Basic Electrophysiology Part 1

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Module Objectives: Upon completion of this module the learner will be able to:

State in correct order the movement of the electrical impulse through the conduction system.
Describe the anatomy of the AV junction.
Name two types of accessory AV connections.
Define automaticity, excitability, conductivity, refractoriness, and contractility.
Differentiate depolarization from repolarization.
Discuss the refractory periods in ventricular conduction.
Describe the ventricular action potential.
Describe two types of arrhythmia mechanisms.
State the inherent or intrinsic rate of the various parts of the conduction system.

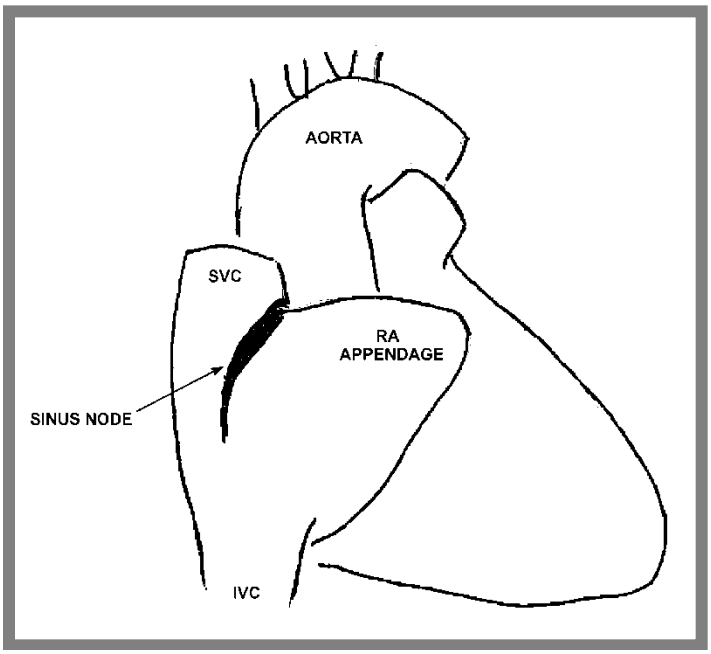
I. Specialized Conduction System of the Heart

The heart has a specialized conduction or electrical system. The conduction system is responsible for the movement of an electrical impulse from the top to the bottom of the heart, stimulating the heart so that it squeezes or contracts. Electrical impulses are normally initiated by the sinus or **sinoatrial (SA) node**. The SA node is located in the right atrium next to the superior vena cava (SVC). The SA node is a sizable structure with a crescent shape. The SA node is actually a band of tissue that lies between the spot where the SVC attaches to the RA and a portion of the RA called the RA appendage. It is more an epicardial structure than endocardial. There are no documented specialized connections linking the SA node to the **AV node**. The SA node is often referred to as the pacemaker of the heart because in the normal, healthy heart the SA node will "fire" automatically more quickly than any other part of the conduction system, at a rate between 60 – 100 beats per minute (though some texts state that 50 is the low value for normal and 110 is the upper limit).

The SA node is richly innervated by the sympathetic and parasympathetic (vagus) nervous system. The effect of sympathetic stimulation would cause the SA rate to increase, while the parasympathetic effect would cause the rate to slow.

The following figure shows the location of the SA node from a frontal view. It should be noted that the SA node is structurally a band of tissue. A rhythm disorder called **SA node reentry** can occur in which an impulse recirculates through portions of the SA node and causes a "sudden

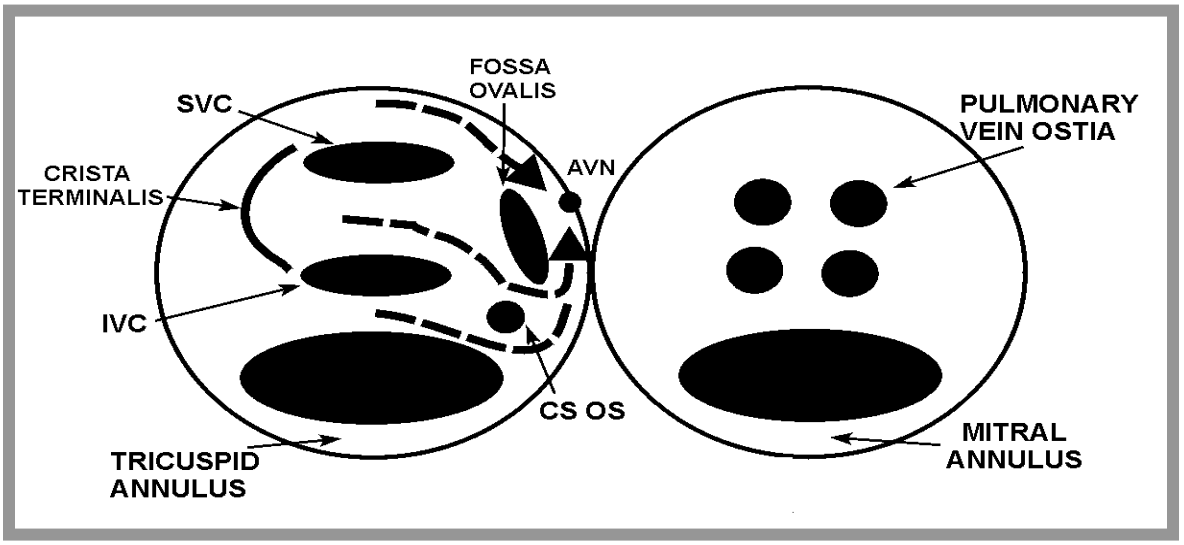
onset" sinus tachycardia at a rate of 100 – 150. The treatment for this rhythm problem may be an "**SA node modification**" via radiofrequency (RF) catheter ablation.



