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# Physiology

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James P. Ryan ■ Michael B. Wang

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# Physiology

## PreTest® Self-Assessment and Review

## Tenth Edition


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PreTest® Self-Assessment and Review

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**Physiology: PreTest® Self-Assessment and Review, Tenth Edition**



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## Notice

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# Introduction

Each *PreTest® Self Assessment and Review* allows medical students to comprehensively and conveniently assess and review their knowledge of a particular basic science, in this instance physiology. The 500 questions parallel the subject areas and degree of difficulty presented by the United States Medical Licensing Examination (USMLE) Step 1.

Each question is accompanied by the correct answer and its explanation. The explanation provides the reason why the correct answer is correct and, in many cases, the reason why the wrong answers are wrong. In addition, the explanation provides additional information relevant to the topic the question is designed to test. The references accompanying each question are from the major textbooks purchased by most medical students. The material in the referenced pages will provide a more expansive description of the subject matter covered by the question.

One effective way to use the PreTest® is to answer 150 of the questions in two and a half hours. Write the answers on a separate sheet of paper and then compare your answers to the ones provided in the book. The PreTest® can also be used as a review book. Answer a group of questions covering the same topic, check your answers, and then read the explanations and the referenced text pages. Whichever way you use the PreTest®, the most important part of your review is to be found in the explanations. The information in the explanations is designed to reinforce and expand on the material covered by the questions.

The High-Yield Facts found at the beginning of the book are not meant to be a complete list of all of the important facts, concepts, and equations necessary for understanding physiology. However, those that are included offer a solid foundation of the subjects they do cover and should be included in your review of physiology in preparation for a class test or for the Step 1 Examination. If you are not familiar with a section of the material presented in the High-Yield Facts, you should plan to do more reading. If, on the other hand, you have a good grasp of this material, you can feel confident in your knowledge of that topic.

Good luck on your exam.

# Contents

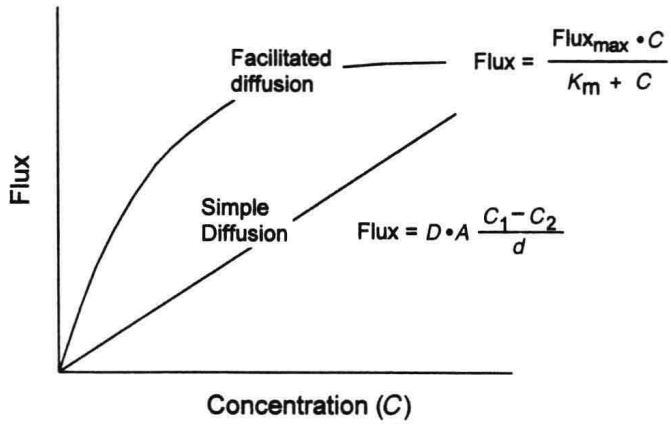
## Neurophysiology

# High-Yield Facts in Physiology

## CELLULAR PHYSIOLOGY

- Ions, nutrients, and waste material are transported across cell membranes by diffusion, osmosis, and active transport.

Simple diffusion is described by the Fick equation. Carrier-mediated diffusion, called *facilitated diffusion,* is described by the Michaelis-Menton equation (see figure below).




The flow of water through membranes by osmosis is described by the osmotic flow equation. Material dissolved in the water is carried across the membrane by solvent drag.

$$\text{Flow} = \sigma \cdot L \cdot (\pi_1 - \pi_2)$$

$\sigma$ = reflection coefficient
$L$ = hydraulic conductivity
$\pi$ = osmotic pressure

The reflection coefficient ($\sigma$)

$$\sigma = 1 - \frac{P_{solute}}{P_{water}}$$

is an index of the membrane's permeability to the solute and varies between 0 and 1. Particles that are impermeable to the membrane have a reflection coefficient of 1. Particles that are freely permeable to the membrane have a reflection coefficient of 0.

