parts lie laterally, while those from higher levels form successive layers towards the centre
(4) Receives afferent sensory fibres of the dorsal root, which belong to type Aβ fibres, which ascend directly in the dorsal columns	Receives afferent sensory fibres, which are thin myelinated type Aδ fibres or unmyelinated type C fibres. These travel in Lissauer's tract and relay in the dorsal horn, to give rise to the anterolateral pathway
(5) Has a very fast velocity of transmission (i.e. 30–110 metres/s)	Has a relatively slow velocity of transmission (8–40 metres/s)
(6) Has great ability to transmit rapidly repetitive sensations (i.e. vibration sense)	Has poor ability to transmit rapidly repetitive sensations
(7) Is limited to transmission of mechanoreceptive sensations only (i.e. fine touch and pressure sense, vibration sense, position sense)	Has the ability to transmit a broad spectrum of modalities (i.e. pain, thermal sensation, crude touch and pressure, itch and tickle sensations, sexual sensations)

Role of the thalamus and the sensory cortex in the appreciation of sensation

All sensory tracts, except the olfactory pathway, synapse in the thalamus on their way to the cerebral cortex. When impulses mediating a given sensation reach the thalamus, the subject becomes crudely aware of the sensation but he cannot perceive all of its fine details: e.g. a person will be aware of a change in temperature if he contacts a hot object but he will not be able to indicate how hot the object is. Gradations and other spatial and temporal characteristics are appreciated at the level of the sensory cortex and not at the level of the thalamus (Fig. 17.16). Pain, however, seems to be the only sensation that seems to be fully appreciated at the thalamic and probably even at the reticular formation level or even lower. Still, interpretation of the quality and localization of pain occurs at the level of the cerebral cortex.

↑ ↑

The Somatosensory cortex (area 3,1,2)

* at Post-central gyrus (Parietal lobe)
* Stimulated by impulses arriving from the contralateral half of the body with the Exception of the face which is bilaterally represented in both hemispheres.
* body → represented upside down (legs on top and head at bottom of gyrus)
* representation of body parts → related to the density of receptors in the Pare and not to its size → lips represented by the greatest area, followed by the face & thumb.

Sensory cortex → concerned with 3 discriminative functions

(1) Spatial recognition This includes tactile Localization (localization of the site of Stimulus) and two-point discrimination ⇒ both are lost in a lesion of Post. columns and somatosensory cortex

(2) *Recognition of relative intensities of different stimuli.* An increase in intensity of stimuli is transmitted to the brain in the form of an increase in the number of afferent fibres stimulated and increased frequency of action potentials in these fibres. These two features are perceived as an indication of the strength of the stimulus.

(3) *Stereognosis.* This is defined as the ability to recognize objects by touch without the aid of vision. Loss of this ability is called *astereognosis*, which may occur due to a dorsal column or a parietal lobe lesion. Due to the former, other sensations subserved by the dorsal columns are also lost (i.e. position, vibration and fine pressure sense). When it is due to a parietal lobe lesion, position sense and light touch are normal but tactile discrimination is lost.

(I) Protopathic sensations

These are CRUDE Sensations that are perceived by the thalamus. They include Crude Pain & tactile sensations & extremes of temperature (above 38°C is perceived as hot and below 24°C is perceived as cold & between these 2 temperatures, the thalamus is thermally insensitive ⇒ the protopathic sensations are of high threshold (require strong stimuli to be produced)

(II) Epicritic (Cortical) sensations

These are fine sensations that are perceived by the cerebral corter e.g
- tactile localization & discrimination
- Stereognosis
- fine grades of temp.
- these sensations of low threshold

In discussing features that accompany disordered function of the sensory system, it is helpful to recall that, at the level of the spinal cord: (i) the spinothalamic pathway is crossed; and (ii) the dorsal column pathway is uncrossed. It is also of importance to note that crossing of the dorsal column lemniscal system occurs higher up in the medulla. Due to the crossing of the two major sensory tracts, sensory information from one half of the body goes to the cerebral hemisphere of the opposite side.

Localization of the site of lesion in disorders of the sensory system

Lesion of a peripheral nerve In such a case, all sensations are lost in the area supplied by the nerve. When many peripheral nerves are diffusely affected, as in polyneuritis or polyneuropathy, all forms of common sensation are impaired in the distal parts of the limbs (e.g. glove-and-stocking anaesthesia).

Lesion of the dorsal root Here, all sensations are lost in the relative dermatome, i.e. area of skin supplied by the dorsal root. The tendon reflexes mediated by fibres in the root are also lost.

