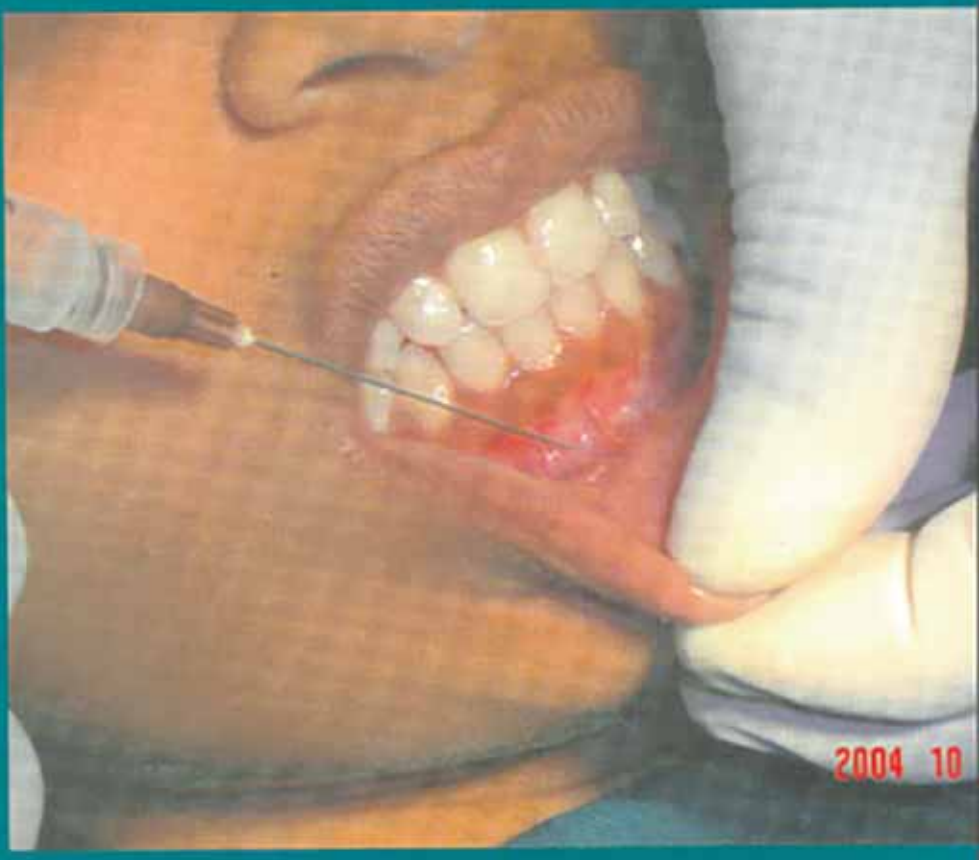
Essentials of Local Anesthesia with MCQs



KG Ghorpade

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Contents

2 Nervous System

STRUCTURE OF NERVOUS SYSTEM

The nervous system comprises of neurons which are specialized tissues, with the ability to co-relate, integrate and rapidly conduct sensations such as touch, pain and temperature to the nerve centers and carry the responses back to the viseral and other organs of the body. A neuron is made up of a cell body, which gives out number of processes called dendrites. Apart from these dendrites, a long process extends from the cell body called axon (Fig. 2-1). The axon can extend to various lengths through the body. Axon contains gelatinous axoplasm encased in a membrane, which separates it from extracellular fluid.

The vital difference between the dendrite and the axon is that, the impulses from the dendrites travel up to the cell body, whereas an axon transmits the impulses away from the cell body. The axon is covered by a rolled myelin sheath, which intercepts at regular intervals. Between two segments of myelin sheaths, the axon membrane is exposed to tissue fluid. The area of interceptions are called as nodes of Ranvier. The myelin layer is covered by a layer of cytoplasm called neurilemma, which encloses a Schwann cell at the outer part (Fig. 2-2). This Schwann cell is responsible for the formation of the myelin sheath. A connective tissue layer called endoneurium holds together a number of nerve fibers forming a bundle or fasciculi. Several such fasciculi are covered and held together by a dense layer of connective tissue called epineurium (Fig. 2-3). The size of the nerve fiber varies from thick to fine. The thick

