ARRHYTHMIAS

Revised Physiology Notes:

Mark Haigney, MD

mcph@AOL.com

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Introduction

300,000-500,000 deaths each year in the U.S. are attributable to arrhythmias, making sudden cardiac death the leading killer. An understanding of basic cardiac physiology is essential to comprehending the therapy of arrhythmias.

Definition: An **arrhythmia** is any cardiac rhythm which is **not normal sinus rhythm.**

Normal sinus rhythm = rate between 60 and 100 bpm originating in sinus node.

Rate >100=TACHYCARDIA

Rate<60=BRADYCARDIA

*These rates are arbitrary cutoffs since normal heart rates actually run 50-90 bpm, but standard medical usage defines normal as 60-100.

For a rapid review of the surface EKG, I recommend this website
http://endeavor.med.nyu.edu/courses/physiology/courseware/ekg_pt1/ekgmenu.html

Review: The Cardiac Action Potential

Cardiac tissue, like nervous tissue, is excitable, ie. it is capable of responding to stimulation with a large, rapid shift in membrane voltage (potential). This potential change triggers mechanical action in the heart, hence is called an action potential **(AP)**.

The AP in cardiac muscle is different from the AP in the pacemaker tissue of the SA and AV nodes. Remember that the speed of conduction through tissue is a function of the slope and amplitude of phase 0 (faster, taller upstroke= faster conduction). Also, remember that the duration of the **refractory period** (the time during the AP when the cell is not excitable) is roughly equal to the AP duration.

Because cardiac **muscle** must contract in a rapid, synchronized manner, the **APs have rapid phase 0 upstrokes (due to opening of Na+ channels)** which allow relatively fast conduction of impulses from cell to cell. And because these cells need to remain at rest until stimulated, **phase 4 is flat, without spontaneous depolarization.**

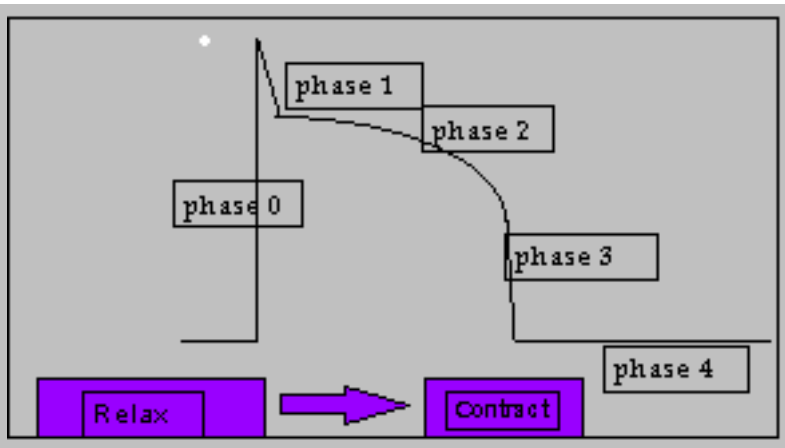


Figure 1 Ventricular Muscle AP

Pacemaker tissue differs in that the upstroke of phase 0 is slow. Na channels are **absent** in this tissue; phase 0 is dominated by calcium channels that open slower and pass less current than Na channels. This slow upstroke results in **delayed conduction through the AV node**, allowing the atria time to empty before the ventricles begin contraction. Phase 4 is demonstrates a slow depolarization (rise in voltage) resulting in spontaneous triggering of an action potential. This property is "automaticity" and controls the heart rate in the SA node. The higher the slope of phase 4, the higher heart rate. Vagal stimulation slows phase 4 depolarization, catecholamines speed it.

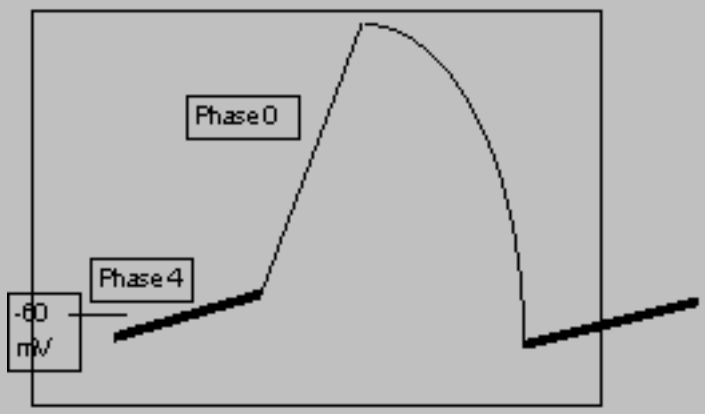


Figure 2 Nodal tissue Action Potential

Purkinje fibers are specialized, elongated conducting cells linking the AV node with the ventricular myocardium and comprise the His bundle and bundle branches. These cells have Na channels enabling fast conduction from the AV node to both ventricles, but also have a limited capacity for automaticity and can supply a **slow escape rhythm of 20-40** bpm in complete heart block.

