
Pain Sensation

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Pain

- An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage"

International Association for the Study of Pain

Pain Sensation

- ❑ **Protective** mechanism.
 - ❑ Damage to body tissues.
 - ❑ Removal from the pain stimulus
 - ❑ Pain Receptors: **Free nerve endings**.
 - ❑ Superficial layers of the skin, periosteum, the arterial walls, joint surfaces, falx cerebri, and tentorium of the cranial vault.
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Stimuli that can excite Pain Receptors

- Mechanical, thermal, and chemical
 - Chemicals include:
 - Bradykinin, Serotonin & Histamine
 - K ions & Acids
 - Acetylcholine
 - Proteolytic enzymes
 - In addition, PGs and substance P also enhances the sensitivity of pain endings but do not directly excite them.
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Non-adapting Nature of the Pain Receptors

- ❑ **Adapt very little** and some times not all.
 - ❑ Continued stimulation → greater excitation of pain fibers becomes → **Hyperalgesia**
 - ❑ Little adaptation → person apprised of a damaging stimulus.
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Tissue Ischemia & Pain

- ❑ Blocking blood flow → pain within few minutes.
 - ❑ **Why?** The greater the rate of metabolism of the tissue the more rapidly the pain appears.
 - ❑ Caused by:
 - A. Ischemia → large amounts of lactic acids.
 - B. Chemical agents (bradykinin and proteolytic enzymes after the cell damage).
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Muscle Spasm & Pain

- ❑ **Direct effect:** muscle spasm → stimulating mechano-sensitive pain receptors.
 - ❑ **Indirect effect:** muscle spasm → compress blood vessels → ischemia.
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Fast Pain

- ☐ Sharp, pricking, acute and electric pain.
 - ☐ Felt within 0.1 sec.
 - ☐ Not felt in most of the deeper tissues.
 - ☐ Elicited by mechanical & thermal stimuli.
 - ☐ Localized
 - ☐ Transmission: type A δ with CV= 6-30 m/sec
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Transmission of Fast Pain

- **Type A δ** nerve ending secrete **glutamate** (period of action = **few milliseconds** & terminate on neurons in the dorsal horn.
 - Signals pass via **neospinothalamic tract** fibers and terminate in **lamina I (Lamina Marginalis)** of the dorsal horn.
 - Second-order neuron cross through anterior commissar and pass upward in anterolateral column.
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Transmission of Fast Pain

- Neospinothalamic tract terminate into:
 - 1) Few fibers: Reticular areas of brainstem.
 - 2) Most fibers: Thalamus (Ventrobasal complex)
 - 3) Few fibers: **Posterior nuclear group** of the thalamus → other basal areas of the brain → somatic sensory cortex.
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Slow Pain

- ❑ Slow burning, throbbing, nauseous & chronic.
 - ❑ **Felt after** 1 second → slowly ↑ over several seconds or minutes.
 - ❑ Felt in **most** of skin **and deeper tissues** of & associated with tissue damage.
 - ❑ Stimuli: Mechanical, **chemical**, and thermal.
 - ❑ Poorly localized.
 - ❑ Transmission: type C with $CV = 0.5-2 \text{ m/sec}$.
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Transmission of Slow Pain

- Type C secrete **glutamate** and **substance P** (released & building up slowly) & terminate on neurons in the dorsal horn.
 - Signals pass via **paleospinothalamic tract** fibers and terminate in **lamina II and III (Substantia Gelatinosa)** of the dorsal horn.
 - Most signals pass through one or more short fiber neurons before entering **Lamina V through VIII**.
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Transmission of Slow Pain

