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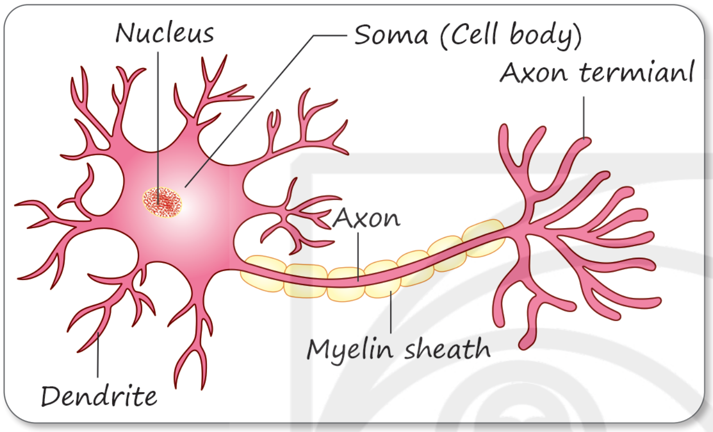
3. NERVE MUSCLE PHYSIOLOGY

STRUCTURE OF A NEURON



PARTS OF A NEURON ALONG WITH THEIR CHARACTERISTICS

Parts of a neuron	Characteristics
Cell body	• Contains nucleus • Metabolic center
Nissl bodies	• Rough endoplasmic reticulum of neurons
Dendrites	• Receive information • Has small knobby projections called **dendritic spines**
Axon hillock	• Thickened area of the cell body
Axon	• Transmits propagated impulses to the nerve endings
Initial segment	• Site where propagated action potentials are generated
Nerve endings	• Site where neurotransmitters are stored and released

CLASSIFICATION OF NEURONS ALONG WITH THEIR CHARACTERISTICS

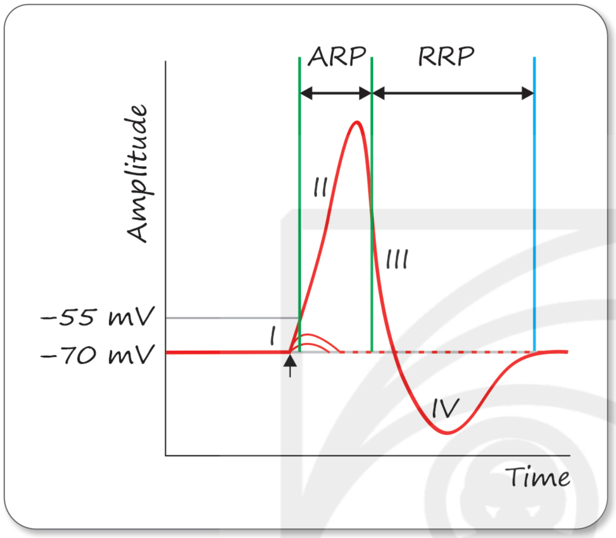
Neuron type	Characteristics
Unipolar neurons	• Have a single process originating from the cell body. • Example: Invertebrate neurons
Bipolar neurons	• Have two types of processes that are functionally specialized– dendrite and axon. • Examples: Bipolar retinal neurons, olfactory neurons
Pseudo unipolar neurons	• Are variants of bipolar cells in which one end goes to the spinal cord and the other end goes to peripheral skin • Example: Dorsal root ganglion cell
Multipolar neurons	• Have single axon and many dendrites • These are the most common types of neurons in CNS • Examples: Spinal motor neuron, Purkinje cells of cerebellum, sympathetic ganglia

Contd...

MYELIN

- Myelin insulates nerve fibers and fastens conduction of impulses.
- Myelin speeds impulse conduction by permitting action potentials to jump between naked regions of axons called nodes of Ranvier. Such a type of nerve conduction is called **Saltatory conduction.**
- Myelin is a lipid protein complex. Lipid component is Sphingomyelin; and protein components are Myelin Basic Protein (MBP) and Myelin Oligodendrocyte Glycoprotein (MOG). Autoantibodies are directed against these proteins in a demyelinating disorder called **multiple sclerosis.**
- Myelin is formed by oligodendrocytes in central nervous system and Schwann cells in the peripheral nervous system.
- Oligodendrocyte can myelinate multiple neurons at a time but Schwann cell can myelinate only one neuron at a time.
- **Luxol fast blue:** Special stain for staining myelin sheath.

