



Neuromuscular Junction



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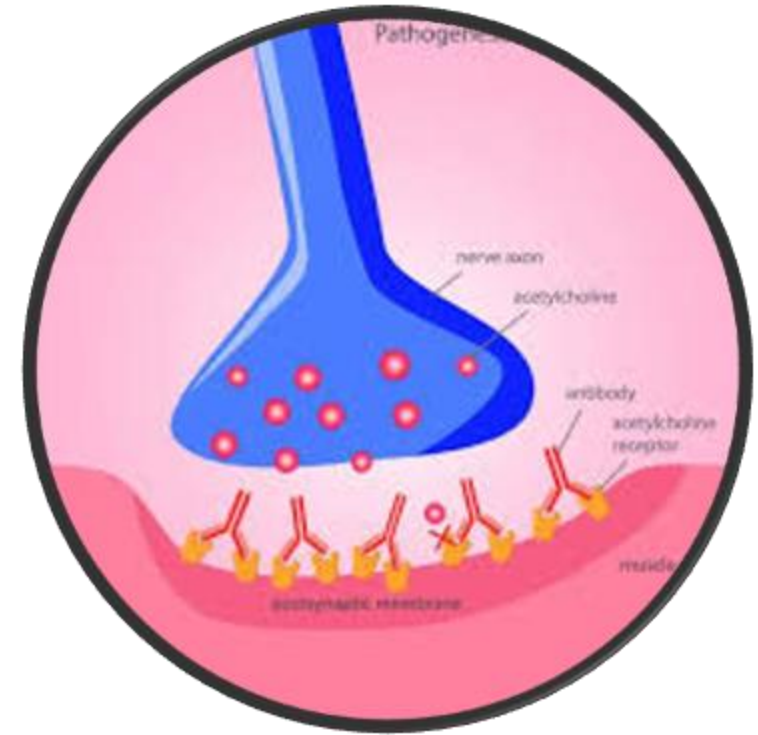
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Introduction



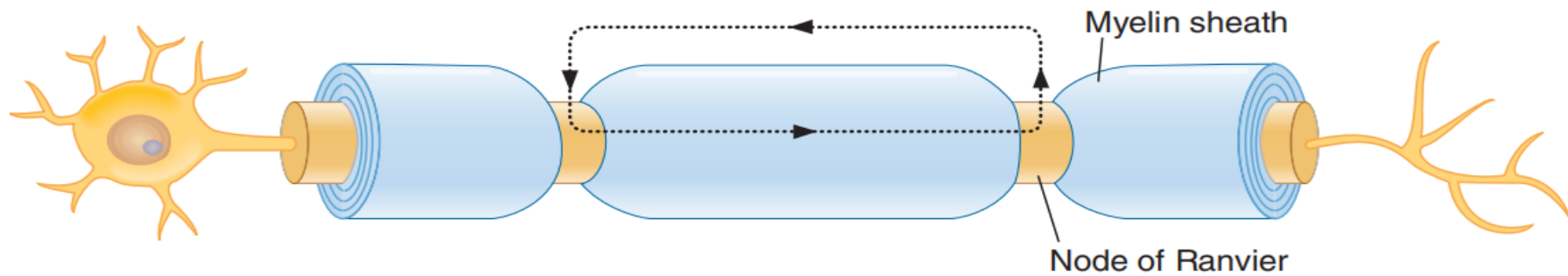


FIGURE 1-8 Myelinated axon. Action potentials can occur at nodes of Ranvier.

- An action potential in a motor neuron is rapidly propagated from the **cell body within the CNS to the skeletal muscle** along **the large myelinated axon** (efferent fiber) of the neuron.
- As the axon approaches a muscle, it divides and loses its myelin sheath.
- Each of these axon terminals forms a special junction, a neuromuscular junction, with one of the many muscle cells that compose the whole muscle.

Introduction

- Each branch innervates only one muscle cell; therefore, each muscle cell has only one neuromuscular junction.
- Both the neural and muscular components make up the neuromuscular junction.
- A single muscle cell, called a muscle fiber, is long and cylindrical.

