ontrol of HR
NS & SNS CO must be responsive to Δs in metabolic demands, ex. exercise CO may ↑ 4 xs, to 20 L/min.
is normally accomplished by approximately doubling both the HR and the SV.
s in HR are accomplished by reflex controls mediated by ANS, inc. its SNS and PNS divisions.
he PNS impulses (via vagus nerve) can ↓cardiac rate. SNS impulses ↑ it. Effects result from action on SA node
alance bt these two reflex control systems normally determines the HR.
R is ↑d by the SNS nervous system through an ↑d level of circulating catecholamines (from adrenal gland) and by excess TH, which produces a
atecholaminelike effect.
aroreceptors Also HR is affected by CNS and baroreceptor activity (nerve cells in aortic arch & internal carotid
aroreceptors are sensitive to changes in BP
uring sig ↑ in BP, these cells ↑ their rate of discharge, transmitting impulses to the cerebral medulla.
his initiates PNS activity and inhibits SNS response, lowering the HR and the BP.
pposite is true during ↓ BP, results in less baroreceptor stim, prompting a ↓ in PNS inhibitory activity in the SA node, allowing for enhanced SNS activity.
he resultant vasoconstriction and ↑d HR elevate the BP.
ontrol of SV primarily determined by three factors: preload, afterload, and contractility.
reload refers to the degree of stretch of the ventricular cardiac muscle fibers at the end of diastole.
nd of diastole is period when filling volume in ventricles is highest & degree of stretch on muscle fibers is greatest
olume of blood w/in ventricle at end of diastole determines preload, which directly affects SV.
reload is commonly referred to as left ventricular end-diastolic pressure (LVEDP).
s volume of blood returning to the heart ↑s, muscle fiber stretch also ↑s (↑d preload), =s ↑ contraction & greater SV. This relationship (Frank-Starling law)
s maintained until the physiologic limit of the muscle is reached.
rank-Starling law based on fact that, w/in limits, the greater the initial length or stretch of the cardiac muscle cells (sarcomeres), the greater the degree of
hortening that occurs. rubber band example
esult is caused by ↑d interaction between the thick and thin filaments within the cardiac muscle cells.
reload is ↓d by a reduction in the volume of blood returning to the ventricles.
iuresis, venodilating agents (eg, nitrates), excessive loss of blood, or dehydration reduce preload.
reload is ↑d by increasing the return of circulating blood volume to the ventricles.
ontrolling loss of blood or body fluids and replacing fluids are examples of ways to ↑ preload.
fterload, or resistance to ejection of blood from the ventricle, is the second determinant of SV.
esistance of the systemic BP to left ventricular ejection is called **systemic vascular resistance**.
esistance of the pulmonary BP to right ventricular ejection is called **pulmonary vascular resistance**.
elationship between afterload and SV is inverse. For example, afterload is ↑d by arterial vasoconstriction, which leads to ↓d SV.
pposite is true w/ arterial vasodilation: Afterload ↓s because there is less resistance to ejection, and SV ↑s.
ontractility refers to the force generated by the contracting myocardium.
ontractility is enhanced by catecholamines, SNS neuronal activity, & medications (eg, digoxin [Lanoxin], dopamine [Intropin], or dobutamine [Dobutrex]).
d contractility results in ↑d SV.
ontractility is depressed by hypoxemia, acidosis, & medications (eg, beta-adrenergic blocking agents such as atenolol [Tenormin]).
he heart can achieve an ↑ in SV (eg, during exercise) if preload is ↑d (through ↑d venous return), if contractility is ↑d (through SNS nervous system
discharge), and if afterload is ↓d (through peripheral vasodilation with ↓d aortic pressure).
jection fraction: % of end-diastolic blood volume that is ejected with each heartbeat
jection fraction of normal left ventricle is 55% to 65%. Right ventricular ejection fraction is rarely measured.
jection fraction is used as a measure of myocardial contractility.
jection fraction of less than 40% indicates that the patient has ↓d left ventricular function & likely requires tx for HF

hase 0: Cellular depolarizationis initiated, atrial and ventricular myocytes rapidly epolarize as N moves into the cells through Na+ fast channels. The myocyte have a ıst response action potential. In contrast, the cells of the SA and AV node depolarize hen Ca++ enters these cells through Ca++slow channels. These cells have a slower esponse .

hase 1: Early cellular repolarization begins during this phase as K+ exits the cell.

hase 2: plateau phase , rate of repolarization slows. Ca++ ions enter the cell.