Significance of Arrhythmias

Cardiac output remains preserved across a wide range of heart rates, but extreme values (ie. <50 or >150 in elderly patients) result in depression of cardiac output with reduced perfusion of the brain, pulmonary congestion, and possibly myocardial ischemia, sometimes with fatal results.

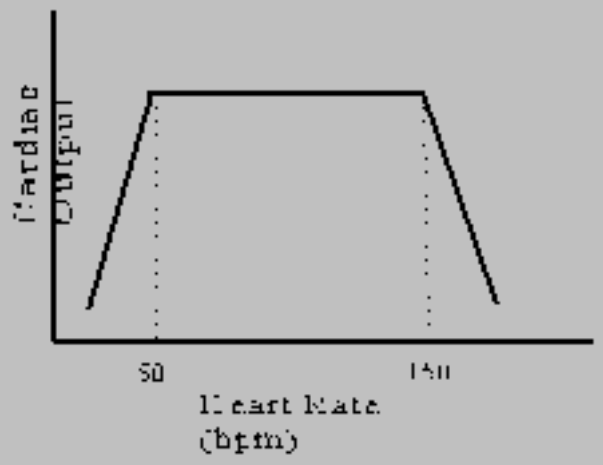


Figure 3 Effect of Heart Rate on Cardiac Output

Tachycardias- Leading Cause of Sudden Cardiac Death (SCD)

The mechanism of the vast majority of clinically relevant tachycardias is **reentry**. Reentry is a self perpetuating pattern of depolarization involving any cardiac tissues and does **NOT** depend on altered automaticity. Instead, this phenomenon depends on heterogeneities between areas of the heart allowing conduction in one area and block in another.

Three Necessary Conditions Required for Reentry

These conditions may exist only transiently due to alterations in tissue properties caused by ischemia, drugs, electrolytes, etc.

1. Heterogeneous refractoriness- there must be a difference in the refractory periods in cadiac tissue. Usually this means that the AP durations in two conduction paths surrounding an obstacle are different. This allows a premature stimulus to hit refractory tissue in one area (long AP) but conduct in another (short AP). The obstacle is some nonconducting structure which may be dead tissue (such as after a heart attack), a normal structure (like the tricuspid valve annulus), or temporarily refractory tissue.

2. **Slow conduction:** one pathway must conduct slowly enough that excitability can be regained in the other pathway before the depolarizing wave can make a complete cycle through the circuit.