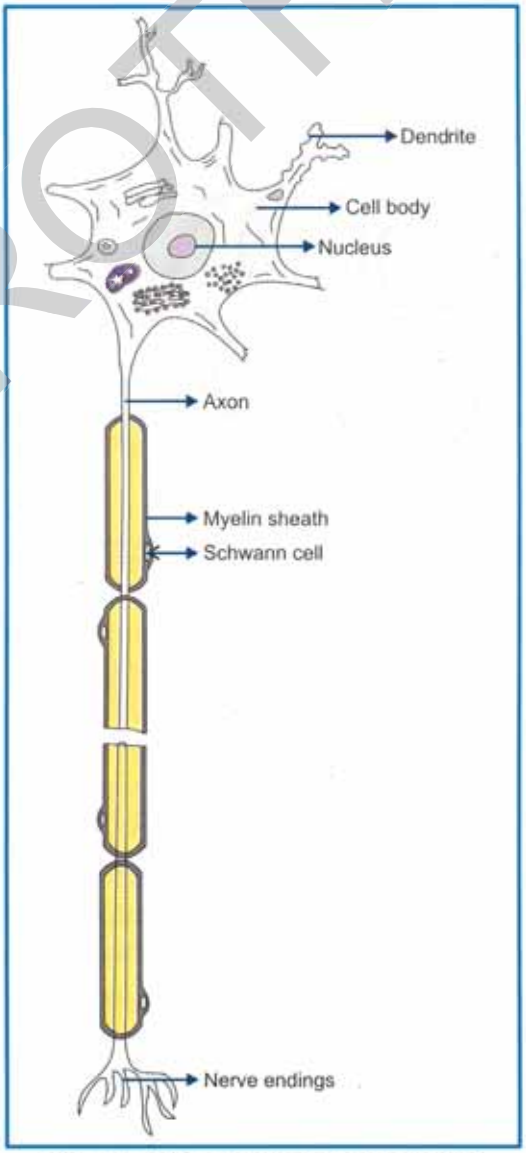

Fig. 2-1: Diagram of a multipolar neuron

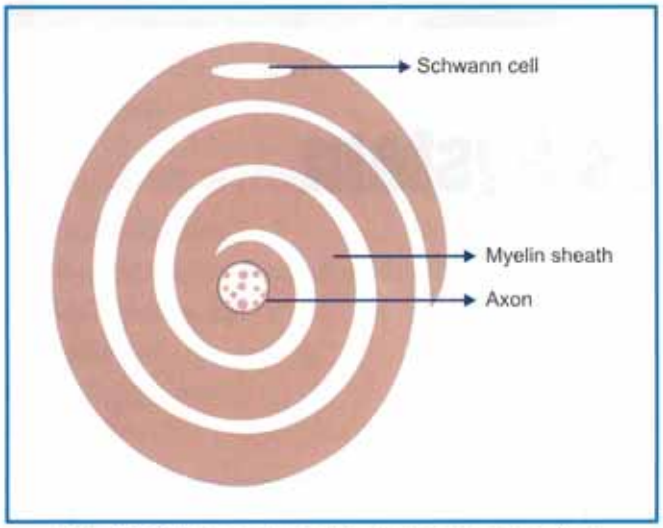

Fig. 2-2: Cross section of a myelinated nerve fiber

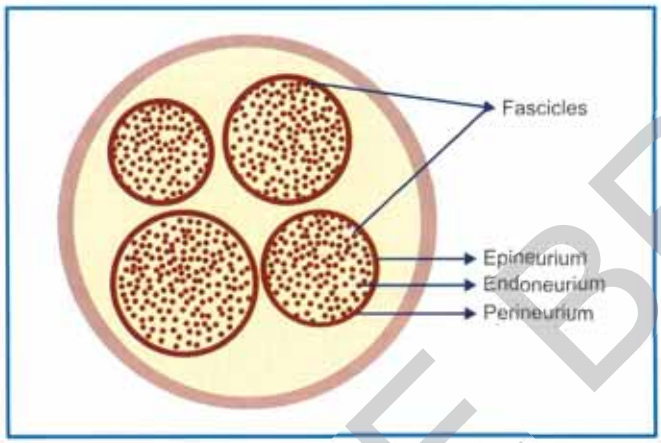

Fig. 2-3: Cross section of a nerve bundle

nerve fibers are myelinated and thinner fibers are unmyelinated. Based on the diameter of the nerve fibre they can be classified as A, B, and C. 'A' fibers are further classified as alpha (α)- 15-20 μm, beta (β) – 8-15 μm, gamma (γ) – 4-8 μm, and delta (δ)- 3-4 μm. A and B are myelinated fibers. C fibers (0.5 to 4 μ) are thin and unmyelinated. Type 'A' fibers supply muscles, spindles, and tendons. Type 'B' fibers are preganglionic afferent or efferent fibers which transmit impulses from viscera, skin and mucous membrane. Type 'C' are fibers in the postganglionic autonomic nervous system and carry sensation of pain, touch and temperature.

