

Heart: Cardiac Function & ECGs

Adapted From:

Textbook Of Medical Physiology, 11th Ed.
Arthur C. Guyton, John E. Hall
Chapters 9, 10, 11, 12, & 13

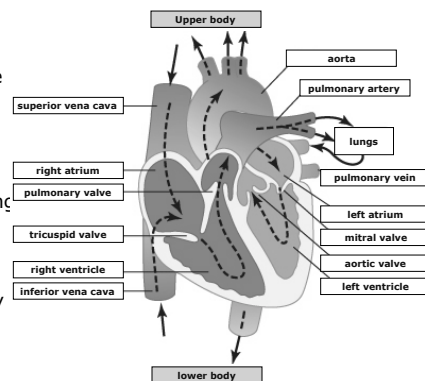
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The Heart and Circulatory System

Introduction

- The heart is two separate pumps
 - A right heart that pumps blood through the lungs
 - A left heart that pumps blood through the peripheral organs
- Each heart pump is a pulsatile two-chamber pump composed of an atrium and a ventricle
 - The atrium is a weak primer pump, helping to move blood into the ventricle
 - The ventricle then supplies the main pumping force that propels the blood
 - Through the pulmonary circulation by the right ventricle
 - Through the peripheral circulation by the left ventricle



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Physiologic Anatomy of Cardiac Muscle

Introduction

- The heart is composed of three major types of cardiac muscle
- Atrial muscle and ventricular muscle contract in a manner similar to skeletal muscle, except that the duration of contraction is much longer
- Specialized excitatory and conductive fibers contract only feebly because they contain few contractile fibrils
 - However, they exhibit automatic rhythmical electrical discharge in the form of action potentials, providing an excitatory system that controls the rhythmical beating of the heart

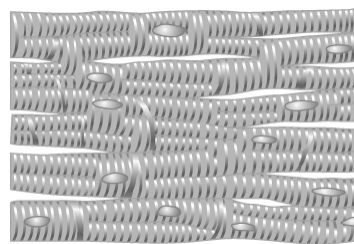


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Physiologic Anatomy of Cardiac Muscle

Cardiac Muscle as a Syncytium

- Cardiac muscle is striated in the same manner as in typical skeletal muscle
 - Cardiac muscle has typical myofibrils that contain actin and myosin filaments
- The dark areas crossing the cardiac muscle fibers are called intercalated discs
- These discs are actually cell membranes that separate individual cardiac muscle cells from one another
- Cardiac muscle fibers are made up of many individual cells connected in series and in parallel with one another



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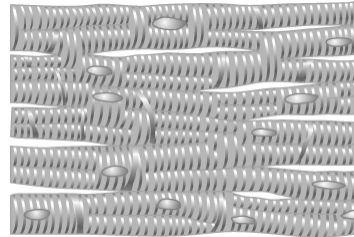


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Physiologic Anatomy of Cardiac Muscle

Cardiac Muscle as a Syncytium

- At each intercalated disc the cell membranes fuse with one another, forming gap junctions
 - Ions move with ease in the intracellular fluid along the longitudinal axes of the cardiac muscle fibers
 - Action potentials travel easily from one cardiac muscle cell to the next
- Thus, when one cardiomyocyte becomes excited, the action potential spreads to all of them, spreading from cell to cell throughout the latticework interconnections



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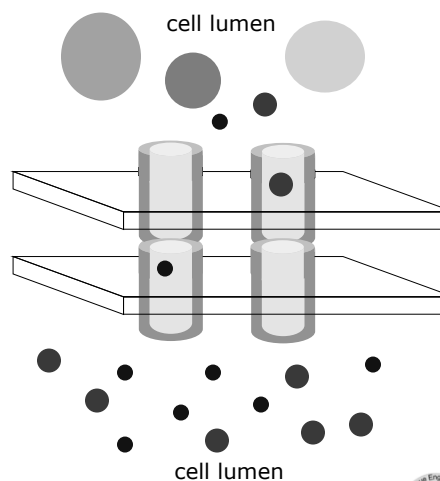
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Cell Junctions

Communicating Junctions

- Most notable type of communicating junction is the gap junction
- Gap junctions provide a mechanism for regulated exchange of molecules between adjacent cells
- Formed by the coordinated assembly of transmembrane proteins
- Gap junctions in the liver, two adjacent cells each contribute 6 proteins (connexins) to form a 12 transmembrane protein complex (connexon) that allows small molecules (< 1.2 kDa) to move from one cell to another



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Physiologic Anatomy of Cardiac Muscle

Cardiac Muscle as a Syncytium

- The heart actually is composed of two syncytiums
 - The atrial syncytium that constitutes the walls of the two atria
 - The ventricular syncytium that constitutes the walls of the two ventricles
- The atria are separated from the ventricles by fibrous tissue that surrounds the atrioventricular (A-V) valves between the atria and ventricles
 - Action potentials are not conducted from the atrial syncytium into the ventricular syncytium directly through this fibrous tissue
 - Action potentials conducted only by way of a specialized conductive system called the A-V bundle
- This division allows the atria to contract a short time ahead of ventricular contraction, allowing for effective heart pumping

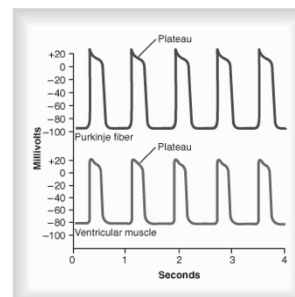


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Action Potentials Cardiac Muscle

Introduction

- The magnitude of an action potential recorded in a ventricular muscle fiber averages about 105 mV
- After the initial spike, the membrane remains depolarized for about 0.2 sec in a plateau, followed by abrupt repolarization
- The action potential plateau causes ventricular contraction to last as much as 15 times as long in cardiac muscle as in skeletal muscle



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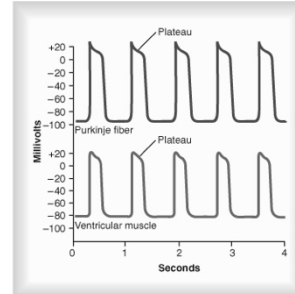


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Action Potentials Cardiac Muscle

Introduction

- The prolonged action potential in cardiac muscle is caused by opening of two types of channels
- The same fast Na^+ channels as those in skeletal muscle
- Slow Ca^{++} channels, which are also called $\text{Ca}^{++}\text{-Na}^+$ channels
 - Channels are slower to open and remain open for several tenths of a second
 - A large quantity of both Ca^{++} and Na^+ ions flows through these channels to the interior of the cardiac muscle fiber, causing the plateau in the action potential
 - Further, Ca^{++} that enter during this plateau phase activate the muscle contractile process



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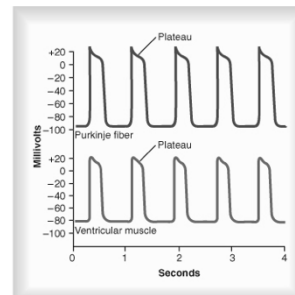


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Action Potentials Cardiac Muscle

Introduction

- Immediately after the onset of the action potential, the permeability of the cardiac muscle membrane for K^+ decreases about fivefold
- This decreased K^+ permeability greatly decreases the outflux of positively charged potassium ions during the action potential plateau and thereby prevents early return of the action potential voltage to its resting level
- When the slow $\text{Ca}^{++}\text{-Na}^+$ channels do close, K^+ permeability also increases rapidly
 - This rapid loss of K^+ immediately returns the membrane potential to its resting level, thus ending the action potential



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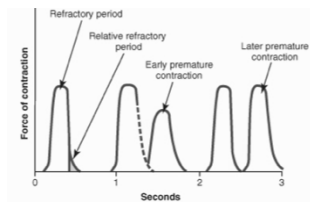


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Action Potentials Cardiac Muscle

Velocity of Signal Conduction in Cardiac Muscle

- The velocity of conduction of the excitatory action potential signal along both atrial and ventricular muscle fibers is about 0.3 to 0.5 m/sec
 - 1/250 the velocity in very large nerve fibers
 - 1/10 the velocity in skeletal muscle fibers
- The velocity of conduction in the Purkinje fibers - the specialized heart conductive system - approaches 4 m/sec



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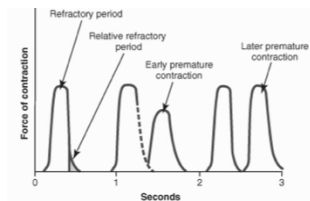


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Action Potentials Cardiac Muscle

Refractory Period of Cardiac Muscle

- Cardiac muscle is refractory to restimulation
- The normal refractory period of the ventricle is 0.25 to 0.30 sec, about the duration of the prolonged plateau action potential
 - An additional relative refractory period of about 0.05 sec occurs, during which the muscle is more difficult to excite
- The refractory period of atrial muscle is much shorter than that for the ventricles, approximately 0.15 sec



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Action Potentials Cardiac Muscle

Excitation - Contraction Coupling

- Excitation-contraction coupling: an action potential causes cardiac myofibrils to contract in a manner similar to skeletal muscle
 - AP spreads to the interior of cardiac muscle fiber along T tubules, activated SR releases Ca^{++} , Ca^{++} diffuse into the myofibrils, and Ca^{++} catalyze actin and myosin contraction
- Differences from skeletal muscle
 - Extra Ca^{++} also diffuses into the sarcoplasm from the T tubules
 - Without the extra Ca^{++} from the T tubules the strength of cardiac muscle contraction would be diminished as the SR does not store enough Ca^{++}
 - T tubules have 5x diameter and 25x volume of those in skeletal muscle tubules
 - T tubules contain mucopolysaccharides that are electronegatively charged and bind and store Ca^{++} for action potentials



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Action Potentials Cardiac Muscle

Excitation - Contraction Coupling

- The strength of contraction depends upon Ca^{++} concentration in the extracellular fluids
 - Openings of the T tubules pass directly through the cardiac muscle cell membrane into the extracellular spaces surrounding the cells
 - Alternatively, the strength of skeletal muscle contraction is hardly affected by changes in extracellular Ca^{++} because skeletal muscle contraction is caused almost entirely by Ca^{++} release from the SR
- Duration of contraction
 - Cardiac muscle begins to contract a few milliseconds after the action potential begins and continues to contract until a few milliseconds after the action potential ends
 - The duration of contraction of cardiac muscle is mainly a function of the duration of the action potential



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The Cardiac Cycle

Introduction

- The cardiac cycle describes the events that occur from the beginning of one heartbeat to the beginning of the next
- Each cycle is initiated by spontaneous generation of an action potential in the sinus node
- This node is located in the superior lateral wall of the right atrium near the opening of the superior vena cava, and the action potential travels from here rapidly through both atria and then through the A-V bundle into the ventricles
- Thus there is a delay of more than 0.1 second during passage of the cardiac impulse from the atria into the ventricles - allowing the atria to contract ahead of ventricular contraction, thereby pumping blood into the ventricles before the strong ventricular contraction begins

