

PHYSIOLOGY OF CARDIAC MUSCLE

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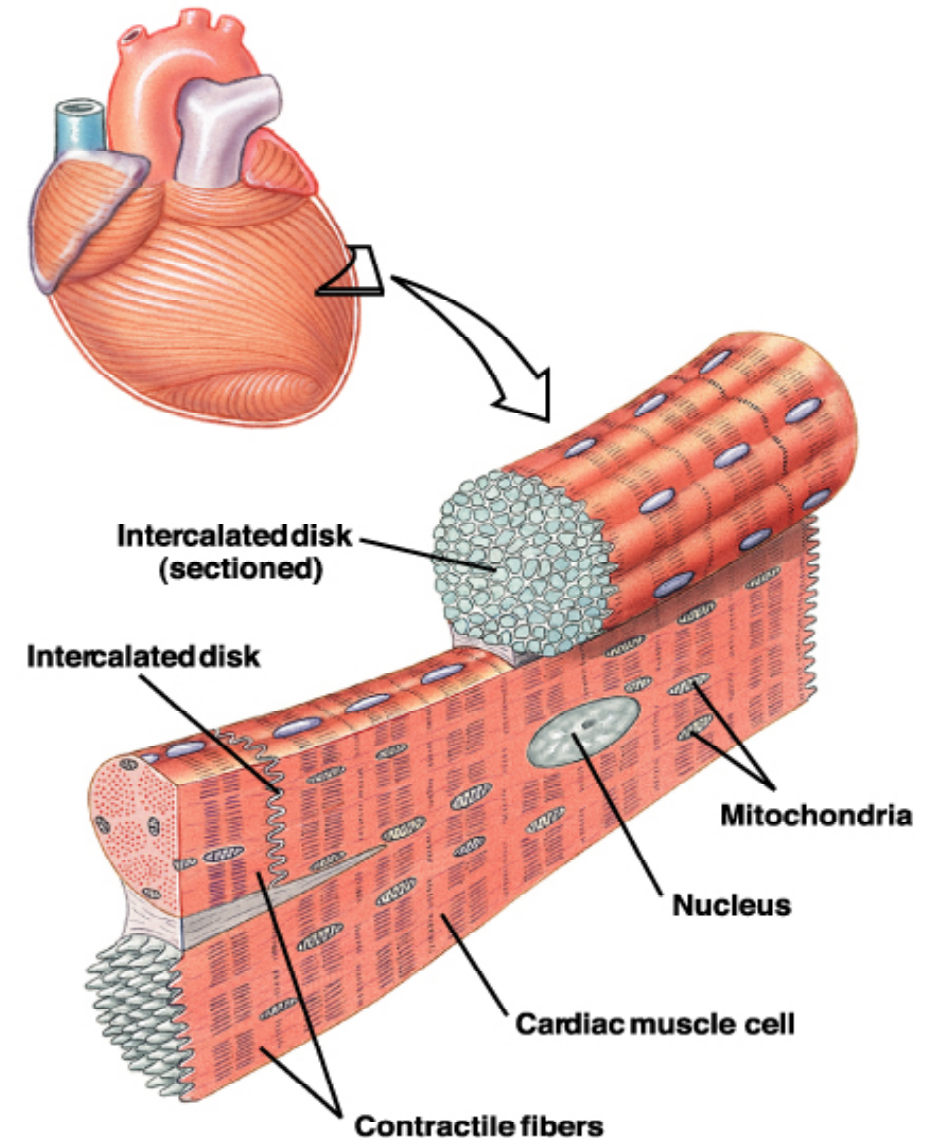
INTRODUCTION

- Cardiac muscle, like skeletal muscle & neurons, is an excitable tissue with the ability to generate action potential.
- The heart is composed of three major types of cardiac muscle:
 - Atrial muscle,
 - Ventricular muscle, and
 - Specialized excitatory and conductive muscle fibers.
- The atrial and ventricular types of muscle contract in much the same way as skeletal muscle, except duration of contraction is much longer.
- The specialized excitatory and conductive fibers of the heart, however, contract only feebly because they contain few contractile fibrils.

- The heart can contract without an outside signal because the signal for contraction is ***myogenic, originating within the heart itself.***
- The heart contracts, or beats, rhythmically as a result of action potentials that it generates by itself, a property called **auto-rhythmicity** (*auto* means “self”).
- The signal for myocardial contraction comes NOT from the nervous system but from specialized myocardial cells also called **auto-rhythmic cells.**
- These cells are also called **pacemaker cells** because they set the rate of the heart beat.