Spinal cord lesions The features in spinal cord lesions depend on the location of damage. The three commonest lesions are:

1 *Brown-Séquard syndrome (hemisection of the spinal cord).* In this condition one-half of the spinal cord is damaged (Fig. 17.18). The patient will show the following:

(a) Sensory disturbances at the level of the

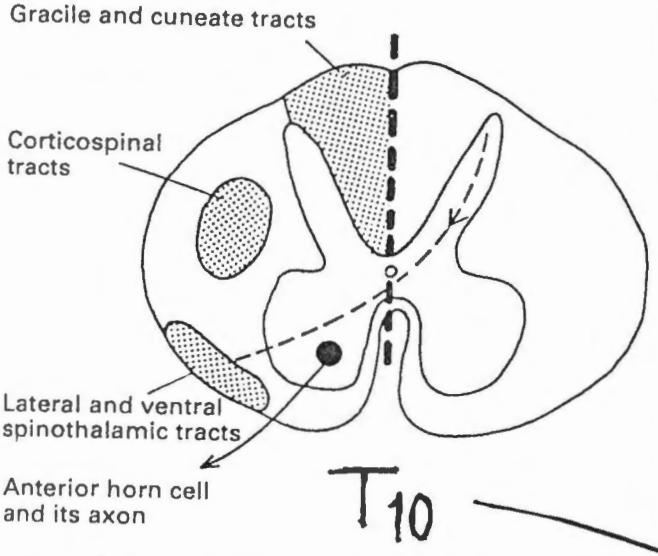
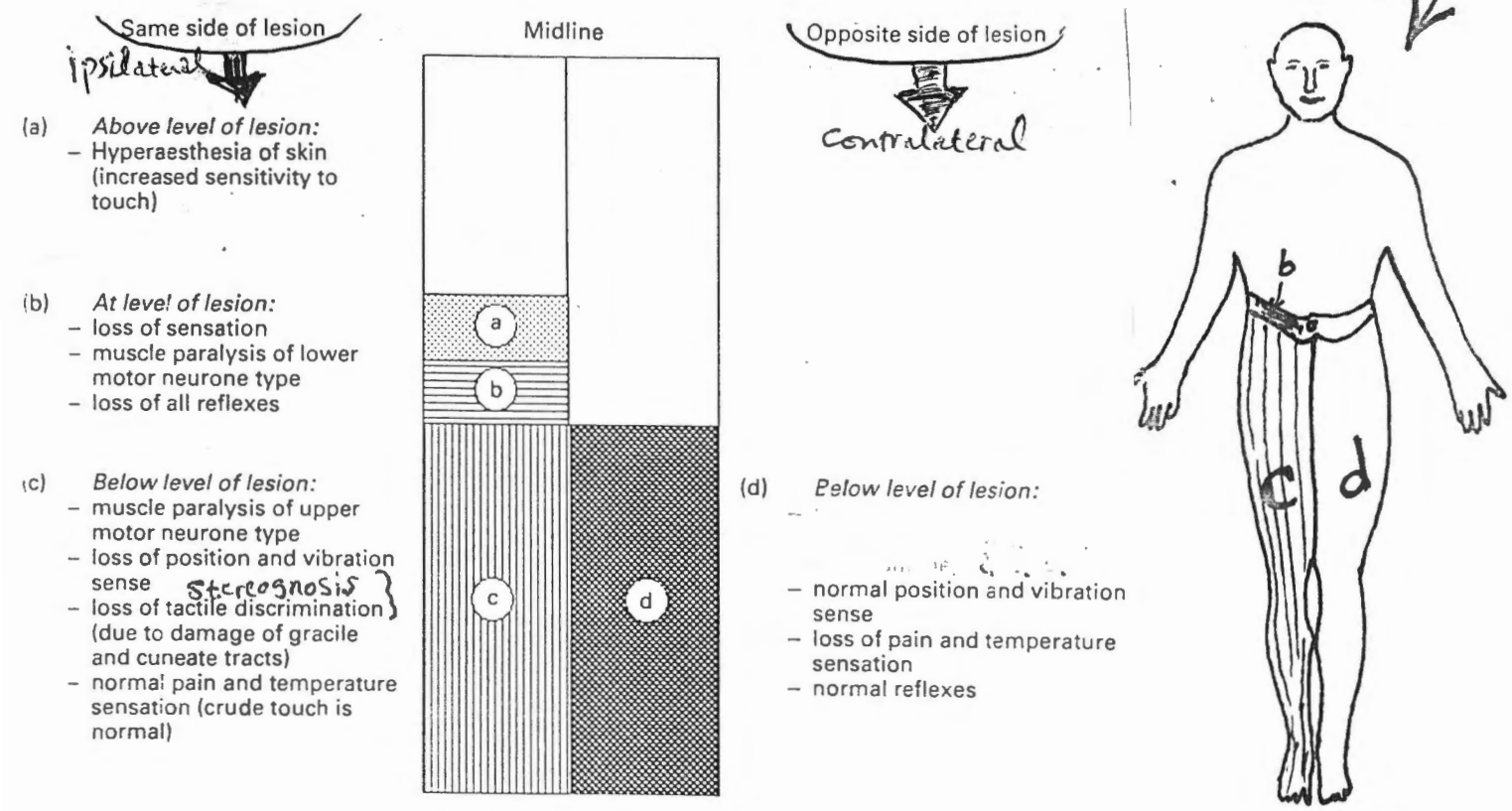


Fig. 17.18 Brown-Séquard syndrome. The pathways (tracts) damaged in Brown-Séquard syndrome are shown by the shaded areas. The thick broken line indicates the limits of the lesion.