The nerve membrane which plays an important role in impulse transmission is made up of double layered phospholipid molecules. The hydrophilic

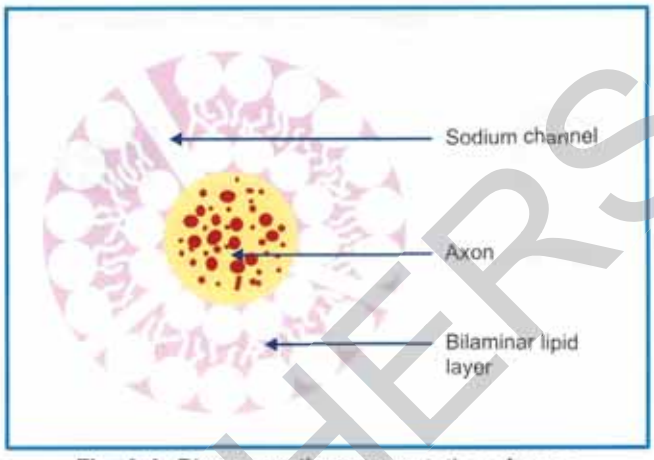

Fig. 2-4: Diagrammatic representation of cross section of a neuron

head faces outwards and the hydrophobic ends faces inwards (Fig. 2-4). Proteins are embedded in the lipid matrix. Ion channels lined by protein globules traverse the membrane. These channels are dynamically active and open and close influenced by external electrical or chemical forces.

PATHWAY OF PAIN SENSATION

Sensations of pain, touch, pressure and temperature from the facial region are carried from the peripheral nerves to the semi-lunar ganglion of the trigeminal nerve (Fig. 2-5). The central fibers from the ganglion enter the pons and 50% of these fibers divide into: a) ascending, and b) descending fibers. The rest of the 50% along with ascending fibers terminate in the main sensory nucleus. The descending fibers terminate in the spinal tract nucleus. The ascending fibers carry sensations of tactile sensibility and the descending fibers carry the sensation of pain and temperature. The proprioceptive sensation from the muscles of mastication and facial muscles are carried by the motor nerve of the trigeminal nerve, pass through motor nucleus and terminate in mesencephalic nucleus. The ascending fibers from the spinal tract nucleus join the other sensory fibers in the mesencephalic nucleus, cross the median plane, ascend and terminate in the postventral nucleus of thalamus. The fibers from the thalamus are carried to the cerebral cortex.

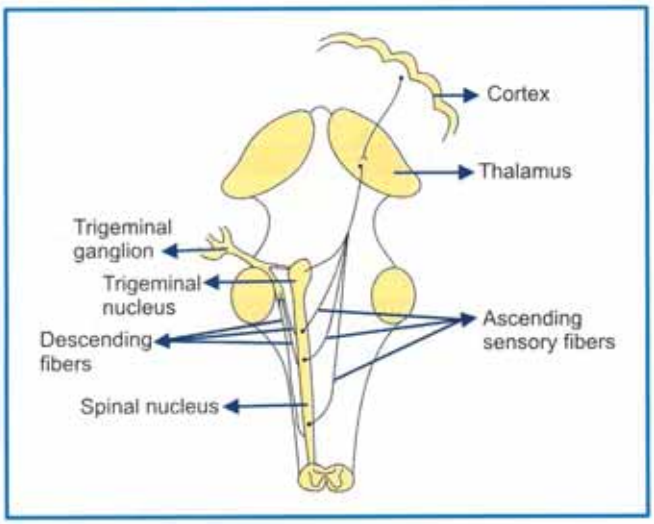

Fig. 2-5: Pain pathway

PHYSIOLOGY OF NERVE IMPULSE CONDUCTION

Propogation of a nerve impulse in response to a stimulus occurs by a complex physiologic activity, which can be explained thus: In a resting nerve there is low sodium ion concentration and a higher potassium concentration. Outside the nerve membrane, the tissue fluid concentration of Na^+ ions is higher. This creates a concentration gradient of electrolytes across the membrane which is usually around – 70 mV. Apart from this, there is low concentration of Cl^- and Ca^{++} ions within the neural tissue. There is a continuous passive exchange of ions across the membrane with a tendency for sodium ions to diffuse into the nerve. The diffused sodium ions are continuously being removed by sodium pump, which regulates the constant tissue gradient. This state in the nerve is termed as resting potential (Fig. 2-6).