Structure of Cardiac Muscle fiber

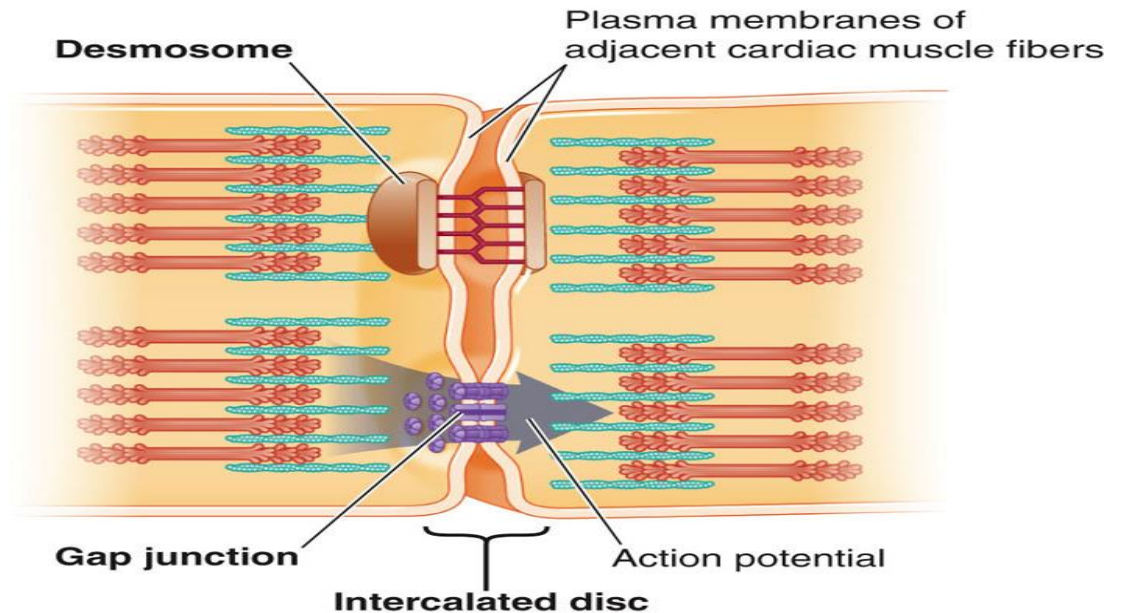
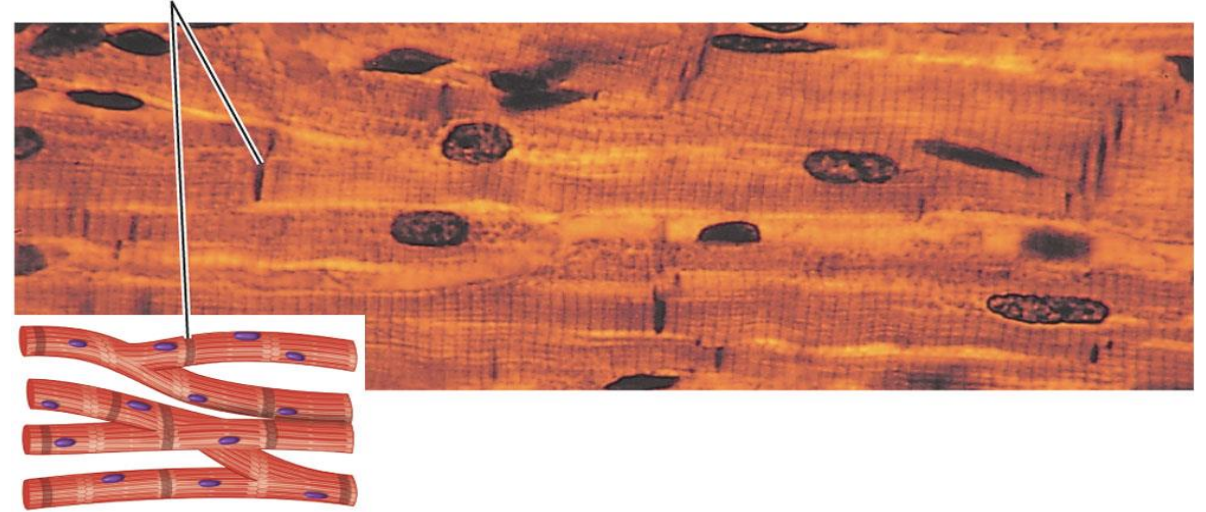
- Cardiac muscle fibers are **striated** in appearance.
- Has typical **myofibrils** that contain actin and myosin filaments
- Functional unit is called **Sarcomere**.
- Fibers are **branched**; connect to one another at **intercalated discs**.
- The discs contain several **Gap Junctions**.
- Fibers are not anchored at ends; allows for greater sarcomere shortening and lengthening.
- **Nuclei** are **centrally** located.
- **Abundant Mitochondria**.
- Sarcoplasmic Reticulum is less abundant than in skeletal muscle, but greater in density than smooth muscle.
- Sarcolemma has specialized ion channels (**Voltage-gated Ca^{2+} channels**) that skeletal muscle does not.



Intercalated discs

- Intercalated discs are cell membranes that separate individual cardiac muscle cells from one another.
- At each intercalated disc the cell membranes fuse with one another to form permeable “communicating” junctions (**gap junctions**) that allow rapid diffusion of ions.
- Ions move with ease in the intracellular fluid along the longitudinal axes of the cardiac muscle fibers, so that action potentials travel easily from one cardiac muscle cell to the next past the intercalated discs.

Intercalated discs



- Cardiac muscle is a syncytium of many heart muscle cells in which the cardiac cells are so interconnected that when one cell becomes excited, the action potential rapidly spreads to all of them.
- The heart actually is composed of two syncytiums:
 - **the atrial syncytium**, which constitutes the walls of the two atria, and
 - **the ventricular syncytium**, which constitutes the walls of the two ventricles.
- The atria are separated from the ventricles by **fibrous tissue** that surrounds the **atrioventricular (A-V) valvular openings** between the atria and ventricles.
- Normally, potentials are not conducted from the atrial syncytium into the ventricular syncytium directly through this fibrous tissue.

CARDIAC MUSCLE PROPERTIES

The cardiac muscle cells are responsible for the electrical stimulation which leads to mechanical function.

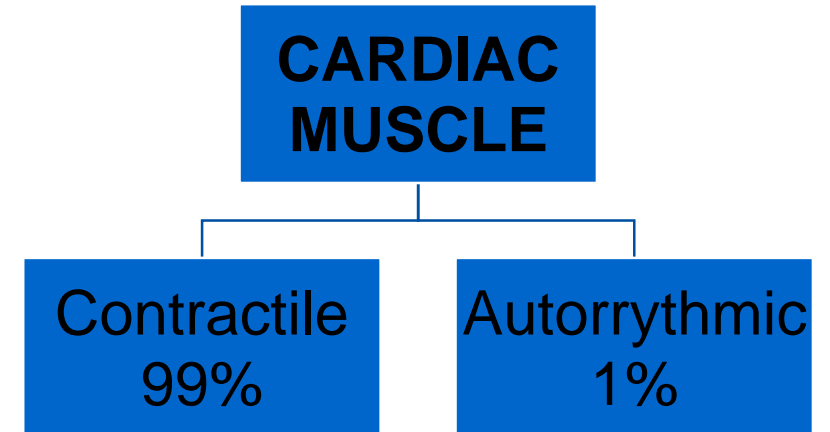
The electro-physiologic properties of cardiac muscles are:

1. **Automaticity:** Ability to spontaneously generate an electrical impulse.
2. **Excitability:** Ability to respond to an electrical impulse.
3. **Conductivity:** Allows transmission of electrical impulse to another cardiac cell.
4. **Contractility:** Ability to contract after electrical impulse response
5. **Rhythmicity:** Ability to send electrical impulses in a regularly manner.

Types of Cardiac Muscle Cells

Two specialized types of cardiac muscle cells:
Each of these 2 types of cells has a distinctive action potential.