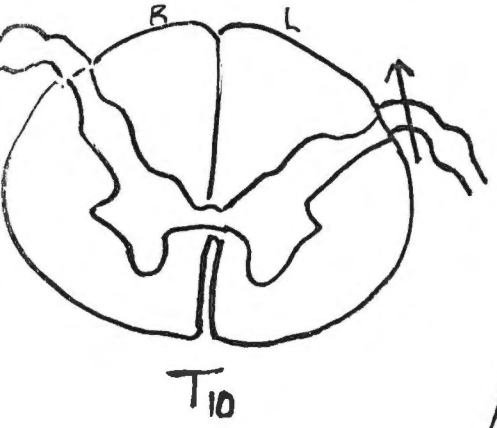


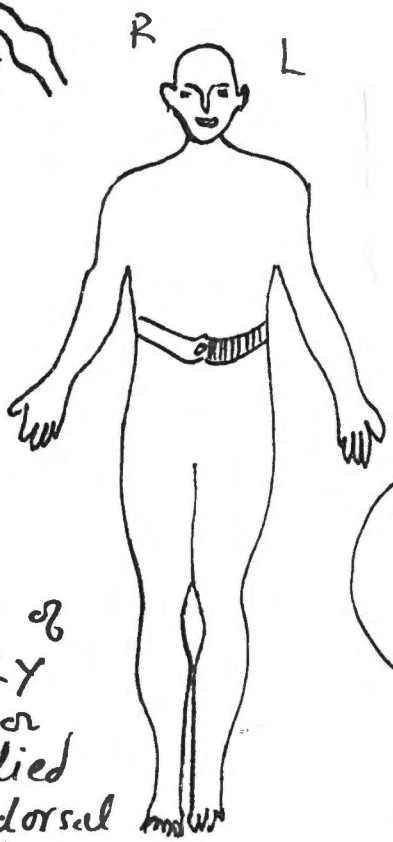
A schematic diagram showing the manifestations of Brown-Séquard syndrome.

lesion: loss of all sensations from the area supplied by the dorsal roots that enter the spinal cord at the damaged segments on the ipsilateral side.

(b) sensory disturbances [below] the level of the lesion → loss of sense of position + movement (Kinesthesia), loss of stereognosis + 2 point discrimination ON IPSILATERAL SIDE of lesion
- Loss of pain + temperature sense on the contralateral side

Motor disturbances include
- LMNL (lower motor neuron lesion) i.e Flaccid paralysis (at) the level of the lesion on the same side
- ipsilateral UMNL (upper motor neuron lesion) i.e Spastic Paralysis (BELOW) the level of the lesion
  i.e - Paresis or paralysis
  - spasticity
  - hyperreflexia
  - +ve Babinski sign
  - clonus



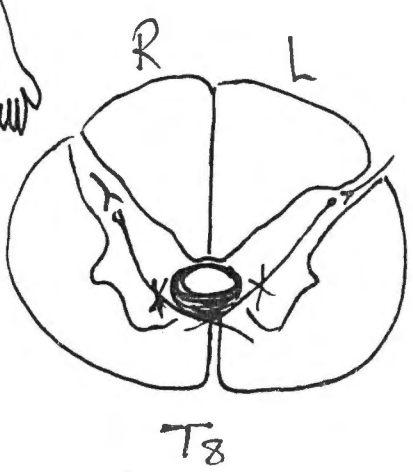


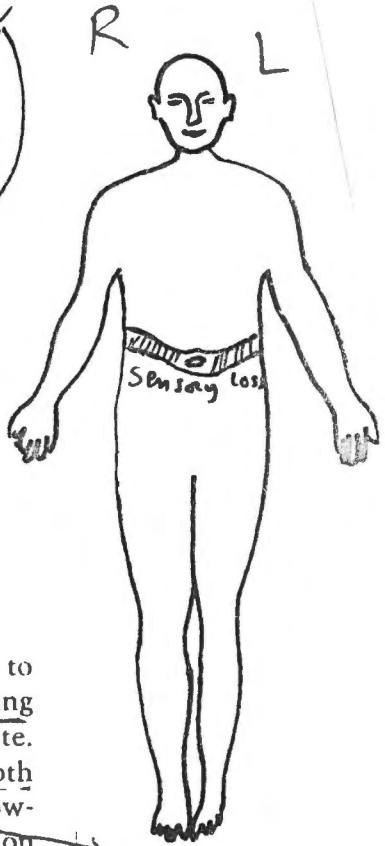
Bustami

(b) Lesion of the dorsal (Posterior) ROOTS

⇓

Loss of all types of Sensations in ONLY the dermatome or dermatomes supplied by the affected dorsal roots. Thus if a lesion involves dorsal root T10 → sensory loss in the skin around the umbilicus on the SAME SIDE as the dorsal root affected





3 *Syringomyelia.* In this condition, damage is to the central part of the cord, where the crossing fibres of pain, temperature and touch decussate. This leads to loss of these sensations on both sides of the body at the [ ] segments. However, fine touch including tactile discrimination and position sense are not affected, as they are carried in the dorsal column/lemniscal pathway. Thus, the result is dissociated sensory loss.

