

Cardiac Muscle and the Cardiac Conduction System

Electrical and Contractile Activity of the Heart



**Al-Farabi Kazakh
National
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Medicine**



LEARNING OUTCOMES

As a result of the lesson you will be able to:

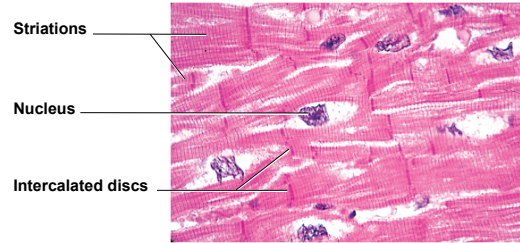
- ❑ *Describe the unique metabolic characteristics of cardiac muscle;*
- ❑ *Explain the functional significance of the intercellular junctions between cardiac muscle cells;*
- ❑ *Describe the heart's pacemaker and internal electrical conduction system and the pathway of impulses through this system.*
- ❑ *Describe the nerve supply to the heart and explain its role*
- ❑ *Explain why the SA node fires spontaneously and rhythmically*
- ❑ *Describe the unusual action potentials of cardiac muscle and relate them to the contractile behavior of the heart;*

Structure of Cardiac Muscle

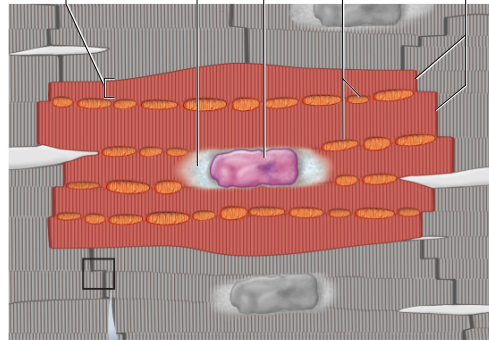
- **cardiocytes** - striated, short, thick, branched cells, one central nucleus surrounded by light staining mass of glycogen
- **intercalated discs** - join cardiocytes end to end
 - **interdigitating folds** – folds interlock with each other, and increase surface area of contact
 - **mechanical junctions** tightly join cardiocytes
 - **fascia adherens** – broad band in which the actin of the thin myofilaments is anchored to the plasma membrane
 - each cell is linked to the next via transmembrane proteins
 - **desmosomes** - weldlike mechanical junctions between cells
 - prevents cardiocytes from being pulled apart
 - **electrical junctions - gap junctions** allow ions to flow between cells – can stimulate neighbors
 - entire myocardium of either two atria or two ventricles acts like single unified cell
- repair of damage of cardiac muscle is almost entirely by **fibrosis** (scarring)

Structure of Cardiac Muscle Cell

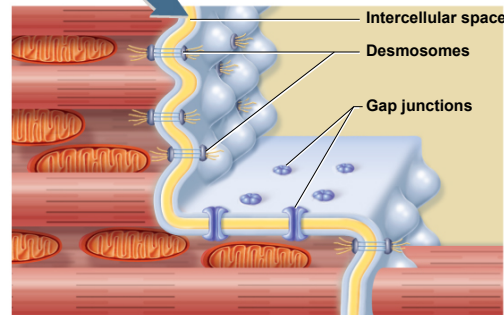
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Striated myofibril Glycogen Nucleus Mitochondria Intercalated discs



(b)