SA node from the frontal view

Atrial Floor Plan

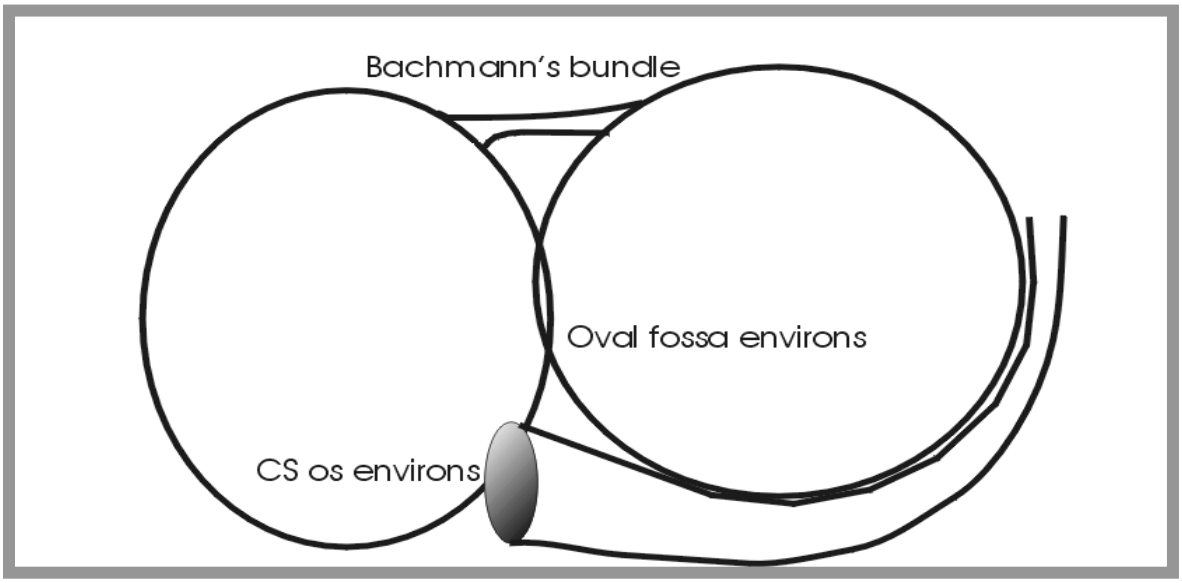
The routes of impulse conduction throughout the right and left atria are largely determined by the regions of ordinary atrial myocardium left over when you exclude the spaces taken up by holes (orifices of great vessels emptying into the atrium) and nonconducting borders (the annuli).



Atrial Floor Plan

The above diagram is a rough illustration of the atrial floor plan as if the atria were flattened into an equal area map and is intended to show how we might envision the "preferential" routes of conduction from sinus to AV node. In general, these routes contain muscle fibers which maintain a reasonably parallel alignment for a considerable distance, thereby contributing to the appearance of "preferential" conduction through these routes.

The actual area of contact between the right and left atrium is relatively small. The major connection to the left atrium is via the coronary sinus os and **Bachmann's bundle**, a rather bulky appearing muscular band coursing over the roof of the of the right atrium, connecting to the left above the interatrial septum.



Connections to the left atrium

The coronary sinus is the other pathway an impulse could follow from the right to the left atrium. The figure above, which looks down into the atria, highlights the Bachmann's bundle connection and the pathway an impulse could follow from the right to the left atrium along the coronary sinus.

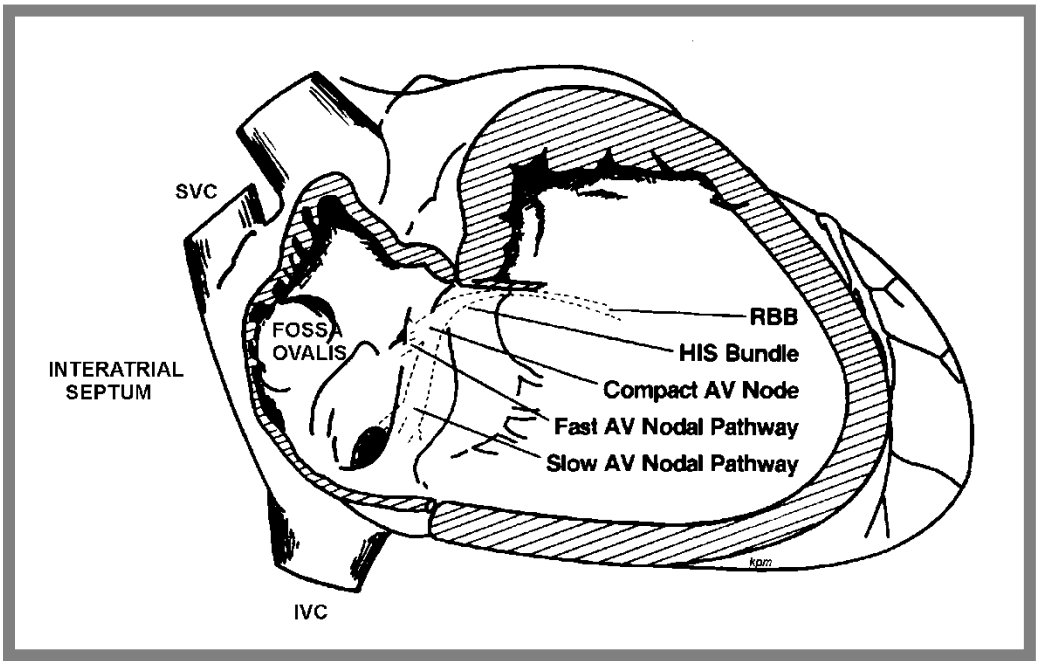
Historically drawings of the conduction system portrayed internodal tracts, displayed in such a manner as to suggest that they bear some resemblance to the bundle branches/Purkinje system. Such anatomic suggestions, including the inaccurate representation of the AV junction, are still found in current textbooks.