NERVE ACTION POTENTIAL



- Phase I: Local potential (–70 mV to –55 mV). Due to slow sodium influx
- Phase II: Depolarization–Rapid sodium influx through voltage gated sodium channels
- Phase III: Repolarization–Potassium efflux
- Phase IV: Hyperpolarization–Delayed closure of potassium channels and also due to chloride influx
- Absolute refractory period (ARP): from firing level (–55 mV) until repolarization is about one-third complete. No stimulus, no matter how strong, will not excite the nerve due to inactivation of sodium channels
- Relative refractory period (RRP): Begins from the remaining part of repolarization to the end of action potential. Stronger than normal stimulus (suprathreshold stimulus) produces action potential

CLASSIFICATION OF NERVE FIBERS

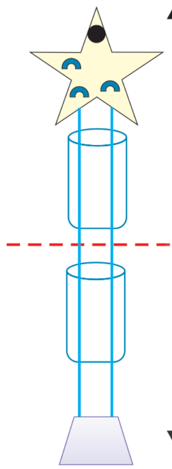
Erlanger and Gasser Classification

Fiber type	Myelin present or absent	Diameter (μm)	Conduction velocity (m/s)	Functions
A α	Myelinated	12–20 (**Largest**)	70–120 (**Highest**)	Proprioception; somatic motor
A β	Myelinated	5–12	30–70	Touch, pressure
A γ	Myelinated	3–6	15–30	Motor to muscle spindles
A δ	Myelinated	2–5	12–30	Pain, temperature
B	Myelinated	<3	3–15	Preganglionic autonomic
C, dorsal root	Unmyelinated	0.4–1.2 (**Smallest**)	0.5–2 (**lowest**)	Pain, temperature
C, Sympathetic	Unmyelinated	0.3–1.3	0.7–2.3	Postganglionic sympathetic

Lloyd Classification

Number	Origin	Fiber type
Ia	Muscle spindle, annulo spiral ending	A α
Ib	Golgi tendon organ	A α
II	Muscle spindle, flower spray ending, touch pressure	A β
III	Pain and cold receptors	A δ
IV	Pain and temperature receptors	C

NERVE INJURY



Retrograde (or) proximal degeneration:

- Withing 48 hours of injury and continues up to 15-20 days
- Nucleus is usually pushed toward the periphery
- Rough endoplasmic reticulum of neurons is called Nissl bodies. They undergo degeneration and dissolution. This phenomenon is called chromatolysis

Axonal injury

Wallerian (or) distal degeneration:

- Occurs distal to the site of injury
- Usually begins within 24-36 hours after injury
- Axonal degeneration is the earliest to occur followed by degeneration of myelin sheath
- Macrophages and Schwann cell clear the debris following degeneration

Nerve regeneration:

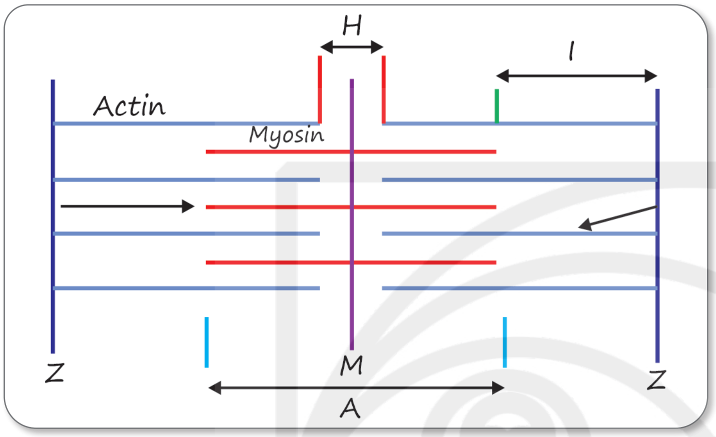
- With 96 hours of the injury
- Nerve regeneration occurs at the rate of 1-3 mm/day
- **Tinel Sign:** It is done by tapping distal to proximal along the injured nerve. positive sign means it produces **tingling sensation** along the course of the nerve. It is indicative of **nerve regeneration.**