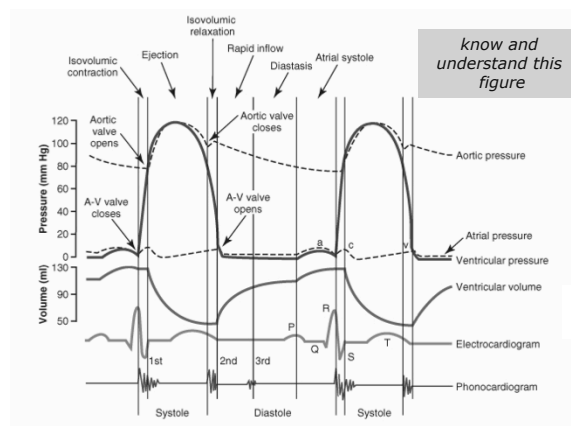


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The Cardiac Cycle

Diastole and Systole

- The cardiac cycle consists of a period of relaxation called diastole, during which the heart fills with blood, followed by a period of contraction called systole



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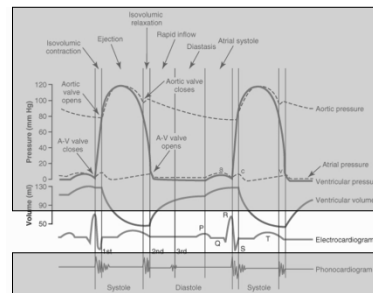


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The Cardiac Cycle

Electrocardiogram

- The electrocardiogram depicts electrical voltages generated by the heart and recorded by the electrocardiograph from the surface of the body
- P wave is caused by atrial depolarization and is followed by atrial contraction, which causes a slight rise in the atrial pressure
- About 0.16 second after the onset of the P wave, the QRS waves appear as a result of ventricular depolarization, which initiates contraction of the ventricles and causes the ventricular pressure to begin rising
- T wave represents ventricular repolarization and relaxation of the ventricular muscle fibers begin to relax



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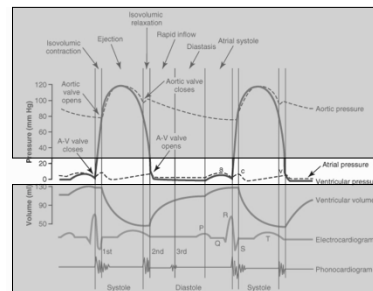


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The Cardiac Cycle

Function of the Atria as Primer Pumps

- Blood normally flows continually from the great veins into the atria
 - About 80% of blood flows directly through the atria into the ventricles even before the atria contract
 - Atrial contraction usually causes an additional 20% filling of the ventricles
- The heart can continue to operate under most conditions even without atrial filling because it normally has the capability of pumping 300 to 400% more blood than is required by the resting body
- Atrial dysfunction is unlikely to be noticed unless a person exercises



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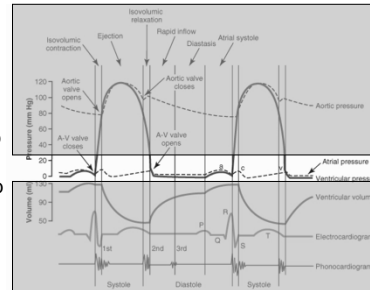


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The Cardiac Cycle

Atrial Pressure Waves

- Three minor pressure elevations, called the a, c, and v atrial pressure waves, are noted
- The a wave is caused by atrial contraction
 - Right atrial pressure increases 4 to 6 mm Hg during atrial contraction
 - Left atrial pressure increases about 7 to 8 mm Hg
- The c wave occurs when the ventricles begin to contract
 - Results from a slight backflow of blood into the atria at the onset of ventricular contraction, but mainly by bulging of the A-V valves into the atria
- The v wave occurs toward the end of ventricular contraction
 - Results from slow flow of blood into the atria from the veins while the A-V valves are closed during ventricular contraction



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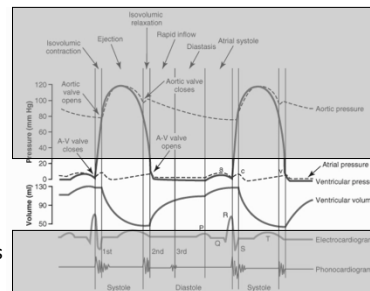


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The Cardiac Cycle

Filling the Ventricles

- During ventricular systole, large amounts of blood accumulate in the right and left atria because of the closed A-V valves
- After systole, the moderately increased atrial pressures immediately push the A-V valves open and allow blood to flow rapidly into the ventricles
 - The period of rapid filling lasts for about the first third of diastole
- During the middle third of diastole, a small amount of blood normally flows into the ventricles
 - This is blood passes directly from the veins into the ventricles
- During the last third of diastole, the atria contract and to give additional blood flow
 - This accounts for about 20% of the filling of the ventricles during each heart cycle



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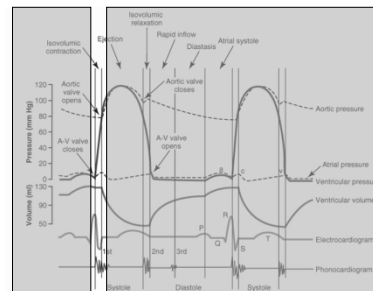


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The Cardiac Cycle

Period of Isovolumic (Isometric) Contraction

- Immediately after ventricular contraction begins, the ventricular pressure rises abruptly, causing the A-V valves to close
- After 0.02 to 0.03 sec of building pressure, the semilunar (aortic and pulmonary) valves open against the pressures in the aorta and pulmonary artery
- Therefore, during this period, contraction is occurring in the ventricles, but there is no emptying - isovolumic or isometric contraction



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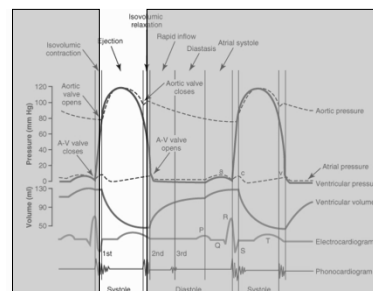


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The Cardiac Cycle

Period of Ejection

- When the left ventricular pressure rises slightly above 80 mmHg and the right ventricular pressure slightly above 8 mmHg, ventricular pressures push the semilunar valves open
- Immediately, blood begins to pour out of the ventricles
 - Rapid ejection - 70% of the blood emptying occurs during the first 1/3 of ejection
 - Slow ejection - the remaining 30% of the blood emptying occurs during the latter 2/3 of ejection



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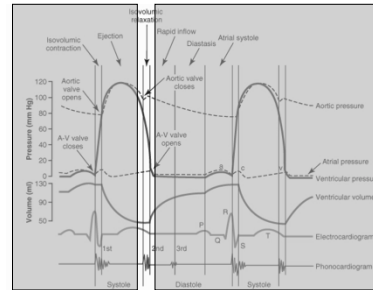


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The Cardiac Cycle

Period of Isovolumic (Isometric) Relaxation

- At the end of systole, ventricular relaxation begins, allowing intraventricular pressures to decrease rapidly
- The pressure in the just filled arteries pushes blood back toward the ventricles, which snaps the aortic and pulmonary valves closed
- For another 0.03 to 0.06 sec, the ventricular muscle continues to relax, even though the ventricular volume does not change
- Intraventricular pressures decrease rapidly back to their low diastolic levels
- Then the A-V valves open to begin a new cycle of ventricular pumping



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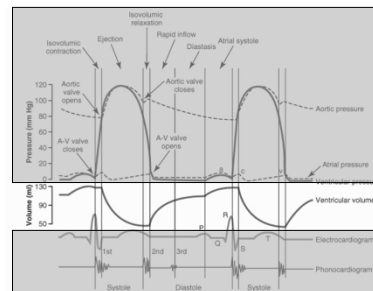


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The Cardiac Cycle

Cardiac Volume Changes

- During diastole, normal filling of the ventricles increases the volume of each ventricle to about 110 to 120 ml - end-diastolic volume
- As the ventricles empty during systole, the ejected volume is about 70 ml - stroke volume output
- There is 40 to 50 ml remaining in each ventricle - end-systolic volume
- The fraction of the end-diastolic volume that is ejected is about 60% - ejection fraction
- When the heart contracts strongly, an increasing end-diastolic volume and decreasing end-systolic volume can double the stroke volume output



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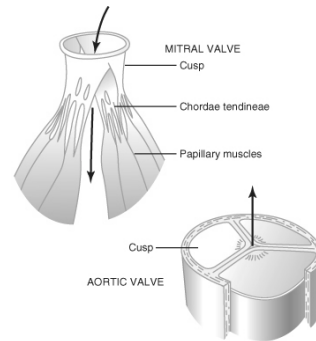


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The Cardiac Cycle

Function of the Valves

- The atrioventricular valves (the tricuspid and mitral valves) prevent backflow of blood from the ventricles to the atria during systole
- The semilunar valves (the aortic and pulmonary artery valves) prevent backflow from the aorta and pulmonary arteries into the ventricles during diastole
- These valves close and open passively
 - Closed when a backward pressure gradient pushes blood backward
 - Opened when a forward pressure gradient forces blood in the forward direction
- The thin, filmy A-V valves require almost no backflow to cause closure
- Heavier semilunar valves require rather rapid backflow for a few milliseconds



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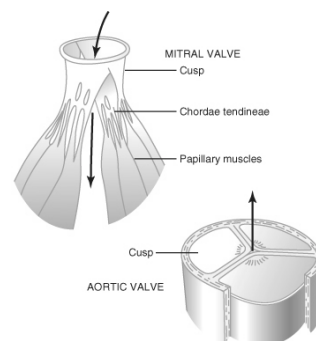


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The Cardiac Cycle

Function of the Papillary Muscles

- Papillary muscles attach to the vanes of the A-V valves by the chordae tendineae
- The papillary muscles contract when the ventricular walls contract, but do not help the valves to close
- Instead, they pull the vanes of the valves inward toward the ventricles to prevent their bulging too far backward toward the atria during ventricular contraction
 - If the chordae tendineae are ruptured or paralyzed, the valve bulges far backward during ventricular contraction, sometimes so far that it leaks severely and results in severe or even lethal cardiac incapacity



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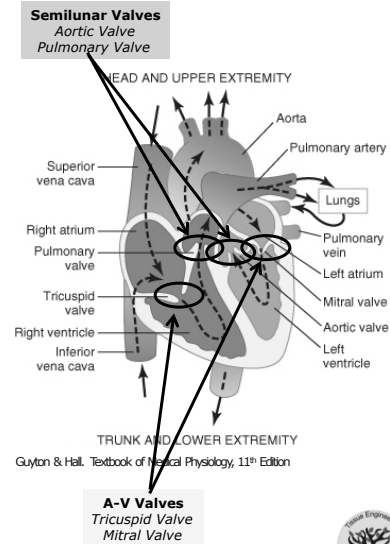


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The Cardiac Cycle

Semilunar Valves vs. A-V Valves

- The high pressures in the arteries at the end of systole cause the semilunar (aortic and pulmonary) valves to snap to the closed position
 - A-V (tricuspid and mitral) valves close more softly
- Blood ejection velocity is great through the semilunar valves
 - Velocity is much lower through the A-V valves
- Due to rapid closure and ejection, the edges of the semilunar valves are subjected to significant mechanical abrasion
- Semilunar valves are not supported
 - A-V valves are supported by the chordae tendineae



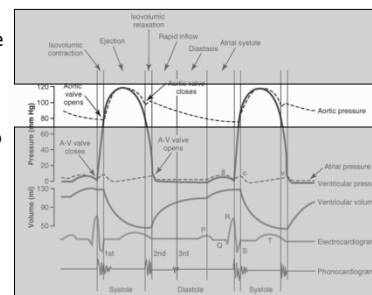
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The Cardiac Cycle