(c)

a: © Ed Reschke

Figure 19.11 a-c

Metabolism of Cardiac Muscle

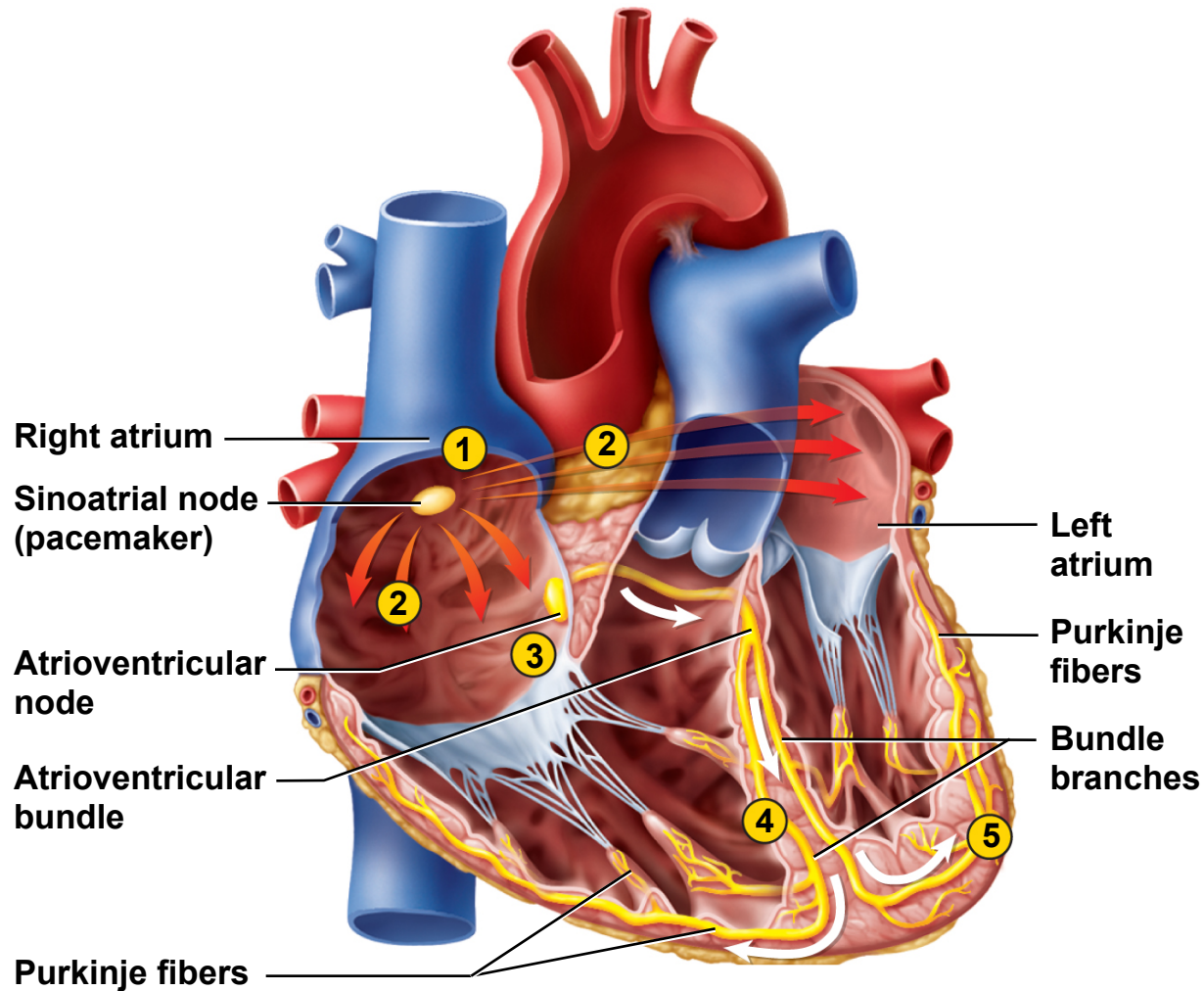
- **cardiac muscle** depends almost exclusively on **aerobic respiration** used to make ATP
 - rich in myoglobin and glycogen
 - huge mitochondria – fill 25% of cell
- **adaptable to organic fuels** used
 - fatty acids (60%), glucose (35%), ketones, lactic acid and amino acids (5%)
 - more vulnerable to oxygen deficiency than lack of a specific fuel
- **fatigue resistant** since makes little use of anaerobic fermentation or oxygen debt mechanisms
 - does not fatigue for a lifetime