Key

Clinical detail

Throughout the text, simplified outlines of the major anatomical–clinical syndromes are presented diagrammatically following the conventional neurological examination of cranial nerves, motor function, reflexes, sensation and coordination and mental state described in textbooks of clinical methods (Fig. 1.26).

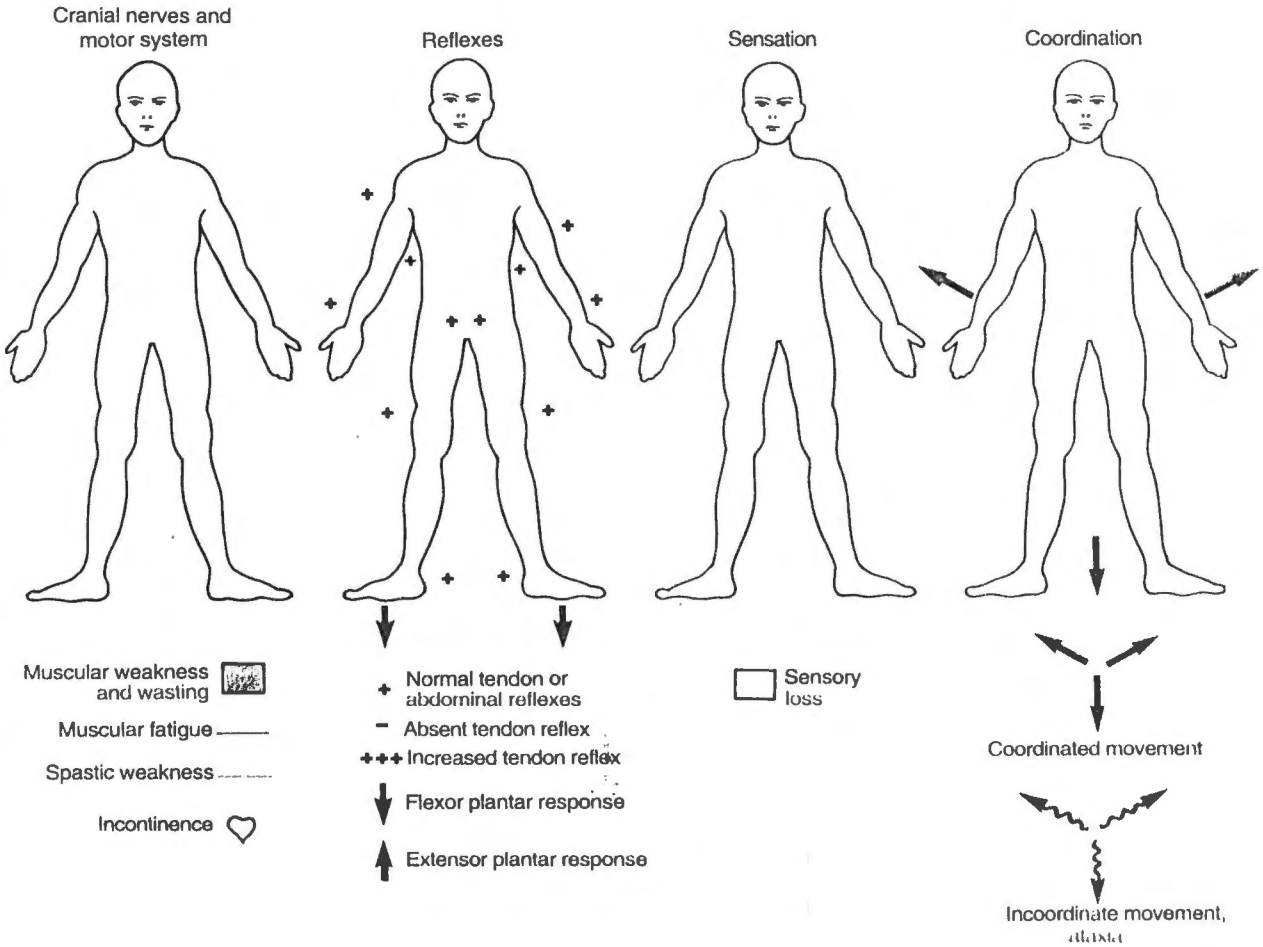
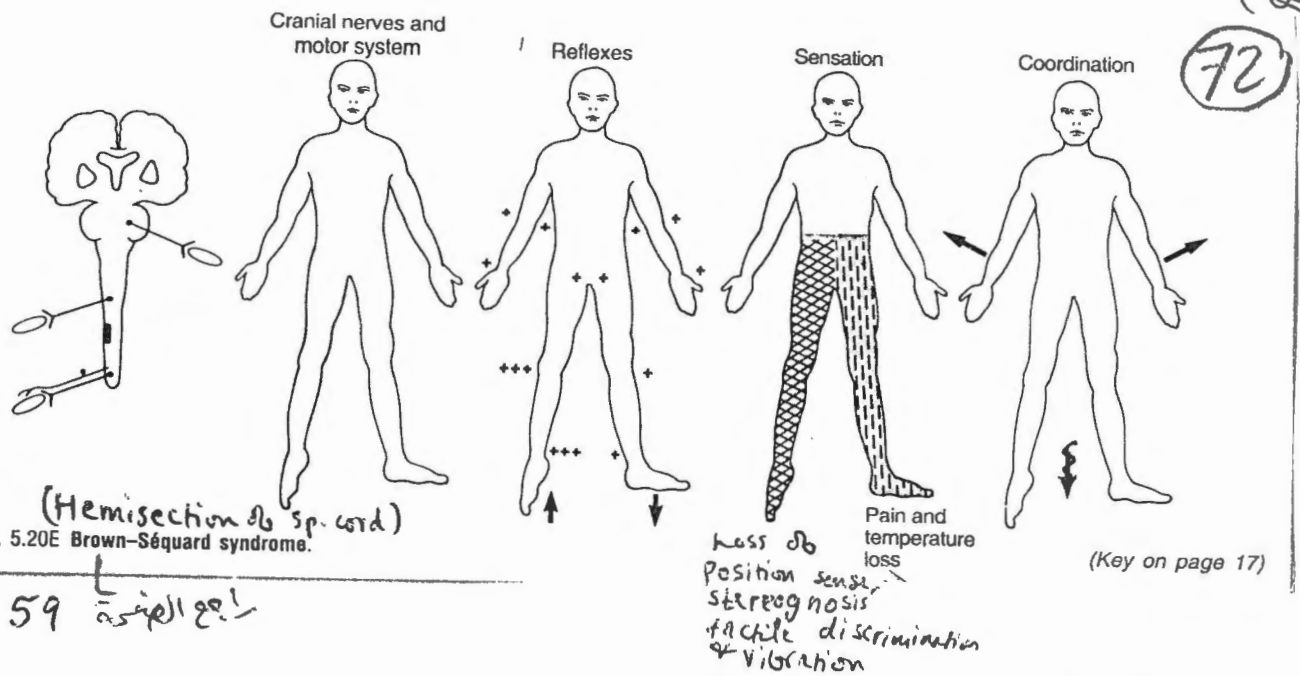