The AV Junction

The **AV junction** could be the most interesting structure within the heart. As the sole normal connection between atria and ventricles, it is responsible for introducing complexity to the cardiac rhythm. Without it, the classification of arrhythmias would be rudimentary, including only tachycardia, bradycardia and fibrillation -with emphasis on the ventricles since the atria would

assume a secondary importance. Because of the AV junction, there are a wide variety of AV and VA interactions of single beats or rhythms arising from the sinus node, atria and ventricles that impart complexity to rhythm analysis. The AV junction is located at the anterosuperior portion of the interatrial septum near the tricuspid annulus. The inherent rate for the AV junction to pace the heart is 40-60 beats per minute (slower than the sinus rate of 60-100). The AV junction is capable of being a 'backup' pacemaker should the SA node fail. The AV junction is innervated by both sympathetic and parasympathetic fibers, as the SA node is. Electrical impulses which arrive in the AV junction are slowed briefly to allow atrial contents to empty into the ventricles prior to ventricular contraction.
The AV junction can be considered as having three component parts:

1. the compact AV node
2. the anterosuperior approaches to the node located in the anterosuperior interatrial septum (**fast AV nodal pathway**)
3. the posteroinferior approaches extending from the compact node to the coronary sinus os (**slow AV nodal pathway**)



AV nodal anatomy as seen from the RAO view

Terminology varies depending on who you read, but the term "junction" is best because it allows distinction between the compact AV node and its atrial approaches while at the same time minimizes confusion about what is meant by the "node". Some authors may be referring to the entire AV junction as the AV node while others may infer only parts.

The fast and slow pathways converge onto the compact AV node in a way that promotes proper orientation for conduction into and through the node. The compact node is the primary site of conduction delay, and is responsible for the PR interval. Normal AV conduction proceeds via the fast pathway, termed fast because nodal conduction is quicker than when it conducts through the

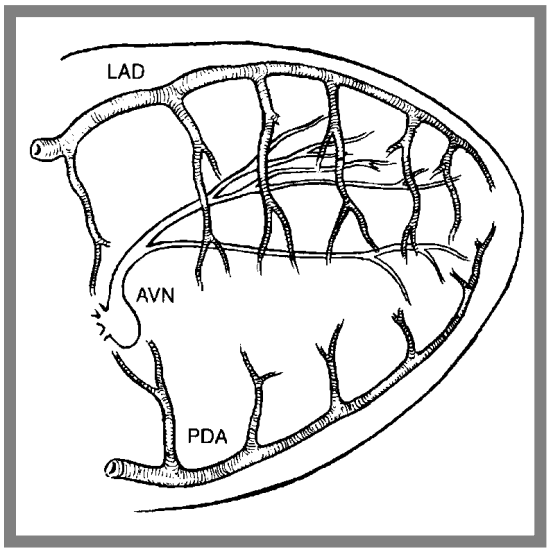
node via the slow pathway. Thus, the normal PR interval of 0.12-0.20 sec reflects preferential conduction over this faster route.

Although the spectrum of AV conduction behavior in the majority of patients suggests two pathways, hence the well-known reference to "dual physiology", many variations on the theme exist. At one extreme, there may be no detectable evidence for a slow pathway (fast pathway dominance) while at the other extreme multiple slow pathways can be found. Fast pathway dominance is unusual, accounting for less than 5-10% of patients. An arrhythmia called **AV node reentry (AVNRT)** can occur when an impulse travels down one pathway and up the other in a circle causing a ventricular rate of about 150-250 per minute.

His-Purkinje System

The **bundle of His** represents the beginning of the conduction system in the ventricles. The bundle of His divides into two main branches, the **right bundle branch** and the **left bundle branch**. The right bundle branch is long and slender and delivers the electrical impulse to the right ventricle. A blockage of impulse through this structure is called a **right bundle branch block (RBBB)**. During RBBB an impulse must travel down the left bundle and cross the ventricular septum to provide stimulation to the right side so that the right ventricle will contract.

The left bundle branch (LBB) is broad and fanlike. The LBB delivers the electrical impulse to the ventricles. In most individuals there are three divisions (fascicles) to the LBB. These divisions are the **posteroinferior**, the **anterosuperior** and the **septal**. There are identifiable EKG criteria for a blockage of the anterosuperior or posteroinferior divisions. Blockages of these divisions are called left anterior hemiblock and left posterior hemiblock. The following figure shows the location of this left bundle branch toward the upper right from the AV nodal area and the right bundle branch below. The LAD is the left anterior descending coronary artery and the PDA is the posterior descending artery.



The bundle branches

The last stop for the electrical impulse in the ventricle is the **Purkinje** fibers. The ventricles are then stimulated sufficiently to contract. The ventricles may also act as a backup pacemaker for the heart if the areas above fail; however the rates would be in the range of 10-40, depending on which site served as the backup.

Self-Study Questions:

1. Describe the shape of the SA node.
2. What is SA node reentry?
3. What is the basic or inherent rate for the SA node?
4. What is Bachmann's Bundle?
5. How does the electrical impulse travel?
6. What is the inherent rate for the AV junction?
7. Describe the fast and slow AV nodal pathways.
8. How does AV node reentry occur?
9. Describe the conduction system components that are found in the ventricles.
10. What is right bundle branch block?
11. What is the most common treatment for AV node reentry?

II. Accessory Atrioventricular Connections (Pathways, Bypass Tracts)

Accessory atrioventricular connections (also known as pathways or **bypass tracts**) are connections between the atria and ventricles which are not present in everyone. They provide an alternative route for the travel of the electrical impulse instead of the AV junction. Thus these bypass tracts may be responsible for alterations in QRS width or changed P wave/QRS relationships.