Aortic Pressure Curve

- When the left ventricle contracts, the ventricular pressure increases rapidly until the aortic valve opens, and blood immediately flows out of the ventricle into the aorta and the systemic distribution
- Entry of blood into the arteries causes the arterial walls to stretch and arterial pressure to increase to about 120 mm Hg
 - At the end of systole, the elastic walls of the arteries maintain a high pressure in the arteries, even during diastole
- A so-called incisura occurs in the aortic pressure curve when the aortic valve closes
 - A short period of backward flow of blood immediately before closure of the valve, followed by sudden cessation of the backflow



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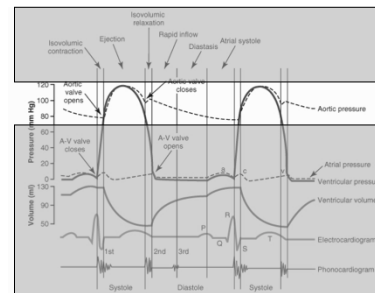
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The Cardiac Cycle

Aortic Pressure Curve

- After the aortic valve has closed, the pressure in the aorta decreases slowly throughout diastole because the blood stored in the distended elastic arteries flows continually through the peripheral vessels back to the veins
- Before the ventricle contracts again, the aortic pressure usually has fallen to about 80 mm Hg (diastolic pressure), which is two thirds the maximal pressure of 120 mm Hg (systolic pressure) that occurs in the aorta during ventricular contraction
- The pressure curves in the right ventricle and pulmonary artery are similar to those in the aorta, except that the pressures are only about one sixth as great



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The Cardiac Cycle

Relationship of the Heart Sounds to Heart Pumping

- When the ventricles contract, one first hears a sound caused by closure of the A-V valves with a stethoscope
 - The vibration is low in pitch and relatively long-lasting and is known as the first heart sound
- When the aortic and pulmonary valves close at the end of systole, one hears a rapid snap because these valves close rapidly, and the surroundings vibrate for a short period
 - This sound is called the second heart sound



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Work Output of the Heart

Introduction

- The stroke work output of the heart is the amount of energy that the heart converts to work during each heartbeat while pumping blood into the arteries
- Minute work output is the total amount of energy converted to work in 1 minute
 - Minute work output equals stroke work output times the heart rate per minute
- The major proportion of work is used to move the blood from the low-pressure veins to the high-pressure arteries
 - This is called volume-pressure work or external work
- A minor proportion of the energy is used to accelerate the blood to its velocity of ejection through the aortic and pulmonary valves
 - This is the kinetic energy of blood flow component of the work output

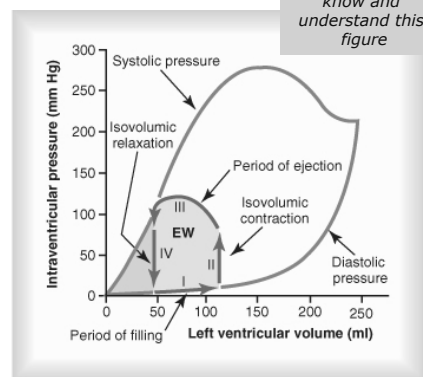


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Work Output of the Heart

Graphical Analysis of Ventricular Pumping

- Consider the pumping mechanics of the left ventricle
- The diastolic pressure curve is determined by filling the heart with progressively greater volumes of blood and then measuring the diastolic pressure immediately before ventricular contraction occurs, which is the end-diastolic pressure of the ventricle
- The systolic pressure curve is determined by recording the maximum systolic pressure achieved during ventricular contraction at each volume of filling, and without any outflow of blood from the heart



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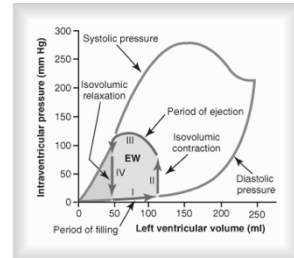


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Work Output of the Heart

Graphical Analysis of Ventricular Pumping

- Phase I: Period of filling
- Initially, ventricular volume of about 45 ml and a diastolic pressure near 0 mm Hg
 - 45 ml is the amount of blood that remains in the ventricle after the previous heartbeat and is called the end-systolic volume
- As venous blood flows into the ventricle from the left atrium, the ventricular volume normally increases to about 115 ml, called the end-diastolic volume
- Diastolic pressure rises to about 5 mm Hg



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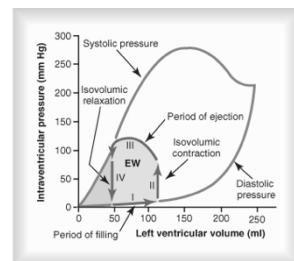


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Work Output of the Heart

Graphical Analysis of Ventricular Pumping

- Phase II: Period of isovolumic contraction
- The volume of the ventricle does not change because all valves are closed
- The pressure inside the ventricle increases to equal the pressure in the aorta, at a pressure value of about 80 mm Hg



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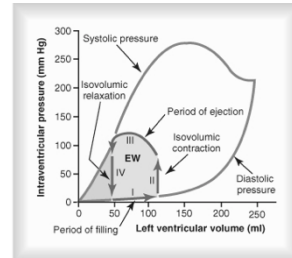


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Work Output of the Heart

Graphical Analysis of Ventricular Pumping

- Phase III: Period of ejection
- The systolic pressure rises even higher because of still more contraction of the ventricle
- At the same time, the volume of the ventricle decreases because the aortic valve has now opened and blood flows out of the ventricle into the aorta



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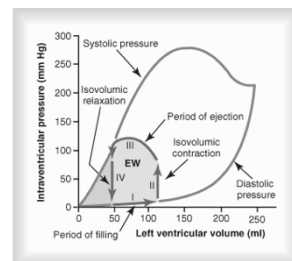


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Work Output of the Heart

Graphical Analysis of Ventricular Pumping

- Phase IV: Period of isovolumic relaxation
- At the end of the period of ejection, the aortic valve closes, and the ventricular pressure falls back to the diastolic pressure level
- The ventricle returns to its starting point, with about 45 ml of blood left in the ventricle and at an atrial pressure near 0 mm Hg



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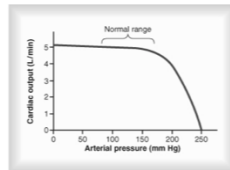


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Regulation of Heart Pumping

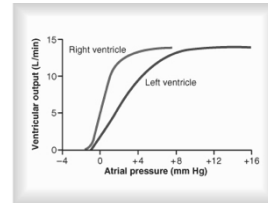
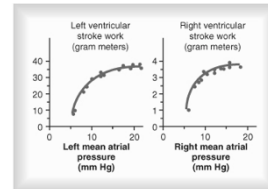
Frank-Starling Mechanism

- Fundamental Concept: The amount of blood pumped by the heart each minute (venous return) is determined by the rate of blood flow into the heart
 - Peripheral tissue controls blood flow
- The greater the heart is stretched during filling, the greater it contracts and the greater quantity of blood is pumped into the aorta
- The intrinsic ability of the heart to adapt to increasing volumes of inflowing blood is called the Frank-Starling Mechanism



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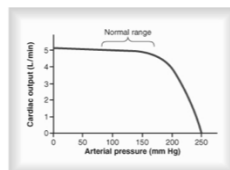
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Regulation of Heart Pumping

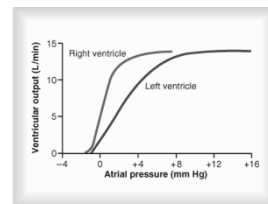
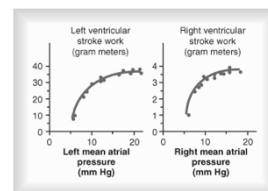
Frank-Starling Mechanism

- Two explanations of Frank-Starling
 - Increased stretch of the heart muscle optimizes the overlap of actin and myosin filaments, which then increases the force of contraction
 - Increased stretch of the heart muscle also increases heart rate
- Frank Starling is demonstrated by ventricular function curves, where increases in arterial pressure cause an increase in ventricular output - up to a critical point



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Rhythmical Excitation of the Heart

Introduction

- In addition to contraction, the heart possesses other functions
 - Generation of rhythmical electrical impulses to cause rhythmical contraction of the heart muscle
 - Conduction of these impulses rapidly through cardiac muscle
- Major results of this electrical signaling system include
 - Atria contraction ahead of ventricular contraction
 - Near spatial homogeneity of ventricular contraction
- This rhythmical and conductive system of the heart is susceptible to damage by ischemia

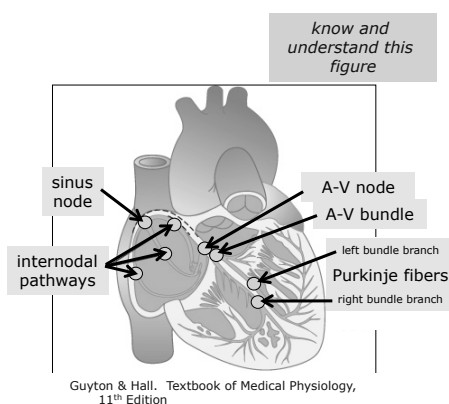


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Specialized Excitatory and Conductive System

Introduction

- The sinus node (also called sinoatrial or S-A node) generates the normal rhythmical impulse
- The internodal pathways that conduct the impulse from the sinus node to the atrioventricular (A-V) node
- The A-V bundle conducts impulses from the atria into the ventricles
- The left and right bundle branches of Purkinje fibers conduct the cardiac impulse to all parts of the ventricles

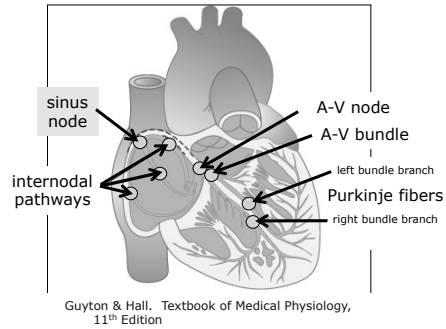


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Specialized Excitatory and Conductive System

Sinus Node

- A small, flattened, ellipsoid strip of specialized cardiac muscle
 - About 3 mm wide, 15 mm long, and 1 mm thick
 - Located in the superior posterolateral wall of the right atrium immediately below and slightly lateral to the opening of the superior vena cava
- Fibers have almost no contractile muscle filaments
- Fibers connect directly with the atrial muscle fibers, so that any action potential that begins in the sinus node spreads immediately into the atrial muscle wall

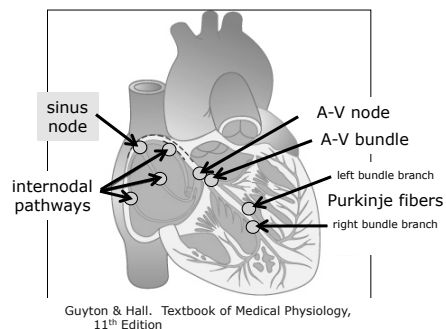


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Automatic Electrical Rhythmicity of the Sinus Fibers