When the nerve is stimulated by any of the causes the nerve membrane becomes momentarily highly permeable to Na^+ ions (Fig. 2-7) which brings down the tissue gradient from –70 mV to + 20 or + 40 mV. This reversal of Na^+ ion concentration initiates an action potential which transmits the nerve impulse along the nerve fibers. This process is called depolarization (Fig. 2-8).

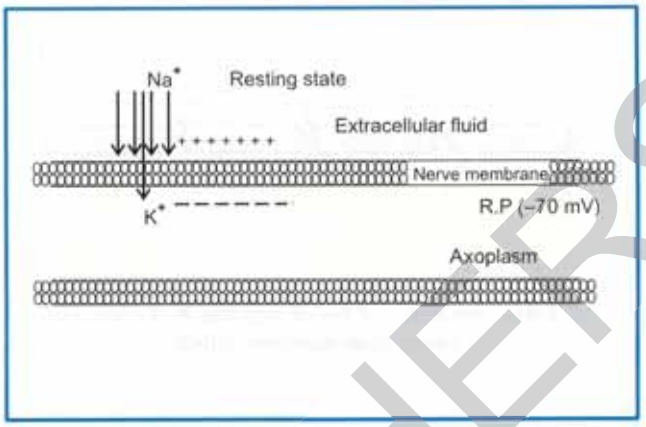

Fig. 2-6: Diagrammatic representation of resting state of a nerve

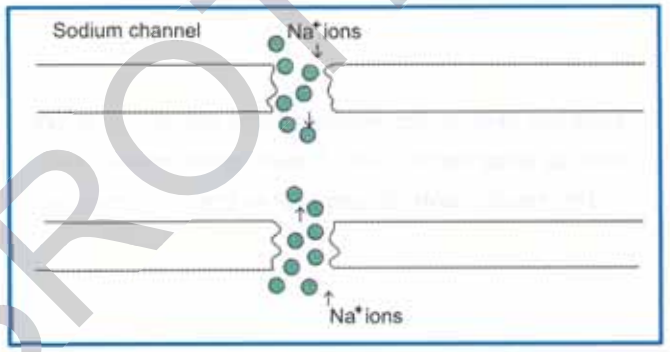

Fig. 2-7: Na^+ ions entering the nerve through the sodium chanel

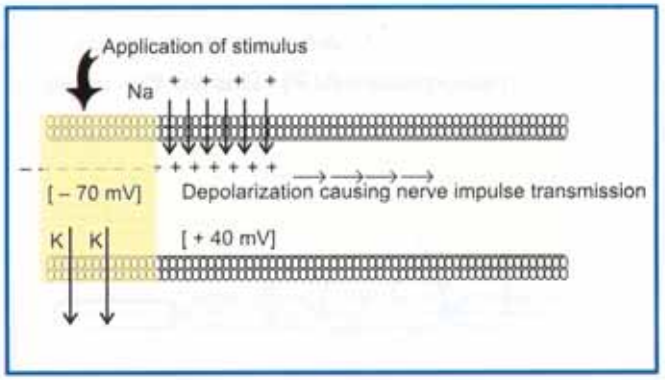

Fig. 2-8: Diagrammatic representation of depolarization

In a non-myelinated nerve fiber, the impulse travels along the nerve by segmental depolarization of adjacent nerve length while repolarization follows rapidly. This results in slow conduction of the nerve impulse. In a