Rapidly Conduction Accessory AV Pathways

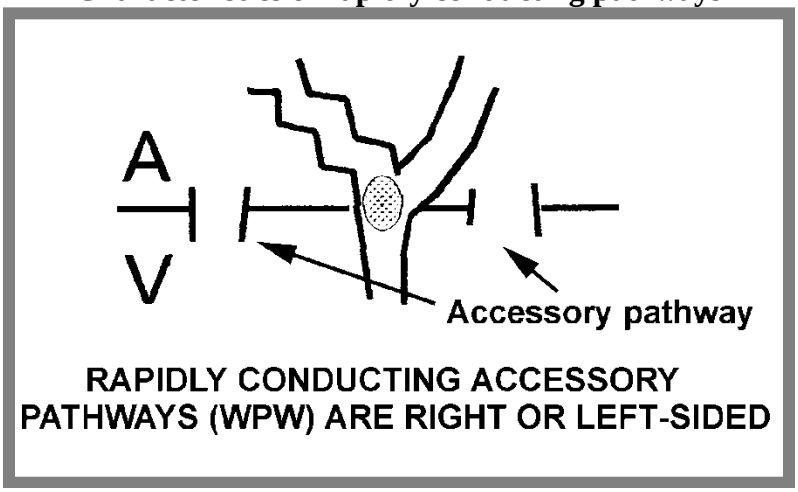
Rapidly conducting accessory AV pathways are the pathways which connect the atria and ventricle in the **Wolff-Parkinson-White (WPW) Syndrome**. These pathways exist because of an abnormality in development of the AV groove of the heart in utero. The AV groove separates the atria from the ventricle electrically, except for the AV node, thus making impulse travel from the atria to ventricle orderly.

With WPW, there are stray pieces of muscle which still connect the atria to the ventricle, thus allowing an alternative route for the electrical impulse. This may result in the triad of short PR interval, wide QRS and a delta wave (gradual incline at the onset of the QRS), which 'preexcites' the ventricle. Or its presence may provide the pathway for an impulse to circulate down the AV node and up the pathway, or down the pathway and up the AV node. This is called a reentrant tachycardia (**AV reentry tachycardia, AVRT**, or **circus movement tachycardia**).

Rapidly conducting accessory pathways exist on either the left or right side of the heart. They are usually capable of **antegrade** and **retrograde** conduction. About 75% of patients exhibit **preexcitation** on a 12 lead ECG during normal sinus rhythm. About 25% will conduct retrograde only (concealed accessory pathways) and less than 2-4% conduct antegrade only.

- ❖Exist on left or right side
- ❖Rapidly conducting
- ❖Usually capable of antegrade and retrograde conduction
- ❖Preexcitation may or may not be present on 12 lead ECG

Characteristics of rapidly conducting pathways



Rapidly conducting accessory pathways (WPW) are right or left-sided

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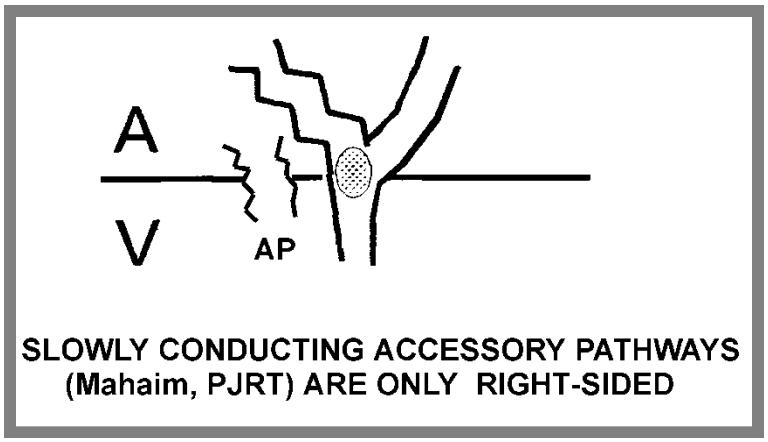
Slowly Conducting Accessory AV Pathways

Slowly conducting accessory AV pathways exist only on the right side of the heart. Those that are capable of only **antegrade** conduction are called **Mahaim fibers**. Preexcitation will be evident on the 12 lead ECG only during tachycardia. The clinical syndrome is known as **Mahaim PSVT**.

The slowly conducting accessory AV pathways capable of only retrograde conduction are responsible for a permanent form of **junctional reciprocating tachycardia (PJRT)**. With this pathway, preexcitation is never evident on the 12 lead ECG in normal sinus rhythm or during PSVT. The slowly conducting accessory AV pathways are diagramed below.



Slowly conducting accessory pathways (Mahaim, PJRT) are only right-sided

- ❖Slowly conducting pathways
- ❖Located on right side
- ❖Conduct antegrade only
- ❖Preexcitation on 12 lead ECG only during PSVT

Summary of characteristics for Mahaim fibers

- ❖Slowly conducting pathways
- ❖Located on right side
- ❖Conduct retrograde only
- ❖Preexcitation never evident on 12 lead ECG

Summary of characteristics for PJRT pathway

Self-Study Questions

1. What is an accessory pathway?
2. What are rapidly conducting accessory pathways called?
3. Describe the aberrant anatomy that is known as WPW.
4. What are the three abnormal findings that may be present on the EKG complex in WPW during normal sinus rhythm?

5. On what side(s) (L or R) of the heart will WPW pathways be found?
6. Describe antegrade and retrograde conduction.
7. Name two slowly conducting pathways.
8. Describe preexcitation.

III. Properties of Cardiac Function

Five properties of cardiac function should be reviewed to adequately understand electrical and mechanical events. The first of these is **automaticity**. Automaticity is the ability of certain cells in the heart to initiate electrical impulses spontaneously. This property exists in all cells known as pacemaker cells. Areas of the conduction system thus capable of initiating impulses includes the SA node and atrium, the AV junction and the His-Purkinje system. Normal automaticity for each of these regions would thus elicit the following rates: SA node 60-100 bpm; AV junction 40-60 bpm; and His-Purkinje system 10-40 bpm. Altered automaticity occurs when these areas perform more slowly (depressed automaticity) or more rapidly (enhanced automaticity). These abnormal situations may be the cause for some of the arrhythmias that we see.