- Some cardiac fibers have the capability of self-excitation, causing automatic rhythmical discharge and contraction
 - Particularly the fibers of the sinus node
- Thus, the sinus node ordinarily controls the rate of beat of the entire heart



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Mechanism of Sinus Nodal Rhythmicity

- The resting membrane potential of the sinus nodal fiber between discharges is relatively high at -55 to -60 mV
 - The cause of this lesser negativity is that the cell membranes of the sinus fibers are naturally leaky to Na^+ and Ca^{++}
- Remember cardiac muscle has three critical ion channels
 - Fast Na^+ channels
 - Slow $\text{Na}^+-\text{Ca}^{++}$ channels
 - K^+ channels
- In the sinus nodal fiber
 - The fast Na^+ channels are mostly inactivated
 - Only the slow $\text{Na}^+-\text{Ca}^{++}$ channels can open and initiate an action potential
 - As a result, the atrial nodal action potential is slower to develop than the action potential of the ventricular muscle
 - Also, the return of the potential to its negative state occurs slowly

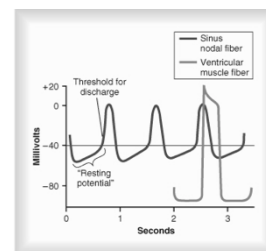


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Self-Excitation of Sinus Nodal Fibers

- Due to high extracellular Na^+ and some open Na^+ channels, Na^+ tends to leak to the inside the cardiac muscle fiber
- Between heartbeats, Na^+ influx causes a slow rise in the resting membrane potential in the positive direction
- When the potential reaches a threshold voltage of about -40 mV, $\text{Na}^+-\text{Ca}^{++}$ channels are activated, causing an action potential
- Repolarization then results
 - $\text{Na}^+-\text{Ca}^{++}$ channel inactivation within about 100 to 150 msec after opening
 - Nearly simultaneously, K^+ channels open allowing K^+ to diffuse out of the fiber
- Finally, K^+ channels begin to close allowing Na^+ leak to take over



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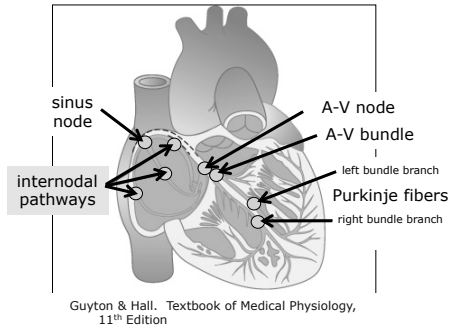


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Internodal Pathways

- The ends of the sinus nodal fibers connect directly with surrounding atrial muscle fibers
- Action potentials originating in the sinus node travel outward into these atrial muscle fibers, allowing an action potential to spread through the entire atrial muscle mass and to the A-V node
 - Velocity of conduction is about 0.3 m/sec in most atrial muscle, but about 1 m/sec in small bands of atrial fibers
- The anterior interatrial band, passes through the anterior walls of the atria to the left atrium

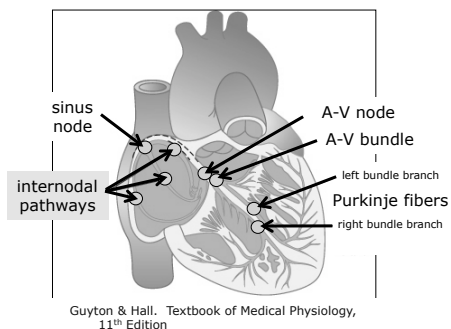


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Internodal Pathways

- The anterior, middle, and posterior internodal pathways are formed from three bands that curve through the anterior, lateral, and posterior atrial walls and terminate in the A-V node
- The cause of more rapid velocity of conduction in these bands is the presence of specialized conduction fibers

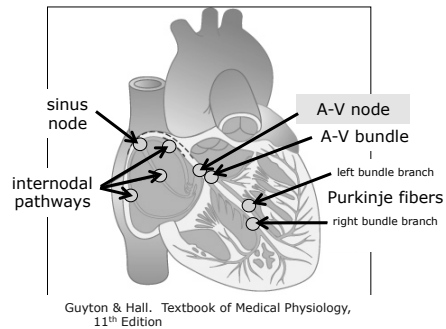


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Atrioventricular Node

- The atrial conductive system is organized so that the cardiac impulse does not travel from the atria into the ventricles too rapidly
- Delay allows time for the atria to empty their blood into the ventricles before ventricular contraction begins
- The A-V node and its adjacent conductive fibers delay this transmission into the ventricles

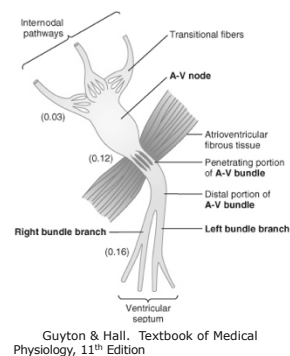


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Organization of the A-V Node

- The A-V node is located in the posterior wall of the right atrium immediately behind the tricuspid valve
- After traveling through the internodal pathways, an impulse reaches the A-V node about 0.03 sec after its origin in the sinus node
- There is a delay of another 0.09 sec before the impulse enters the penetrating portion of the A-V bundle, where it passes into the ventricles
- A final delay of another 0.04 sec occurs mainly in this penetrating A-V bundle, which is composed of multiple small fascicles passing through the fibrous tissue separating the atria from the ventricles
 - The total delay in the A-V nodal and A-V bundle system is about 0.13 sec
- A total delay of 0.16 sec occurs before the excitatory signal finally reaches the contracting muscle of the ventricles

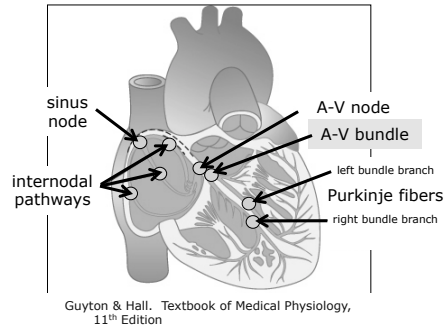


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Cause of the Slow Conduction

- The slow conduction in the transitional, nodal, and penetrating A-V bundle fibers is caused mainly by diminished numbers of gap junctions between successive cells in the conducting pathways
- Less gap junctions implies a greater resistance to conduction of excitatory ions from one conducting fiber to the next



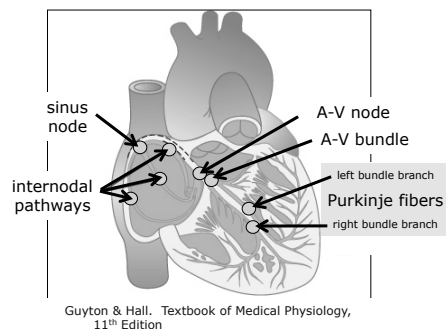
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Rapid Transmission in the Purkinje System

- Special Purkinje fibers lead from the A-V node through the A-V bundle into the ventricles
- They mostly have functional characteristics that are quite the opposite of those of the A-V nodal fibers
- They are very large fibers and they transmit action potentials at a velocity of 1.5 to 4.0 m/sec allowing almost instantaneous transmission of the cardiac impulse throughout the ventricular muscle
 - About 6x that in ventricular muscle
 - About 150x that in A-V nodal fibers



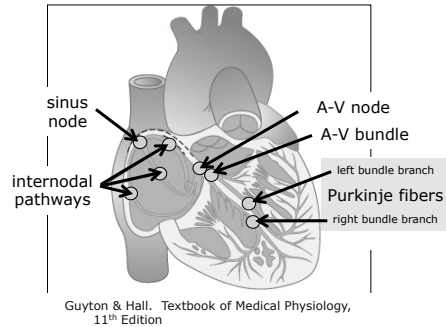
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Rapid Transmission in the Purkinje System

- The rapid transmission by Purkinje fibers is believed to be caused by a very high level of permeability of the gap junctions
 - The Purkinje fibers also have very few myofibrils, which means that they contract little or not at all during the course of impulse transmission

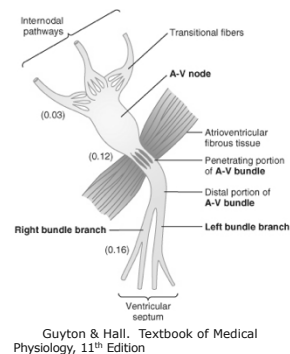


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Conduction Through the A-V Bundle

- A special characteristic of the A-V bundle is the inability of action potentials to travel backward from the ventricles to the atria
- This prevents re-entry of cardiac impulses by this route from the ventricles to the atria, allowing only forward conduction from the atria to the ventricles
- Everywhere except at the A-V bundle the atrial muscle is separated from the ventricular muscle by a continuous fibrous barrier
- The barrier acts as an insulator to prevent passage of the cardiac impulse between atrial and ventricular muscle through any other route besides forward conduction through the A-V bundle itself



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Distribution of the Purkinje Fibers in the Ventricles

- After penetrating the fibrous tissue between the atrial and ventricular muscle, the distal portion of the A-V bundle passes downward in the ventricular septum for 5 to 15 mm toward the apex of the heart
- Then the bundle divides into left and right bundle branches that lie beneath the endocardium on the two respective sides of the ventricular septum
 - Each branch spreads downward toward the apex of the ventricle, progressively dividing into smaller branches
 - These branches progress sidewise around each ventricular chamber and back toward the base of the heart
 - The ends of the Purkinje fibers penetrate about 1/3 the way into the muscle mass and finally become continuous with cardiac muscle fibers
- Once the cardiac impulse enters the ventricular Purkinje conductive system, it spreads almost immediately to the entire ventricular muscle mass



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Transmission of the Cardiac Impulse in the Ventricular Muscle

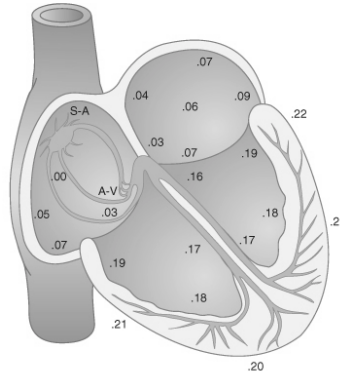
- Once the impulse reaches the ends of the Purkinje fibers, it is transmitted through the ventricular muscle mass by the ventricular muscle fibers themselves
 - The velocity of transmission is now only 0.3 to 0.5 m/sec, 1/6 that in the Purkinje fibers
- The cardiac muscle wraps around the heart in a double spiral, with fibrous septa between the spiraling layers
 - The cardiac impulse angulates toward the surface along the directions of the spirals
- Transmission from the endocardial surface to the epicardial surface of the ventricle requires as much as another 0.03 second



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Specialized Excitatory and Conductive System

Summary of Cardiac Impulse Transmission



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Control of Excitation and Conduction in the Heart

Introduction

- Cardiac impulses normally arise in the sinus node
 - The normal rate of rhythmic discharge in the sinus node is 70 to 80 /min
- However, other portions of the heart can exhibit intrinsic rhythmic excitation
 - Unstimulated A-V nodal fibers discharge at an intrinsic rhythmic rate of 40 to 60 /min
 - Unstimulated Purkinje fibers discharge at a rate somewhere between 15 and 40 /min
- The sinus node is virtually always the pacemaker of the normal heart
 - The discharge rate of the sinus node is considerably faster than the natural self-excitatory discharge rate of either the A-V node or the Purkinje fibers
 - Each time the sinus node discharges, its impulse is conducted into both the A-V node and the Purkinje fibers, also discharging their excitable membranes
 - The sinus node discharges again before either the A-V node or the Purkinje fibers can reach their own thresholds for self-excitation