- $1/10^{\text{th}}$ - $1/4^{\text{th}}$ fibers pass to thalamus.
 - The rest instead terminate into:
 - 1) Reticular nuclei of MO, pons and mesencephalon.
 - 2) Tectal area of mesencephalon.
 - 3) Periaqueductal gray region surrounding aqueduct of sylvius
 - 4) From brainstem: multiple short fibers relay upward into intralaminar and central lateral nuclei of thalamus → hypothalamus.
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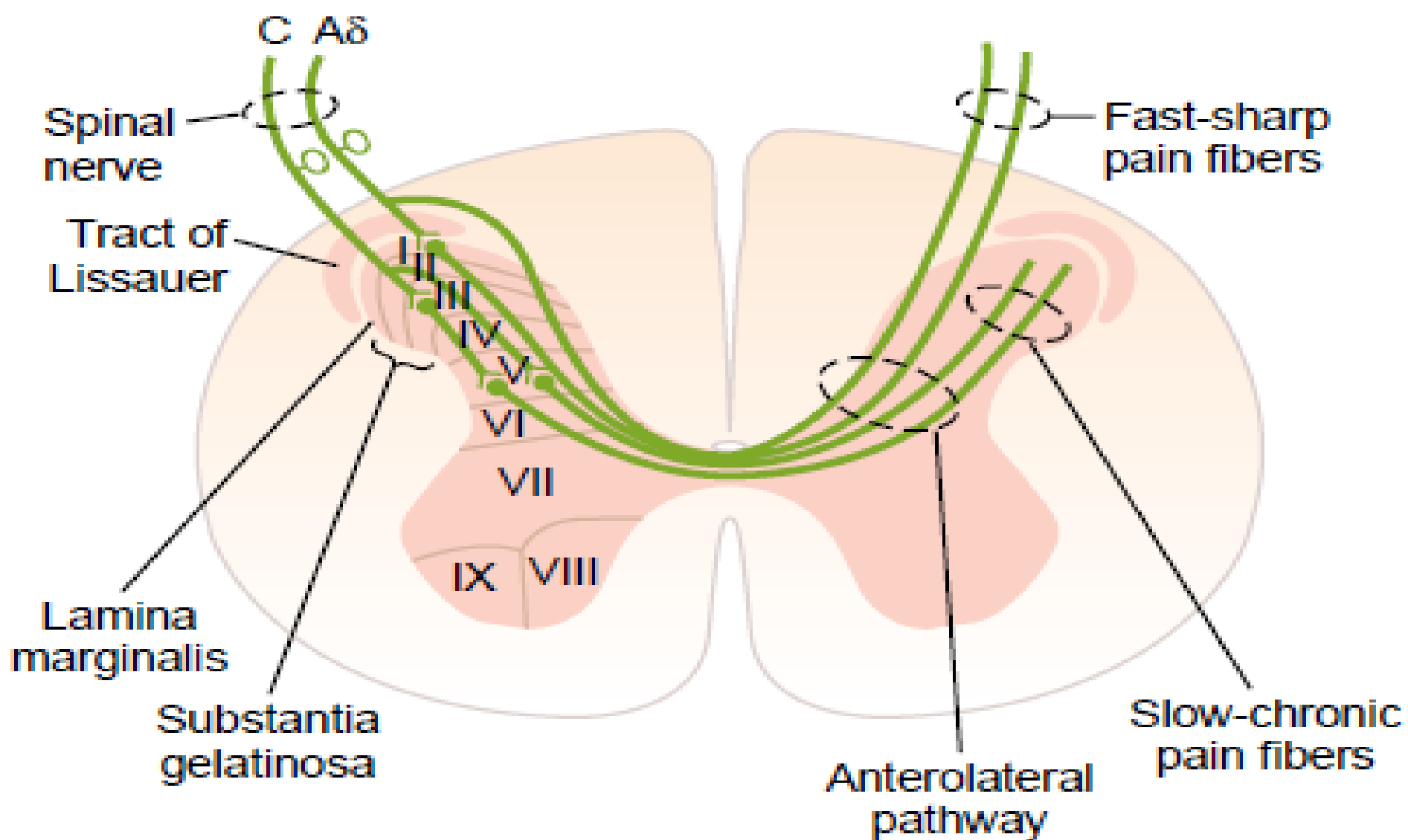


Figure 48-2

Transmission of both “fast-sharp” and “slow-chronic” pain signals into and through the spinal cord on their way to the brain.