Excitability is the ability of cardiac cells to respond to stimulation. A cell must be properly oxygenated and have electrolytes and ph within normal range to respond in a normal fashion. When an impulse makes its way to an individual cell, the impulse must be strong enough to change a cell from predominately electrically negative to electrically positive. The success of the impulse to achieve this is also determined by its ability to measure up to the "threshold level" for that cell. The threshold level is analogous to a bar over which the impulse must jump. If the threshold level or bar is lowered, weaker impulses may be able to affect the cell and depolarize it. A lowering of the bar occurs with low serum potassium levels and low tissue oxygen levels. Thus during these situations arrhythmias are allowed to occur which one normally would not see. A raising of the bar on the other hand, may prohibit even the normal impulse from the SA node from causing the cell to depolarize. An example of this would be the situation caused by high serum potassium levels, which in the extreme could cause asystole.

Automaticity -	ability of certain cells in the heart to initiate electrical impulses spontaneously.
Excitability -	ability of cardiac cells to respond to stimulation.
Conductivity -	the ability to transmit an impulse through specialized conduction system and atrial/ventricular muscle.
Refractoriness-	inability to undergo repeat stimulation until after a certain period of time has elapsed.
Contractility -	ability of the fibers to shorten when stimulated, resulting in muscle contraction (pump action).

Conductivity is the ability to transmit an impulse through the specialized conduction system and the atrial and ventricular musculature. Conductivity is analogous to traveling or the taking of a trip. We measure the 'trip' of an impulse in three different parts of the ECG complex. The first 'trip' we measure is that taken from the SA node to the ventricles, and the time that trip takes is normally **0.12 to 0.20 seconds** and is known as the PR interval. The second trip is that taken through the ventricles, and corresponds with the QRS width. That trip is usually **less than 0.12 seconds**. The third trip that we measure is the combined trip of ventricular depolarization and repolarization, and this is known as the QT interval. The QT interval normal varies for men and children versus women. A general yardstick for measuring however, is that normal is probably half of an individual's R to R interval.

Some conditions that may alter conductivity include increased sympathetic or parasympathetic influence, disease related factors such as ischemia, cardiomegaly, calcification, congenital disorders, fibrosis, neurological disorders, tumors, metabolic disorders, myocarditis, and aging. Medications such as digoxin and antiarrhythmics may also cause conduction problems.

Refractoriness is the inability of a cell or tissue to undergo repeat stimulation until after a certain period of time has elapsed. This means that once a cell is stimulated and responds to an impulse, the cell must recover before it can be stimulated again. State of refractoriness is a moving target, changing from moment to moment. We ask where in the cycle we are at any given point.

Finally, **contractility** is the ability of cardiac muscle fibers to shorten when they are stimulated, resulting in muscle contraction (pump action). Cardiac muscle cells must be healthy with normal elasticity to make pumping efficient. Muscle cells that are dilated and that have experienced excessive stretch will not contract in a normal fashion and this pump action will be decreased. This is the situation we see with heart failure.

We can use drugs or devices to alter contractility. Bi-ventricular pacing is an effort to enhance contractility. Giving inotropic drugs enhances contractility. Sometimes we give drugs to depress contractility. For example, in intractable angina we may give beta blockers or calcium channel blockers to decrease contractility and therefore reduce pain. Also, for those with hypertrophic cardiomyopathy beta blockers and calcium channel blockers reduce contractility, causing the left ventricular muscle to relax a bit and decrease aortic valve obstruction, thus allowing more blood to be ejected with each contraction.

Self-Study Questions

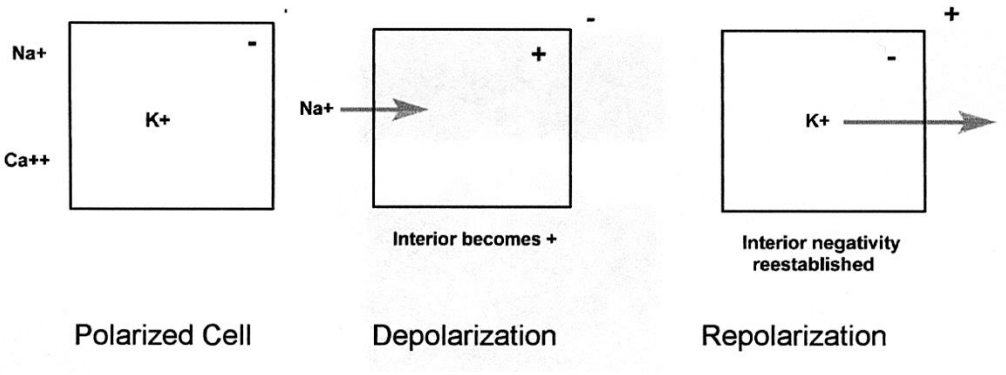
1. Describe the principle of automaticity and its relationship to the SA node.
2. In order for a cell to be excitable, what must be true?
3. Name the three parts of the EKG complex in which conductivity may be measured.
4. Define refractoriness.
5. Name a diagnosis in which contractility is impaired.

IV. Depolarization and Repolarization

Cardiac cells can exist in three major states electrically. The first is that of the **polarized cell**. This is the cell at rest. The interior of the cell is negative with respect to the outside. Potassium and sodium are maintained with normal distribution.

The next cell state is **depolarization**. With depolarization the electrical impulse arrives at the cardiac cell and the cell is stimulated. Stimulation will occur appropriately if tissue oxygenation and electrolytes are at normal levels. With depolarization, sodium enters the cell, producing an inward electrical current. Calcium enters the cells soon after. This process proceeds from the inner muscle layer, the endocardium, to the outer layer, epicardium, and begins with the ventricular septum. The impulse moves from cell to cell as depolarization occurs successfully.