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Control of Excitation and Conduction in the Heart

Abnormal Pacemakers

- A pacemaker elsewhere than the sinus node is called an "ectopic" pacemaker
- Some parts of the heart may develop a rhythmical discharge rate that is more rapid than that of the sinus node
 - The pacemaker of the heart then shifts away from the sinus node, typically to the A-V node or the Purkinje fibers
 - Under rarer conditions, a place in the atrial or ventricular muscle develops excessive excitability and becomes the pacemaker
- A blockage of impulse transmission from the sinus node can occur
 - The atria continue to beat normally under sinus node control
 - A new pacemaker also usually develops in the Purkinje system of the ventricles and drives the ventricular contraction
 - Stokes-Adams syndrome: After sudden A-V bundle block, the Purkinje system does not take over for 5 to 20 sec during which the ventricles fail to pump blood, and the person faints
 - If the delay period is too long, it can lead to death



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Control of Excitation and Conduction in the Heart

Role of the Purkinje System

- The Purkinje system allows a cardiac impulse to arrive at almost all portions of the ventricles within a narrow span of time
 - The first ventricular muscle fiber is excited only 0.03 to 0.06 sec ahead of the last ventricular muscle fiber
- This causes all portions of the ventricular muscle in both ventricles to begin contracting at almost the same time and then to continue contracting for about another 0.3 sec
- Effective pumping by the two ventricular chambers requires this synchronous type of contraction
 - If the cardiac impulse should travel through the ventricles slowly, much of the ventricular mass would contract before contraction of the remainder, in which case the overall pumping effect would be greatly depressed

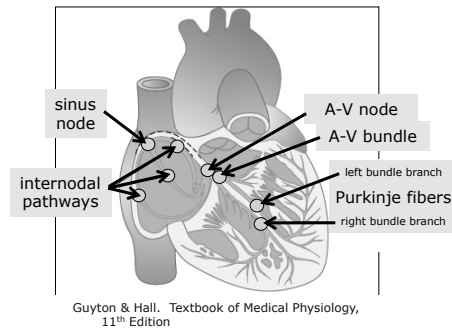


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Control of Excitation and Conduction in the Heart

Control of Heart Rhythmicity and Impulse Conduction

- The heart is supplied with both sympathetic and parasympathetic nerves
- The parasympathetic nerves (vagi) are distributed mainly to the S-A and A-V nodes
 - Less so to the muscle of the two atria
 - Very little directly to the ventricular muscle
- The sympathetic nerves are distributed to all parts of the heart



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Control of Excitation and Conduction in the Heart

Parasympathetic (Vagal) Stimulation

- Stimulation of the parasympathetic nerves to the heart causes acetylcholine release at the vagal endings
 - Acetylcholine decreases the rate of rhythm of the sinus node
 - Acetylcholine decreases the excitability of the A-V junctional fibers between the atrial musculature and the A-V node, thereby slowing transmission of the cardiac impulse into the ventricles
- Weak to moderate vagal stimulation slows the rate of heart pumping
- Strong stimulation of the vagi can stop completely the rhythmical excitation by the sinus node or block completely transmission of the cardiac impulse from the atria into the ventricles through the A-V node
 - Ventricular escape: Under strong vagi stimulation, rhythmical excitatory signals are no longer transmitted into the ventricles, ventricles stop beating for 5 to 20 sec, then Purkinje fibers take control, and causes ventricular contraction at a rate of 15 to 40/min



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Control of Excitation and Conduction in the Heart

Mechanism of the Vagal Effects

- Acetylcholine release increases the permeability of the fiber membranes to K^+ , allows K^+ leakage, increasing negativity inside the fibers (hyperpolarization), and reducing tissue excitability
- Hyperpolarization decreases the resting membrane potential of the sinus nodal fibers and thus the initial rise of the sinus nodal membrane potential caused by inward Na^+ and Ca^{++} leakage requires much longer to reach the threshold potential for excitation
 - This greatly slows the rate of rhythmicity, possibly stopping the rhythmical self-excitation of this node
- In the A-V node, hyperpolarization makes it difficult for the small atrial fibers entering the node to generate enough electricity to excite the nodal fibers
 - Therefore, the safety factor for transmission of the cardiac impulse through the transitional fibers into the A-V nodal fibers decreases
 - A moderate decrease simply delays conduction of the impulse, but a large decrease blocks conduction entirely



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Control of Excitation and Conduction in the Heart

Effect of Sympathetic Stimulation

- Sympathetic stimulation causes essentially the opposite effects on the heart to those caused by vagal stimulation
 - First, sympathetic stimulation increases the rate of sinus nodal discharge
 - Second, sympathetic stimulation increases the rate of conduction as well as the level of excitability in all portions of the heart
 - Third, sympathetic stimulation increases greatly the force of contraction of all the cardiac musculature, both atrial and ventricular
- In short, sympathetic stimulation increases the overall activity of the heart



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Control of Excitation and Conduction in the Heart

Mechanism of the Sympathetic Effect

- Stimulation of the sympathetic nerves releases the hormone norepinephrine at the sympathetic nerve endings
- It is hypothesized that norepinephrine increases the permeability of the fiber membrane to Na^+ and Ca^{++} , creating a more positive resting potential, increasing the rate of upward drift of the diastolic membrane potential, accelerating self-excitation, and increasing the heart rate
- In the A-V node and A-V bundles, increased Na^+ - Ca^{++} permeability makes it easier for the action potential to excite each succeeding portion of the conducting fiber bundles, thereby decreasing the conduction time from the atria to the ventricles
- The increase in Ca^{++} permeability is at least partially responsible for the increase in contractile strength of the cardiac muscle, due to Ca^{++} role in exciting myofibril contraction



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The Normal Electrocardiogram

Introduction

- As cardiac impulses pass through the heart, a small proportion spread to the surface of the body
- If electrodes are placed on the skin on opposite sides of the heart, electrical potentials generated by the heart can be recorded
- The time recording of the electropotentials is known as an electrocardiogram

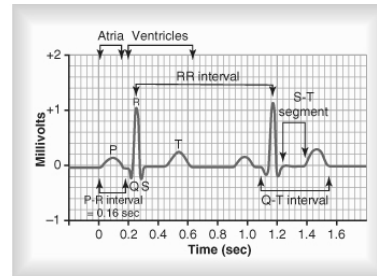


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The Normal Electrocardiogram

Characteristics of the ECG

- The normal ECG has a P wave, QRS complex, and a T wave
 - The QRS complex is normally composed of a Q wave, R wave, and S wave
- P wave is caused by electrical potentials generated by atrial depolarization
- QRS complex is caused by potentials generated by ventricular depolarization
- T wave is caused by potentials generated by ventricular repolarization



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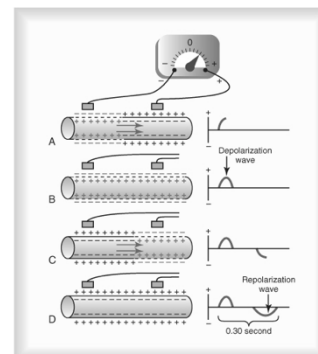


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The Normal Electrocardiogram

Depolarization and Repolarization Waves

- During depolarization, a positive electrical potential is first generated due to the differences in charge between the measuring electrodes
- Then the electrical potential difference returns to zero as the muscle fiber is equally negative across the two measuring electrodes
- A similar, though opposite, series of events give rise to the repolarization wave



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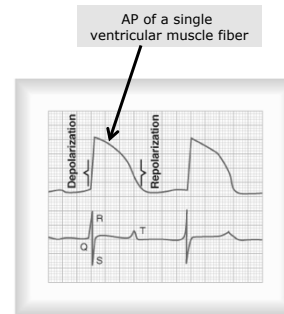


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The Normal Electrocardiogram

Relationship Between the Ventricular Action Potential and QRS / T Waves

- A ventricular action potential shows a very quick depolarization, followed by an increasingly quick repolarization
- The QRS wave reflects the very quick depolarization, and the T waves reflects repolarization
- No potential is recorded when the ventricle is either completely polarized or depolarized
- Recordings are only observed during a change in polarization



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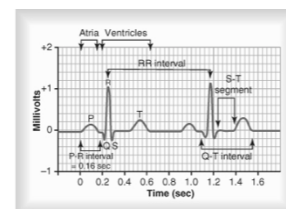


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The Normal Electrocardiogram

Contraction and the ECG

- Muscle contraction occurs after depolarization
- Thus, the P wave occurs at the beginning of atrial contraction, the QRS waves occurs at the beginning of ventricular contraction, and the T wave occurs just before the end of ventricular contraction
- Atrial repolarization (atrial T wave) occurs 0.15 to 0.20 sec after the P wave and is usually obscured by the QRS wave
- Ventricular repolarization (T wave) occurs 0.20 through 0.35 sec after the QRS wave and therefore is usually a broad wave
 - Note T wave voltage is less the QRS wave voltage



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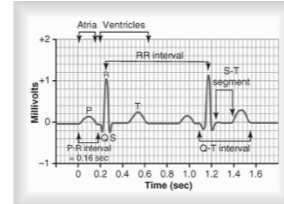


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The Normal Electrocardiogram

Voltage and the ECG

- Voltage measurement depends upon the placement of the measuring electrodes
 - Position 1: Direct cardiac muscle
 - QRS has a voltage of 110 mV
 - Position 2: Heart and any second site
 - QRS has a voltage of 3 - 4 mV
 - Position 3: Arm and arm / arm and leg
 - QRS has a voltage of 1 mV
 - P has a voltage of 0.1 - 0.3 mV
 - T has a voltage of 0.2 - 0.3 mV



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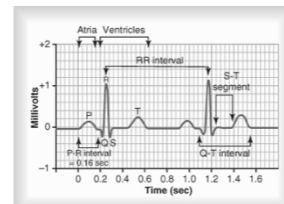


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The Normal Electrocardiogram

Voltage and the ECG

- P-Q / P-R Interval
 - Defines time between atrial contraction and ventricular contraction
 - Typically 0.16 sec
 - Sometimes called P-R interval as Q wave is often absent
- Q-T Interval
 - Defines length of ventricular contraction
 - Typically 0.35 sec
- Heart Rate
 - Easily measured from a ECG, and typically the interval between two QRS complexes is 0.83 sec and therefore heart rate is approximately 72 beats per sec



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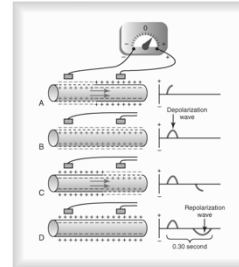
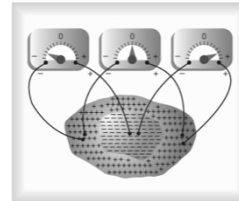


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The Normal Electrocardiogram

Current Flow During Cardiac Cycle

- At rest, a syncytial mass of cardiac muscle is positively charged extracellularly and negatively charged intracellularly
- If an area is depolarized, there will exist an electrical potential between the depolarized and polarized areas of the muscle
- Depending upon the placement of measuring electrodes, this electrical potential will be observed with a variety of different outcomes