Introduction

- Within a neuromuscular junction, the **axon terminal splits** into **multiple fine branches**, each of which ends in an **enlarged knoblike** structure called the **terminal button, or bouton**.
- The entire axon terminal ending **fits into a shallow depression**, or **groove**, in the underlying muscle fiber, this depression is called **motor end plate**.

Spinal cord (section)

Axons of two efferent motor neurons

Muscle fibers innervated by *red* motor neuron

Muscle fibers innervated by *blue* motor neuron

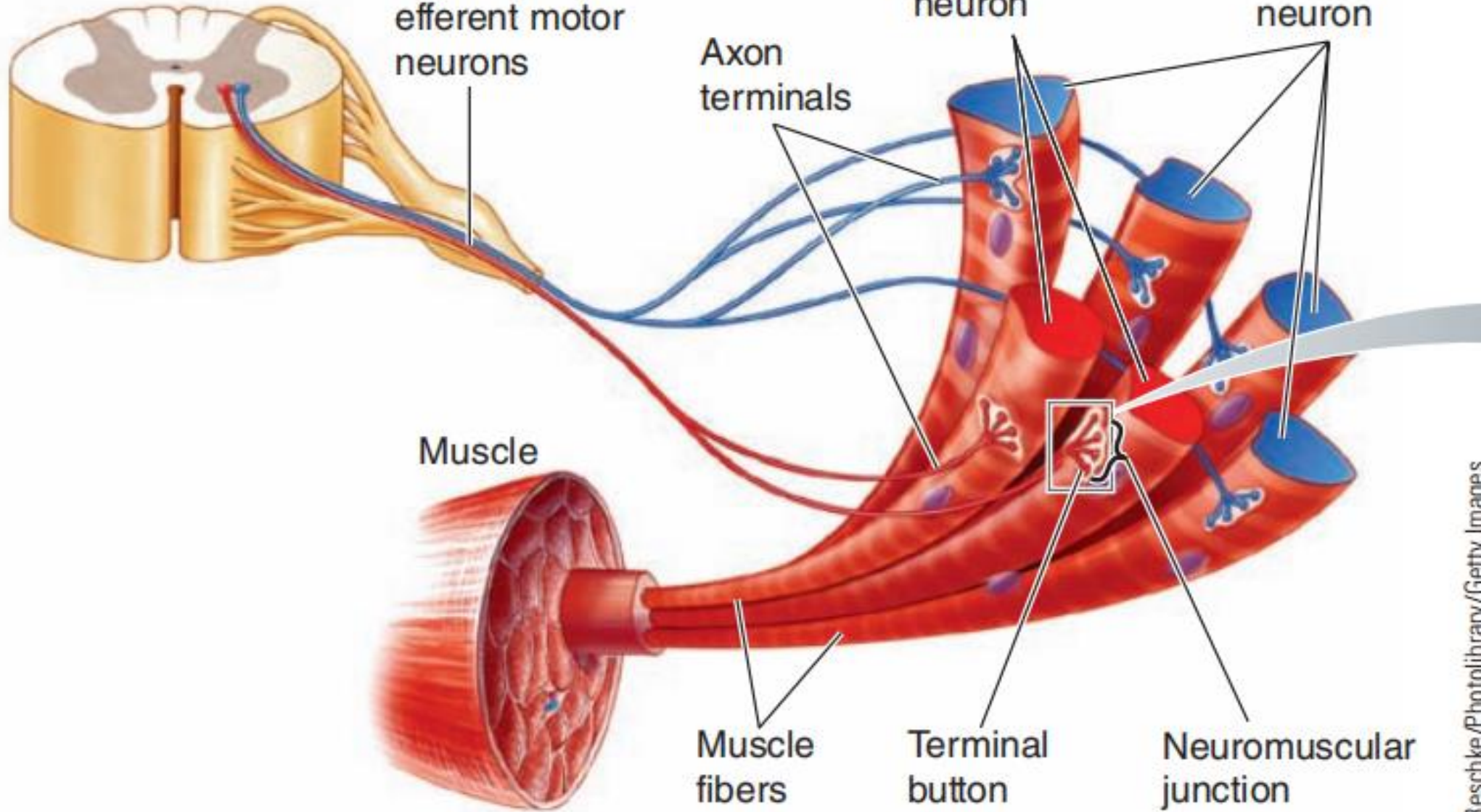
