



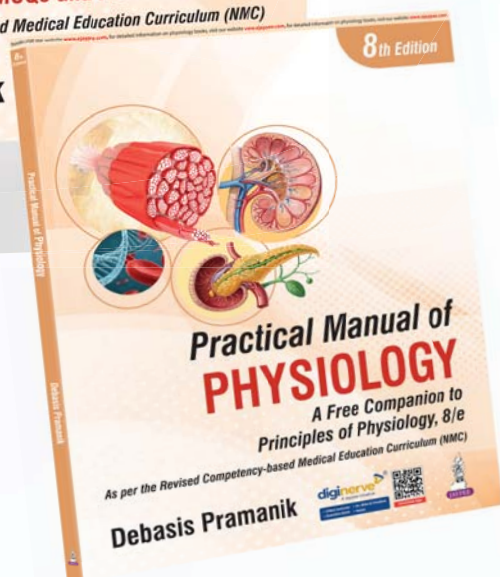
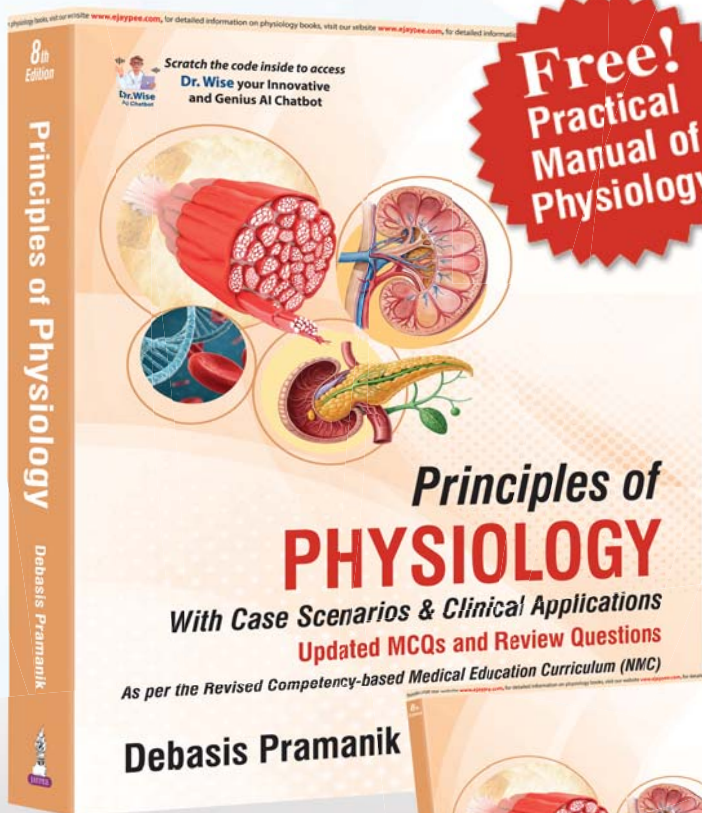
Principle of PHYSIOLOGY

With Case Scenarios & Clinical Applications

Updated MCQs and Review Questions

As per the Latest Complementary-based Medical Education Curriculum (NMC)

8th Edition Debasis Pramanik



TOC & Sample Chapter

MRP ₹ TBA



Why to Buy this Book ?

- It includes a concise coverage of **physiological principles** and **competencies** enumerated in **CBME** curriculum.
- New formatting style for **easier memorization** of keywords has been used.
- New **tables** and **summaries** have been added for easy retention of complex topics.
- A few important chapters have been rewritten entirely with improved presentation.
- A list of wide-ranging **MCQs** have been retained with helpful explanations while a list of **probable questions** including short notes have been added as per **NMC** guidelines.
- The usual **student-friendly** approach and **lucid language** have been maintained.

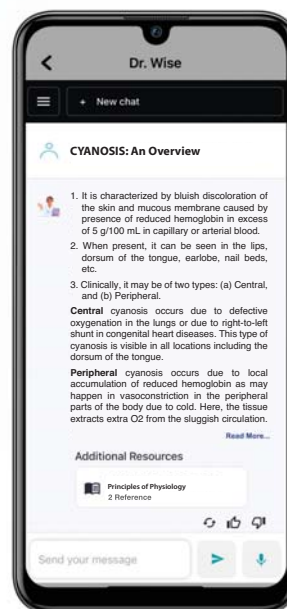
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Dr. Wise

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Clinical MCQs

Case Scenarios

Short Notes

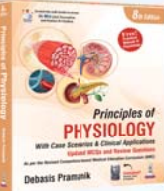
Long Notes

Images

Flashcards

Comparison Tables

Drugs



SAMPLE PAGES

Main Book

Chapters start with specific **Learning Objectives** that highlight the main ideas to be covered.