Cardiac Conduction System

- coordinates the heartbeat
 - composed of an **internal pacemaker** and **nervelike conduction pathways** through myocardium
 - generates and conducts rhythmic electrical signals in the following order:
- **sinoatrial (SA) node** - modified cardiocytes
 - initiates each heartbeat and determines heart rate
 - signals spread throughout atria
 - **pacemaker** in right atrium near base of superior vena cava
- **atrioventricular (AV) node**
 - located near the right AV valve at lower end of interatrial septum
 - electrical gateway to the ventricles
 - fibrous skeleton acts as an insulator to prevent currents from getting to the ventricles from any other route
- **atrioventricular (AV) bundle (bundle of His)**
 - bundle forks into right and left bundle branches
 - these branches pass through interventricular septum toward apex
- **Purkinje fibers**
 - nervelike processes spread throughout ventricular myocardium
- signal pass from cell to cell through **gap junctions**

Cardiac Conduction System

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- 1 SA node fires.
- 2 Excitation spreads through atrial myocardium.
- 3 AV node fires.
- 4 Excitation spreads down AV bundle.
- 5 Purkinje fibers distribute excitation through ventricular myocardium.

Figure 19.12

Nerve Supply to Heart

- **sympathetic nerves (raise heart rate)**
 - sympathetic pathway to the heart originates in the **lower cervical to upper thoracic segments** of the spinal cord
 - continues to adjacent **sympathetic chain ganglia**
 - some pass through **cardiac plexus** in mediastinum
 - continue as **cardiac nerves** to the heart
 - fibers terminate in SA and AV nodes, in atrial and ventricular myocardium, as well as the aorta, pulmonary trunk, and coronary arteries
 - increase heart rate and contraction strength
 - dilates coronary arteries to increase myocardial blood flow
- **parasympathetic nerves (slows heart rate)**
 - pathway begins with **nuclei of the vagus nerves** in the **medulla oblongata**
 - extend to **cardiac plexus** and continue to the heart by way of the **cardiac nerves**
 - fibers of **right vagus nerve** lead to the **SA node**
 - fibers of **left vagus nerve** lead to the **AV node**
 - little or no vagal stimulation of the myocardium
 - parasympathetic stimulation reduces the heart rate

Cardiac Rhythm

- cycle of events in heart – special names
 - **systole** – atrial or ventricular contraction
 - **diastole** – atrial or ventricular relaxation
- **sinus rhythm** - normal heartbeat triggered by the SA node
 - set by SA node at 60 – 100 bpm
 - adult at rest is 70 to 80 bpm (vagal tone)
- **ectopic focus** - another parts of heart fires before SA node
 - caused by hypoxia, electrolyte imbalance, or caffeine, nicotine, and other drugs

Abnormal Heart Rhythms

- spontaneous firing from some part of heart not the SA node
 - **ectopic foci** - region of spontaneous firing
 - **nodal rhythm** – if SA node is damaged, heart rate is set by AV node, 40 to 50 bpm
 - **intrinsic ventricular rhythm** – if both SA and AV nodes are not functioning, rate set at 20 to 40 bpm
 - this requires pacemaker to sustain life
- **arrhythmia** – any abnormal cardiac rhythm
 - failure of conduction system to transmit signals (heart block)
 - bundle branch block
 - total heart block (damage to AV node)

Cardiac Arrhythmias

- **atrial flutter** – ectopic foci in atria
 - atrial fibrillation
 - atria beat 200 - 400 times per minute
- **premature ventricular contractions (PVCs)**
 - caused by stimulants, stress or lack of sleep
- **ventricular fibrillation**
 - serious arrhythmia caused by electrical signals reaching different regions at widely different times
 - heart can't pump blood and no coronary perfusion
 - kills quickly if not stopped
 - **defibrillation** - strong electrical shock whose intent is to depolarize the entire myocardium, stop the fibrillation, and reset SA nodes to sinus rhythm