Fig. 1.26 **Prototypical figure for illustration of major syndromes of the neuromuscular system.**

Lesions of the spinal cord

Focal lesions of the spinal cord and the nerve roots produce clinical manifestations in two ways:

- the lesion destroys function at the segmental level
- the lesion interrupts descending motor and ascending sensory tracts.



(Hemisection of sp. cord)

Fig. 5.20E **Brown–Séquard syndrome.**

(Key on page 17)

Loss of position sense, stereognosis, fine tactile discrimination & vibration

Peripheral sensorimotor neuropathies

Peripheral sensorimotor neuropathies are characterised by muscular weakness and wasting (especially of distal muscles), distal areflexia and a 'glove and stocking' distribution of sensory loss (Fig. 3.6). Peripheral neuropathies may be due to systemic disease, vascular disease, heredodegenerative disorders, infection, immune disorders. In general there are two pathological types. **Demyelinating neuropathies** predominantly damage Schwann cells and myelin sheaths. **Axonal neuropathies** primarily cause axonal degeneration. Recovery from neuropathy requires remyelination and regeneration of axons.

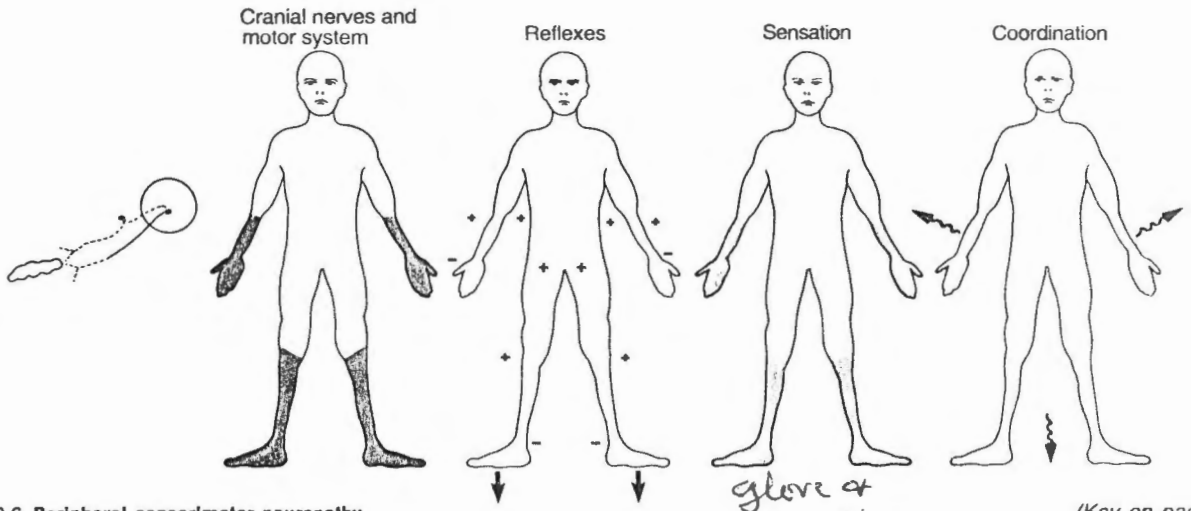


Fig. 3.6 **Peripheral sensorimotor neuropathy.**

(Key on page 17)

glove & stocking anaesthesia

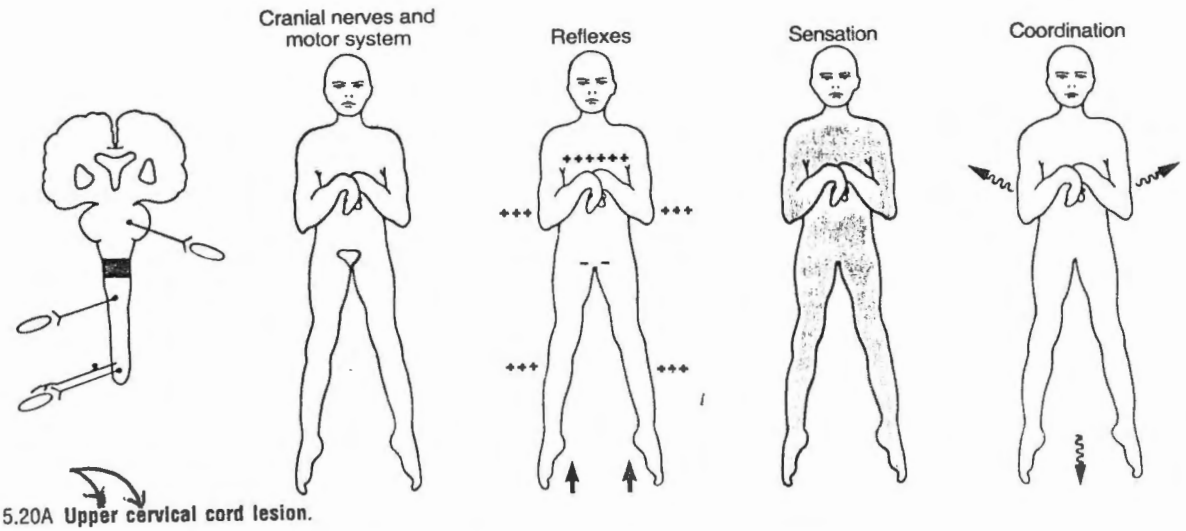


Fig. 5.20A **Upper cervical cord lesion.**

Upper cervical cord lesion. A high cervical cord lesion causes spastic tetraplegia with hyperreflexia, extensor plantar responses (upper motor neurone lesion), incontinence, sensory loss below the level of the lesion and 'sensory' ataxia (Fig. 5.20A).

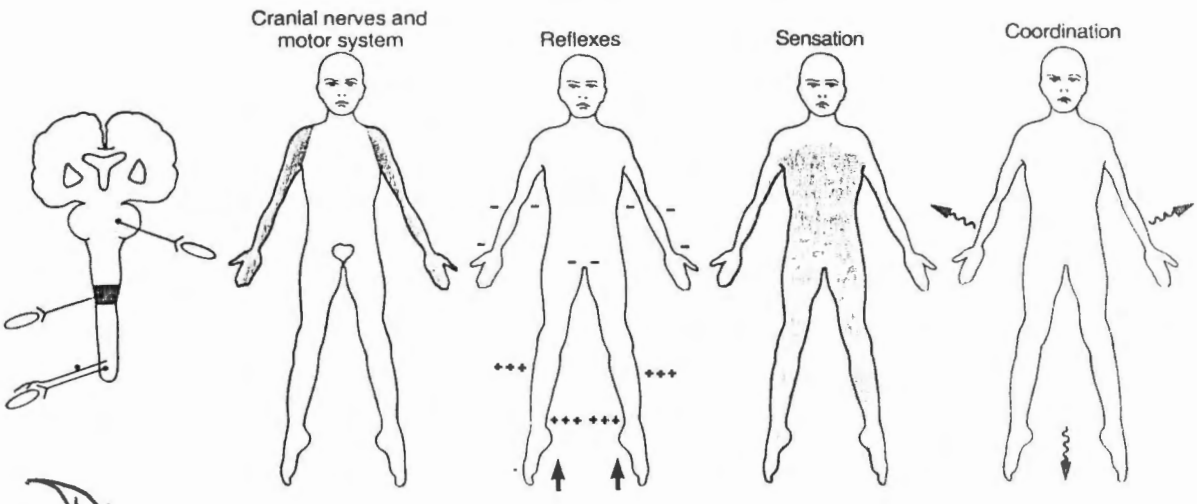


Fig. 5.20B **Lower cervical cord lesion.**

(Key on page 17)