Sunderland Classification with its Equivalent Seddon Classification Terminology and Features

Sunderland classification	Equivalent Seddon classification terminology	Features
First degree	Neuropraxia	• Nerve in continuity • Due to compression or ischemia • Only local conduction block seen • Spontaneous recovery in minutes
Second degree	Axonotmesis	• Injury to axon • Encapsulating structures intact • Wallerian degeneration occurs • Recovery at 1-3 mm/day
Third degree	Neurotmesis	• Injury to axon • Endoneurium disrupted, epineurium and perineurium intact • Wallerian degeneration occurs
Fourth degree	Neurotmesis	• Injury to axon • Endoneurium and perineurium disrupted, only epineurium intact • Wallerian degeneration occurs • Requires surgical intervention for recovery
Fifth degree	Neurotmesis	• Injury to axon • Disruption in all encapsulating layers • Wallerian degeneration occurs • Requires surgical intervention for recovery

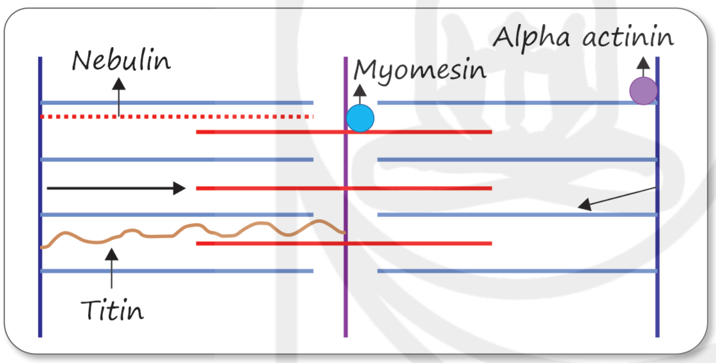
SKELETAL MUSCLE

Sarcomere (Functional Units)



Structure of sarcomere

- Sarcomere—area between two Z lines
- I band—actin filaments (Thin filaments)
- A band—mainly myosin (Thick filaments)
- M line—runs exactly through center of A band
- H band—nonoverlapping part of myosin
- During muscle contraction—Z lines come closer,. Length of H band and I band decreases, A band length remains constant



Sarcomere proteins

- Titin
  - Largest known protein present in mammals
  - Muscle spring responsible for "Elasticity"
  - Connects Z line to the M line
- Nebulin
  - Runs along the length of actin
  - Regulates actin length
- Myomesin
  - Attach myosin to M line
- Alpha actinin
  - Attach actin to Z line

Skeletal Muscle Proteins

- **Contractile Proteins:** Actin and Myosin
- **Structural Supporting Proteins:** Titin, Desmin, Dystrophin
- **Regulatory Proteins:** Tropomyosin and Troponin.
  - **Troponin C:** Contains the binding sites for Ca^{2+}.
  - **Troponin I:** Inhibits the interaction of myosin with actin.
  - **Troponin T:** Binds the troponin components to tropomyosin
- **Relaxation Protein: SERCA pump:** Takes up calcium ions leading to muscle relaxation

NEUROMUSCULAR TRANSMISSION AND EXCITATION CONTRACTION COUPLING

- RMP of Neuron is -70 mV

Contd...

- Opening of Voltage gated Na+ channels-Sodium influx leads to RMP change from -70 mV to -40 mV
- Opening of Voltage gated Ca^{2+} channels-Calcium influx
- Exocytosis of Acetylcholine
- Acetylcholine attaches to Nicotinic Ach receptor in Post synaptic end muscle endplate
- Sodium influx into motor endplate—Endplate potential
- Endplate potential summates to produce action potential
- Action potential activates Dihydropyridine Receptor (DHPR)
- DHPR and Ryanodine receptor are mechanically linked and opening of ryanodine receptor leads to calcium release and actin myosin interaction and muscle contraction
- Muscle relaxation is brought about by reuptake of calcium by SERCA pump