The osmotic pressure (in units of mmHg) is calculated with the van't Hoff equation:

$$\pi = c \cdot R \cdot T$$

Cells shrink when placed in hypertonic solutions and swell when placed in hypotonic solutions. *Tonicity* is the concentration of nonpermeable particles. The following equation is used to calculate the steady state volume of the cell:

$$\pi_{initial} \cdot V_{initial} = \pi_{final} \cdot V_{final}$$

Active transport processes may be primary or secondary. Primary active transport processes, such as the Na-K pump, use the energy derived from the hydrolysis of ATP to transport materials against their electrochemical gradient. Secondary active transport processes, such as those that transport glucose and amino acids into renal or intestinal epithelial cells, use the energy from the $Na^+$ electrochemical gradient.

- All cells have membrane potentials. The magnitude of the membrane potential is determined by the membrane permeability and ion concentration gradient of the ions that are permeable to the membrane.

In the resting state, the membrane is primarily permeable to $K^+$, and, therefore, the resting membrane potential is close to the equilibrium potential for $K^+$.

The equilibrium potential is calculated with Nernst's equation:

$$\left(E_{ion} = -60 \cdot \log \frac{Conc_{in}}{Conc_{out}}\right)$$

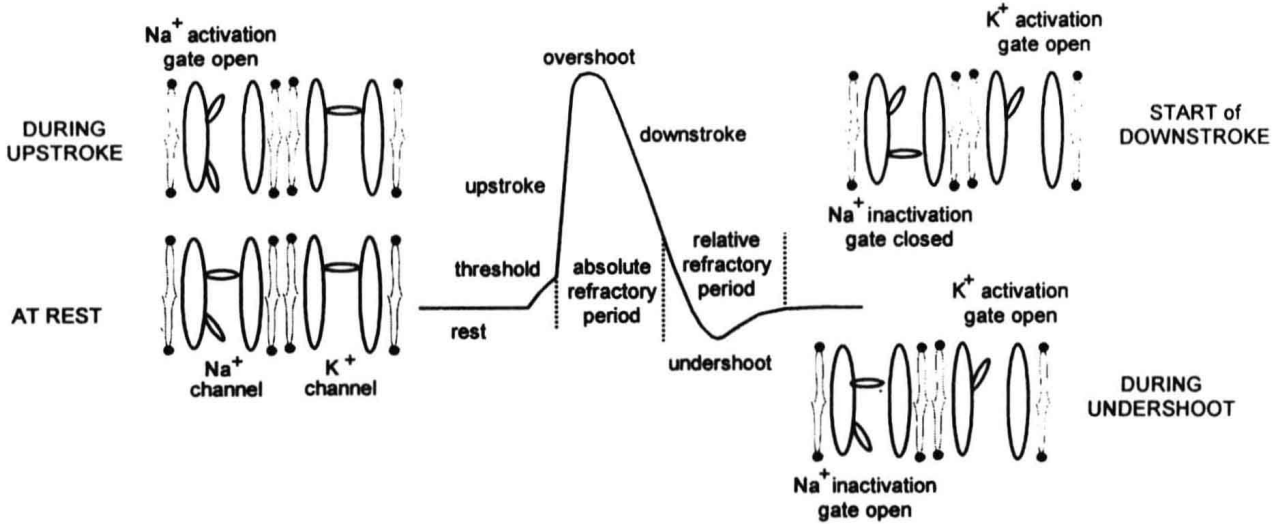
The resting potential is calculated with the transference equation:

$$\left(\begin{aligned} E_M &= T_K \cdot E_{Na} + T_{Na} \cdot E_{Na} \\ T_K &= \frac{g_K}{g_{Na} + g_K};\ T_{Na} = \frac{g_{Na}}{g_{Na} + g_K} \end{aligned}\right)$$

or with the Goldman equation:

$$E_M = -61 \cdot \log \frac{P_{Na} \cdot [Na]_{in} + P_K \cdot [K]_{in}}{P_{Na} \cdot [Na]_{out} + P_K \cdot [K]_{out}}$$

Action potentials are produced by voltage and time-dependent gates covering ion selective channels. Nerve and skeletal muscle membranes contain $Na^+$ and $K^+$ ion selective channels (see figure on p 3).