Axon terminals

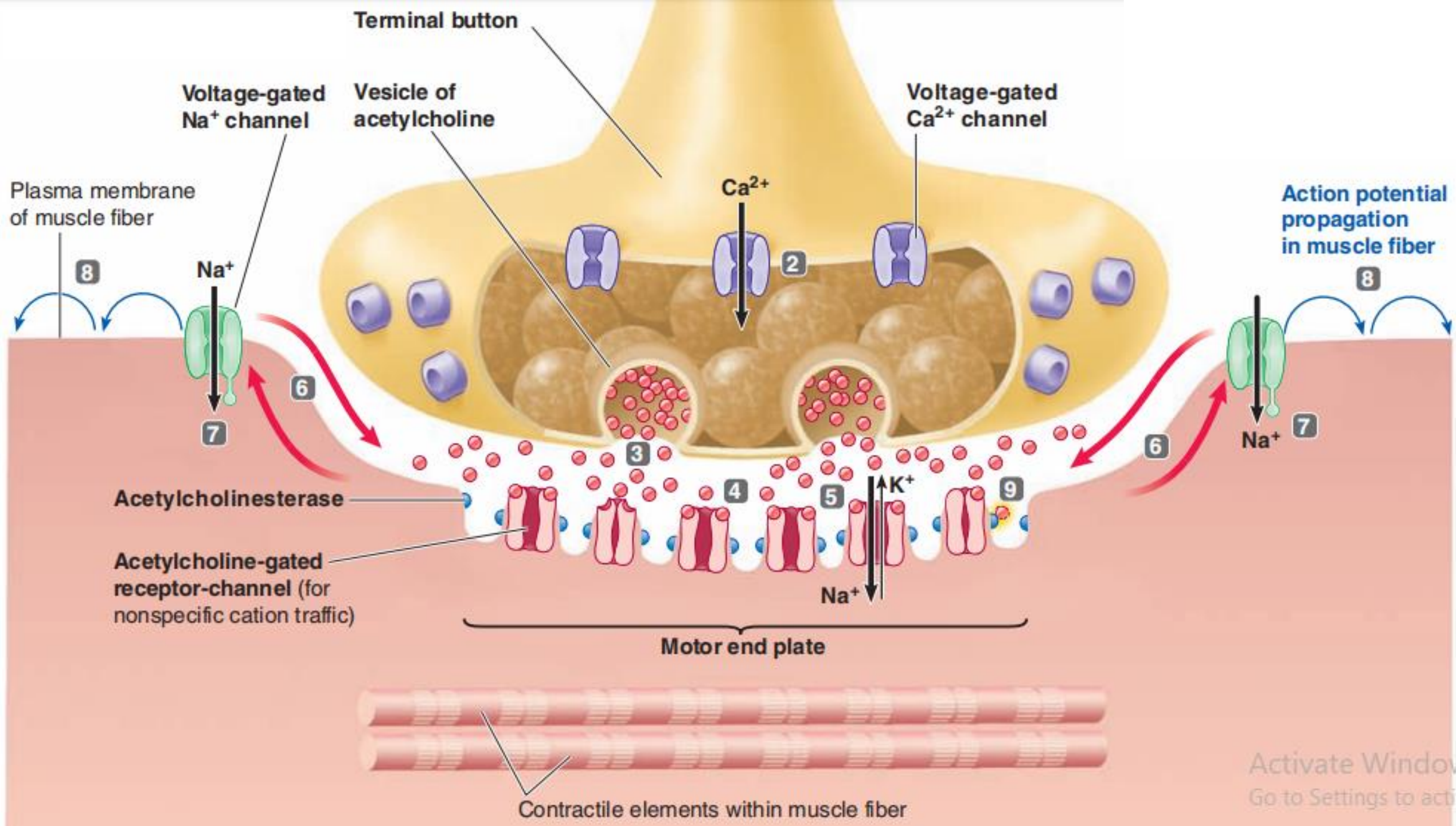
Muscle

Muscle fibers

Terminal button

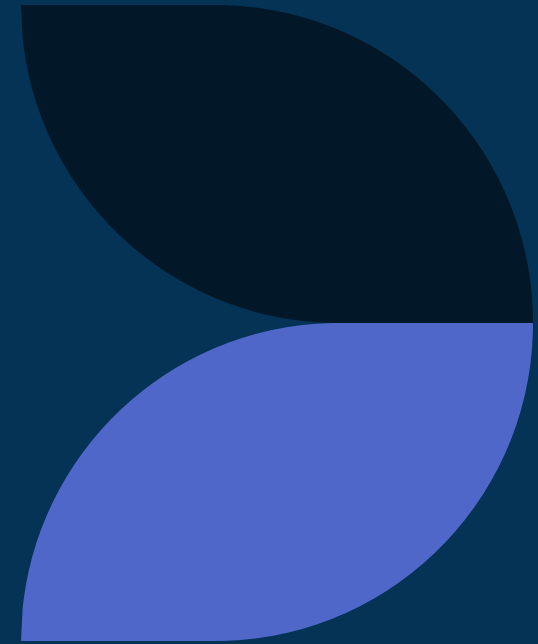
Neuromuscular junction





Introduction

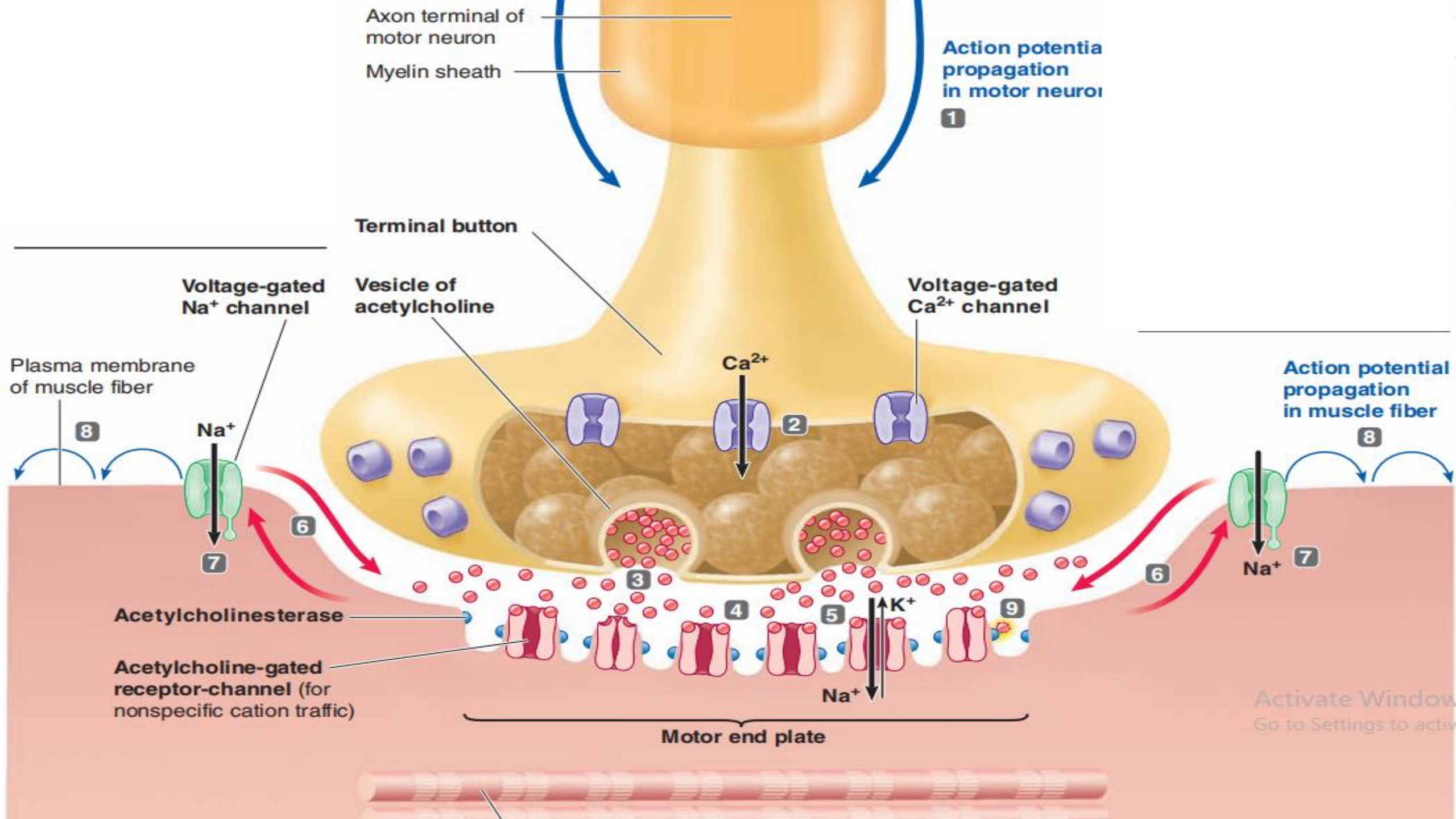
- Nerve and muscle cells do **not come into direct contact** at a neuromuscular junction.
- The space, or **(synaptic cleft)**, between these two structures is **too large for electrical transmission** of an impulse between them.
- A chemical messenger carries the signal between a terminal button and the muscle fiber. **This neurotransmitter is Ach.**