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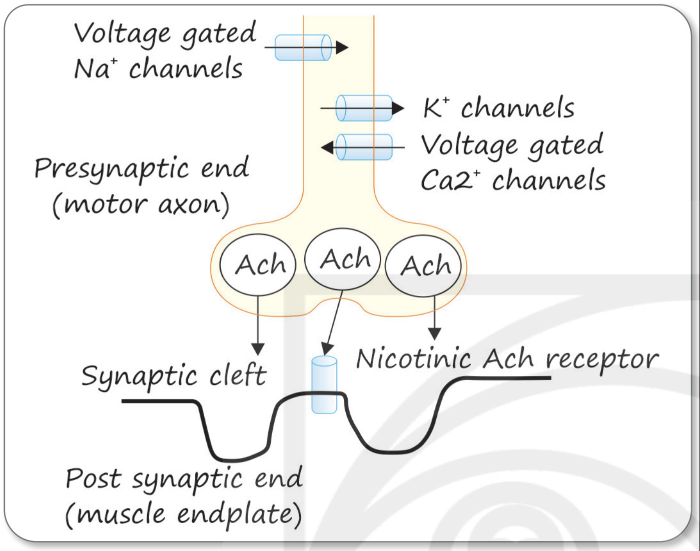


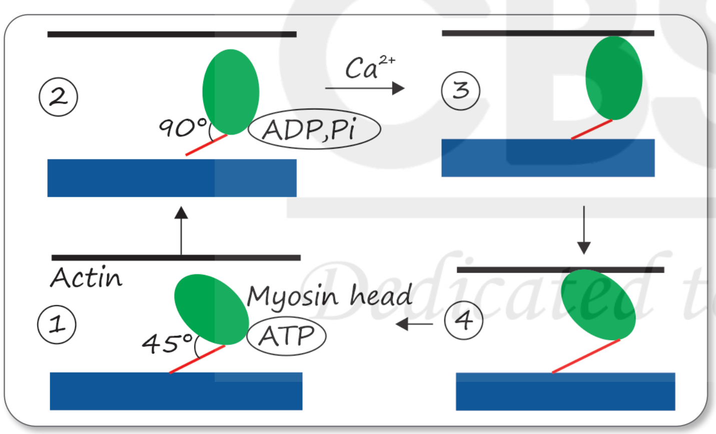
NEUROMUSCULAR BLOCKING AGENTS AND THEIR MECHANISMS OF ACTION

Neuromuscular blocking agent	Mechanism of action
Tetrodotoxin (source—Puffer fish)	Presynaptic voltage-gated sodium channel blocker
Dendrotoxin (source—Mamba snake)	Presynaptic voltage-gated potassium channel blocker
Conotoxin (source—Snail)	Presynaptic voltage-gated calcium channel blocker
Botulinum Toxin	Inhibits release of Acetylcholine leading to flaccid paralysis

NEUROMUSCULAR JUNCTION AND DISEASES

Myasthenia Gravis	Lambert Eaton Myasthenia Syndrome
• Myasthenia gravis (MG) is an autoimmune disease due to auto antibodies directed against nicotinic acetylcholine receptors (AChRs). It is a postsynaptic disorder • The cardinal features are weakness and fatigability of muscles but deep tendon reflexes are preserved • The amount of ACh released per impulse normally declines on repeated activity. It is the reason behind **decremental response** seen in repeated nerve stimulation in myasthenia gravis called **myasthenic fatigue.**	• Lambert Eaton Myasthenia Syndrome (LEMS) is a presynaptic disorder • LEMS is caused by autoantibodies directed against P/Q-type calcium channels at the motor nerve terminals • It is distinguished from myasthenia gravis by two important reasons. One, LEMS have depressed or absent reflexes and two, high frequency repetitive nerve stimulation causes incremental response in LEMS

MUSCLE CONTRACTION: SLIDING FILAMENT THEORY



Muscle contraction, sliding filament theory

Step 1

- **Myosin head at 45°** during resting state
- ATP binding to myosin head activates myosin ATPase
- ATP hydrolysis to ADP and Pi