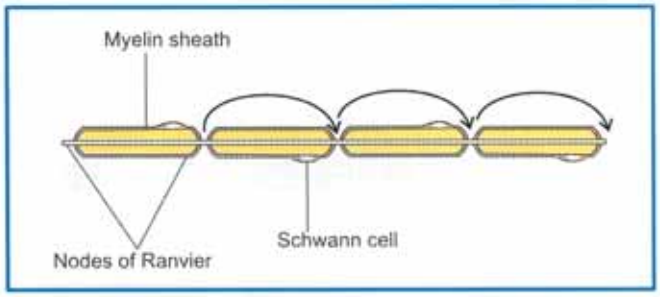

Fig. 2-9: Conduction of nerve impulse in a myelinated nerve fiber-saltatory effect

myelinated nerve fiber, the sodium diffusion is at the nodes of Ranvier. Hence the action potential hops from one node to the other which is termed as saltatory effect (Fig. 2-9). In saltatory action the ion current at each successive nodes becomes smaller until the firing threshold is reached. To effectively block the transmission of impulses in a myelinated nerve the anesthetic solution should at least bathe 2 or 3 successive nodes (Fig. 2-10). The conduction of impulse is faster in myelinated fibers. As the distance between two nodes of Ranvier increases the speed of conduction also increases.

After the influx of Na^+ ions during depolarization, potassium ions diffuse out of the nerve down the concentration gradient bringing back the potential to normal. This is termed as repolarization (Fig. 2-11). During repolarization, Na^+ ions present in excess within the nerve is removed by the sodium pump using the adenosine triphosphate (ATP). Due to the oxidative

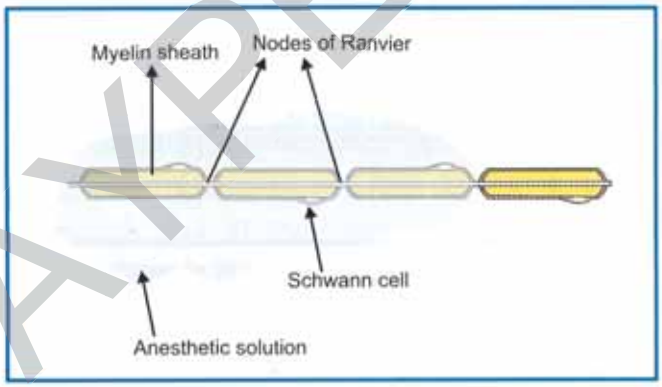

Fig. 2-10: Diagrammatic representation of anesthetic solution covering three consecutive nodes of Ranvier

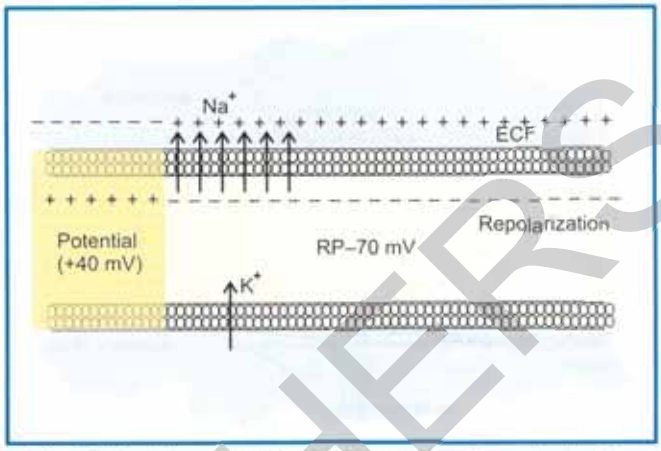

Fig. 2-11: Diagrammatic representation of repolarization

metabolism of ATP the necessary energy is created to activate the sodium pump. At the same time potassium ions enter back into the nerve. The nerve returns to the resting potential of –70 mV.

MODE OF ACTION

A number of theories have been put forward to explain the mechanism of action of anesthetic drugs on the nerve membrane.

Humoral Theory

It is postulated that acetylcholine is responsible for the transmission of the impulse conduction. The lack of any definitive evidence of a chemical action and since there is dissimilarity between acteylcholine and local anesthetics. This is not an acceptable theory.

Calcium Displacement Theory

Role of calcium in nerve block is still being disputed. However it is beyond doubt that it does cause certain amount of nerve membrane excitability. The theory postulates that the local anesthetic molecules displace the calcium from their receptor sites and in turn get attached to them. This binding of the anesthetic molecules to the receptor sites is supposed to prevent sodium ion influx into the nerve.