Pain Transmission In Spinothalamic Tract

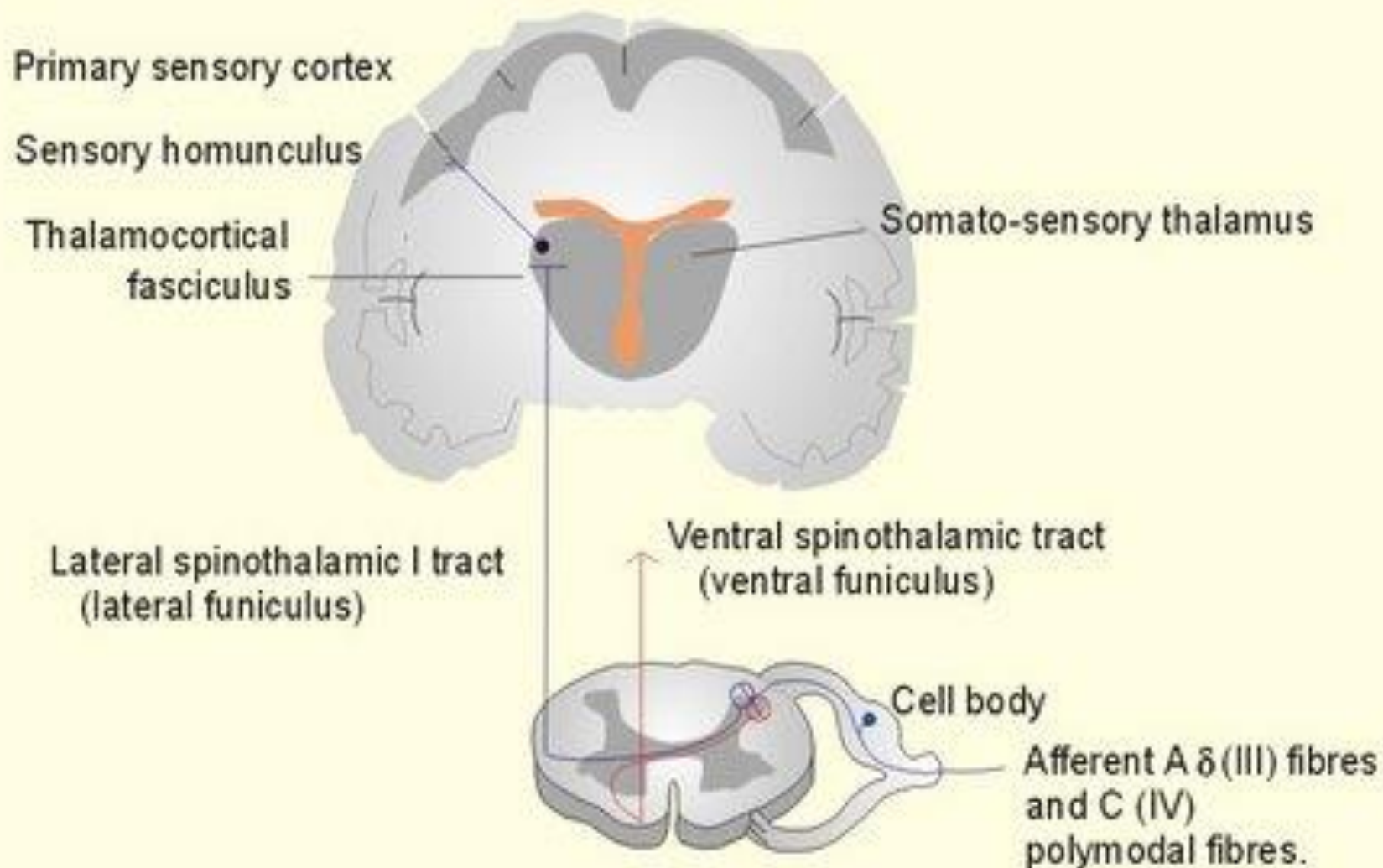


Fig. 3-9

Pain Suppression System in the Brain and Spinal Cord

- ❑ Person reaction to pain **varies**.
 - ❑ Brain suppress pain signals inputs to NS by activating pain control system called "**analgesia system**".
 - ❑ Transmitters involved like **enkephalins** and **serotonin**.
 - ❑ **3** major components:
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Analgesia System

1) Periaqueductal & periventricular areas

- Mesencephalon and upper pons.
- Neurons secrete enkephalin at their endings

2) Raphe magnus nucleus & Nucleus reticularis paragiganto cellularis

- Lower pons and upper medulla oblongata.
 - secrete serotonin.
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Analgesia System