Release of ACh at the Neuromuscular Junction

- Each **terminal button** contains thousands of **vesicles** that **store ACh**.
- **1-** Propagation of an **action potential** to the axon terminal **triggers** the **opening of voltage-gated calcium (Ca^{2+}) channels** in its terminal buttons.
- **2-** When **Ca^{2+}** channels open, **Ca^{2+}** diffuses into the terminal button from its higher extracellular concentration, **3-** which in turn **causes release of ACh** by **exocytosis** from several hundred vesicles into the **synaptic cleft**.





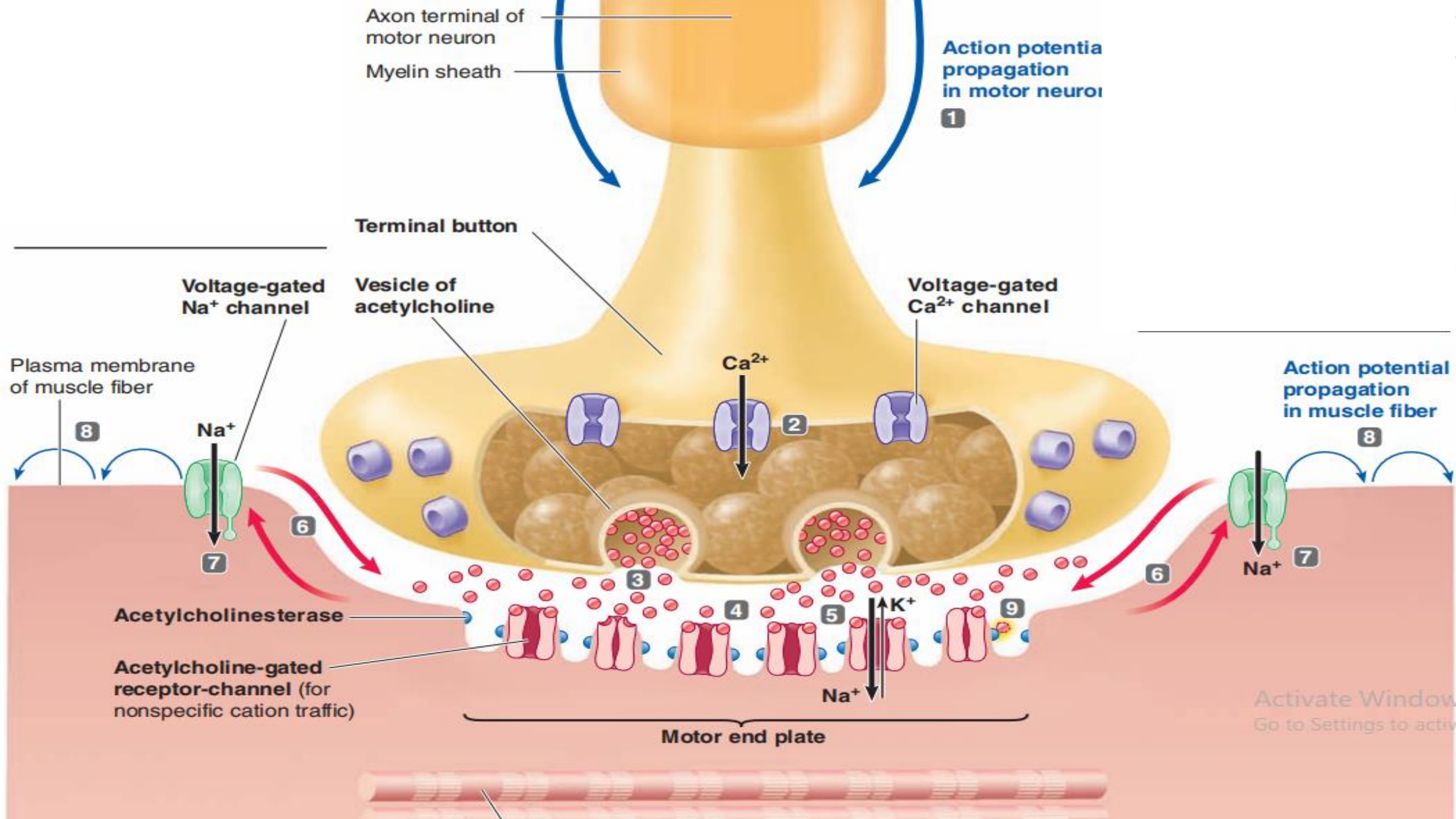
Formation of an End-Plate Potential



- **4-** The released ACh diffuses across the synaptic cleft and binds with chemically gated receptor channels (**Ligand-gated channels**) of the cholinergic nicotinic type on the motor end-plate portion of the muscle fiber membrane.
- **5-** Binding with ACh causes these receptor-channels to open.
- They are nonspecific cation channels that permit both Na⁺ and K⁺ traffic through them.



(more Na⁺ moves inward than K⁺ moves outward, leading to **depolarizing the motor end plate**)



Formation of an End-Plate Potential

- The **collective potential change** resulting from these ion movements across **all of the terminal buttons** within a neuromuscular junctions is called the **end-plate potential (EPP)**.