3. Unidirectional block: one pathway must be capable of conducting in one direction only.

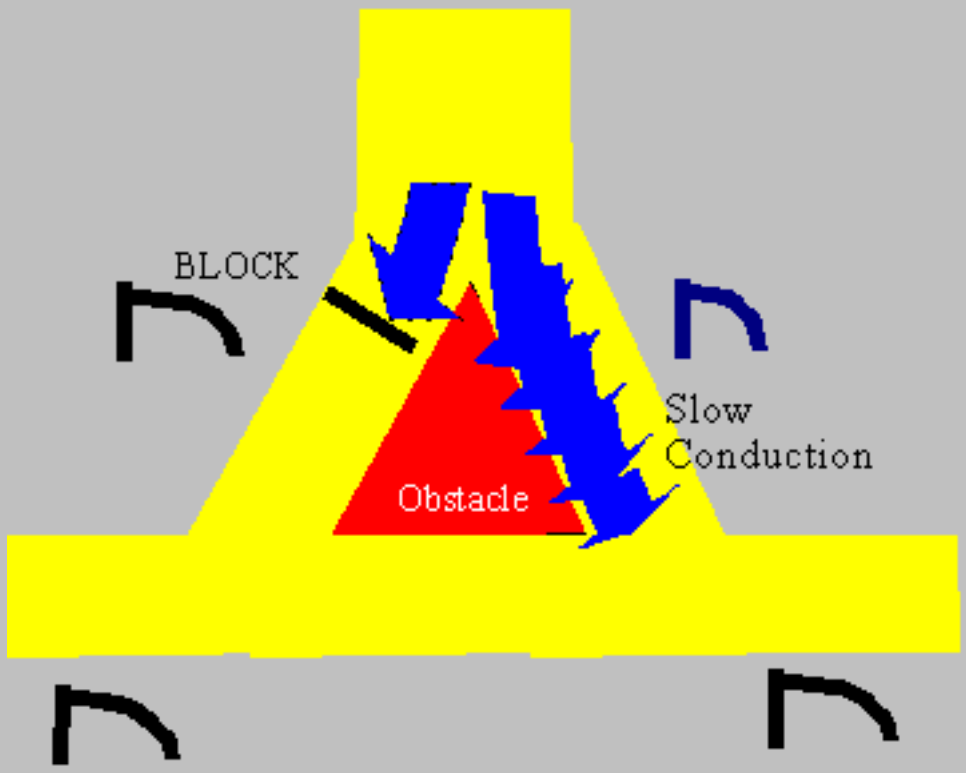


Figure 4 Unidirectional block and slow conduction around a non-conducting obstacle

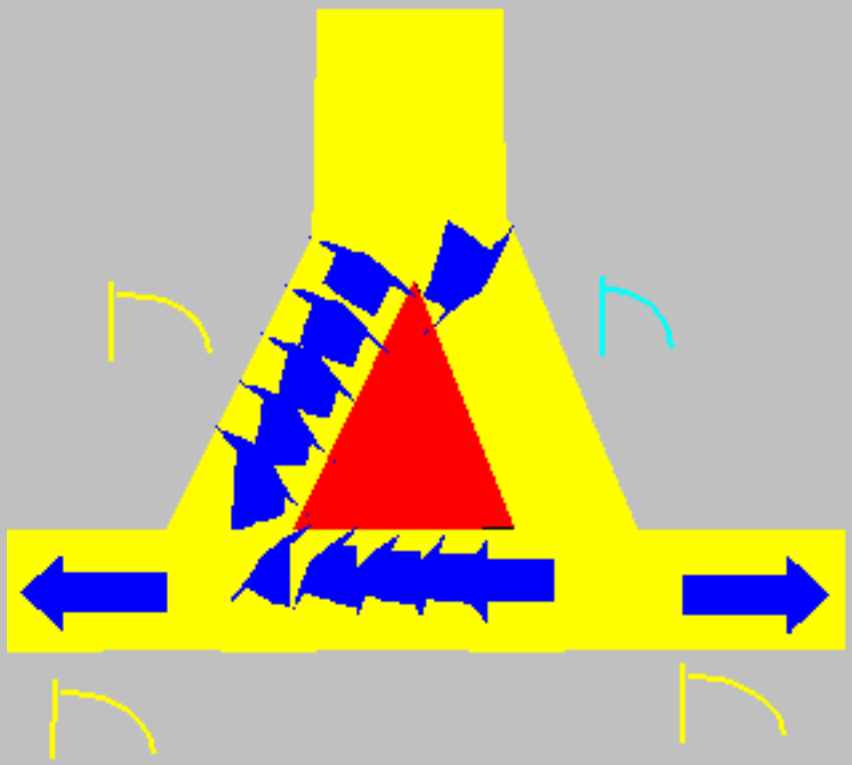


Figure 5 Completion of one cycle of reentry; cycle about to repeat with excitation of slowly conducting path

2. Types of Reentry- reentry is classified based on the nature of the obstacle.

1. Anatomic Reentry- obstacle is a **fixed** structure like the tricuspid anulus or a scar. Typically associated with tachycardias which have a regular rate and unchanging, monotonous EKG pattern due to the stable nature of circuit.

a. Macroreentry- Obstacle is a macroscopic structure, ie. tricuspid annulus, mitral annus**. Examples- atrial flutter; Wolff-Parkinson-White syndrome**.

b. Microreentry- Obstacle is small, like AV node or scar from an infarct. **Examples- AV node reentry, ventricular tachycardia**, sinus node reentry.

2. Functional Reentry- Obstacle is a **moving** area of temporary block resulting from diffuse abnormalities in the tissue. Usually seen in severely enlarged atria or ventricles or large areas of ischemia. Results in rapid, irregular rates with chaotic, unpredictable EKG patterns**. Examples- Atrial fibrillation, ventricular fibrillation**, and (possibly) torsades de pointes.