3) Pain inhibitory system

- spinal cord.
 - Serotonin neurons → local spinal cord neurons to secrete enkephalin.
 - Enkephalin → pre- and post-synaptic inhibition of both incoming (**type C and Aδ**) pain fibers where they synapse in the dorsal horn.
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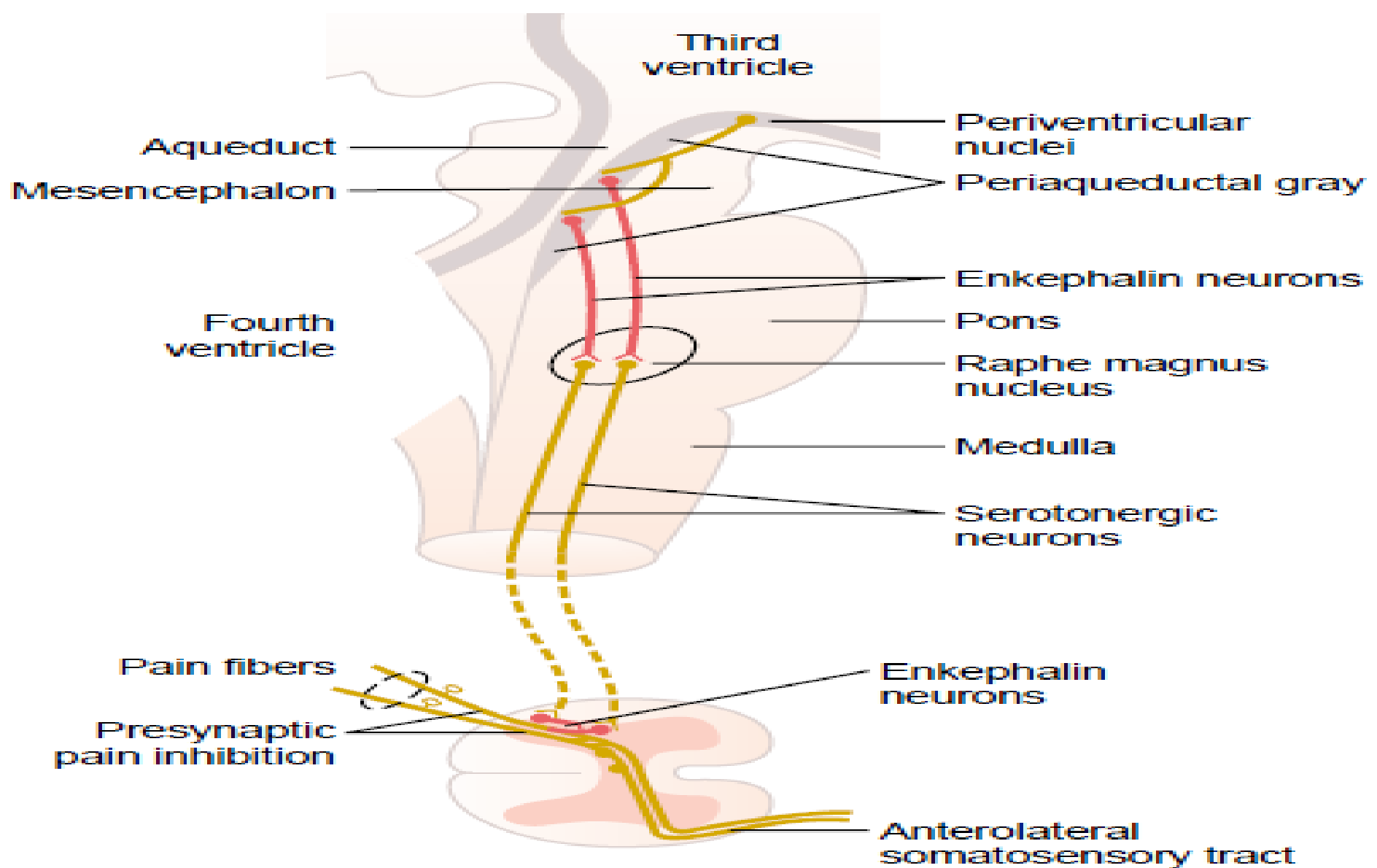


Figure 48–4

Analgesia system of the brain and spinal cord, showing (1) inhibition of incoming pain signals at the cord level and (2) presence of *enkephalin-secreting neurons* that suppress pain signals in both the cord and the brain stem.