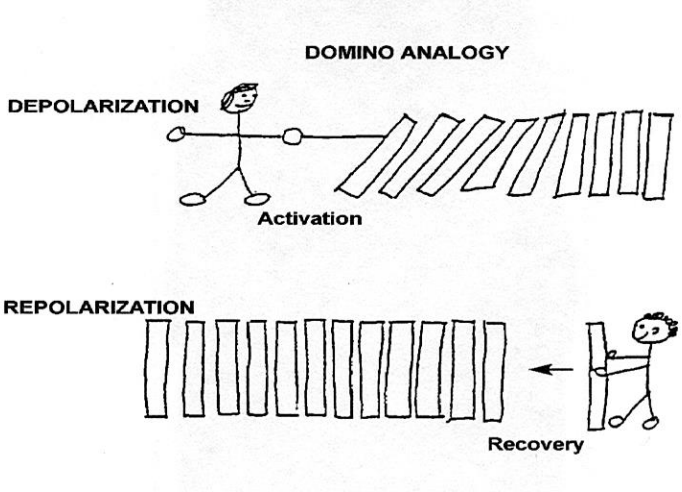
The third cell state is **repolarization**. With this process the cell returns to the resting state. Potassium leaves the cell and an outward electrical current is produced. Eventually the sodium-potassium pump plays a roll and the polarized state is again reached.



The three electrical states of the cell

Notice that we are speaking of sodium, potassium, and calcium. Three of the major classes of antiarrhythmic drugs act by blocking these very electrolytes. The drug actions occur at the cellular level. These drugs therefore alter depolarization and repolarization in different ways.

The next figure further explains the principles of depolarization and repolarization. In the Domino Analogy one starts with a group of dominos stood on end, representing polarized cells. The depolarization or activation man arrives and knocks over the first domino which in turn knocks down the second, etc. The recovery or repolarization man arrives and begins to stand the dominos up again. The key point to understand here is that activation or depolarization cannot occur again until recovery has taken place.



The domino analogy

The other analogy for depolarization and repolarization that has been made is that of flushing a toilet (this one you'll remember!!). Flushing corresponds with depolarization. The filling of the tank corresponds with repolarization. One cannot flush the toilet again until an adequate amount of water has filled the tank.

V. Refractory Periods

Refractoriness refers to the inability of cells to undergo repeat stimulation until after a certain period of time has elapsed. With this elapsing time are certain further periods to note. These can be seen in the next figure.

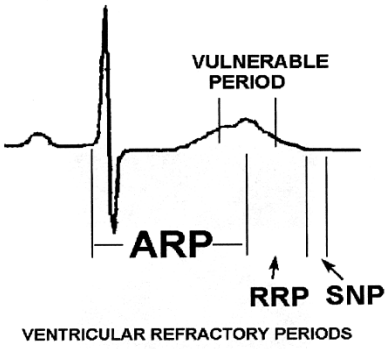
The **absolute refractory period** (ARP) is a relatively long period of time following excitation during which cells cannot respond to another stimulus, regardless of its strength. One remembers this through the idea that *'absolutely nothing else'* can happen here. The ARP period roughly corresponds to the duration of systole.

The **relative refractory period** (RRP) is a narrow window of time near the beginning of the ARP during which stimulus strength must be above normal to cause depolarization and the response elicited will be less than normal (not all the dominos are back up).

The **vulnerable period** is not a refractory period, but is a window of time during the refractory phase in which the heart is prone to develop fibrillation in response to a premature beat delivered at that time. This roughly corresponds to the top of the T wave. When a PVC hits on the top of the T and causes ventricular fibrillation, this is called R or T phenomenon. Another situation which may also cause this is when a pacemaker is improperly sensing and delivers a pacing stimulus on a T wave and causes fibrillation. A third way in which this may occur would be in the setting of a cardioversion in which the synchronized button is not pressed and instead defibrillation occurs on a T wave, thus sending a patient with an atrial flutter or atrial fibrillation into a ventricular fibrillation. Synchronized cardioversion ensures that electricity is delivered only within the period of the QRS, thus protecting against the accidental delivery of current on

the T wave. With cardioversion you also need to check the height of the T wave on your monitor screen before delivering the shock, because if the T wave is as tall as the QRS, a cardioversion shock could be delivered on the T wave. If this equal height exists, you need to put the patient in a lead where the QRS is the tallest complex.

Finally, the **supernormal period** (which is really not a refractory period), is an interval of time during the latter portion of the T wave when it may be possible for a subthreshold stimulus (premature beat) to conduct.



Self-Study Questions

1. Describe the polarized cell.
2. What happens during depolarization?
3. Define repolarization.
4. Name electrolytes involved with depolarization and repolarization.
5. Will the ventricles respond to another impulse during the absolute refractory period?
6. Why may the vulnerable period be a time for concern?
7. What is the super-normal period?

VI. Action Potential 101

The next figure demonstrates the activities within a single ventricular cell during depolarization and repolarization. The upper figure shows the various numbered phases of electrical activity within the cell. The lower figure corresponds with the phases and tells the story of electrolyte movement within these phases. It should be noted that potassium appears to be quite active throughout these phases. This is because there are at least seven different potassium currents which have been identified, each behaving in a slightly different manner.

The upper figure (the action potential diagram) contains a horizontal line called the threshold potential. This line defines the strength an impulse must possess in order to successfully depolarize a cell. A weak impulse will not be able to 'jump' this high. The height of the line

may be lowered with low serum potassium level or low tissue oxygenation levels, allowing weak impulses to cause ectopic beats or rhythms. A high serum potassium will 'raise the bar' and possibly prevent even a normal SA node impulse from depolarizing the cell, leading to asystole.