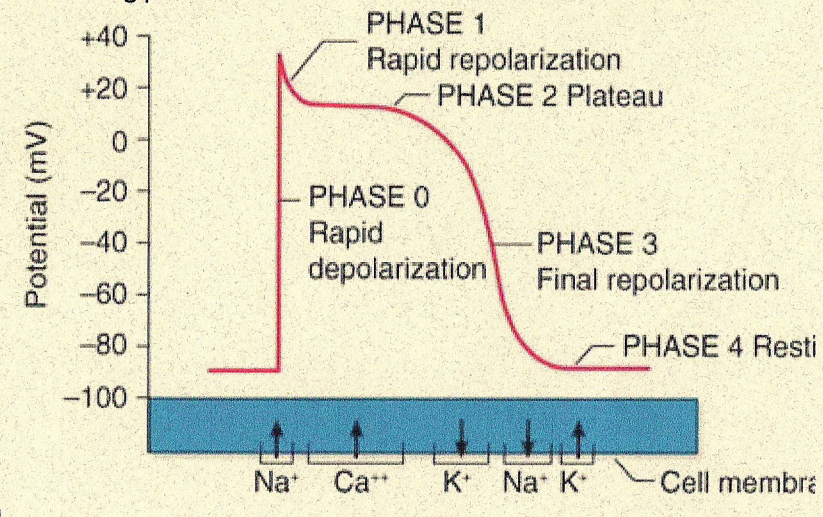
hase 3: marks the completion of repolarization and return of the cell to its resting state.

hase 4: This phase is considered the resting phase before the next depolarization.

/HY THIS MATTERS

Myocardial cells completely repolarize before they depolarize again: refractory period. Two phases:effective (or absolute) refractory period and the relative refractory period. During effective refractory period, cell is completely unresponsive to any electrical stim effective refractory period occurs w/ the time in phase 0 to the middle of phase 3 During the relative refractory period if an electrical stimulus is stronger than normal, the cell may depolarize prematurely. This period corresponds with the end of phase 3. Early depolarizations of atrium or ventricle cause premature contractions, ↑risk for **dysrhythmias**. Premature ventricular contractions in certain situations, such as the presence of myocardial ischemia, can trigger **life-threatening dysrhythmias**, including ventricular tachycardia or ventricular fibrillation. Several circumstances make the heart more susceptible to early depolarization during the relative refractory period.

Cardiac action potential Purkinje fiber. The arrows indicate the approx time and direction of movement of each ion in ncing membrane potential. Ca^{++} movement out of cell is thought to oc during phase 4



ardiac conduction system generates and transmits electrical impulses that stimulate contraction of the myocardium.

CCS first stims contraction of the atria and then the ventricles, allows ventricles to fill completely prior to ejection,maximizing CO.

Three physiologic characteristics of two types of special electrical cells (nodal & the Purkinje) provide this synchronization:

Automaticity: ability to initiate an electrical impulse *Excitability:* ability to respond *Conductivity:* ability to transmit

Sinoatrial (SA) node and **atrioventricular (AV) node** are composed of nodal cells

SA node is primary pacemaker of the heart

located at the junction of the superior vena cava and the right atrium

fires at a rate of 60 to 100 impulses per minute, rate changes in response to the metabolic demands

impulses initiated by SA node are conducted along myocardial cells of the atria via Internodal pathways causing atrial contrxn

impulses are then conducted to the AV node, which is located in the right atrial wall near the tricuspid valve

AV node coordinates the incoming electrical impulses from the atria, and after a slight delay (allowing the atria time to contract and complete ventricular fil relays the impulse to the ventricles.

impulse is conducted through a bundle of specialized conducting tissue, referred to as the bundle of His, which then divides into the right bundle branch (conducting impulses to the right ventricle) and the left bundle branch (conducting impulses to the left ventricle).