Initiation of an Action Potential

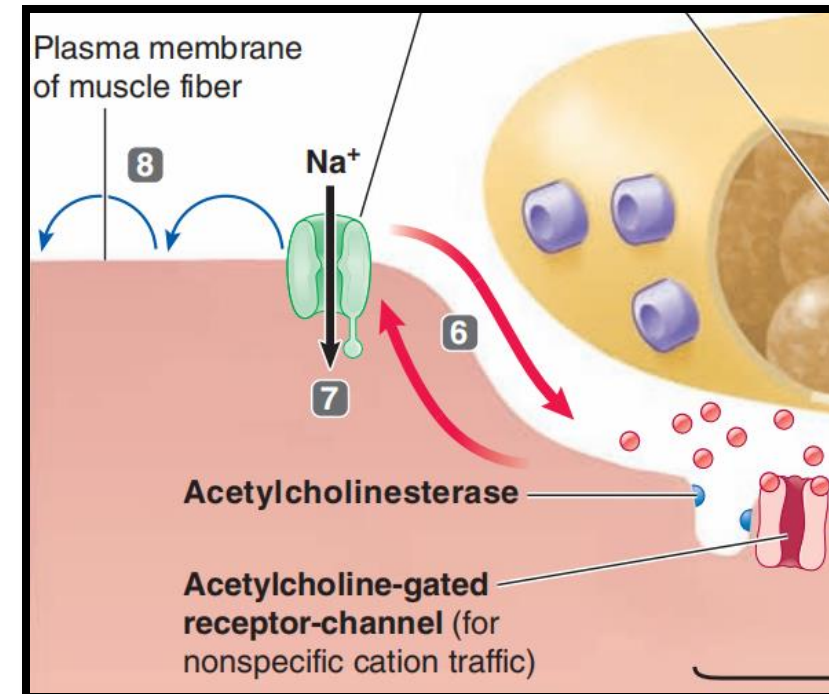


- The motor end-plate region itself does **not have a threshold potential**, so an action potential cannot be initiated at this site.
- The neuromuscular junction is usually in **the middle of the long, cylindrical muscle fiber**.
- When an EPP takes place, local current flow occurs between the depolarized end plate and the adjacent, resting cell membrane in both directions, opening voltage-gated Na⁺ channels and thus reducing the potential to threshold in the adjacent areas



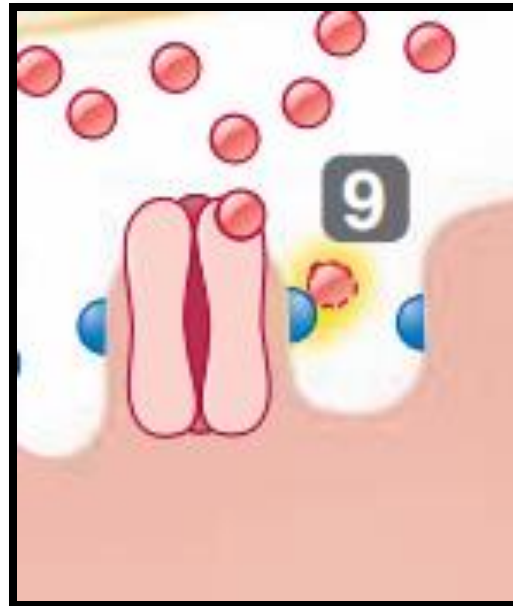
Initiation of an Action Potential

- **6-** When an **EPP** takes place, **local current flow occurs between the depolarized end plate and the adjacent resting cell membrane** in **both directions**, **7-** **opening voltage-gated Na^+ channels** and thus reducing the potential to threshold in the adjacent areas.