- 1. Contractile cells**, which are 99% of the cardiac muscle cells, do the mechanical work of pumping. These working cells normally do not initiate their own action potentials.
- 2. Auto-rhythmic cells**, do not contract but instead are specialized for initiating and conducting the action potentials responsible for contraction of the working cells.

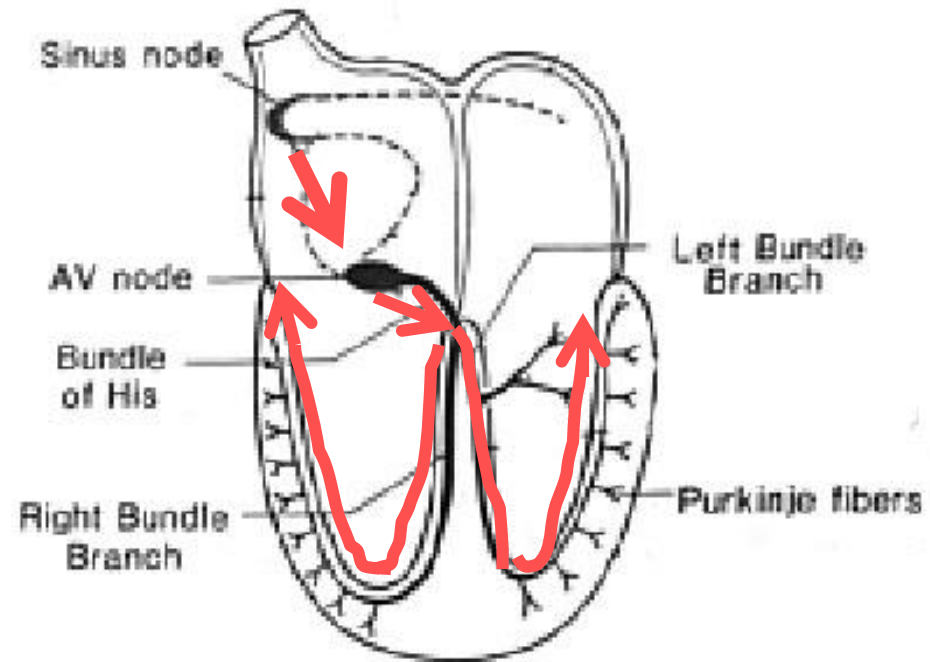
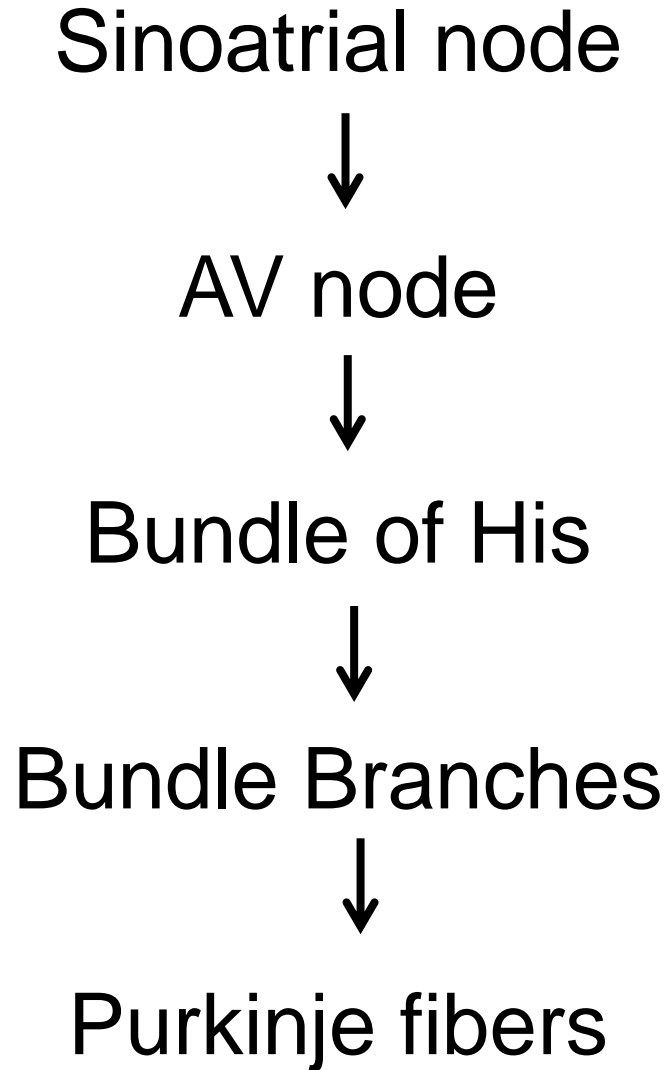


Autorhythmic cells

The specialized noncontractile cardiac cells capable of autorhythmicity lie in the following specific sites:

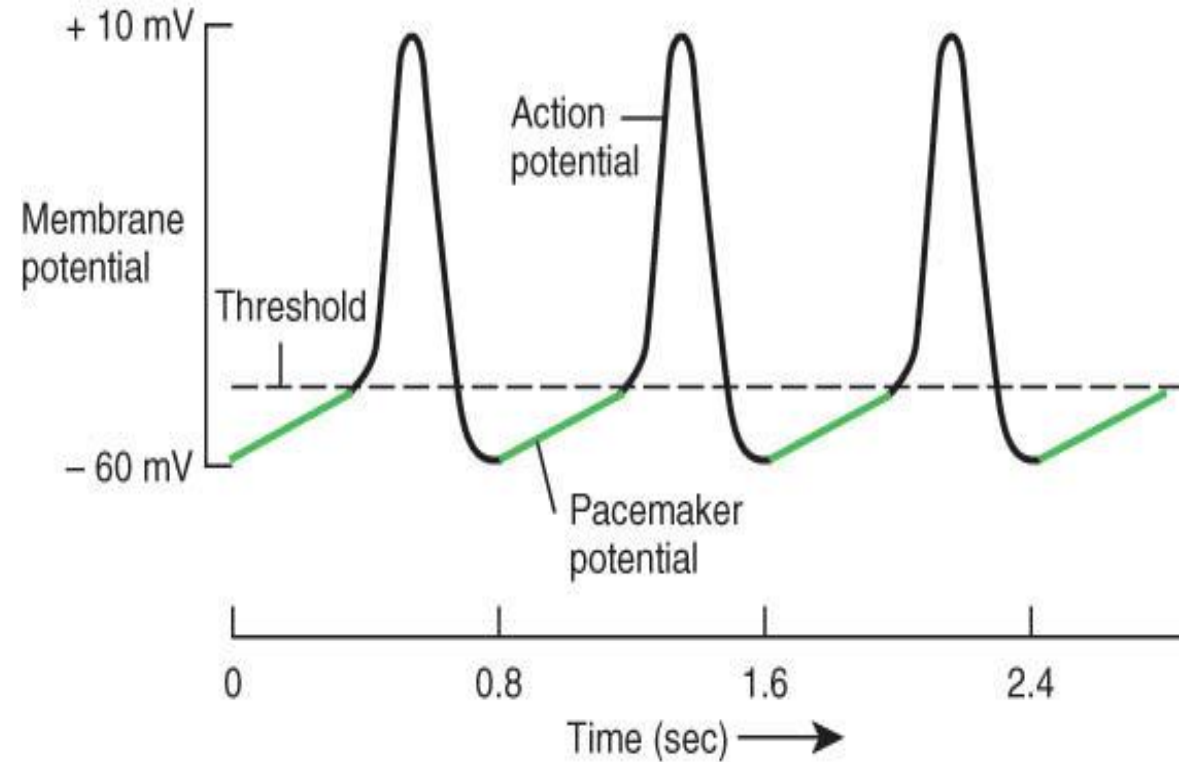
1. **The sinoatrial node (SA node)**, a small, specialized region in the right atrial wall. Primary pacemaker of the heart. Generates spontaneous action potentials due to the presence of funny channels.
2. **The atrioventricular node (AV node)**, a small bundle of specialized cardiac muscle cells located at the base of the right atrium near the septum. Delays the conduction of action potentials, allowing for proper atrial contraction before ventricular contraction. Protects the ventricles from excessively fast impulses.
3. **The bundle of His**, a tract of specialized cells that originates at the AV node and enters the septum between the ventricles. Here, it divides to form the **right and left bundle branches** that travel down the septum, curve around the tip of the ventricular chambers, and travel back toward the atria along the outer walls.
4. **Purkinje fibers**, small terminal fibers that extend from the bundle of His and spread throughout the ventricular myocardium much like small twigs of a tree branch. Rapidly conduct action potentials to ensure synchronous ventricular contraction. High conduction velocity due to fewer gap junctions

Normal Impulse Conduction



Action Potential of the Autorhythmic cardiac cells

- The auto rhythmic cells **do not** have a stable resting membrane potential like the nerve and the skeletal muscles.
- Instead they have an unstable membrane potential that starts at -60mV and slowly drifts upwards towards threshold.
- Because the membrane potential never rests at a constant value, it is called a ***Pacemaker Potential*** rather than a resting membrane potential.



(b) Pacemaker potentials and action potentials in autorhythmic fibers of SA node

Ionic Basis of Action Potential of Autorhythmic Cells

Phase 1: Pacemaker Potential:

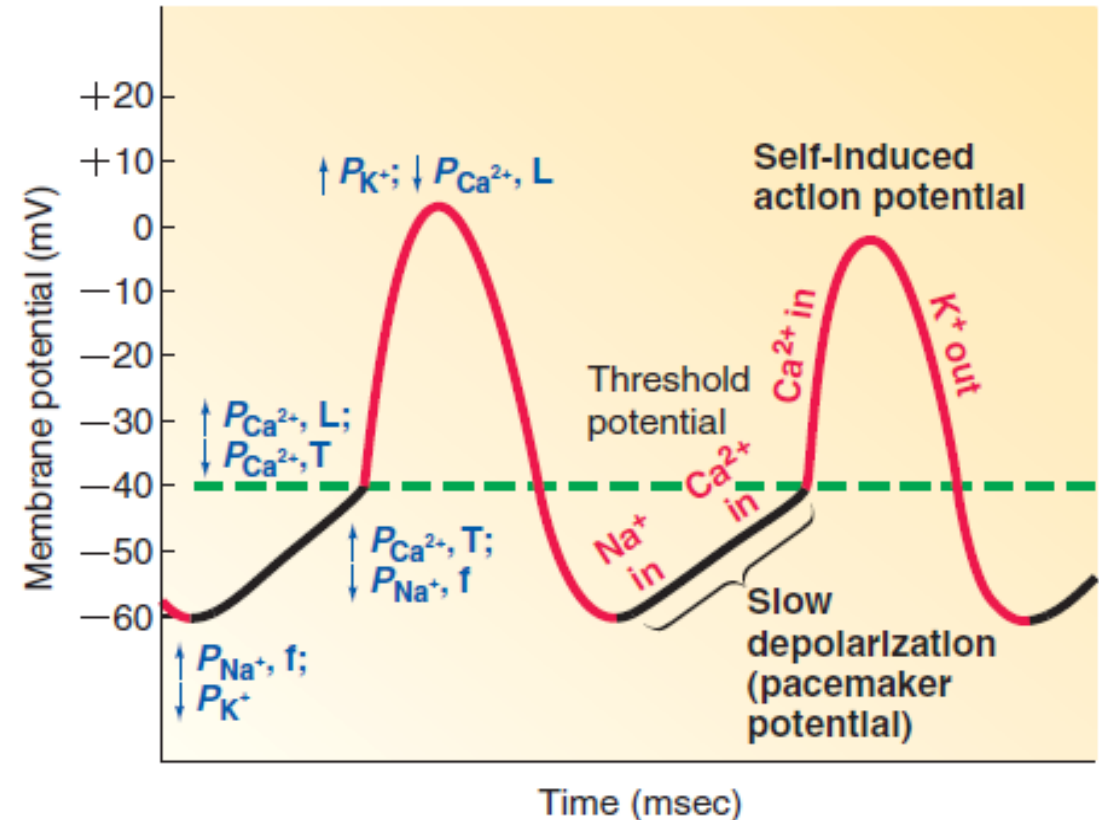
- Opening of voltage-gated Sodium channels called Funny channels (I_f or f channels).
- Closure of voltage-gated Potassium channels.
- Opening of Voltage-gated Transient-type Calcium (T-type Ca^{2+} channels) channels.