The content uses a redesigned, memory-friendly layout that **incorporates Figures** to support better recall and **reinforce learning**.

CHAPTER 81 Sensory System

LEARNING OBJECTIVES

After reading this chapter, the student should be familiar with:

- Sensory areas in cerebral cortex
- Disposition of gray matter-Rexed lamina
- White matter
- Bell-Magendie's law
- Dermatome
- Sensory coding
- Physiology of touch sensation
- Pain sensation
- Afferent nerve fiber of pain
- Types of pain
- Pathways of pain
- Syringomyelia
- Referred pain
- Dermatomal rule
- Role of experience
- Theories explaining referred pain
- Different instances of "analgesia" (loss of pain sensation)
- Pain-inhibiting mechanisms in the body
- Thalamic pain syndrome
- Surgical procedures for relief of pain
- Hyperalgesia and allodynia
- Chronic pain
- Itch sensation
- Tickle sensation
- Temperature sensation
- Spinocerebellar tracts

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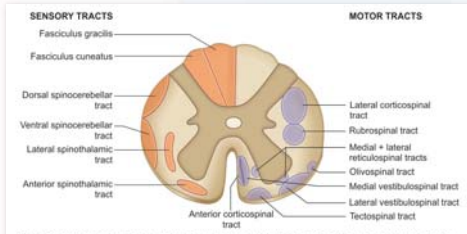


Fig. 81.1: Cross-section of spinal cord showing the sensory (ascending) and motor (descending) tracts.



Fig. 81.2: The somatosensory areas of cortex.

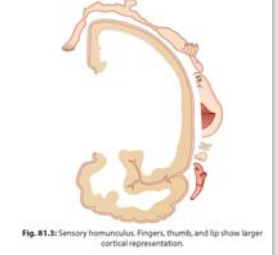


Fig. 81.3: Sensory homunculus. Fingers, thumb, and lip show larger cortical representation.

Note Boxes aid retention of key or specialized topics.

Note: Conscious proprioceptive sense is carried by dorsal column. Although most of the fibers carrying proprioceptive sense end in cerebellum, some pass to the cortex via the thalamus. These latter fibers are believed to carry sensation of conscious proprioception.

Conscious perception of the outline of body image is created from the combined sensory inputs of—(1) slowly adapting spray endings located around joints, (2) Pacinian corpuscles, (3) touch receptors, and (4) muscle spindles.

Note: Blind persons can "read" Braille symbols, which are raised dots separated by a distance of 2.5 mm which is a little greater than "two-point touch threshold" at the fingertips. This enables the blind person to discriminate between adjacent dots and decipher the symbols.

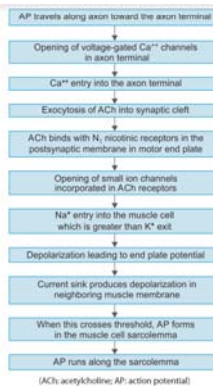
Table 8.1: Mechanism of action of agents affecting neuromuscular transmission.

Botulinum toxin	<ul style="list-style-type: none"> Damages docking proteins, e.g., Synaptobrevin and syntaxin and SNAP-25 Fusion of acetylcholine-containing vesicles with presynaptic membrane does not occur Release of acetylcholine prevented Diminished neuromuscular transmission Competes with/displaces acetylcholine for binding with receptors Diminished binding of acetylcholine with receptor Diminished neuromuscular transmission Inhibits breakdown of acetylcholine Increased acetylcholine concentration at synaptic cleft (reverses block caused by Curare) Organophosphorus compounds cause: <ul style="list-style-type: none"> Increased stimulation of muscarinic receptors by acetylcholine Increased salivation, constriction of pupils, diarrhea, seizures (due to central stimulation)
Curare and pancuronium	<ul style="list-style-type: none"> Competes with/displaces acetylcholine for binding with receptors Diminished neuromuscular transmission Inhibits breakdown of acetylcholine Increased acetylcholine concentration at synaptic cleft (reverses block caused by Curare) Organophosphorus compounds cause: <ul style="list-style-type: none"> Increased stimulation of muscarinic receptors by acetylcholine Increased salivation, constriction of pupils, diarrhea, seizures (due to central stimulation)
Anticholinesterases	<ul style="list-style-type: none"> Inhibits breakdown of acetylcholine Increased acetylcholine concentration at synaptic cleft (reverses block caused by Curare) Organophosphorus compounds cause: <ul style="list-style-type: none"> Increased stimulation of muscarinic receptors by acetylcholine Increased salivation, constriction of pupils, diarrhea, seizures (due to central stimulation)
Succinylcholine	<ul style="list-style-type: none"> Binds with acetylcholine receptors at postsynaptic membrane Persistent depolarization (not broken down by acetylcholinesterase) Inactivation of Na⁺ channels Diminished neuromuscular transmission Muscle relaxation