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If this is confusing, simply remember this

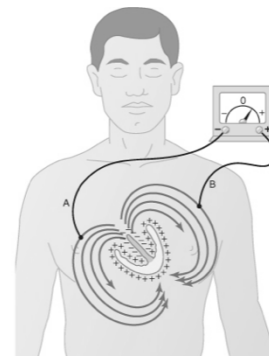


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The Normal Electrocardiogram

Current Flow During Cardiac Cycle

- Remember, cardiac impulses first arrive in the ventricles in the septum and then reach the ventricles endocardial surfaces
- Thus, electronegativity exists on the inside of the ventricles and electropositivity exists on the outside of the ventricles
 - This induces current flow elliptically around the ventricles
 - The average current flow occurs with negativity at the base of the heart and positivity at the apex of the heart throughout most of depolarization
 - Immediately before the end of depolarization, this current flow reverses for 1/100 sec



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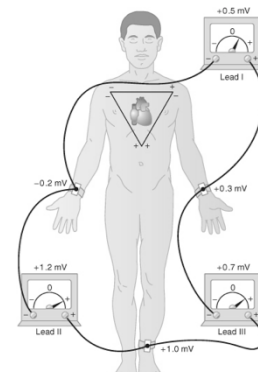


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The Normal Electrocardiogram

Electrocardiographic Leads

- Standard bipolar limb leads, on each arm and the left leg, provide 3 different measurements
- Lead I: Negative terminal at right arm and positive terminal at left arm
 - Positive measurement means right arm is electronegative with respect to left arm
- Lead II: Negative terminal at right arm and positive terminal at left leg
- Lead III: Negative terminal at left arm and positive terminal at left leg



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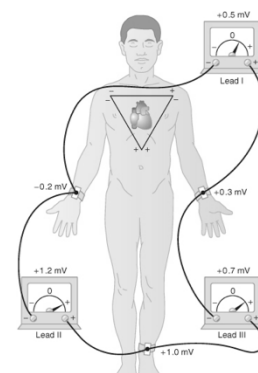


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The Normal Electrocardiogram

Einthoven's Triangle

- A triangle may be drawn around the heart representing the bipolar limb leads
- Einthoven's Law: The sum of the voltage differences in any two leads equals the voltage difference in the third lead
 - Note: The summation must take into account the positive and negative signs of the different leads
 - $0.5 + 0.7 = 1.2$ mV
 - Add the Lead I vector to the Lead III vector and your result is the Lead II vector
 - With this addition approach, all vectors are read "positively" regardless of their +/- values



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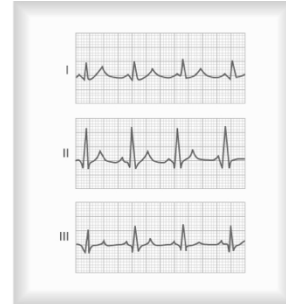


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The Normal Electrocardiogram

ECG from Standard Bipolar Limb Leads

- All leads depict the P wave, QRS complex, and T wave
- Further, the sum of potentials in leads I and III equals the potential observed in lead II
- Cardiac arrhythmias (temporally irregular heart beats) are observed in all leads
- Ventricular or artiral muscle damage as well as conducting tissue damage is observed significantly differently between the various leads



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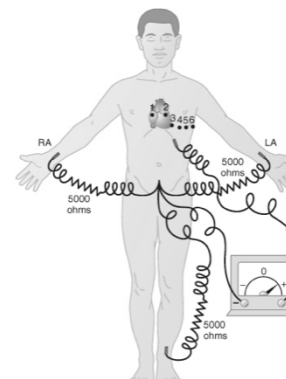
The Normal Electrocardiogram

Precordial Leads

- Often ECGs are measured with one positive electrode over the heart at one of six possible points and one negative electrode at either the arms or left leg
- The result is six different ECGs reflecting the cardiac tissue beneath each different lead
 - V1 and V2: QRS is mainly negative as these leads are nearer the base of the heart
 - V4, V5, and V6: QRS is mainly positive as these leads are nearer the apex



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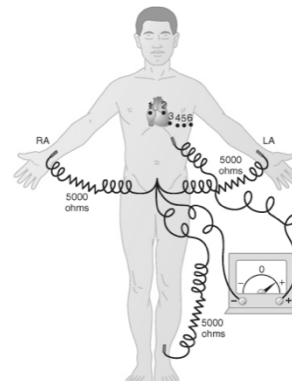
The Normal Electrocardiogram

Augmented Unipolar Limb Leads

- Two limbs are connected through electrical resistances to the negative terminal, and the third limb is connected to the positive terminal
 - aV_R : positive on right arm
 - aV_L : positive on left arm
 - aV_F : positive on left leg



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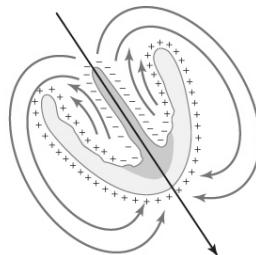


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Electrocardiographic Interpretation

Principles of Vector Analysis in Electrocardiograms

- Remembering vectors in electrical circuits, a vector points in the direction of electrical potential with the arrowhead in the positive direction and the length of the arrow depicting the voltage of the potential
- Thus, the instantaneous mean vector depicting the generated potential within the partially depolarized heart can be seen below



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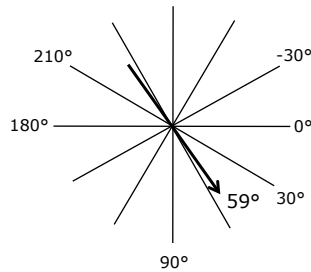


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Electrocardiographic Interpretation

Principles of Vector Analysis in Electrocardiograms

- The mean QRS vector, which is the average direction of the heart vector during the spread of the depolarization wave, is approximately $+59^\circ$



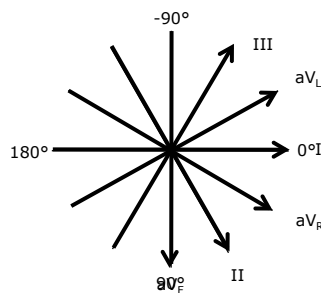
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Electrocardiographic Interpretation

Principles of Vector Analysis in Electrocardiograms

- Each standard bipolar limb lead, as well the aV_R lead, aV_L , and aV_F leads, can also be depicted as vectors
 - aV_R : two limbs at negative terminals, right arm at positive terminal
 - aV_L : two limbs at negative terminals, left arm at positive terminal
 - aV_F : two limbs at negative terminals, left leg at positive terminal



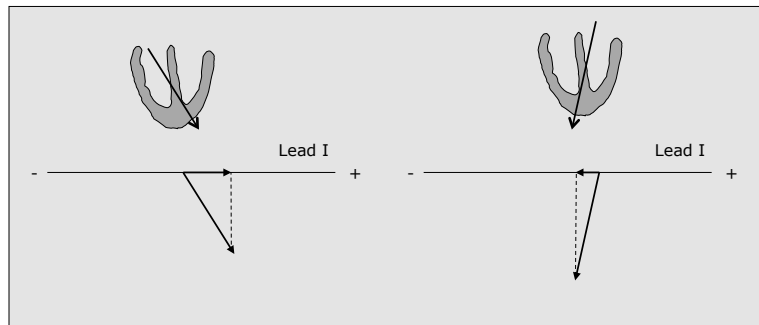
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Electrocardiographic Interpretation

Principles of Vector Analysis in Electrocardiograms

- A vector representing the instantaneous mean direction of current flow may be projected onto any axis to determine the observation of that axis



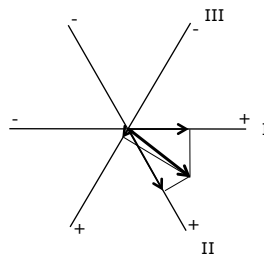
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Electrocardiographic Interpretation

Principles of Vector Analysis in Electrocardiograms

- Similarly, a vector representing the instantaneous mean direction of current flow may be projected onto all three leads to determine their relative contributions



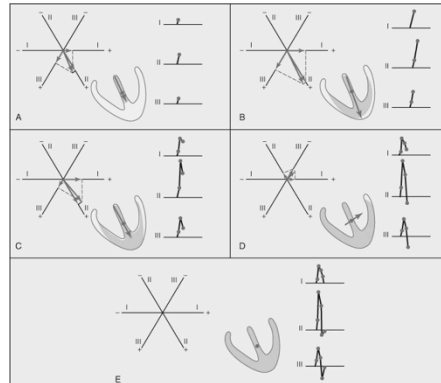
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Electrocardiographic Interpretation

Principles of Vector Analysis in Electrocardiograms

- Finally, this vector analysis may be applied to ventricular vectors during depolarization and compared to the ECG's QRS wave



know and understand this figure

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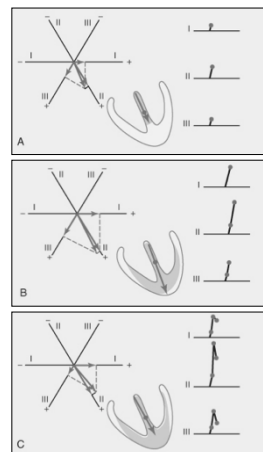
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Electrocardiographic Interpretation

Vector Analysis During Depolarization

- Approximately 0.01 sec after the initiation of depolarization
 - The vector is short as only a small portion of tissue is depolarized
 - Most of the vector is along lead II
- Approximately 0.02 sec after the initiation of depolarization
 - Vector is significantly longer in length due to the increased amount of depolarized tissue
- Approximately 0.035 sec after the initiation of depolarization
 - Vector length decreases as some tissue becomes repolarized
 - Vector begins to point left as the left ventricle depolarizes slightly after the right



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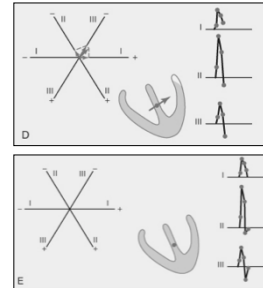
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Electrocardiographic Interpretation

Vector Analysis During Depolarization

- Approximately 0.05 sec after the initiation of depolarization
 - The vector is short as only a small of the left ventricle remains depolarized
 - Vector points left, toward the base of the left ventricle
- Approximately 0.06 sec after the initiation of depolarization
 - Vector is zero as the cardiac muscle is completely depolarized and thus there is no current flow



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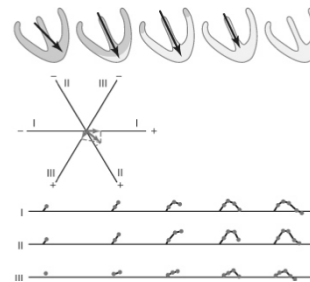


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Electrocardiographic Interpretation

Vector Analysis During Repolarization

- A similar analysis may be performed during ventricular repolarization and the T wave
- Important concepts here include
 - Repolarization occurs 0.15 sec after the end of depolarization and is complete 0.35 sec after the onset of the QRS wave
 - Repolarization occurs first at the outer surface of the ventricles and then in endocardial areas
 - Exactly opposite to depolarization
 - Perhaps due to reduced endocardial blood flow during contraction
 - Thus vector is similar to depolarization vector
 - Thus T wave is positive