Lower cervical cord lesion. A lower cervical cord lesion causes weakness, wasting and fasciculation of muscles, and areflexia of the upper limbs (lower motor neurone lesion). In addition, there is spastic paraparesis, hyperreflexia and extensor plantar responses (upper motor neurone lesion) in the lower limbs, incontinence, sensory loss below the level of the lesion and 'sensory' ataxia (Fig. 5.20B).

Lesions of spinal cord (*continued*)

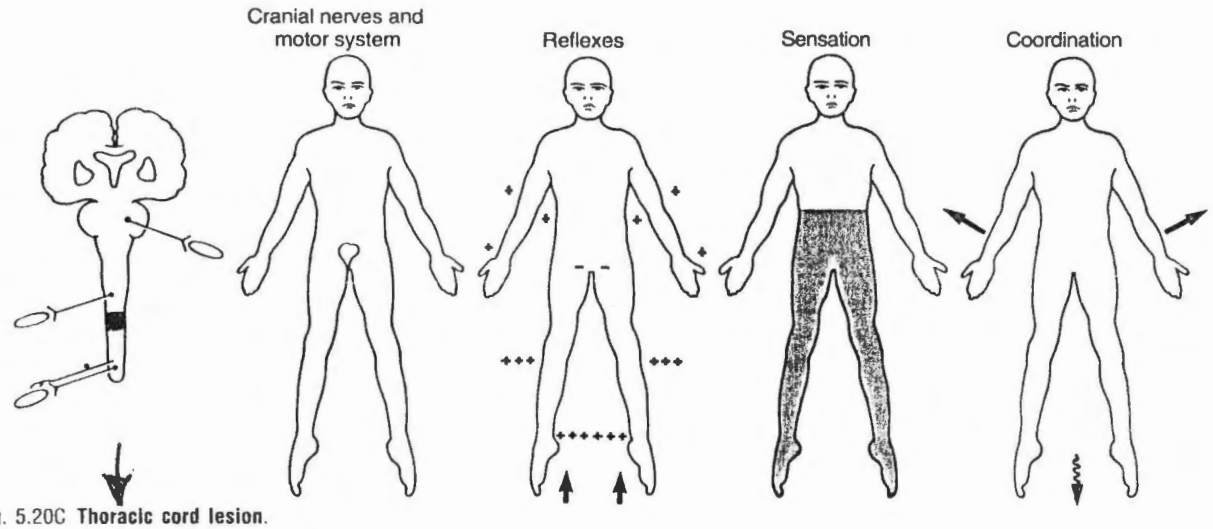


Fig. 5.20C **Thoracic cord lesion.**

Thoracic cord lesion. A thoracic cord lesion causes a spastic paraparesis, hyperreflexia and extensor plantar responses (upper motor neurone lesion), incontinence, sensory loss below the level of the lesion and 'sensory' ataxia (Fig. 5.20C).