Step 2

- Myosin head moves from 45° to 90°
- Myosin head contains ADP and Pi

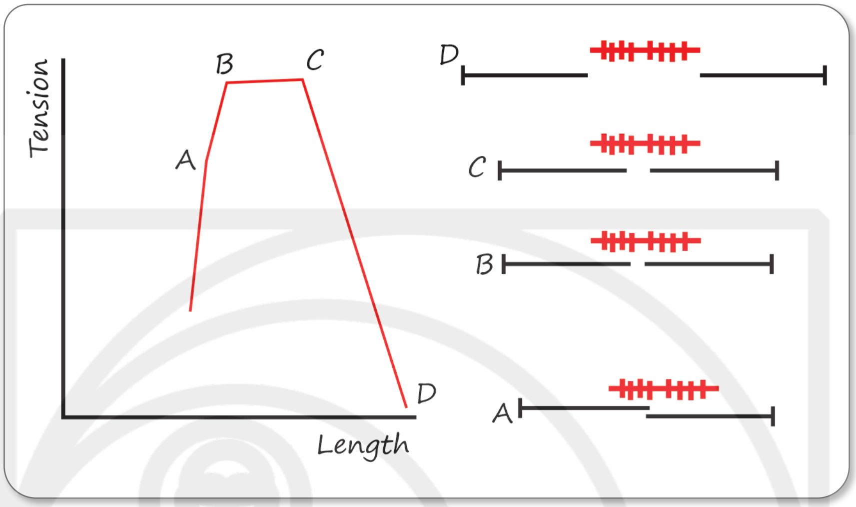
Step 3

- In presence of calcium, actin and myosin interacts
- Formation of crossbridge

Step 4

- **ADP and Pi are released from myosin head**
- **Myosin head tilts back to 45°** (Power Stroke)
- Myosin head detachment from actin require a new molecule of ATP attachment to myosin head
- After death—No ATP—No myosin head detachment leads to RIGOR MORTIS (Sustained Contraction)

LENGTH-TENSION RELATIONSHIP: SKELETAL MUSCLE



- Maximum tension generated by the muscle is between point B and C
- It corresponds to the sarcomere length of 2.2 micrometers.
- It Is at this segment BC where there is optimal overlap between actin and myosin
- On either side of BC, tension produced by the muscle decreases

SKELETAL MUSCLE FIBER TYPES

Characteristics of Skeletal Muscle Fibers

Characteristics	Type I fibers	Type II fibers	
		Type II a	Type II b
Other names	Slow oxidative	Fast oxidative glycolytic	Fast glycolytic
Myoglobin content	High	High	Low
Color	Red	Red	White
Myosin ATPase activity	Slow	Fast	Fast
Ca^{2+} pumping capacity of SR	Moderate	High	High
Diameter	Small	Large	Large
Glycolytic capacity	Moderate	High	High
Oxidative capacity	High	Moderate	Low
Associated with motor unit type	Slow	Fast resistant to fatigue	Fast fatigable
Recruitment order	First	Second	Third
Activities best suited for	Maintaining posture, Endurance type activities (running a marathon)	Walking	Sprinting
Mitochondria	Many	Many	Few
Capillaries	Many	Many	Few

Motor Unit

- Each single motor neuron and the muscle fibers it innervates constitute a motor unit.
- Motor unit innervates very few (three to six) muscle fibers in muscles concerned with fine, precise, graded movements like extra ocular muscles and hand muscles. In leg muscles, motor unit innervates up to 600 muscle fibers.

Size Principle

- Small diameter slow type I fibers are always recruited first. They are followed by recruitment of Type II a fatigue resistant fiber.
- Last to be recruited are the Type II b fast fatigable fibers.

CARDIAC MUSCLE

- Striated, Involuntary
- Have Intercalated Disk-Cardiac Gap junctions
- **Connexins:** Proteins present in Gap Junction
- **Functional syncytium:** cardiac muscle fibers contract together all at the same time because of the presence of Gap junctions
- **Calcium-induced calcium release (CICR):** Calcium from extracellular source should come first to release calcium from intracellular source in cardiac muscle.