Na+ activation gate open
DURING UPSTROKE
AT REST
Na+ channel
K+ channel
overshoot
upstroke
downstroke
threshold
rest
absolute refractory period
relative refractory period
undershoot
K+ activation gate open
START of DOWNSTROKE
Na+ inactivation gate closed
K+ activation gate open
DURING UNDERSHOOT
Na+ inactivation gate open

- Cardiac muscle membranes contain $Na^+$, $K^+$, and $Ca^{2+}$ ion selective channels.
- The upstroke (phase 0) is produced by the activation of $Na^+$ channels.
- The initial repolarization (phase 1) is produced by inactivation of $Na^+$ channels.
- The plateau (phase 2) is caused by the activation of $Ca^{2+}$ channels and the closing of inward rectifying (anomalous) $K^+$ channels.
- The downstroke (phase 3) is caused by the activation of the delayed rectifier $K^+$ channels and the inactivation of $Ca^{2+}$ channels (see figure on p 5).
- Increasing extracellular $K^+$ concentration depolarizes the membrane and reduces its excitability. Excitability is reduced because the depolarization inactivates $Na^+$ channels. The reduced excitability can lead to muscle weakness and cardiac arrhythmias.
- Synaptic transmission is used to transmit information from one cell to another cell. The synaptic transmitter, released from the presynaptic cell by exocytosis, diffuses across a synaptic cleft and binds to a receptor on the postsynaptic cell. The effect produced on the postsynaptic cell depends on the identity of the synaptic transmitter and the receptor (see figure on p 6).

Acetylcholine, which binds to the end plate of skeletal muscle cells, and glutamate and GABA, which bind to the postsynaptic membranes of many central nervous system membranes, open ion selective channels.

Norepinephrine and acetylcholine, which bind to the postsynaptic membranes of smooth muscle cells, produce their effect by activating a G protein which, in turn, activates an enzyme-mediated response.

- Muscle contraction is produced by repetitive cycling of the myosin cross-bridges on thick filaments. The cross-bridges attach to actin molecules on the thin filaments and cause the thin filaments to slide over the thick filaments toward the center of the sarcomere (skeletal or cardiac muscle) or cell (smooth muscle; see figure on p 7).

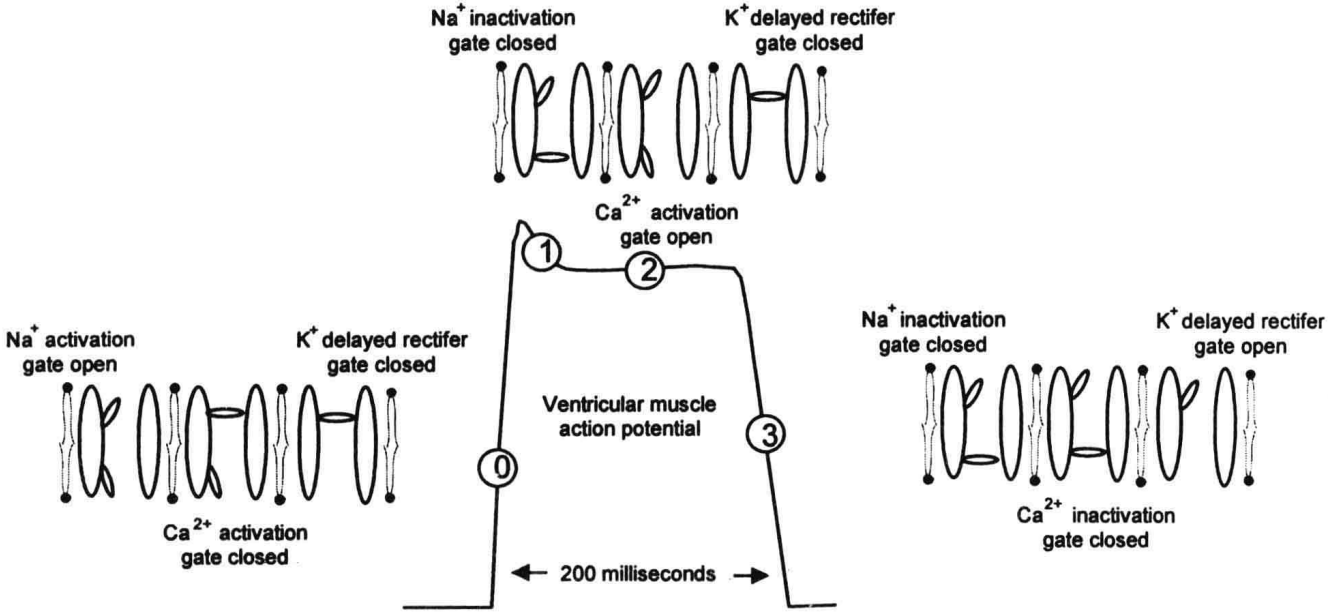
The initiation of contraction is called *excitation-contraction coupling.* In striated muscle, excitation-contraction coupling is initiated when $Ca^{2+}$ binds to troponin. Troponin causes tropomyosin to move, thereby exposing the actin binding site to myosin (see figure on p 8).