MECHANISM OF NEUROMUSCULAR TRANSMISSION (FLOWCHART 8.1)

1. When an AP is transmitted via the motor nerve (involving the neuromuscular junction), it ultimately

Flowchart 8.1: Steps of neuromuscular transmission.



A wide range of **Flowcharts, Tables, Applied Physiology Sections, and Summaries** are included to simplify and support retention of complex topics.

Chapter 82: Neuromuscular Junction

2. Alpha receptors, present in skeletal muscle in certain birds, normally inactivate acetylcholine receptors of neuromuscular junction, preventing acetylcholine from binding to its receptors. They inhibit neuromuscular transmission and cause muscle paralysis.

MYASTHENIA GRAVIS

1. This is an autoimmune disorder characterized by prolonged muscle weakness caused by defects in neuromuscular transmission.

2. Autoantibodies are originally produced against acetylcholine receptors present in the synaptic cleft. Subsequently, they bind with acetylcholine receptors present in the motor end plate of neuromuscular junction, causing their destruction.

3. This reduces the number of available receptors and consequently neuromuscular transmission is impaired.

4. In this disease, the EPF formed is too weak to produce prolonged enough transmission to activate the patient's motor end plate of respiratory muscle paralysis.

5. Anti-cholinesterases, the anticholinesterases, can improve the condition temporarily, by causing an increase in local acetylcholine concentration.

6. Being an autoimmune disorder, immunosuppressants are often associated with other autoimmune disorders like rheumatoid arthritis, systemic lupus erythematosus.

Strength of an EPF or EPSP is judged by safety factor. Safety factor is the ratio between EPF or EPSP amplitude and its amplitude required to reach the threshold for firing an action potential. So when the safety factor is > 1 (it is well above 1 in the case of neuromuscular junction), the EPF produced matches the threshold for firing an action potential and an action potential is produced.

Normally, the EPF formed in the neuromuscular junction is about 50 mV in magnitude. This is 20 mV more than the 30 mV of depolarization required for reaching the firing level of AP in the muscle.

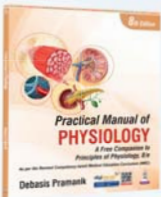
This power to produce 20 mV of excess depolarization increases the value of safety factor to well above 1. This ensures that every AP transmitted through the nerve is capable of producing AP in the muscle. Even if AP in the nerve is repeated in quick succession and the secretion of neurotransmitter is reduced, the safety factor remains above 1 and, an AP is still produced in the muscle.

APPLIED PHYSIOLOGY

Succinylcholine and carbonylcholine are two drugs with a chemical structure similar to acetylcholine. They bind with nicotinic acetylcholine receptors on postsynaptic membrane of neuromuscular junction but unlike acetylcholine, cause prolonged opening of the acetylcholine-gated ion channels, being resistant to hydrolysis by acetylcholinesterase. Although there is depolarization causing muscle contraction initially, prolonged depolarization inactivates Na⁺ channels leading to muscle relaxation and flaccid paralysis. So, they are used as muscle relaxants and they are called depolarizing blockers.

Nicotine, present in tobacco, acts as a central stimulant and has addictive properties as well. As is evident from the neuromuscular, nicotine activates nicotinic acetylcholine receptors in neuromuscular junction. However, the more prominent stimulant actions of nicotine are mediated through acetylcholine receptors in the CNS.

Pancuronium is a synthetically produced competitive antagonist of N₂ nicotinic acetylcholine receptors, having a similar action with curare but it is even more potent. It is used in surgical procedures as a muscle relaxant.



SAMPLE PAGES

Free Book

Table organizes essential information sequentially for better understanding.

Each chapter begins with clear **Learning Objectives** outlining key concepts.