Pacemaker Physiology

- **SA node** does not have a stable resting membrane potential
 - starts at -60 mV and drifts upward from a slow inflow of Na^+
 - gradual depolarization is called **pacemaker potential**
 - slow inflow of Na^+ without a compensating outflow of K^+
 - when reaches **threshold** of -40 mV, voltage-gated **fast Ca^{2+} and Na^+ channels** open
 - faster **depolarization** occurs peaking at 0 mV
 - **K^+ channels** then open and K^+ leaves the cell
 - causing **repolarization**
 - once K^+ channels close, pacemaker potential starts over
- each depolarization of the SA node sets off one heartbeat
 - at rest, fires every 0.8 seconds or 75 bpm
- SA node is the system's pacemaker

SA Node Potentials

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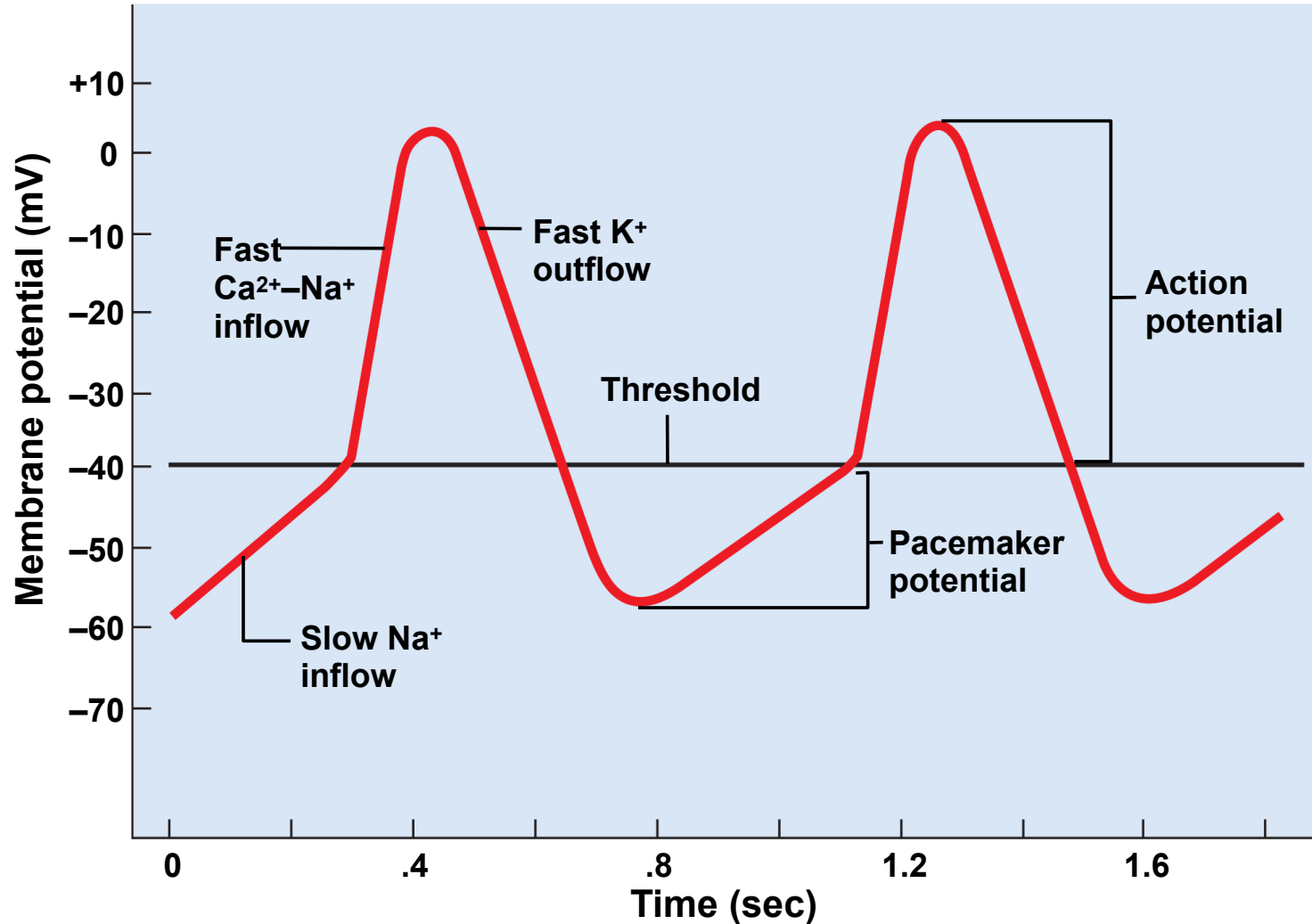