Impulses travel through the bundle branches to reach the terminal point in the conduction system, called the Purkinje fibers. These fibers are composed of Purkinje cells, specialized to rapidly conduct the impulses through the thick walls of the ventricles. This is the point at which the myocardial cells are stimulated, causing ventricular contraction.

The heart rate is determined by the myocardial cells with the fastest inherent firing rate.

SA node has the highest inherent rate (60 to 100 impulses per minute),

AV node has the second-highest inherent rate (40 to 60 impulses per minute),

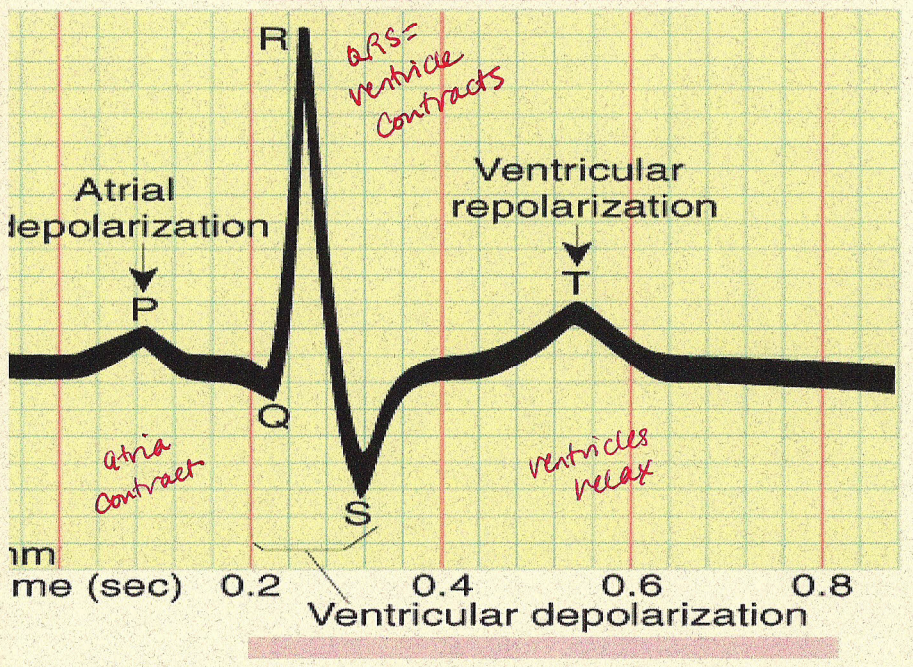
ventricular pacemaker sites have the lowest inherent rate (30 to 40 impulses per minute).

If SA node malfunctions, the AV node generally takes over the pacemaker function of the heart at its inherently lower rate.

Should both SA and AV nodes fail, a pacemaker site in the ventricle will fire at its inherent bradycardic rate of 30 to 40 impulses per minute.