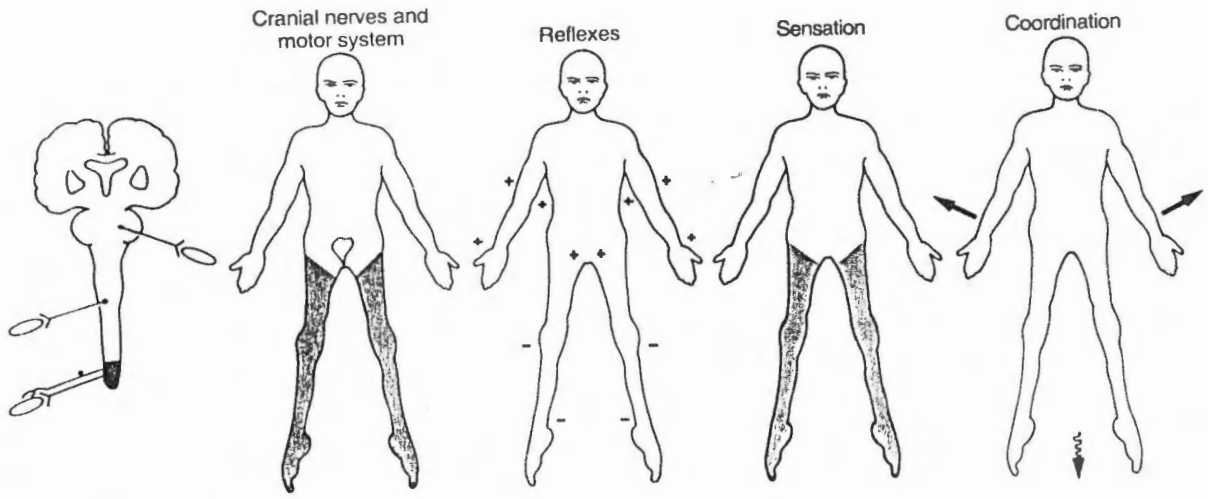
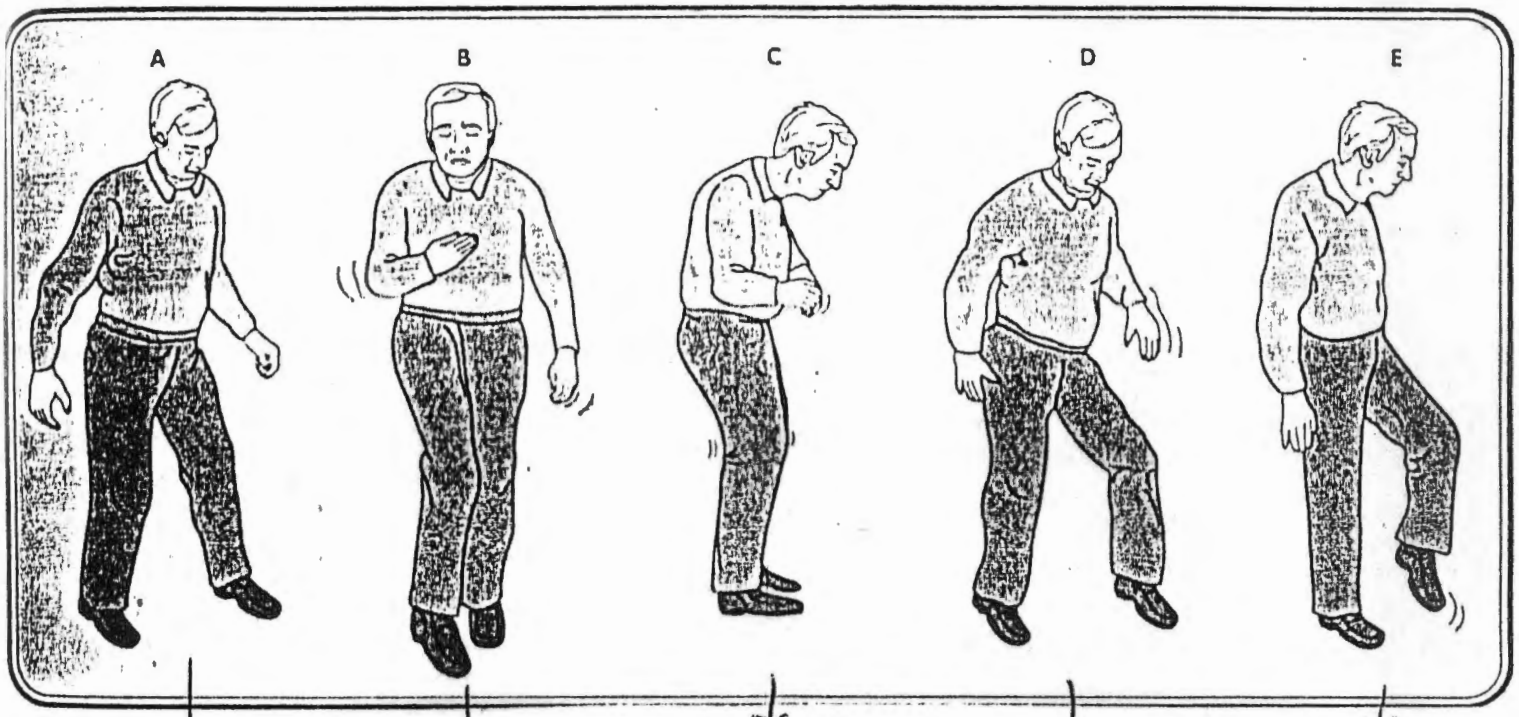


Fig. 5.20D **Lumbar cord lesion.**

Lumbar cord lesion. A lumbar cord lesion causes weakness, wasting and fasciculation of muscles, and areflexia of the lower limbs (lower motor neurone lesion), incontinence, sensory loss below the level of the lesion and 'sensory' ataxia (Fig. 5.20D).

Disorders of Gait



Cerebellar ataxia

Stands & walks on a wide-based gait

Hemiparetic (hemiplegic)

flexion of upper limb & extension of lower limb (spasticity)

Parkinsonian gait

Stooped Posture
+
loss of arm swing
+
Steps are Short & the patient Shuffles
+
difficulty in
- Starting
- Stopping
- turning

Sensory ataxia

arises from impaired proprioception caused by a lesion of:
- → peripheral nerves
- → dorsal roots
- → dorsal columns

↓

the gait is STAMPING

يدوس بقوة

Unilateral foot drop

Steppage gait

↓

arises from weakness of anterior & lateral muscles of the leg

↓

Unable to dorsiflex & evert the foot

↓

the leg is lifted high in walking so that the toes clear the ground