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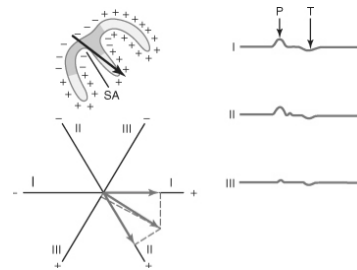


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Electrocardiographic Interpretation

Vector Analysis During Atrial Depolarization and Repolarization

- Again, a similar analysis may be performed during atrial depolarization and the P wave as well as repolarization and the atrial T wave
- Important concepts here include
 - Depolarization begins in the sinus node
 - The direction of the electrical potential vector is slightly to the right of the atrial septum
 - This direction persists throughout depolarization
 - This direction is almost the same as the vector for ventricular depolarization
 - Thus, the P wave is positive
 - Repolarization also begins in the sinus node
 - Opposite to ventricular repolarization
 - Thus vector is in the opposite direction
 - Thus, the atrial T wave is negative



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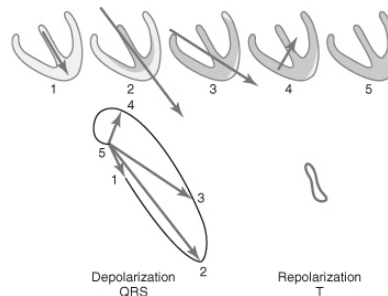


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Electrocardiographic Interpretation

Vectorcardiograms

- The various depolarization and repolarization vectors may be assembled into a vectorcardiogram
 - QRS vectorcardiogram
 - T vectorcardiogram
 - P vectorcardiogram



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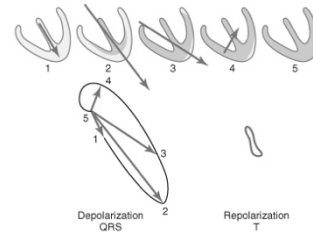


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Electrocardiographic Interpretation

Mean Electrical Axis of the Ventricular QRS

- Throughout ventricular depolarization, the preponderant vector direction is from the base towards the apex of the heart
 - This mean vector is pointing from negative to positive
- This preponderant direction is known as the mean electrical axis or mean QRS vector
- The mean QRS vector is 59° in normal ventricles
- In damaged or diseased ventricles, the mean QRS vector may drift significantly



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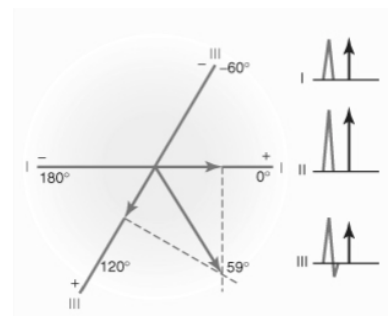


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Electrocardiographic Interpretation

Determining the Mean Electrical Axis

- A standard bipolar limb lead electrocardiogram is performed
- The vectors representing leads I and III are determined and applied to the standard lead axis system
- Perpendicular lines are drawn from the points of the lead I and lead III vectors to assemble the mean electrical axis of the heart
 - Both direction and magnitude are determined



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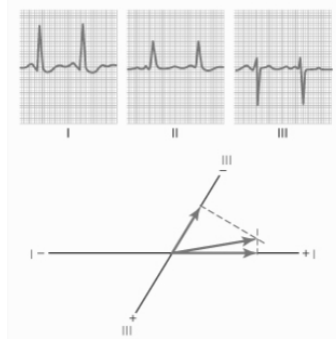


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Electrocardiographic Interpretation

Causes of Axis Deviation

- Left ventricular hypertrophy
 - Enlarged left ventricle causes axis to shift towards the left ventricle
 - Increased cardiac mass
 - Increased depolarization time
- Hypertension
 - Elevated arterial pressure causes ventricular hypertrophy
- Aortic valvular stenosis
- Aortic valvular regurgitation



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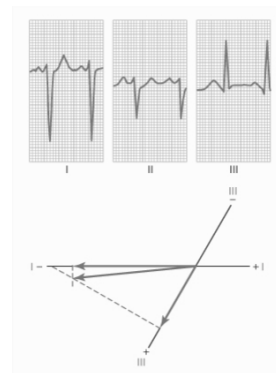


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Electrocardiographic Interpretation

Causes of Axis Deviation

- Right ventricular hypertrophy
 - Enlarged right ventricle causes axis to shift towards the right ventricle
 - Increased cardiac mass
 - Increased depolarization time
- Pulmonary valve stenosis
- Increased pulmonary vascular resistance
- Congenital heart conditions
 - Tetralogy of Fallot
 - Interventricular septal defect



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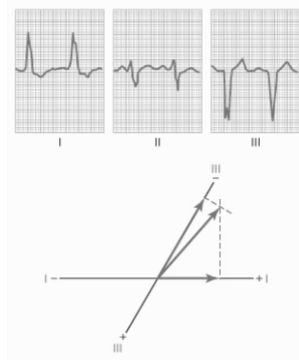


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Electrocardiographic Interpretation

Causes of Axis Deviation

- Bundle branch block
 - Ventricles normally depolarize at the same time due to the quick conduction within the Purkinje system
 - If one side of the bundle branches are blocked, an asymmetric depolarization is observed
- Left bundle branch block
 - Right ventricular depolarization occurs normally, left is slow
 - Left ventricle remains depolarized as right becomes polarized
 - Vector points to the left ventricle
 - Mean electrical axis point is deviated towards the left
 - QRS complex is prolonged due to slow conduction



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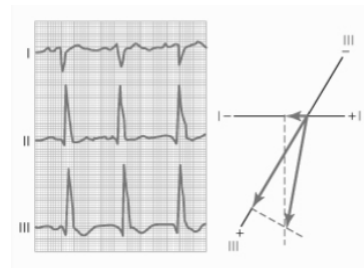


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Electrocardiographic Interpretation

Causes of Axis Deviation

- Right bundle branch block
 - Left ventricular depolarization occurs normally, right is slow
 - Right ventricle remains depolarized as left becomes polarized
 - Vector points to the right ventricle
 - Mean electrical axis point is deviated towards the right
 - QRS complex is prolonged due to slow conduction



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Electrocardiographic Interpretation

Abnormal Voltages in the QRS Complex

- Voltages in the three standard bipolar limb leads, as measured from the peak of the R wave to the bottom of the S wave, vary from 0.5 to 2.0 mV
- When the sum of the voltages of all of the QRS complexes of the three standard leads is greater than 4 mV, a high voltage electrocardiogram is diagnosed
 - High voltage ECGs most often result from hypertrophy
- Depressed voltages occur for a variety of reasons
 - Previous myocardial infarctions that led to diminished cardiac muscle mass
 - Conduction slows and QRS is prolonged
 - Pericardial fluid buildup
 - Pulmonary emphysema



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Electrocardiographic Interpretation

Abnormal Patterns of the QRS Complex

- The normal QRS wave lasts 0.06 to 0.08 sec
- Prolonged QRS waves, 0.09 to 0.12 sec, are a result of prolonged conduction of electrical impulses through the ventricles
 - Perhaps due to hypertrophy or dilation
- Purkinje system blocks
 - Blockage of the Purkinje system forces impulse conduction through cardiac muscle and therefore prolongs conduction by 0.14+ sec
- Unusual QRS waves are usually due to
 - Damage to cardiac muscle
 - Blocks in the Purkinje system



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Electrocardiographic Interpretation

Current of Injury

- Abnormalities can cause a portion of cardiac muscle to remain partially or totally depolarized at all times
 - This is called current of injury
- Under this condition, current flows from the pathologically depolarized areas to the normal areas
 - The injured portion is negatively charged due to depolarization and emits these charges to the surrounding tissue
- Causes of current of injury include
 - Mechanical trauma
 - Infection
 - Ischemia

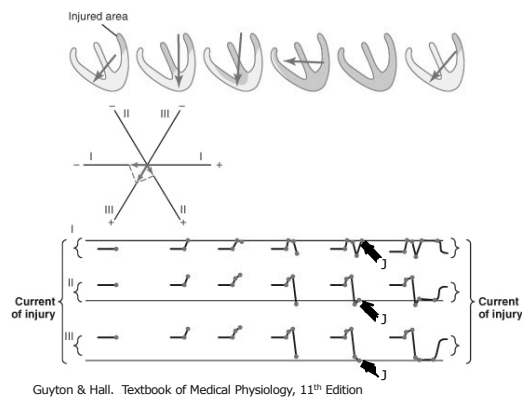


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Electrocardiographic Interpretation

Current of Injury

- Consider a current of injury due to ischemia located in the base of the left ventricle



Notice the definition of the J point

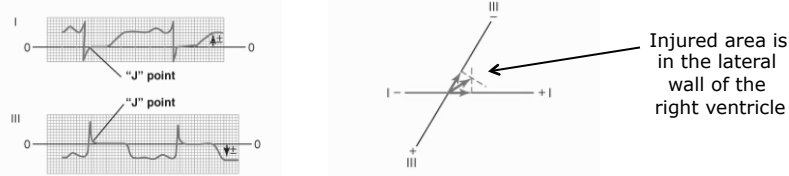


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Electrocardiographic Interpretation

Current of Injury

- A vector analysis can be utilized to identify the location of the current of injury



- Points to consider
 - J point defines zero voltage
 - T-P segment shift, often described as a S-T segment shift
 - Current of injury is a T-P segment shift away from the J point

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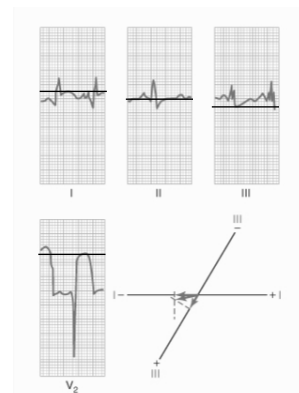


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Electrocardiographic Interpretation

Acute Anterior Wall Infarction

- V2: J point and zero line define a large negative potential during the T-P segment
 - Chest electrode over the front of the heart is highly negative
 - Negative end of the injury potential vector is at the chest wall, current of injury is emanating from the anterior chest wall
 - Anterior wall infarction
- I and III: Negative potential (I) and positive potential (III) related to current of injury means a vector of the current of injury is approx 150°
 - Negative end points at left ventricle and positive end points at right ventricle
- Infarction of the anterior descending limb of the left coronary artery



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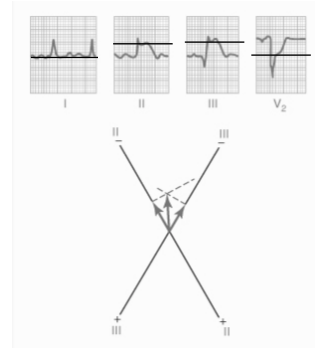


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Electrocardiographic Interpretation

Posterior Wall Infarction

- V2: J point and zero line define a large positive potential during the T-P segment
 - Posterior wall infarction
- II and III: Negative potential (II) and negative potential (III) related to current of injury means a vector of the current of injury is approx -95°
 - Negative end points down and positive end points up
- Infarction of the heart apex on the posterior wall of the left ventricle



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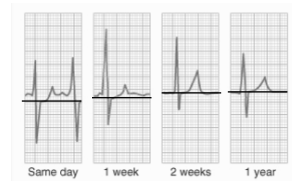