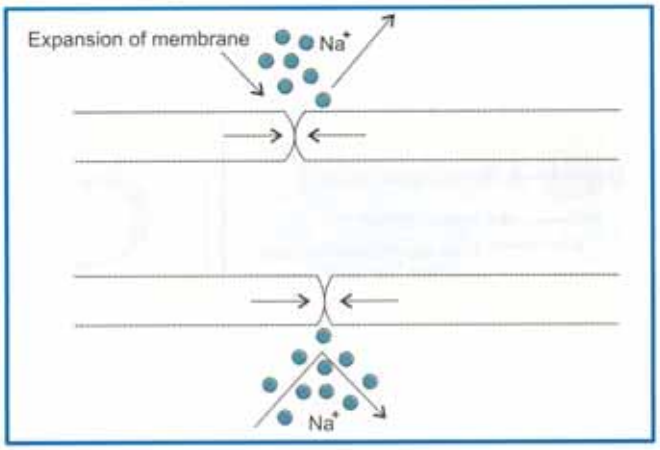

Fig. 2-12: Diagrammatic representation of expansion of membrane resulting in closure of sodium channel thereby preventing Na^+ ion entry into the nerve

Membrane Expansion Theory

According to this theory as the anesthetic solution penetrates the nerve it diffuses through the membrane. This results in expansion of the membrane, which leads to the closure of specific channels through which the Na^+ ions diffuse into the nerve (Fig. 2-12).

Specific Receptor Theory

This has been the most favored theory, which postulates that the anesthetic molecule binds with specific protein receptor in the sodium channel which in turn blocks the diffusion of the sodium ions into the axoplasm. Local anesthetic cations infiltrates to the specific receptor-binding site through the lipophilic transmembrane route. Once the protein binding occurs it blocks the entry of Na^+ ions and thereby blocks conduction.

MODULATION OF PAIN

Gate Control Theory

In 1965 Melzack and Wall explained the Gate control theory, which explains the method of modulation of pain sensation. Pain sensation carried by 'C' fibers are slow in transmission compared to the thicker A-delta fibers. The tactile sensation conducted by thicker fibers can alter the transmission of pain sensation, thus facilitating or preventing its onward transmission. This process can occur either in spinal nucleus or in caudal nucleus of the trigeminal nerve.

Pain suppression (analgesia) system in the brain and spinal cord : Descending control system from the cortical centers are also likely to influence the process of facilitation or inhibition of pain transmission. The degree to which a person reacts to pain varies tremendously. This may be the result from capability of the brain itself to suppress transmission of pain signals by activating a pain control system called analgesic system. Stimulation of the areas in the periaquaductal gray and raphe magnus can stimulate neurotransmitters (encephalins and seratonin) to block the signals of pain being transmitted at the dorsal spinal roots (Fig. 2-13). The neurotransmitters cause both presynaptic and postsynaptic inhibition of pain coming through type 'C' and type 'A' (delta) fibers at the dorsal horn. This is effected by prevention of release of substance 'P' at substantia gelatinosa of the dorsal horn (Figs 2-14A and B).

Limbic system, the site of emotion when stimulated can initiate the pain inhibitory system. This is influenced by psychological fear, previous experience and the threshold of tolerance of pain. A person with higher tolerance and mind set cannot feel the pain for which a

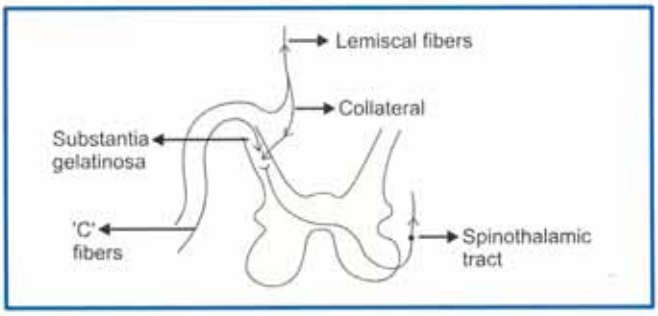

Fig. 2-13: Gate control at the dorsal horn. Lemiscal fibers through collateral branch prevents substance 'P' being liberated