Opiate System of the Brain

- ❑ **Opiate-like substances** are breakdown products of
 - Proopiomelanocortin
 - Proenkephalin
 - Prodynorphin.
 - ❑ Multiple brain areas have opiate receptors (areas of analgesia system).
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Opiate Substances

☐ **Met-enkephalin & Leu-enkephalin**

- found in brainstem & spinal cord.

☐ **β-endorphin**

- present in hypothalamus & pituitary gland.

☐ **Dynorphin**

- found in brainstem & spinal cord (less amount).
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Referred Pain

- ❑ Pain in a part of body that is **remote from tissues causing the pain.**
 - ❑ Initiated in one of visceral organs & referred to an area on body surface.
 - Mechanism:
 - ❑ Branches of visceral pain fibers synapse in spinal cord **on same 2nd order neurons that receive pain fibers from skin.**
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Visceral Pain

- ❑ Viscera have pain sensory receptors **only**.
 - ❑ Differs from surface pain:
 - A. Highly localized damage to viscera seldom cause severe pain
 - surgeons cutting gut while the patient is awake.
 - B. Diffused pain throughout a viscous cause pain that can be severe.
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Causes of Visceral Pain

1) Ischemia of visceral tissues

- Bradykinin & proteolytic enzy. formation.

2) Chemical stimuli

- Ruptured DU → leakage of proteolytic acidic (gastric juice).

3) Spasm of hollow organ →

- **mechanical stimulation** of pain endings
- ↓ blood supply to muscles
- ↑ metabolic need of the muscles for nutrients.

4) Over-distention of a hollow organ →

- collapsed blood vessels.

Cramps

- ❑ Pain increasing to high degree of severity and then subsiding.
 - ❑ Process continues rhythmically (1/few minutes).
 - Mechanism: Rhythmical contraction of smooth muscles.
 - ❑ i.e., gastroenteritis, ureteral obstruction, GB disease, constipation, menstruation, parturition.
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Insensitive Viscera

- Liver parenchyma
 - Exception: **bile duct, liver capsule**
 - Lung alveoli
 - Exception: **bronchi, parietal pleura**
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Parietal Pain caused by Visceral Damage

- ❑ Peritoneum, pleura, or pericardium supplied with extensive pain innervation from spinal nerves.
 - ❑ Affected by diseased viscous.
 - ❑ Pain sensation is transmitted through non-visceral nerve fibers.
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Localization of Referred pain transmitted in Visceral Pathways

- Visceral pain is referred to body surface.
 - Localization in dermatomal segment from which visceral organ originated (embryo)
 - Note: Not necessarily where visceral organ now lies.
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The Heart

- ❑ Origin: Neck & upper thorax → heart visceral pain fibers enter spinal cord **between C₃-T₅ segments.**
 - ❑ Heart pain referred to side of neck, over shoulders, over pectoralis muscle, down the arm, and into substernal area of chest.
 - ❑ These body surface areas send their own sensory nerve fibers into C₃-T₅ segments.
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The Stomach

- ❑ Origin: **7th-9th thoracic segments** of embryo.
 - ❑ Stomach pain referred to anterior epigastrium above the umbilicus
 - ❑ This body surface area served by 7th-9th thoracic segment.
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Visceral & Parietal Transmission Pathways

- ❑ Visceral pain is difficult to localize
 - ❑ **Because:** Brain does not know from firsthand experience that different internal organs exist → **pain originates internally can be localized only generally.**
 - ❑ Abdominal & thorax sensations transmitted through **true visceral pathway** and **parietal pathway** to the CNS.
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Visceral & Parietal Transmission Pathways

- ❑ **True** visceral pain transmitted via sensory fibers **within autonomic nerves**
 - ❑ Sensation is referred to body surface areas far from the painful organ.
 - ❑ Parietal sensations are conducted directly into local spinal nerves and they localized directly over the painful area.
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Parietal Pathway for Abdominal & Thoracic Pain Transmission

- Visceral pain is localized to two body surface areas at the same time.

Mechanism: Dual transmission of pain through referred visceral pathway & direct parietal pathway.