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Electrocardiographic Interpretation

Recovery From Infarction

- After an infarction, a V3 chest lead can depict the change in the current of injury over time
- Results show that the collateral coronary blood flow was sufficient to maintain cardiac muscle health and reestablish most of the affected region



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Electrocardiographic Interpretation

T Wave Abnormalities

- Slow conduction of depolarization
- Prolonged depolarization



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Cardiac Arrhythmias & Electrocardiograms

Introduction

- Many heart conditions result not from heart malfunction, but an abnormal rhythm of the heart
 - Asynchronous arterial - ventricular contraction
- Typical cardiac arrhythmias include
 - Abnormal rhythmicity of the pacemaker
 - Ectopic pacemaker
 - Impulse transmission blockage
 - Abnormal impulse transmission pathways
 - Ectopic impulses



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Cardiac Arrhythmias & Electrocardiograms

Abnormal Sinus Rhythms

- Tachycardia refers to a fast heart rate, greater than 100 beats per minute, and can occur for a variety of reasons
 - Increased body temperature
 - Heart rate increase 10 beats per minute for every °F increase in body temperature, up to 105°F
 - Fever causes tachycardia by increasing sinus node metabolism
 - Sympathetic nerve stimulation
 - Due to massive blood loss
 - Due to weakened heart muscle
 - Toxic heart conditions



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Cardiac Arrhythmias & Electrocardiograms

Abnormal Sinus Rhythms

- Bradycardia refers to a decrease in heart rate, usually less than 60 beats per minute, and can occur for a variety of reasons
 - Athletic physiology
 - Increased cardiac stroke volume due to physical training allows for a decrease in heart rate at rest
 - Parasympathetic (Vagal) stimulation
 - Carotid sinus syndrome: atherosclerosis of the carotid sinus region of the carotid artery increases sensitivity of the baroreceptors, thus a mild pressure increase can cause a significant baroreceptor stimulation and extreme bradycardia



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Cardiac Arrhythmias & Electrocardiograms

Abnormal Impulse Conduction

- Sinoatrial block occurs when the impulse from the sinus node is blocked before it enters the atrial muscle
- In ECGs, S-A block is seen as a cessation of P waves
- AV node picks up as the ventricular pacemaker, thus the QRS wave remains



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Cardiac Arrhythmias & Electrocardiograms

Abnormal Impulse Conduction

- Atrioventricular block can occur in a number of ways
 - Ischemia of the AV node or AV bundle
 - Compression of the AV bundle
 - Inflammation of the AV bundle
 - Excessive parasympathetic stimulation
- First Degree Block
 - The typical P-R (same as P-Q) interval is 0.16 sec, and this decreases with increased heart rate and increases with decreased heart rate
 - At a normal heart rate, a P-R interval of 0.20 sec is said to be a first degree incomplete heart block



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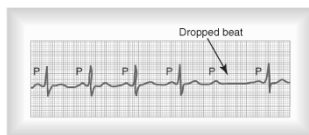
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Cardiac Arrhythmias & Electrocardiograms

Abnormal Impulse Conduction

- Second Degree Block
 - When the P-R interval is 0.25 to 0.45 sec, a second degree incomplete heart block is diagnosed
 - Here, rather than slow conduction, typically the generated impulses are too weak to pass through the A-V bundle
 - Thus, atria are contracting but ventricles are "missing" contractions
- Third Degree Block
 - Complete signal transmission blockage where P wave is decoupled from QRS-T complex which is now driven by the Purkinje fibers (ventricular escape) or even an artificial pacemaker



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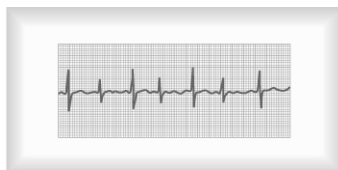
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Cardiac Arrhythmias & Electrocardiograms

Abnormal Impulse Conduction

- Incomplete Intraventricular Block
 - Blockage in the peripheral portions of the ventricular Purkinje system
 - QRS complex can be significantly abnormal
- Electrical Alternans
 - Partial intraventricular block every other heartbeat, often associated with tachycardia



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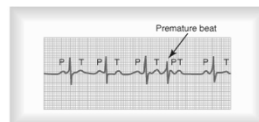
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Cardiac Arrhythmias & Electrocardiograms

Premature Contraction

- Typically premature contraction, or the contraction of the heart before it would normally be expected, results from an ectopic foci where portions of the heart emit abnormal impulses
- Causes of ectopic foci include
 - Local areas of ischemia
 - Calcified plaques that induce inflammation in cardiac muscle
 - Toxic irritation of the conductory system due to drugs, nicotine, or caffeine
- Premature Atrial Contraction: Ectopic origin near the AV node causes a premature P wave, along with a shortened P-R interval and a succeeding delay in the P wave
- AV Nodal / AV Bundle Premature Contraction: Conductance of cardiac signal from ventricles back to atria



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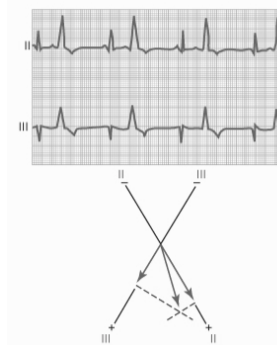
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Cardiac Arrhythmias & Electrocardiograms

Premature Contraction

- Premature Ventricular Contraction
 - QRS complex is usually prolonged as the conducting material is mainly muscle rather than the Purkinje fibers
 - QRS voltage is high since ventricular depolarization is serial (rather than the typically parallel situation), so that depolarization is additive rather than neutralizing
 - T wave polarity is opposite to the QRS complex, since slow conductance reverses the direction of the typical repolarization pattern
- In this example, the vector of premature contraction is negative at the base of the heart and positive towards the apex, indicating the base of the ventricles is the location of the ectopic focus



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Cardiac Arrhythmias & Electrocardiograms

Paroxysmal Tachycardia

- Abnormalities in any portion of the heart can cause the rapid rhythmical discharge of impulses that spread throughout the heart
 - Because of the quick rhythm, this area becomes the pacemaker of the heart
- Paroxysmal implies that these events occur in paroxysm episodes, where the rapid heart rate is quickly established, lasts for seconds, minutes, or hours, and then quickly disappears - allowing the sinus node to take over as pacemaker
- Atrial paroxysmal tachycardia can occur
 - Note inverted P wave
- Ventricular paroxysmal tachycardia can occur
 - Note voltage of QRS complex



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Cardiac Arrhythmias & Electrocardiograms

Ventricular Fibrillation

- The most serious form of cardiac arrhythmia is ventricular fibrillation
- Here, cardiac impulses stimulate one portion of the ventricle muscle, then another, and another, eventually returning to the initial mass and continuing to cycle
- Many small portions of the ventricular muscle will contract at the same time, causing little to no blood flow due to asynchronous contraction
- After 4 to 5 sec, unconsciousness occurs and bodily tissues begin to die
- Ventricular fibrillation can be initiated by
 - Sudden electrical shock of the heart
 - Ischemia of the heart muscle or its conducting system

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Cardiac Arrhythmias & Electrocardiograms

Ventricular Fibrillation

- Phenomena of Re-entry
- A normal cardiac impulse dies out in the ventricle because the all of the ventricular tissue is within its refractory period and the impulse can not be conducted through any of the surrounding tissue
- In an abnormal heart, the cardiac impulse remains conducting throughout the tissue in an irregular and uncontrolled pattern - this may be due to
 - Dilated Hearts: A heart muscle mass that is so large that part of the muscle is released from the refractory period before the impulse has ceased
 - Impulse Blockage: The velocity of the impulse is so slow that part of the muscle is released from the refractory period before the impulse has ceased
 - Drug Stimulation: A refractory period that has shortened, allowing impulse conduction before the impulse has ceased
- The heart is most vulnerable to ventricular fibrillation when some areas are under refractoriness, and other areas are not under refractoriness - *just after cardiac contraction*

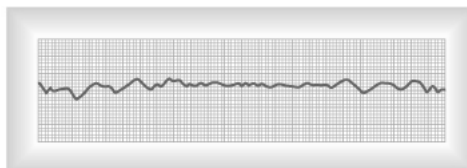


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Cardiac Arrhythmias & Electrocardiograms

Ventricular Fibrillation

- ECGs during ventricular fibrillation are very irregular
- Initially after fibrillation, voltages of 0.5 mV are observed, and these decay to 0.2 - 0.3 mV after 20 to 30 sec, and 0.1 mV for up to 10 min



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Cardiac Arrhythmias & Electrocardiograms

Ventricular Fibrillation

- Electroshock Defibrillation
- A strong electric current passed through the ventricle for a short interval can stop fibrillation by forcing all the ventricular mass into refractoriness
 - All impulses stop, the heart remains quiescent for 3 to 5 seconds, and then the sinus node or other pacemaker takes over
 - However, the same conditions that initiated fibrillation may cause it to return
- Fibrillation can be stopped with a number of approaches
 - 110 V of 60 Hz alternating current applied for 0.1 sec
 - 1000 V of direct current for 0.001+ sec
- After 1+ min of defibrillation, electroshock therapy will likely not work as the cardiac muscle is too weak from lack of blood flow
 - Direct hand contraction of the heart followed by defibrillation can succeed
 - Cardiopulmonary resuscitation can also succeed
 - After 5 - 8 min of blood flow, severe brain damage and/or death occurs



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Cardiac Arrhythmias & Electrocardiograms

Atrial Fibrillation

- Atrial fibrillation can occur in a manner very similar to ventricular fibrillation
 - Ventricular fibrillation can occur alone
 - Atrial fibrillation can occur alone
- Atrial fibrillation likely results from atrial enlargement resulting from heart valve lesions that prevent either (1) the atria from emptying into the ventricles or (2) ventricular failure causing atrial damming
- Under fibrillation, atria will not pump adequately
 - Blood continues to flow into ventricles
 - Primer pump mechanism is lost, reducing ventricular pumping by 20 - 30%
 - A person can live with atrial fibrillation for months or years



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Cardiac Arrhythmias & Electrocardiograms

Atrial Fibrillation

- In atrial fibrillation, the resulting ECG is very irregular
 - No or little P waves are present
 - QRS-T wave complex is unaffected
- Ventricular rhythm, however, is severely affected
 - AV node receives signals irregularly, but will not pass them to the Purkinje system faster than every 0.35 sec
 - In combination with the irregular firing of the atrial impulse, AV nodes may fire from 0.35 sec to 0.95 sec after the previous impulse
 - Irregular heartbeats and a fast heart rate, from 125 - 150 bpm, are clinical indications of atrial fibrillation



Guyton & Hall. Textbook of Medical Physiology, 11th Edition

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Cardiac Arrhythmias & Electrocardiograms

Cardiac Arrest

- A dramatic abnormality of cardiac rhythmicity is cardiac arrest
 - Cessation of all rhythmical impulses in the heart
- Indications
 - Occurs during deep anesthesia leading to severe hypoxia due to inadequate respiration
 - Hypoxia interrupts normal electrolyte concentration differences and thus reduces excitability
 - CPR is quite successful in reestablishing a normal heart rate
 - Occurs with severe myocardial disease
 - Implantable cardiac pacemakers are used successfully for years

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