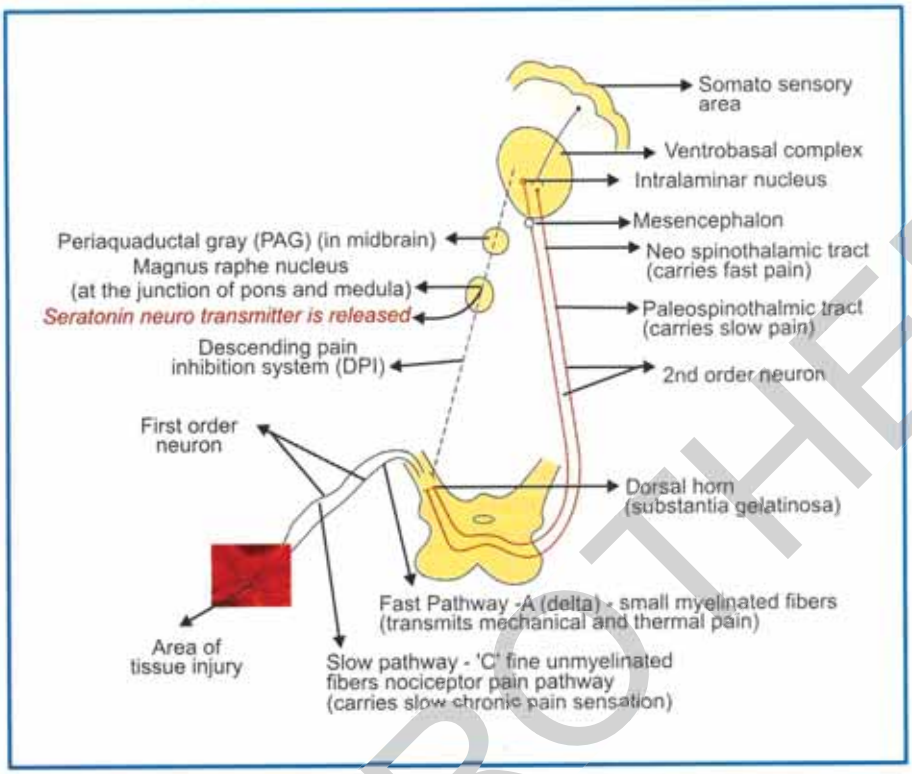

Fig. 2-14A: Diagrammatic representation of descending pain inhibition system. Opioid peptide encephalin related at the dorsal horn by the descending pain inhibition system combines with receptors of afferent pain carrying neuron at the dorsal horn and prevents the release of substance 'P' the neurotransmitter. Thus, the further conduction of pain is inhibited

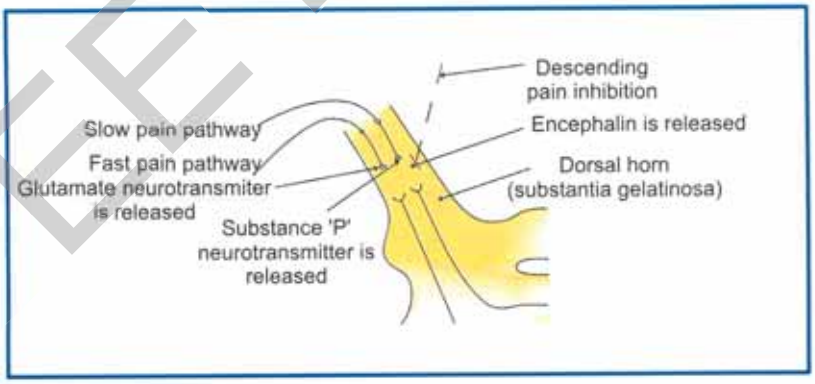

Fig. 2-14B: Detailed view of the dorsal horn

person with lower tolerance level might react intensively. The tolerance level might vary from time to time in an individual. In the author's own experience, a patient had all her teeth removed without any type of anesthesia. This type of high threshold to pain must have been influenced by the descending control system.

Essentials of
Local Anesthesia
with MCQs

This book on Local Anesthesia has been written in view of the needs of dental undergraduates and postgraduates. This should serve as an excellent reference book for private practitioners. This will be the first book on local anesthesia by an Indian author.

The text has been written in easy terms to understand format. Numerous illustrations, diagrams and photographs should enhance understanding of the subject. All aspects of local anesthesia have been elaborately covered. Unnecessary details have been omitted. The anesthetic drugs available, development of armamentarium and the present usage in this country have been emphasized.

To aid the students in competitive and entrance examinations a chapter on multiple choice questions covering all the chapters of local anesthesia has been included.

The author, Dr KG Ghorpade at present is Professor and Head, Oral and Maxillofacial Surgery Dept., Sri Venkateshwara Dental College and Hospital, Bangalore, India. He has been teaching undergraduates and postgraduates in Oral and Maxillofacial Surgery for the last 38 years. He has rich experience in maxillofacial surgical work. He was formerly editor of the Journal of Indian Dental Association.

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