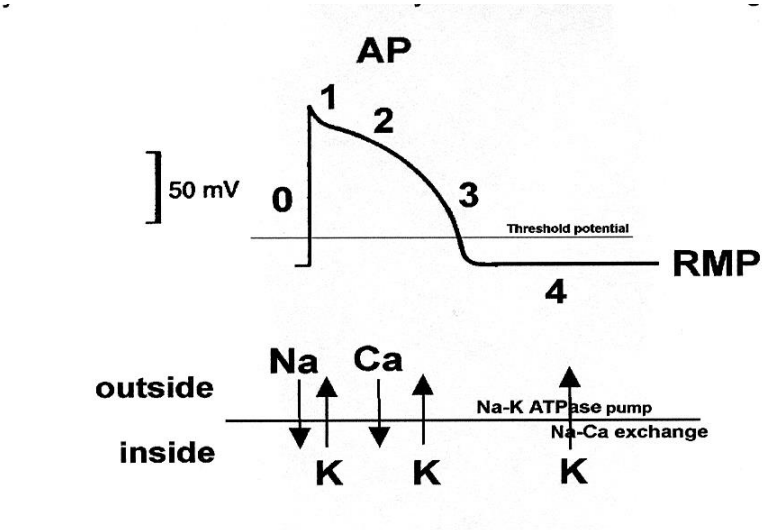
The baseline level of the action potential curve is -90mV until depolarization occurs, at which time the cell becomes electrically positive. The various numbered phases which will be described below are referred to in discussion of antiarrhythmic drugs. Sodium channel blockers alter phase 1 of the action potential. Beta blockers and calcium channel blockers have an effect on phase two of SA node and AV node action potentials. Potassium channel blockers exert changes on the various potassium current activities.

Phase 0 of the action potential is the phase of rapid depolarization. During this phase the cell is stimulated and the cell membrane becomes more permeable to sodium ions. Fast sodium channels open and sodium rushes into the cell.

In **phase 1**, known as the period of initial repolarization, there is a brief very rapid attempt to return to resting membrane potential. During this time sodium influx ends in fast channels and potassium begins to flow out of the cell.

Phase 2 is called the plateau phase. During this phase there is an influx of calcium and sodium through the slow channels (though this sometimes also occurs during phases 0 and 1). This influx is responsible for the refractory period during which the depolarized state is maintained. Calcium also plays a role in maintenance of normal heart muscle contractibility.

Phase 3 is known as the phase of rapid repolarization. Membrane potential is driven to the negative level due to the increased loss of intracellular potassium.



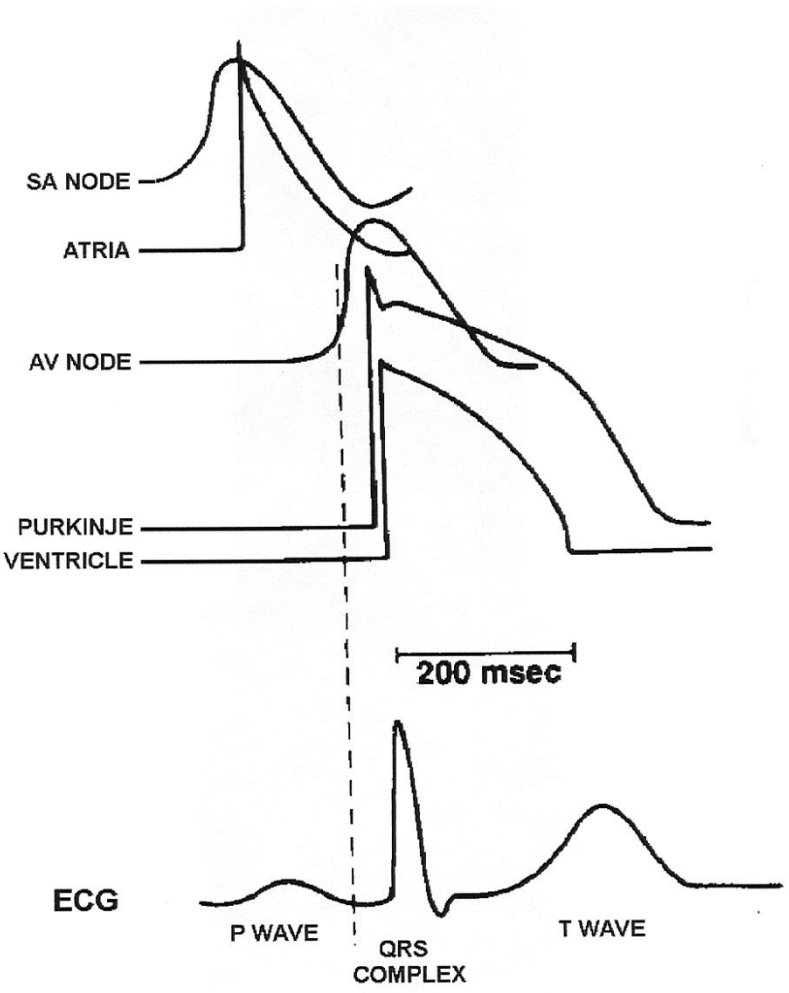
Action potential of a ventricular cell

Phase 4, the quiescent period, is the time when the cell returns to the resting membrane potential level (-90 mV). During this period, normal distribution of sodium and potassium are restored.

The sodium potassium pump plays a roll during these latter phases. ATP is required to fuel this pump and an adequate level of serum magnesium insures that the pump performs properly. Repolarization will be prolonged if the pump malfunctions, thus prolonging the QT interval on the ECG. This may lead to a potentially lethal arrhythmia, torsade de pointes, a polymorphic ventricular tachycardia. Treatment for this arrhythmia is intravenous magnesium.

Local Variations in Action Potentials

The next figure contrasts the action potential curves for each type of pacemaker cell in the heart. Notice that each curve has a somewhat different contour. The phases 0-4 occur with each of these. The SA node and atrial cells depolarize early and repolarize early, thus making these faster areas normally in control of the depolarization impulse, with a rate of 60-100 bpm.



Local variations in action potentials

The table below lists the various currents or channels operational during the cardiac cycle. Notice the various potassium related currents and the timing of their efflux.