Therapy for Reentrant Arrhythmias

Successful reentry requires that the three conditions named above be satisfied; violation of any of these conditions terminates tachycardia. Reentry is in a sense a fragile phenomenon, although rare patients with circuits having areas of very slow conduction may have incessant tachycardia. Indeed, certain class 1C drugs (encainide and flecanide) can slow conduction, and can cause incessant, life-threatening ventricula tachycardia.

Strategies for Preventing Reentry

1. Eliminate heterogeneous refractoriness by equalizing AP durations. Class III drugs like amiodarone and sotalol prolong the AP, hopefully equalizing refractoriness in both conduction paths, causing premature stimuli to block in both limbs.

2. Convert slow conduction to "no conduction." Slow conduction through depolarized, damaged tissue can be blocked entirely by drugs like lidocaine which preferentially bind to depolarized tissue. Again, premature stimuli will be blocked in both limbs of the circuit.

3. Convert unidirectional block to bidirectional block. Similar to "2" above, drugs like quinidine may suppress excitability in an area of the circuit which is conducting weakly.

4. Prematurely depolarize a critical part of the circuit. By depolarizing tissue in the circuit in the circuit before the wave of depolarization reaches it, you may render it inexcitable and prevent conduction of reentry. The simplest way to do this is to administer a large electrical shock to the whole heart. This can depolarize the entire circuit (if large enough) and render all the tissue temporarily inexcitable. With luck, the first area to spontaneously depolarize will be the sinus node, restoring sinus rhythm. **This is the most effective method of terminating reentry.**

Other Causes of Tachyarrhythmias

Triggered Arrhythmias Abnormal depolarizations triggered by a preceding normal depolarization. Occur in settings of drug intoxication (quinidine, seldane, digoxin) and in unusual inherited ion channel abnormalities, ie. congenital long QT interval syndromes. Quite controversial, may only initiate an arrhythmia which then depends on functional reentry.

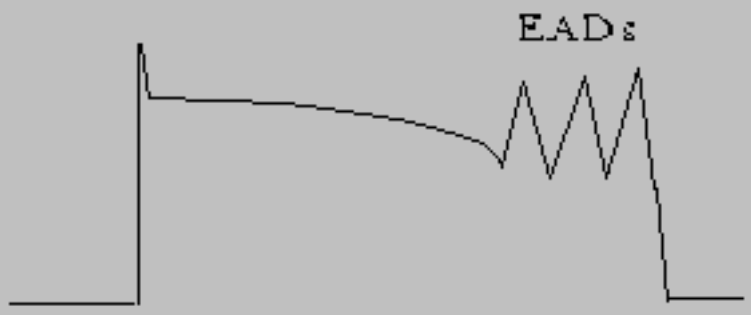


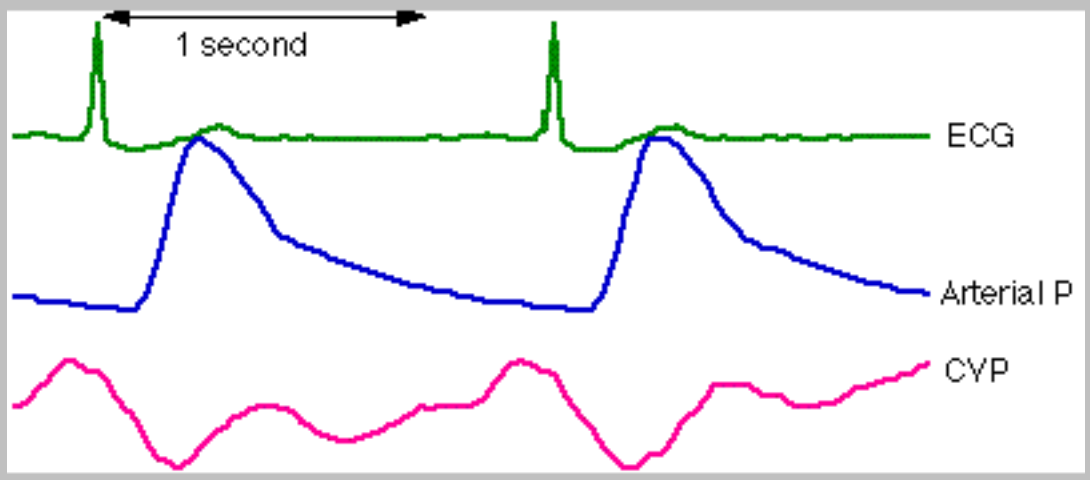
Figure 6 Multiple triggered after-depolarizations in a prolonged action potential

Acquired Automaticity- Occurs when a normally quiescent tissue in the heart develops spontaneous phase 4 depolarization. An unusual cause of clinically significant tachycardia, usually seen in children with congenital heart disease or in acute ischemia.