Phase 2: The Rising Phase or Depolarization:

- Opening of Long-lasting voltage-gated Calcium channels (L-type Ca^{2+} channels).
- Large influx of Calcium.

Phase 3: The Falling Phase or Repolarization:

- Opening of voltage-gated Potassium channels
- Closing of L-type Ca channels.
- Potassium Efflux.

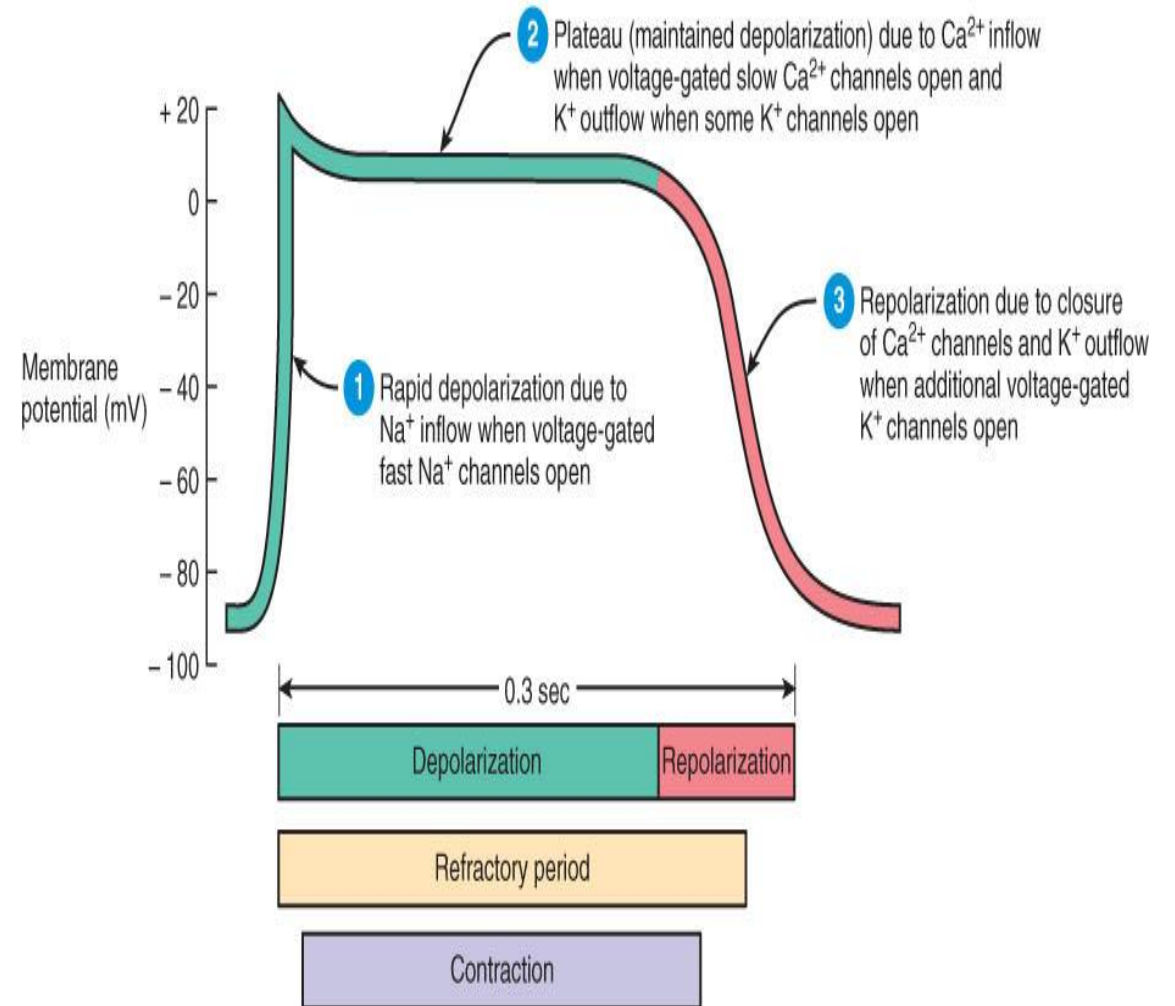


KEY

- f = Funny channels
- T = Transient-type channels
- L = Long-lasting channels

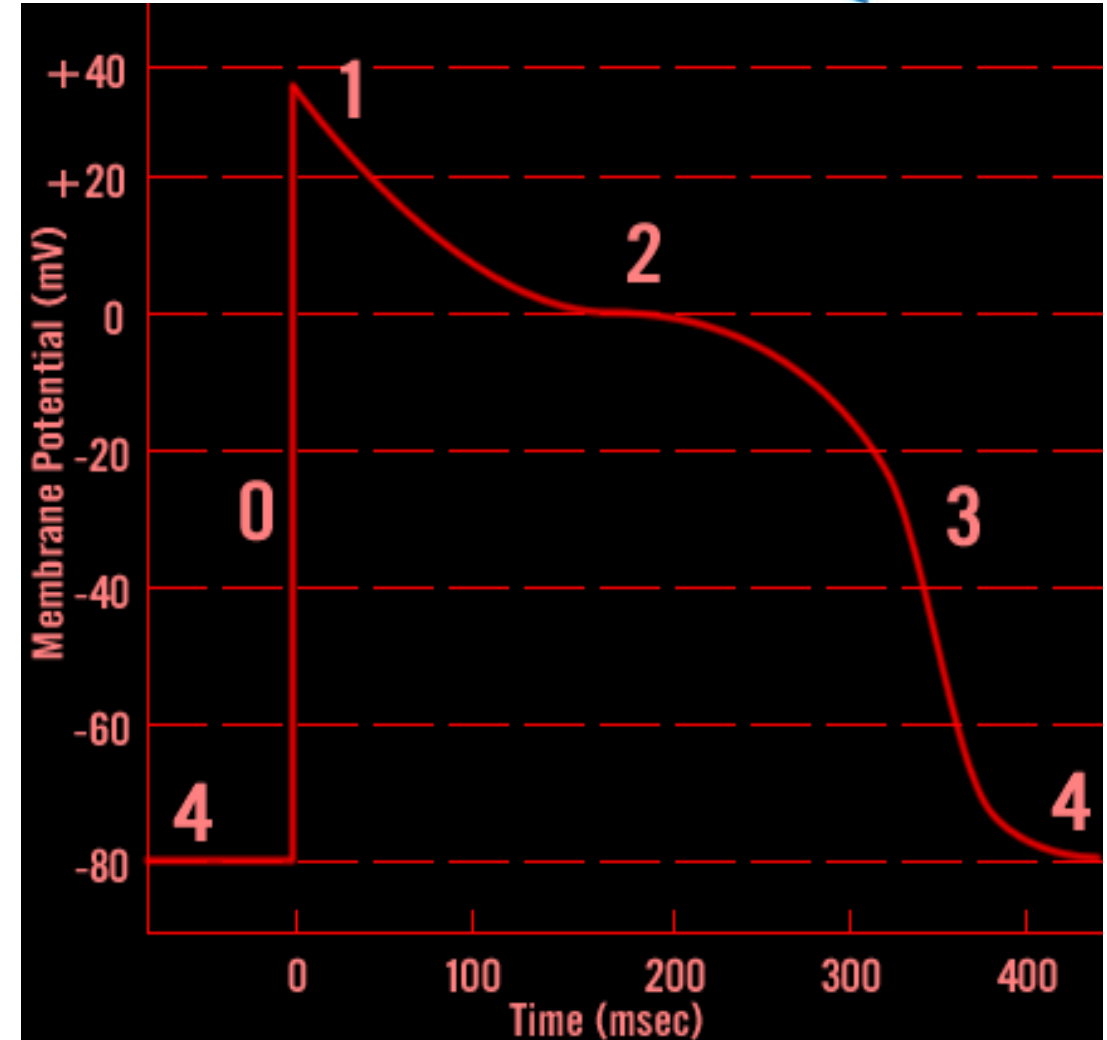
Action Potential of a Contractile Myocardial cell: VENTRICULAR CELL