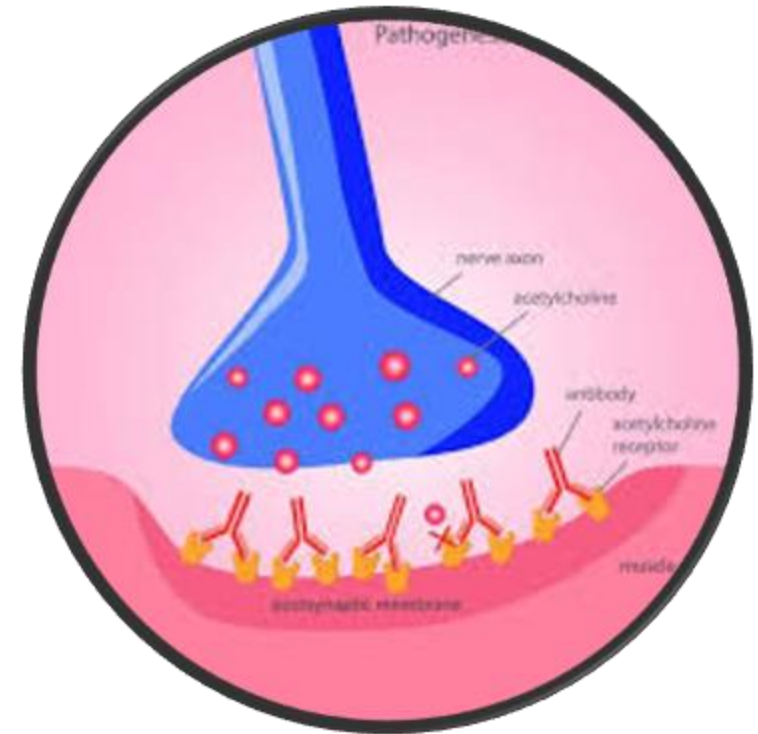


Termination of Ach Activity

- The muscle cell's electrical response is **turned off** by an enzyme in the motor end-plate membrane, **acetylcholinesterase (AChE)**, which **inactivates ACh**.
- **ACh removal ends the EPP.**



Clinical correlation



Black Widow Spider

- Black Widow Spider Venom Causes Explosive Release of Ach from the storage vesicles, not only at neuromuscular junctions but at all cholinergic sites.
- The most harmful result of which is **respiratory failure.**



Organophosphates



- Organophosphates (like **malathion and sarin**) **irreversibly inhibiting AChE**, causing accumulation of acetylcholine, leading to prolonged neuromuscular stimulation followed by paralysis.



Botulinum Toxin

- Botulinum toxin exerts its lethal blow by **blocking the release of ACh** from the terminal button in response to a motorneuron action potential.



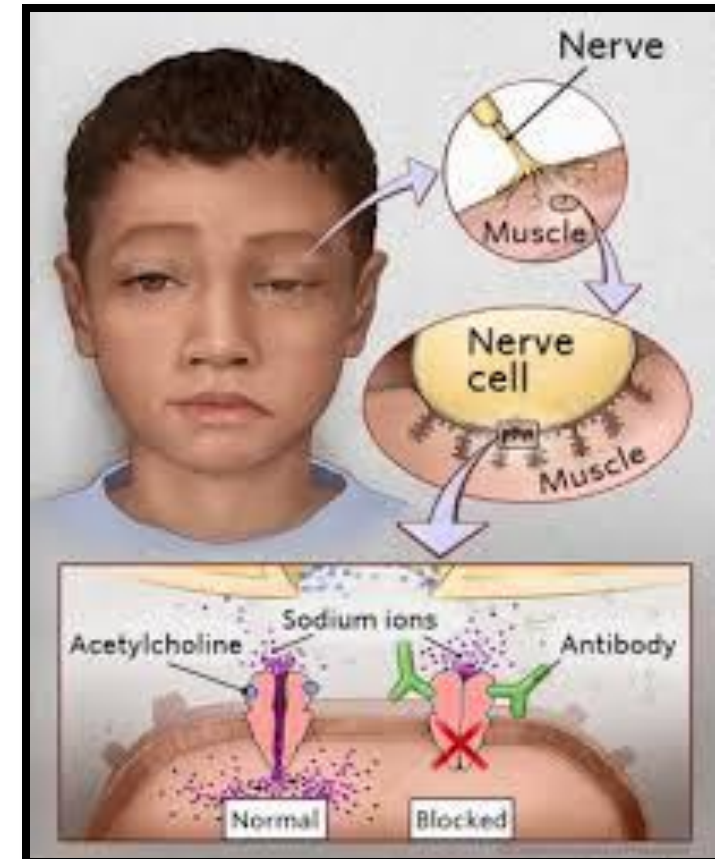
Curare

- Curare, which **reversibly binds to the ACh receptor-channels** on the motor end plate.



Myasthenia Gravis

- Myasthenia gravis, an **autoimmune disease** involving the neuromuscular junction, is characterized by extreme muscular weakness.
- The body erroneously produces **antibodies** against its motor end-plate **ACh receptor-channels**.
- Treatment: **neostigmine that inhibits AChE temporarily.**





Thank you