Figure 19.13

Impulse Conduction to Myocardium

- signal from **SA node** stimulates two atria to contract almost simultaneously
 - reaches AV node in 50 msec
- signal slows down through **AV node**
 - thin cardiocytes have fewer gap junctions
 - delays signal 100 msec which allows the ventricles to fill
- signals travel very quickly through **AV bundle** and **Purkinje fibers**
 - entire ventricular myocardium depolarizes and contracts in near unison
 - papillary muscles contract an instant earlier than the rest, tightening slack in chordae tendineae
- ventricular systole progresses up from the apex of the heart
 - spiral arrangement of cardiocytes twists ventricles slightly
 - like someone wringing out a towel

Electrical Behavior of Myocardium

- cardiocytes have a stable resting potential of -90 mV
- depolarize only when stimulated
 - **depolarization phase** (very brief)
 - stimulus opens voltage regulated Na^+ gates, (Na^+ rushes in) membrane **depolarizes rapidly**
 - action potential peaks at +30 mV
 - Na^+ gates close quickly
 - **plateau phase** lasts 200 to 250 msec, sustains contraction for expulsion of blood from heart
 - Ca^{2+} channels are slow to close and SR is slow to remove Ca^{2+} from the cytosol
 - **repolarization phase** - Ca^{2+} channels close, K^+ channels open, rapid diffusion of K^+ out of cell returns it to resting potential
- has a long absolute refractory period of 250 msec compared to 1 – 2 msec in skeletal muscle
 - prevents wave summation and tetanus which would stop the pumping action of the heart

Action Potential of a Cardiocyte

- 1) Na^+ gates open
- 2) Rapid depolarization
- 3) Na^+ gates close
- 4) Slow Ca^{2+} channels open
- 5) Ca^{2+} channels close, K^+ channels open (repolarization)