Key components of an ECG tracing

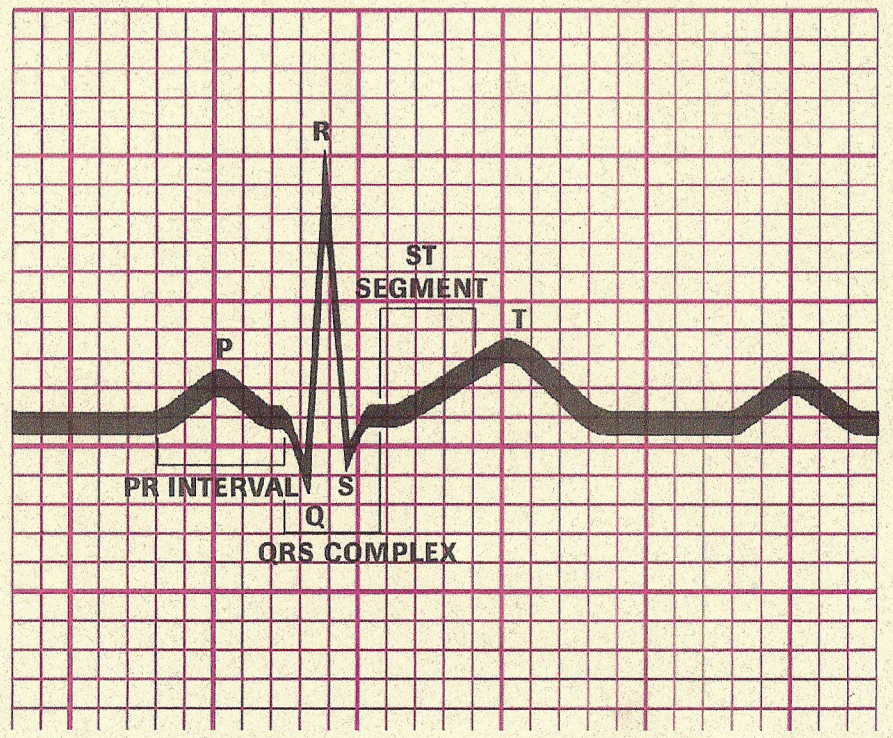
- P wave: reflects initial depolarizing wave in atrial systole
- QRS complex: reflects impulse transmission leading to ventricular systole
- T wave: reflects ventricular repolarization during diastole
- PR interval: reflects the time from the beginning of the P wave to the beginning of the QRS complex
- ST segment: reflects the time from the end of the S wave to the beginning of the T wave



Characteristic PQRST patterns

Electrocardiogram wave patterns are identified by letters; each waveform cor responds to specific electrical events in the cardiac cycle.

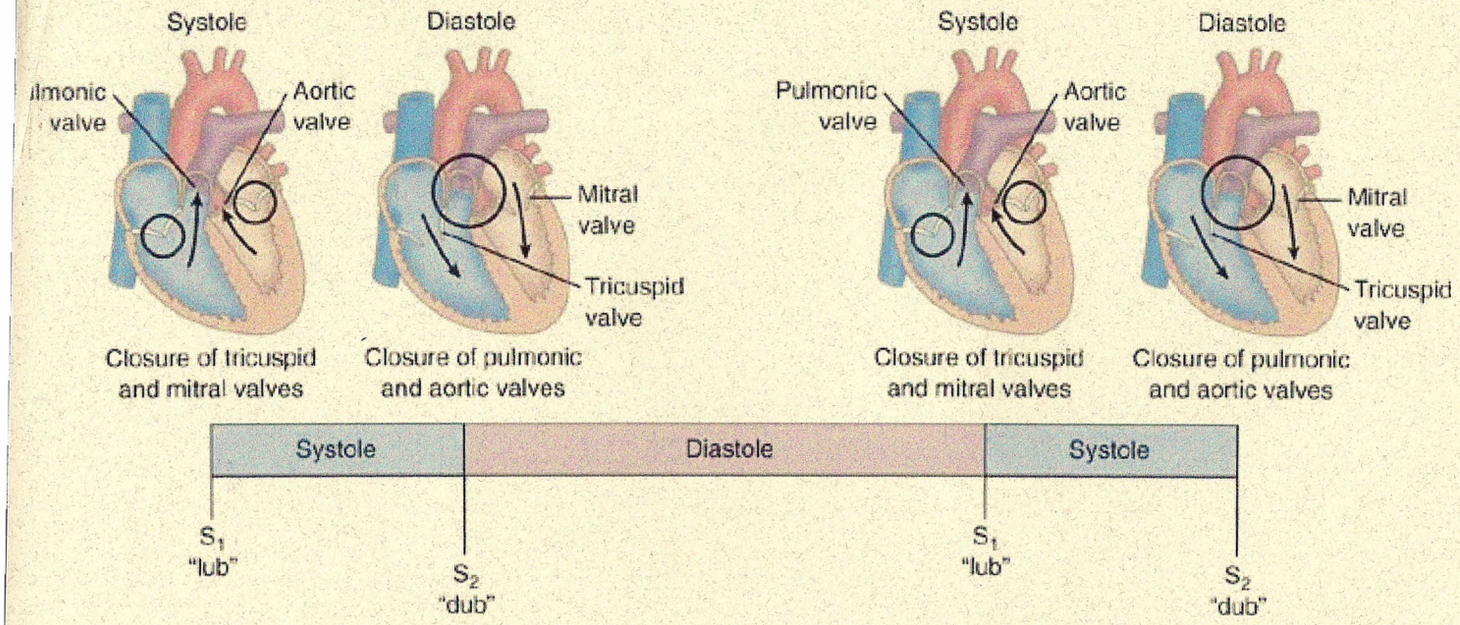
- The P wave reflects the initial wave of depolarization associated with atrial systole.
- The Q, R, and S waves (collectively called the QRS complex) reflect impuls transmission through the right and left bundles into the terminal branches leading to ventricular systole.
- The T wave indicates ventricular repolarization during diastole.
- The PR interval (the time from the beginning of the P wave to the beginnin of the QRS complex) represents the time needed for an impulse to pass fr the atria to the ventricles through the bundle of His.
- The ST segment (the time from the end of the S wave to the beginning of t T wave) represents the time between the end of the spread of the impulse through the ventricle and repolarization of the ventricle.