SMOOTH MUSCLE

- Involuntary
- Single unit and Multi unit—Single unit smooth muscle has Gap junctions.
- Single unit smooth muscle is found in visceral organs namely uterus, intestine, ureter and urinary bladder.
- Multi-unit smooth muscle is found in iris, ciliary body, epididymis, vas deferens, piloerector muscle of skin.
- Blood vessels got both single unit and multi-unit smooth muscle in their walls.
- No Z lines—equivalent is Dense bodies
- Calmodulin—Calcium binding protein. No Troponin
- **Plasticity:** If visceral smooth muscle is stretched, it first exerts increased tension. However, if the muscle is held at the greater length after stretching, the tension gradually decreases. This initial increase in tension later followed by decrease is called plasticity.
- **Latch Bridge Mechanism:** Smooth muscle can be maintained in a prolonged state of partial contraction (tonus) with very little use of ATP. This is due to latch bridge mechanism in which myosin cross-bridges remain attached to actin for some time after the cytoplasmic Ca^{2+} concentration falls.
- The type of **neuromuscular junction in smooth muscle** wherein one neuron innervating multiple smooth muscle cells is called **synapse en passant.**

SYNAPTIC POTENTIALS

- Synaptic potentials can be excitatory postsynaptic potentials (EPSPs) or inhibitory post synaptic potentials (IPSPs).
- EPSP and IPSP can be fast or slow.
- **Fast EPSP:** Due to influx of sodium or calcium (cell interior more positive).
- **Fast IPSP:** Due to influx of chloride (cell interior more negative).
- **Slow EPSP:** Due to reduced potassium efflux (cell interior more positive).
- **Slow IPSP:** Due to increase in potassium efflux (cell interior more negative).
- Slow EPSP and IPSP are commonly seen in autonomic ganglia, cardiac and smooth muscles.

INHIBITION AND FACILITATION AT SYNAPSES

- **Postsynaptic inhibition:** Hyperpolarization is caused by the inhibitory neurotransmitters GABA and Glycine on a postsynaptic neuron. Also called **afferent inhibition.**
- **Presynaptic inhibition:** Occurs at the presynaptic terminals before the signal ever reaches the synapse by GABA. Also called **lateral inhibition.**
- **Renshaw cell inhibition:** Renshaw cells are excited by a motor neuron. In turn, Renshaw cells inhibit the same motor neuron which excites it. Neurotransmitter involved is GLYCINE. Also called "negative feedback inhibition".

NEUROTRANSMITTERS

Some of the major neurotransmitters are as follows:

Acetylcholine	• Functions as transmitter at—neuromuscular junction, autonomic ganglia, and in postganglionic parasympathetic, basal forebrain complex, Ponto mesencephalic cholinergic complex • Involved in regulation of sleep-wake states, learning and memory • Receptors: 2 types • **Muscarinic:** (M_1, M_4, and M_5-CNS), (M_2-Heart), (M_3-glands and smooth muscles) • **Nicotinic:** (N_M- neuromuscular junction), (N_N- CNS and autonomic ganglia)
Norepinephrine	• Location- locus coeruleus • Activates reticular activating system • Responsible for Awake arousal state
Dopamine	• Location—Nigrostriatal system (motor movements), mesocortical system (Ventral tegmental area nucleus accumbens for motivation and addiction) and Tuberoinfundibular system (inhibits prolactin) • Involved in addiction, reward • Receptors: D_1, D_2, D_3, D_4, D_5 (All are G Protein Coupled Receptors)
Serotonin	• Present in highest concentration in blood platelets and in the gastrointestinal tract • Also, in midline raphe nuclei • Responsible for Awake arousal state, platelet aggregation, peristalsis
Histamine	• Location—posterior hypothalamus • Responsible for awake arousal state
Glutamate	• Major excitatory neurotransmitter in CNS • Location—Hippocampus, Subthalamic Nuclei • Important for learning and memory
GABA	• Major Inhibitory neurotransmitter • Causes hyperpolarization due to chloride influx leading to inhibition of neuronal functions
Glycine	• Both inhibitory and excitatory neurotransmitter • Found in Renshaw cells in spinal cord • Antagonist—Strychnine
Nitric Oxide	• Gaseous neurotransmitter found in Hippocampus • Role in learning and memory
Carbon Monoxide	• Gaseous neurotransmitter produced by enzymatic degradation of heme by heme oxygenase • Role in learning and memory, pain processing, olfaction

ONE Touch Physiology

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- **Theory**—A concise form of text (in 100 pages), and most important points to remember are given from the examination point of view. The text is designed in accordance with the recent CBME and NEXT exam curriculum.
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