Bradycardia

In general, bradycardic rhythms result from either:

1. Decreased automaticity- Manifested as an inappropriate decrease in sinus rate without evidence of non-conducted impulses, ie. no extra "p" waves without "QRSs" following. Automaticity in the sinus and AV nodes can be transiently suppressed by an increase in vagal tone or a withdrawal of sympathetic tone. This can occur in otherwise normal patients under the right circumstances. The Bezold-Jarisch reflex causes a paradoxic bradycardia with vasodilation, causing the blood pressure to plummet, often causing loss of conciousness. Also called vaso-depressor or neurocardiogenic syncope, the reflex appears to be caused by an acute decrease in ventricular volume (caused by standing at attention, hemorrhage, etc.) which triggers mechanical receptors to send efferent stimuli to the nucleus tractus solitarius, causing acute sympathetic withdrawal and increased vagal traffic.

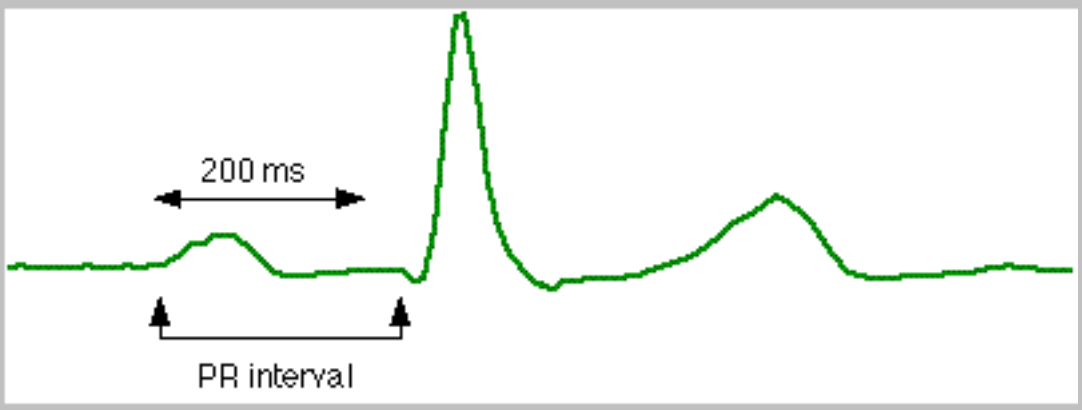


Bradycardia due to decreased automaticity

Drugs such as beta receptor blockers, digoxin, calcium channel blockers, and sodium channel blockers can alter the automaticity of pacemaker tissue and should be immediately suspected in symptomatic bradycardia.

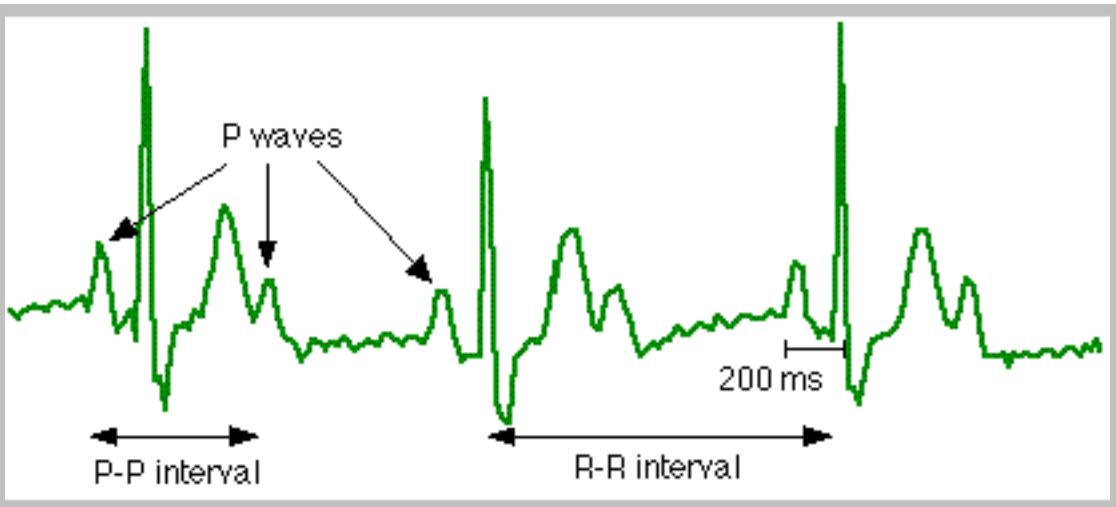
2. Failure of conduction: manifested as either delayed conduction of ÒpÓ waves, ÒpÓ waves without QRS complexes following (AV nodal block), or sudden gaps in the sinus rate which are exact multiples of the sinus rate (SA nodal block). AV nodal blocks are more clinically significant and have four important subtypes:

First degree AV block- prolongation in the conduction time from the atrium to the ventricle manifested as PR interval prolongation, but every "p" is followed by a QRS. Usually due to increased vagal tone slowing AV nodal conduction, and usually not significant.



First degree AV block

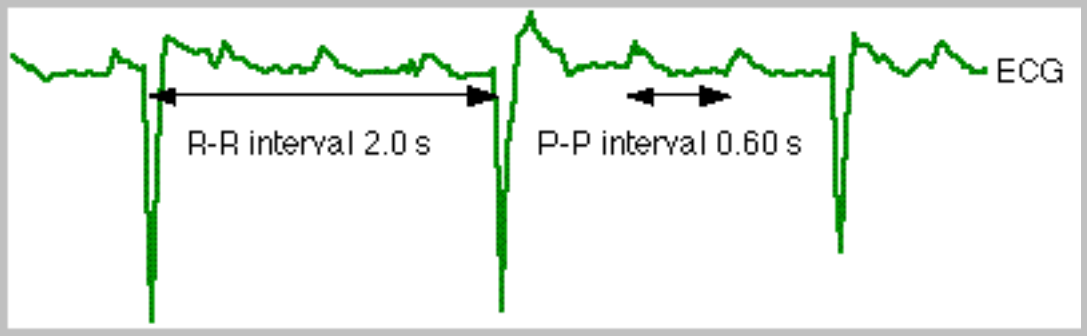
Second degree AV block- second degree AV block is present whenever a "p" wave fails to be followed by a "QRS." This can be due to a normal or a pathologic response by the AV node or His-Purkinje conduction system. Since the AV node conducts decrementally, as the atrial rate speeds up, AV nodal conduction slows down. Depending on the vagal and sympathetic tone of the individual, second degree block may occur in response to a relative tachycardia (see 2:1 conduction below). In highly trained individuals, second degree blok may occur even at slow heart rates due to the increased vagal tone seen in atheletes. Gradual PR interval prolongation is seen prior to the blocked beat. This is **Mobitz 1 or "Wenkebach" block** and can be seen in any condition of high vagal tone, including an inferior wall myocardial infarction, but rarely proceeds to permanent complete AV block. Atropine, which blocks parasympathetic tone, eliminates Mobitz 1 block, as does exercise. **Mobitz 2 block** occurs when there is an abrupt loss of a QRS without a preceding increase in the PR interval. This suggests disease in the His-Purkinje system which can suddenly progress to complete AV block. The presence of **Mobitz 2 block and a wide QRS complex** (>120 ms in duration) suggests serious, widespread degeneration in the conduction system below the AV node**.** Such patients have a high incidence of syncope and sudden death unless a permanent pacemaker (an electronic replacement for the conduction system) is implanted. An anterior wall myocardial infarction or degenerative disease are the usual culprits.



Second degree AV block with 2:1 conduction

Third degree ("Complete") AV Block

Complete failure of conduction from the atrium to the ventricle is called third degree AV block. This usually occurs after a large infarct or in the presence of severe degenerative disease, but rarely a child may be born with congenital CHB. Pacemaker therapy is always indicated unless a reversible cause such as ischemia or drug overdose can be identified.



Complete AV block

Important Principles of Bradycardias

Asymptomatic bradycardias are usually insignificant except in the presence of Mobitz 2 or third degree block. Symptomatic bradycardias need to be evaluated urgently. Search for potentially reversible conditions such as

1) drugs which depress automaticity or conduction (calcium channel blockers, beta-blockers, sodium channel blockers, digoxin)

2) ischemia

3) increased vagal tone

Conclusions

Arrhythmias are the leading cause of sudden death in this country. Understanding the principles of reentry is essential to understanding the therapy for tachycardias. Familiarity with the different types of bradycardias is essential for differentiating normal variants from potentially lethal conditions.