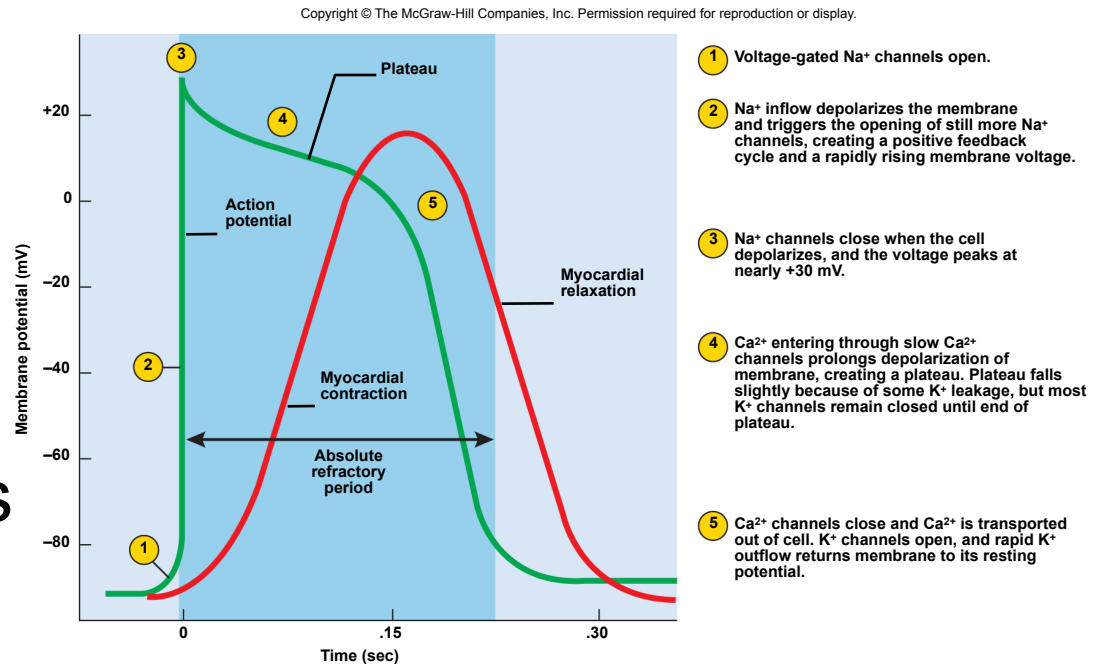


Figure 19.14

Electrocardiogram (ECG or EKG)

- composite of all action potentials of nodal and myocardial cells detected, amplified and recorded by electrodes on arms, legs and chest

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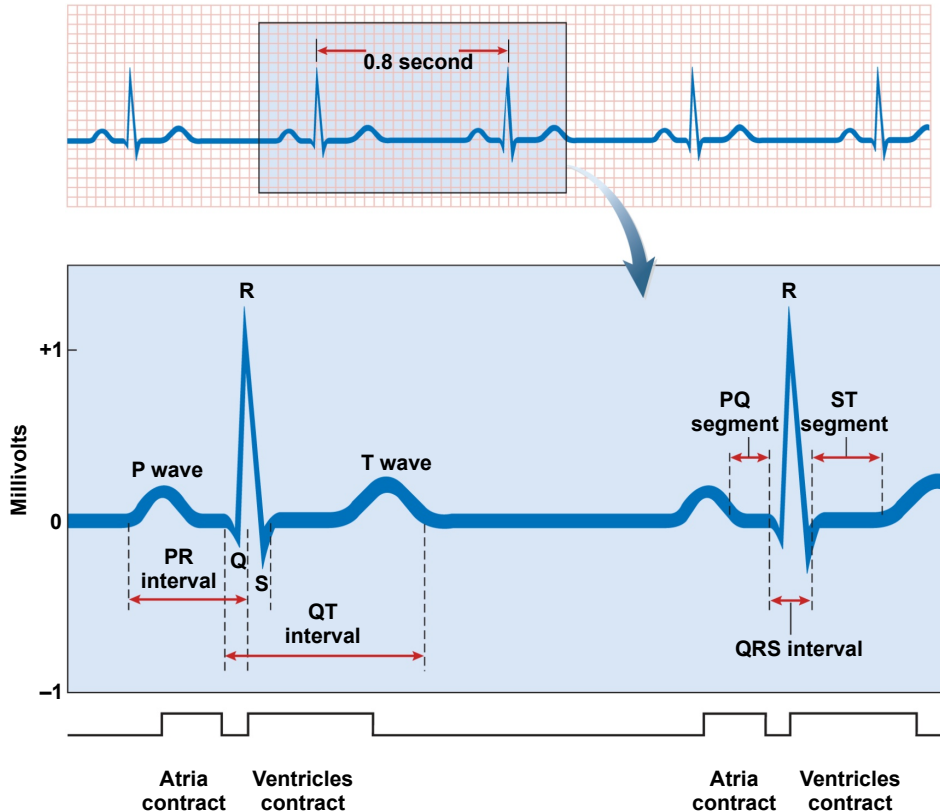