- Unlike the membranes of the autorhythmic cells, the membrane of the contractile cells remain essentially at rest at about -90mV until excited by electrical activity propagated by the pacemaker cells.

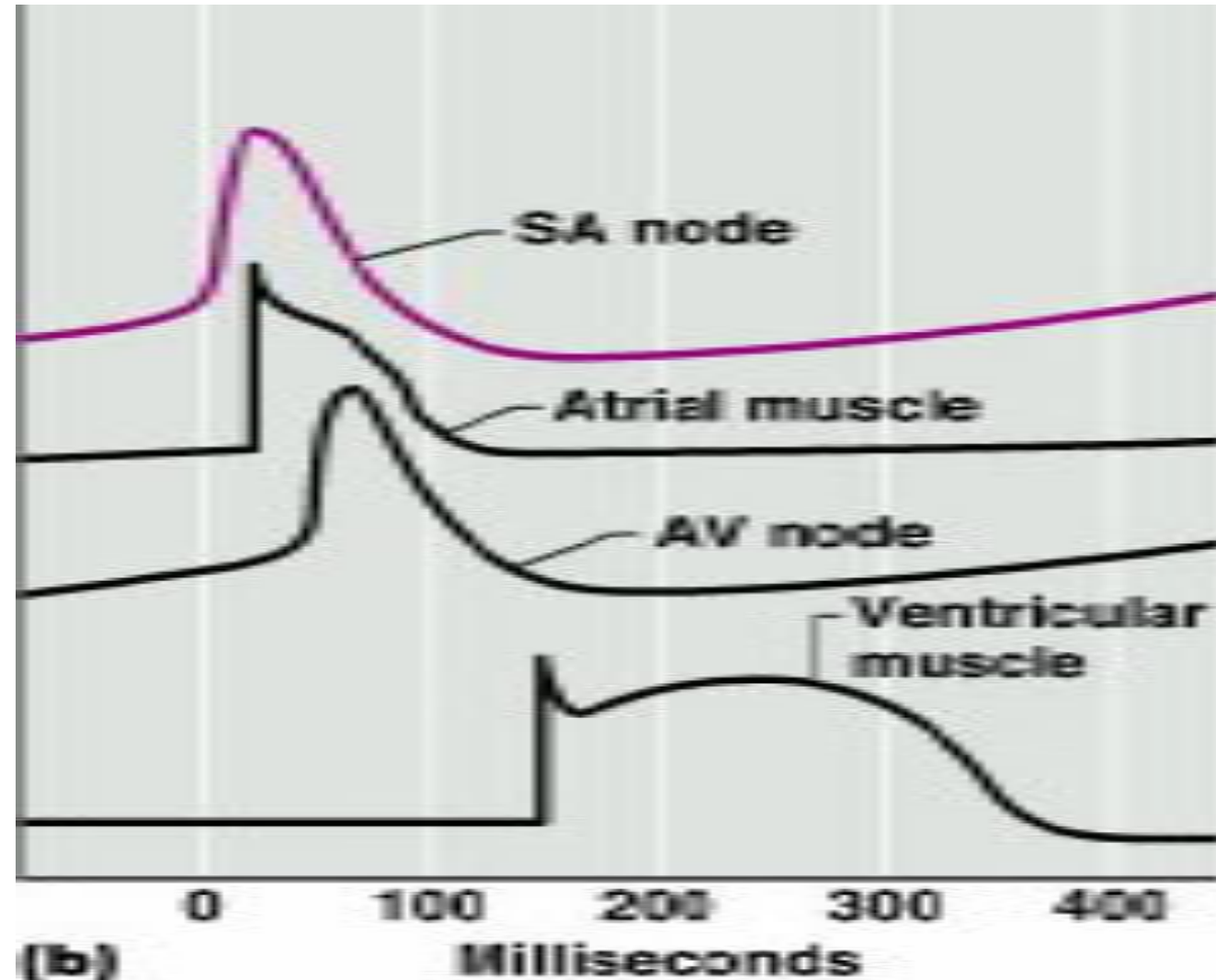


Ionic Basis of Action Potential of Contractile Cells

- **Depolarization(0)**
 - Opening of fast voltage-gated Na^+ channels.
 - Rapid Influx of Sodium ions leading to rapid depolarization.
- **Small Repolarization(1)**
 - Opening of a subclass of Potassium channels which are fast channels.
 - Rapid Potassium Efflux.
- **Plateau phase(2)**
 - 250 msec duration (while it is only 1msec in neuron)
 - Opening of the L-type voltage-gated slow Calcium channels & Closure of the Fast K^+ channels.
 - Large Calcium influx
 - K^+ Efflux is very small as K^+ permeability decreases & only few K channels are open.
- **Repolarization(3)**
 - Opening of the typical, slow, voltage-gated Potassium channels.
 - Closure of the L-type, voltage-gated Calcium channels.
 - Calcium Influx STOPS
 - Potassium Efflux takes place.