CHAPTER 1 Examination of Cardiovascular System

LEARNING OBJECTIVES

After reading this chapter, the student should be familiar with:

Outline of clinical examination	4	Effect of posture on BP	9
Examination of the cardiovascular system	4	Apex beat	10
Arterial pulse	5	Auscultation of heart sounds	11
Measurement of blood pressure	6		

1.1. OUTLINE OF CLINICAL EXAMINATION

Points to Note

Before a clinical examination, one should note the following points:

- The patient should be relaxed and measured. The clinical tests should be properly explained to the patient.
- Adequate sunlight is required for examination. Auscultation cannot be properly performed without adequate sunlight.
- A female patient must not be examined for absence of a female abdominal aortic pulse.
- The body parts to be examined should be adequately exposed. The patient should wear a dressing gown in place of his own clothing.
- A right handed examiner should stand on the right side of the patient and examine the patient with his right hand.

Chapters include **Points to Note** for easy understanding.

Table 1.7: Differences between first heart sound and second heart sound.

	First heart sound	Second heart sound
Pitch	Low-pitched (dull)	High-pitched (sharp)
Duration	Prolonged (0.16 s)	Shorter (0.11 s)
Onomatopoeia	LUBB	DUP
Best heard in areas	Mitral and tricuspid areas	The bases (pulmonary and aortic areas)
Relation with carotid pulse and apex beat	Just precedes/coinides with carotid pulse/apex beat	Just follows carotid pulse/apex beat
Relation with systole	Marks beginning of systole	Marks completion (end) of systole
Physiological splitting	Absent	May be present during deep inspiration

PROCEDURE

- The patient should be requested to lie in a supine position.
- The chest should be bared.
- The stethoscope should be held in the right side of the chest and the subject's left hand should be raised to the left side.

Notes

Original figures and images of instruments are included to enhance student understanding.

Note: The knobs of the earpiece should be directed forwards and medially when inserted into the ear, so that they are aligned in the direction of the auditory canal. The chest piece is usually a combination of a bell, preferred for hearing low-pitched sounds and a diaphragm for better definition of high-pitched sounds.

Section 3: Human Physiology

1. MEASUREMENT OF BLOOD PRESSURE BY SPHYGMOMANOMETRY

1. Kneel pressure or pulse pressure

Although pulse pressure or systolic pressure represents blood pressure from an artery, systolic pressure is a clinical condition, not pressure, and hence pressure is directly proportional to each value. Therefore, systolic pressure is approximately equal to pulse pressure. It is important to understand blood pressure by measuring blood pressure with sphygmomanometry, especially the signal for the pressure. In an earlier case, it is not possible to measure pulse pressure.

2. Sphygmomanometry (Fig. 4.4B) This is, in essence, a mercury manometer connected to an air-tight cuff which is actually an inflatable rubber bag connected with a short connecting tube to a hand pump fitted with a valve. This instrument was invented by Otto von Riva-Rocci and independently modified by Van der Burg.



Fig. 1.4A: Stethoscope.



Fig. 1.4C: Measuring blood pressure.

QUESTIONS

1. What is the course of olfactory nerve?
2. Describe the visual pathway. Mention the various defects of vision in lesions of visual pathway.
3. What is visual acuity? Discuss the conditions with errors of refraction.
4. Name the extrinsic muscles of the eyeball. State their innervation and actions.
5. What is diplopia? How it is caused?
6. What is squint? What are its types?
7. What is ptosis? What are its causes?
8. What is light reflex and accommodation reflex? Describe the pathways. What is Argyll-Robertson pupil?
9. What are the muscles of mastication? How do you test their function?
10. What are Bell's palsy and Bell's phenomenon?
11. What are upper motor neuron and lower motor neuron? What are the effects of lesions in them?
12. How do you distinguish between upper motor neuron lesion and lower motor neuron lesion?
13. What is nystagmus? In which conditions it is found?
14. What is muscle tone? What are the causes of hypertonia and hypotonia?
15. What are the differences between tremor of cerebellar origin and tremor in Parkinson's disease?
16. What is Romberg's sign?
17. What are the different abnormalities in gait?

Probable Questions and Short notes are included as per NMC guidelines.



Fig. 1.4B: Sphygmomanometer.



Fig. 4.13A: Biceps jerk.



Fig. 4.13C: Supinator jerk.



Fig. 4.13B: Triceps jerk.



Fig. 4.13D: Knee jerk (upward).

Section 7: Human Physiology

1. The tip of the lateral canthus should be lightly dabbed on the skin and the patient's eyes should be closed. The lower eyelid should be pulled down to the lower lid. The upper eyelid should be pulled up to the upper lid. The patient's eyes should be closed. The patient's eyes should be closed. The patient's eyes should be closed.