Normal heart sounds

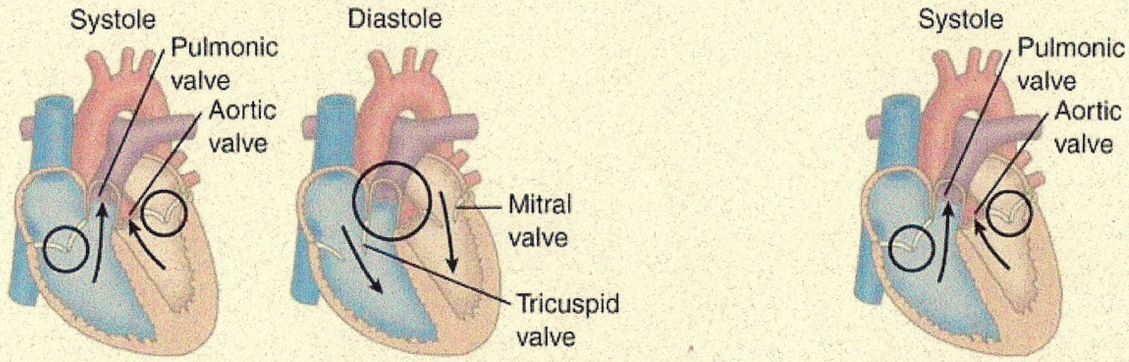
first sou (S1) is produced by closure of the mitral and tricuspid valves "lub"

Second sound (S_2) is produced by closure of the aortic and pulmonic valves "dub"



Diastole
(period of ventricular filling)
all four chambers relax simultaneously, which allows ventricles to fill in preparation fo contraction.

Systole
events in the heart during contraction of the two top chambers (atria) and two bottom chambers (ventricles).
atrial and ventricular systole are not simultaneous events.
Atrial systole occurs first, just at the end of diastole, followed by ventricular systole.
synchronization allows ventricles to completely fill prior to ejectior of blood from their chambers.



Gallop sounds.

S_3 ("DUB") is an abnormal sound heard immediately following S_2 (closure of semilunar valves). generated very early in diastole as blood flowing into the right or left ventricle is met with resistance.

S_4 ("LUB") is an abnormal sound created during atrial systole as blood flowing into the right or left ventricle is met with resistance.