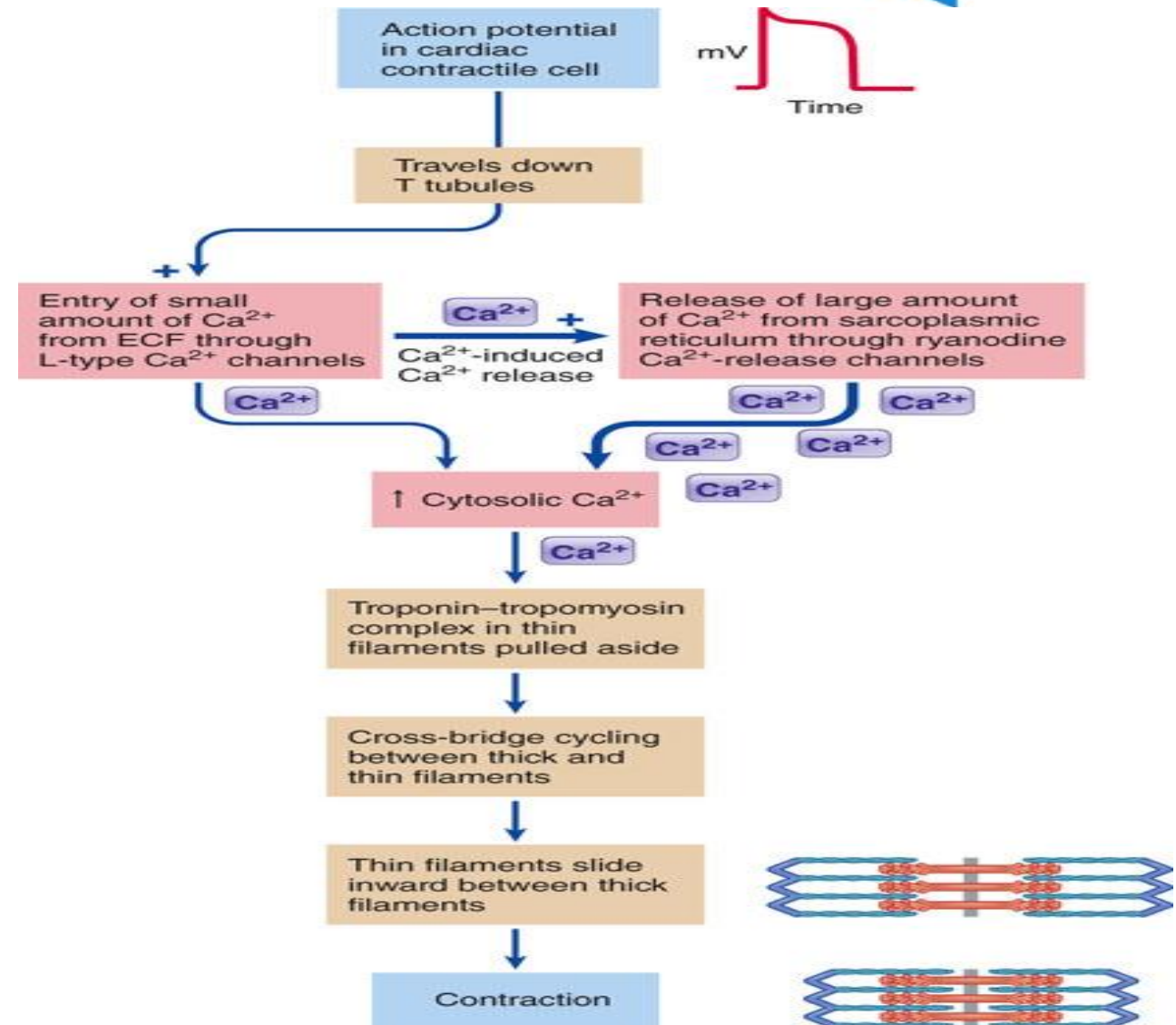


Action Potentials of different cardiac cells:

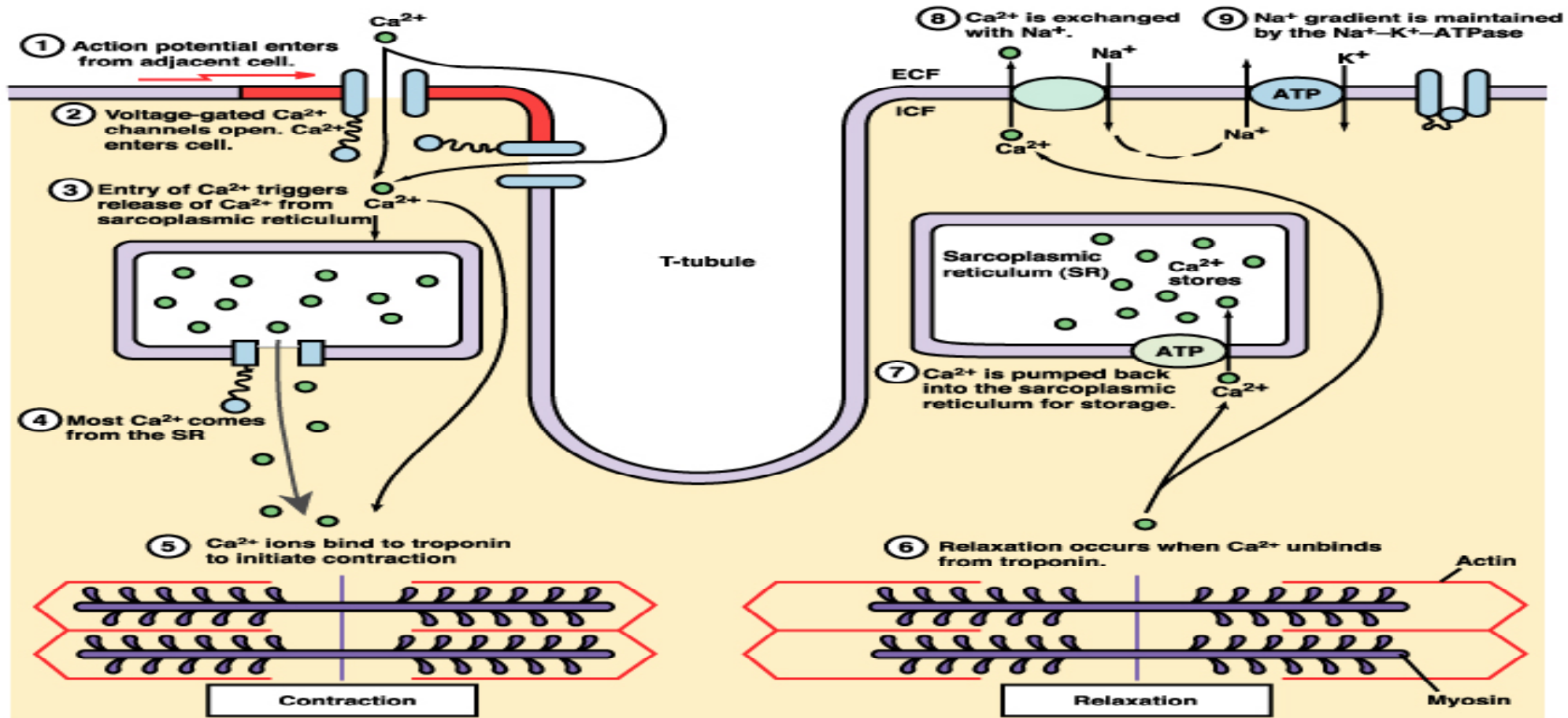


Excitation-Contraction Coupling of Cardiac Muscle

- Excitation of the heart is triggered by electrical impulse rather than neural transmitters.
- Contraction of the heart is triggered by elevation of intracellular calcium influx.



Excitation-Contraction coupling



Calcium ions regulate the contraction of cardiac muscle:

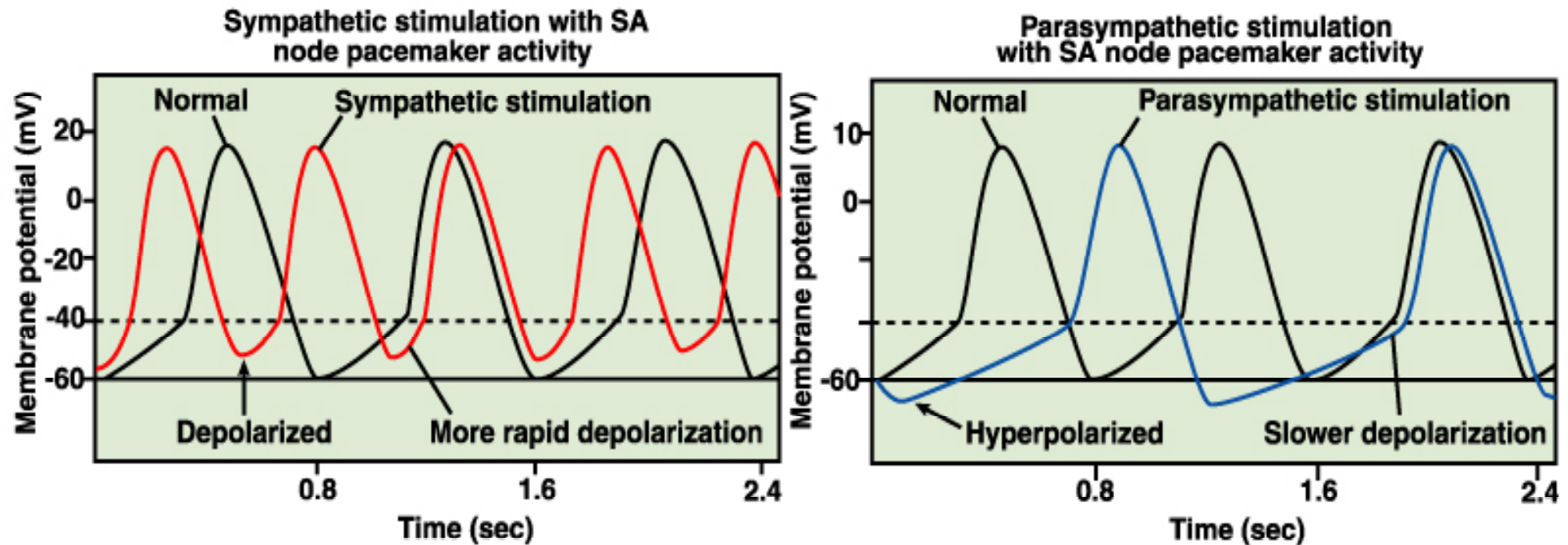
Entry of extracellular calcium ions causes the release of calcium from the sarcoplasmic reticulum (calcium-induced calcium release), source of about 95% of calcium in cytosol.

Factors regulating contractility

Table 14.1 Effects of Autonomic Nerve Activity on the Heart

Region Affected	Sympathetic Nerve Effects	Parasympathetic Nerve Effects
SA node	Increased rate of diastolic depolarization; increased cardiac rate	Decreased rate of diastolic depolarization; decreased cardiac rate
AV node	Increased conduction rate	Decreased conduction rate
Atrial muscle	Increased strength of contraction	Decreased strength of contraction
Ventricular muscle	Increased strength of contraction	No significant effect

Factors regulating contractility



- Autonomic nervous system modulates the frequency of depolarization of pacemaker
- Sympathetic stimulation (neurotransmitter); binds to β_1 receptors on the SA nodal membranes
- Parasympathetic stimulation (neurotransmitter); binds to muscarinic receptors on nodal membranes; increases conductivity of K^+ and decreases conductivity of Ca^{2+}

Clinical Significance

- **Arrhythmias:** Disturbances in the normal sequence of action potentials can lead to arrhythmias. Monitoring and analyzing action potentials are crucial in diagnosing and managing arrhythmias.
- **Drug Effects:** Certain medications, such as antiarrhythmics, affect ion channel activity, influencing action potentials. Understanding these effects is essential for prescribing and monitoring drug therapies.



**THANK YOU
FOR
LISTENING**