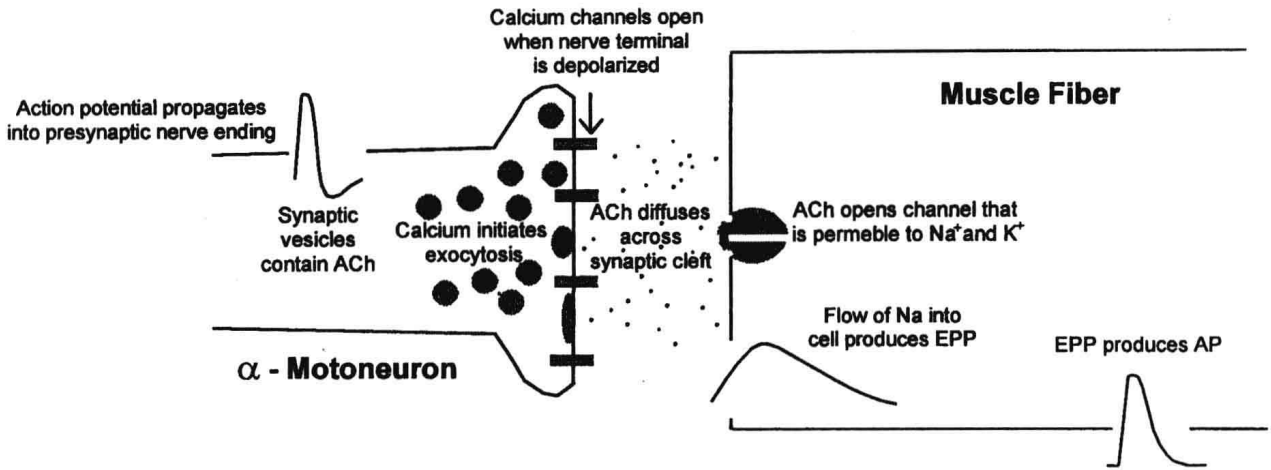
In skeletal muscle, $Ca^{2+}$ is released from the sarcoplasmic reticulum (SR) when the muscle fiber depolarizes.

In cardiac muscle, $Ca^{2+}$ is released from the SR by the $Ca^{2+}$ that enters the cell during the cardiac action potential.

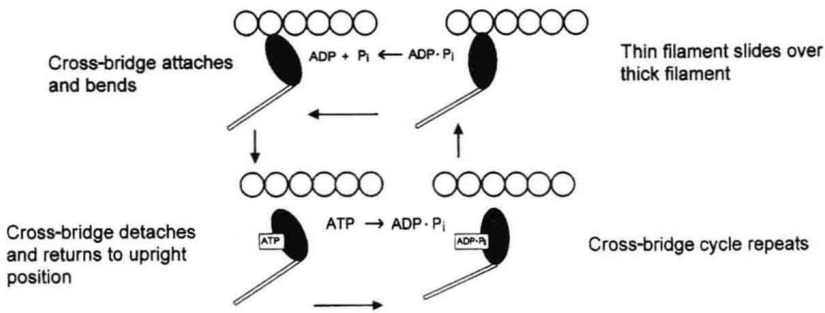
In smooth muscle, excitation-contraction coupling is initiated when $Ca^{2+}$ binds to calmodulin.