Figure 19.15

ECG Deflections

- **P wave**
 - SA node fires, **atria depolarize** and contract
 - atrial systole begins 100 msec after SA signal
- **QRS complex**
 - **ventricular depolarization**
 - complex shape of spike due to different thickness and shape of the two ventricles
- **ST segment - ventricular systole**
 - plateau in myocardial action potential
- **T wave**
 - **ventricular repolarization** and relaxation

Normal Electrocardiogram (ECG)

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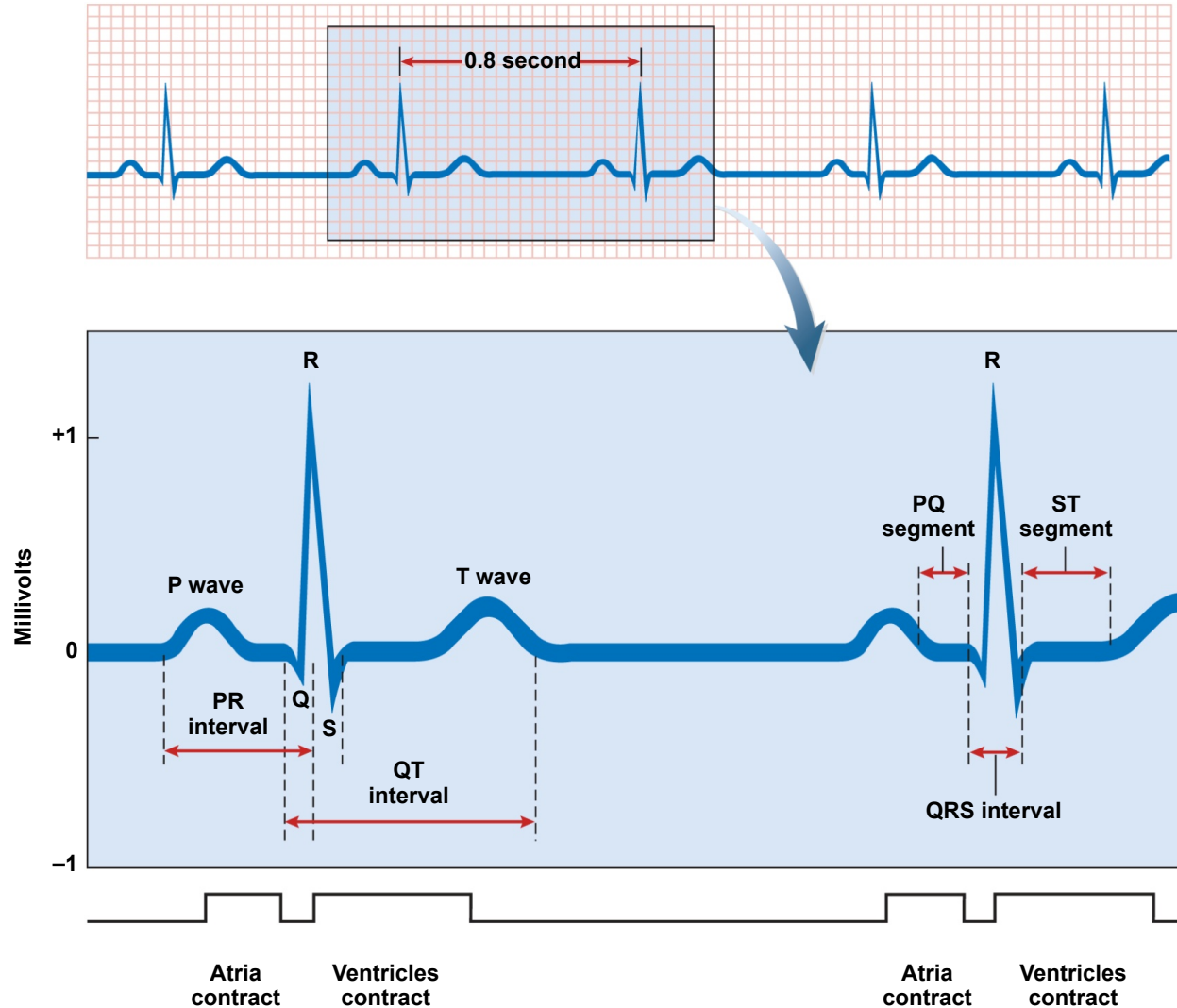