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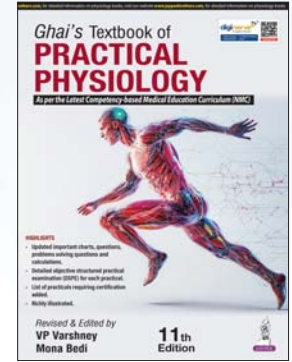
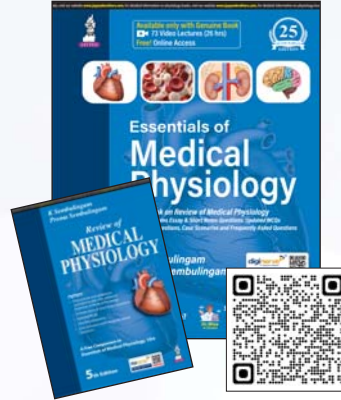
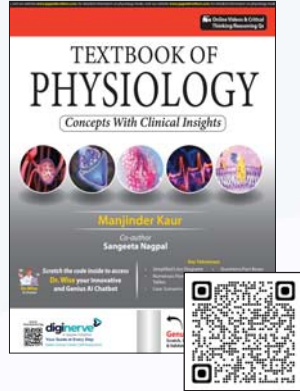
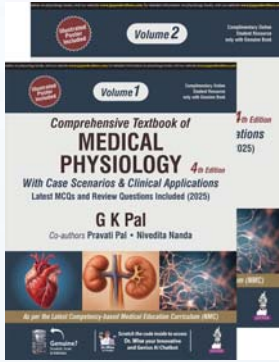
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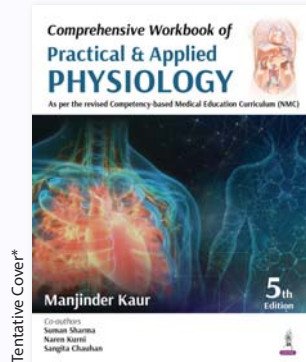
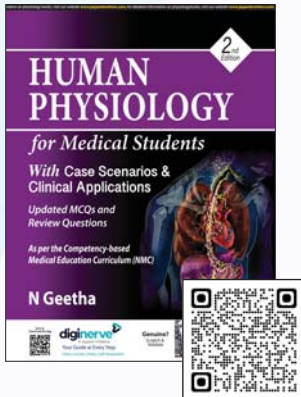
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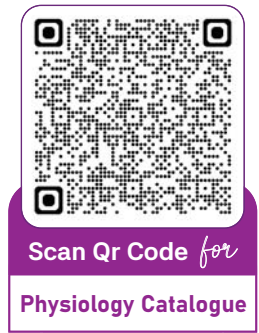
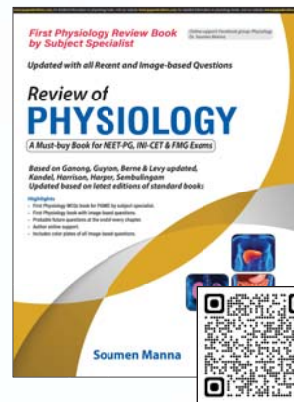
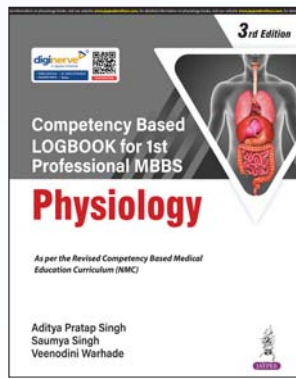
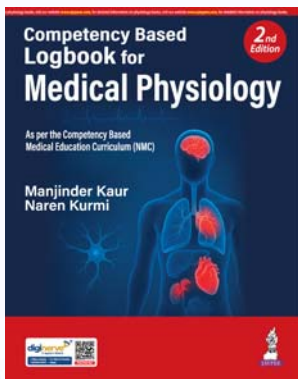
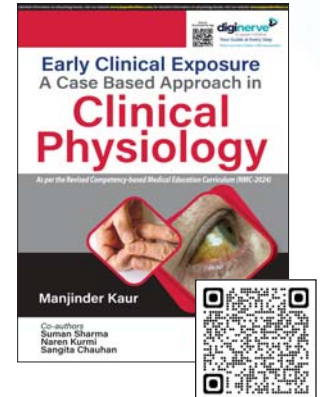
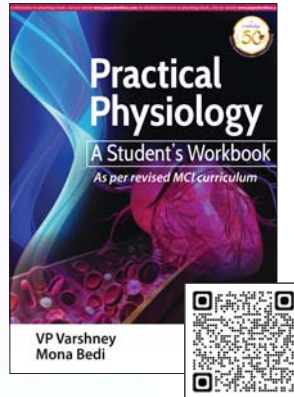
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