- i.e., Inflamed appendix
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Parietal Pathway for Abdominal & Thoracic Pain Transmission

- Pain impulses pass through sympathetic visceral pain fibers into spinal cord at T₁₀₋₁₁.
 - Aching cramping pain referred to an area around umbilicus.
 - Pain impulses from parietal peritoneum where the inflamed appendix touches.
 - Sharp pain directly over the irritated peritoneum in the right lower quadrant of the abdomen.
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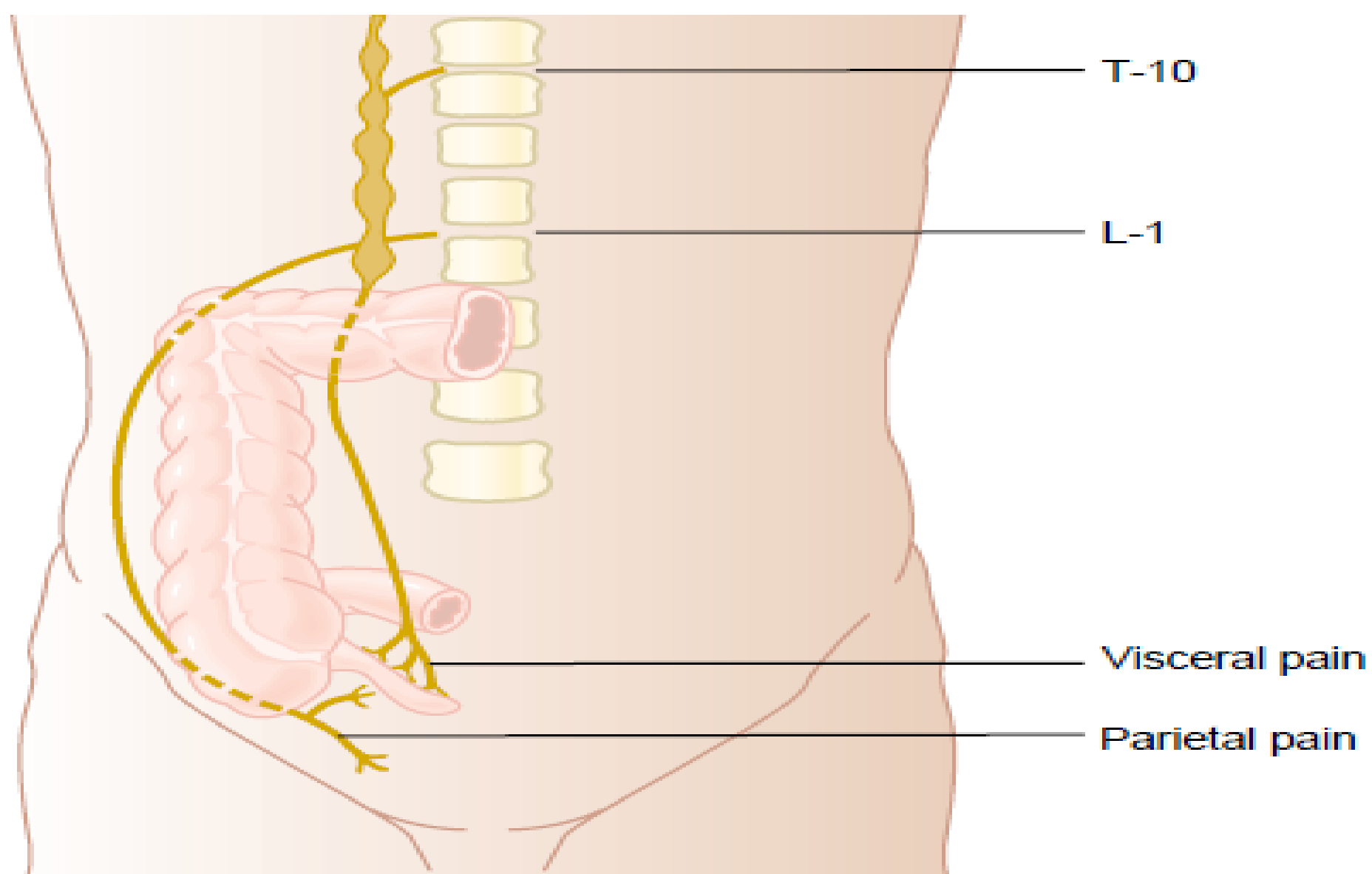


Figure 48-7

Visceral and parietal transmission of pain signals from the appendix.