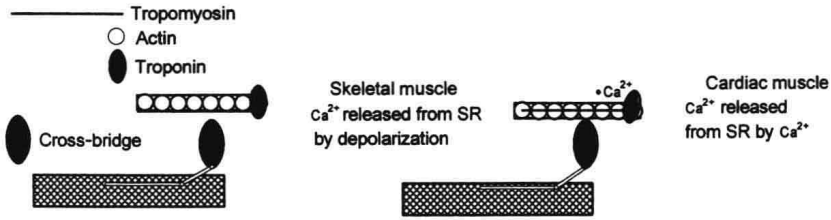
The $Ca^{2+}$-calmodulin complex activates myosin light chain kinase (MLCK) which, in turn, phosphorylates the 20,000-Da myosin light chains ($LC_{20}$). Cross-bridge cycling begins when the myosin light chains are phosphorylated.


Na+ inactivation gate closed
K+ delayed rectifer gate closed
Ca2+ activation gate open
1
2
Na+ activation gate open
K+ delayed rectifer gate closed
Ventricular muscle action potential
3
0
Na+ inactivation gate closed
K+ delayed rectifer gate open
Ca2+ activation gate closed
200 milliseconds
Ca2+ inactivation gate closed


Calcium channels open when nerve terminal is depolarized
Muscle Fiber
Action potential propagates into presynaptic nerve ending
Synaptic vesicles contain ACh
Calcium initiates exocytosis
ACh diffuses across synaptic cleft
ACh opens channel that is permeble to Na+ and K+
Flow of Na into cell produces EPP
EPP produces AP
α - Motoneuron

| Receptor | Enzyme | Enzyme Effect | Postsynaptic Response |
|---|---|---|---|
| Adrenergic Alpha$_1$ ($G_{\upsilon}$) | Activation of phospholipase C | Formation of $IP_3$ and DAG | $IP_3$ releases calcium from SR<br>DAG activates PKC<br>Calcium activates arteriolar smooth muscle cells |
| Adrenergic Alpha$_2$ ($G_i$) | Inhibition of adenylyl cyclase | Reduction in cAMP formation | Relaxes GI smooth muscle cells |
| Adrenergic Beta ($G_s$) | Activation of adenylyl cyclase | Formation of cAMP | cAMP activates PKA which:<br>• Increases $Ca^{2+}$ entry into cardiac muscle cells and increases contractility<br>• Increases sequestration of $Ca^{2+}$ in bronchiole smooth muscle cells and relaxes muscle |
| Cholinergic Muscarinic ($M_1$) | Activation of phospholipase C | Formation of $IP_3$ and DAG | $IP_3$ releases calcium from SR<br>DAG activates PKC<br>Calcium activates GI smooth muscle cells |

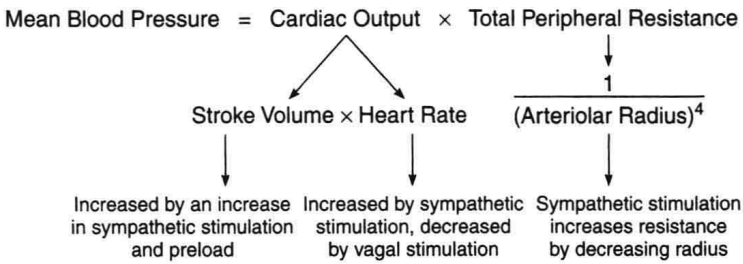





When dephosphorylated, the cross-bridges stay attached (or cycle slowly). The attached, slowly cycling cross-bridges are called latch bridges. Latch bridges allow smooth muscle to maintain force while minimizing energy expenditure.

## CARDIAC AND VASCULAR PHYSIOLOGY

- The heart pumps the blood through the circulation. The cardiac output from the heart must be sufficient to perfuse all the organs and maintain a pressure adequate to perfuse the brain (see figure below).




Cardiac output can be measured using the Fick equation:

$$CO = \frac{\dot{V}O_2 \text{ (oxygen consumption)}}{(\text{Arterial } O_2 \text{ content} - \text{Venous } O_2 \text{ content})}$$

Resistance can be calculated using the Poiseuille equation:

$$R = \frac{8 \cdot \eta \cdot L}{\pi \cdot r^4}$$

$\eta$ = viscosity
$L$ = length
$r$ = radius

Because resistance is inversely proportional to the 4th power of the radius, TPR is controlled by small variations in the diameter of the arterioles.