Figure 19.15

Electrical Activity of Myocardium

- 1) atrial depolarization begins
- 2) atrial depolarization complete (atria contracted)
- 3) ventricles begin to depolarize at apex; atria repolarize (atria relaxed)
- 4) ventricular depolarization complete (ventricles contracted)
- 5) ventricles begin to repolarize at apex
- 6) ventricular repolarization complete (ventricles relaxed)

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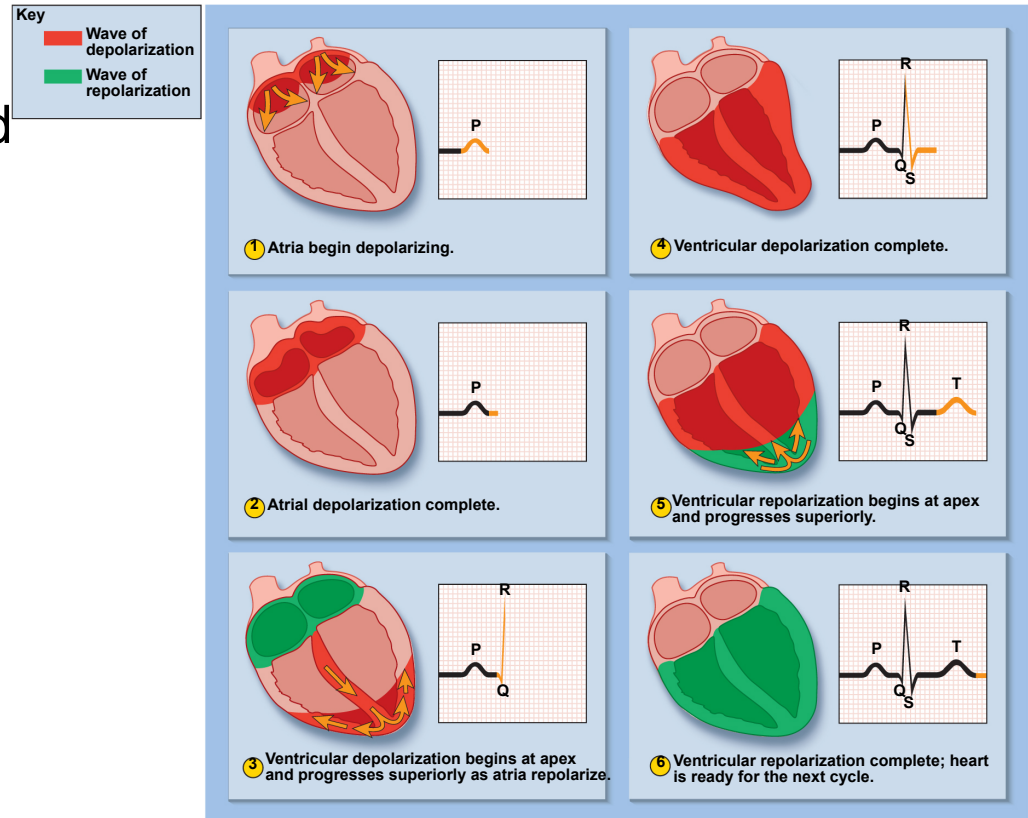


Figure 19.16

Diagnostic Value of ECG

- abnormalities in conduction pathways
- myocardial infarction
- nodal damage
- heart enlargement
- electrolyte and hormone imbalances