- I = current

- I_{na} (slow, medium, fast)
  - -Inward current
  - -Present in atrial, HPS, ventricular cells
  - -Targeted by Class I drugs
    - •Slow conduction
    - •Prolong refractory period

- I_{ca}
  - -Inward current, two types (L and T)
  - -L-type present in SA and AV nodal cells
  - -Target by Class IV drugs

- I_f funny current – automaticity

- I_k
  - -Outward current
  - -Present in atrial, AV nodal, HPS and ventricular cells
  - -Many types of K+ current
    - •Transient outward I_{to}
    - •Delayed rectifier (exist during the plateau)
      - -I_{kr} (rapid) seen in slower heart rate
      - -I_{ks} (slow) seen in faster heart rates

- I_{k1} dominant background K outward current

Currents operational during the cardiac cycle

Self-Study Questions

1. Describe the threshold potential and its importance.
2. What is the role of potassium during the ventricular action potential?
3. What occurs during phase 0 of the action potential?
4. What is the baseline mV level of the ventricular action potential?
5. What occurs during phase 1 of the action potential?
6. What electrolyte is instrumental in causing the activity during phase 2 of the action potential?

7. Describe the activities that occur during phase 4 of the ventricular action potential.
8. Discuss the implications of the difference in action potential timing and shape among the various parts of the conduction system.

VII. Mechanisms of Arrhythmias

There are two major categories of rhythm problems:
those that are fast
those that are slow

Slow

The **slow** rhythm problems are called bradyarrhythmias or **brachycardias**. Two types of mechanisms are said to be responsible for causing bradycardias:

Failure of impulse formation (eg. The SA node fails or automaticity is depressed). These situations may lead to an asystolic situation, presence of a backup rhythm, slower rhythms from the AV junction or ventricles, or the presence of sinus bradycardia.

Failure of impulse propagation. With this problem, the impulse does not conduct normally to the ventricles. Example: AV block, when the impulse is prevented from successfully traveling through the AV node and ventricular rate slows.

Fast

Rhythm abnormalities that are **fast** are called **tachycardias**. There are three mechanisms responsible for tachycardias: **altered automaticity, reentry, and triggered activity**.

These mechanisms are not mutually exclusive: eg. triggered beats can:
Engage a pre-existing reentrant circuit
Create arcs of functional block, generating substrate for reentry

Automatic Rhythms

Automaticity is the ability of cells to 'automatically' initiate an impulse. With **altered automaticity** there is a gradual increase of rate which takes place (a warming up), and then a gradual decrease or 'cooling down'. There may be an 'enhanced normal' as in sinus tachycardia, or an enhanced abnormal, as in atrial tachycardia. If there appears to be a 'warm-up' period, this suggests an automatic focus. The arrhythmia may be transiently suppressed with overdrive, and then re-warm-up. The causes of altered automaticity are often metabolic, and are never due to structural abnormalities of the heart. These causes include acute cardiac ischemia, hypoxemia, hypokalemia, hypomagnesemia, acid-base disorders, increased sympathetic tone, and sympathomimetic agents. Tachycardia caused by altered automaticity should be treated by

treating the identified cause; antiarrhythmics are not effective. These tachycardias cannot be induced by programmed pacing techniques and cannot be studied in the EP lab.

Reentry

Reentry is the most common and most important mechanism for tachyarrhythmias. This mechanism is responsible for many deaths each year. Reentry occurs when an impulse is delayed long enough within a pathway of slow conduction so that it is still active when the surrounding myocardium repolarizes. The same impulse then reenters the surrounding tissue and produces another impulse. Other names are circus movement, reciprocal or echo beats, reciprocating tachycardias. This arrhythmia mechanism can be studied in the EP lab.

The basic requirements for reentry to occur include 2 pathways, and heterogeneous electrophysiologic properties of the pathways that allow conduction to block in one limb and to propagate in the other. There are two general categories of reentry pathways: anatomic and functional.

In **anatomic reentry**, an excitation wave passes around an anatomic obstacle or obstruction. This includes an **excitable gap**. The excitable gap is the excitable myocardium that exists between the head of the reentrant wavefront and the tail of the preceding wavefront. Excitation at any point in gap region will find enough sodium channels available to initiate a propagating impulse. The circuits are actually highly heterogeneous. Conduction velocity, refractoriness, and duration of the excitable gap can all vary along 'the' pathway (which itself may wobble). Examples: AVNRT, Purkinje, BB, Tricuspid Annulus, AV node and AP (AVRT), VT arising in/around remote MI

In **functional reentry** there are functional lines of block, spirals, scrolls, and figures-of-eight. The reentrant circuits may be continuously variable (AF, VF), or may 'settle' into specific pathways (eg atrial flutter). Transient (small) excitable gaps may be demonstrable even in AF and VF

Causes of reentrant pathways include accessory pathways, areas with patches of scar tissue, and conditions which cause decreased membrane potentials, such as ischemia, hypoxemia, hyperkalemia, and pharmacologic agents. Common arrhythmias associated with reentry include WPW-AV reentry tachycardias, PVC's, VT, ventricular bigemini, and PSVT. The most common types of reentry are AVNRT, AVRT, atrial flutter and VT.

1. Initiating impulse, normal sinus or ectopic
2. An area of slow conduction
3. An area of one-way conduction for the return route.

Conditions required for reentry

Triggered activity

Triggered activity is the third cause of tachycardias. Triggered activity has some features of both automaticity and reentry and can be difficult to diagnose in the EP lab. Triggered activity is repetitive firing of a group of abnormal cells brought on by or provoked by one of a series of premature impulses. It can be reproduced in the EP lab by burst pacing. The cellular abnormality is called an afterdepolarization (either early or delayed).

Afterdepolarizations are abnormal oscillations of membrane potential that attend or follow the action potential and depend on preceding transmembrane activity for their initiation. These may be early or delayed. Stimulation of a myocyte (or the heart) may, under pathologic conditions, then give rise to these pathologic, non-driven upstrokes. If these afterdepolarizations are strong enough to engage the rapid sodium channels, another action potential can be generated. In this way it resembles automaticity. On the other hand it resembles reentry by virtue of the fact that it is not spontaneous, and must be provoked by premature beats (inducible by programmed stimulation).

There are two types of afterdepolarizations: early and delayed.

Early Afterdepolarizations (EADs). EADs interrupt phase 3 or retard repolarization.
The arrhythmias are incited by action-potential-drugs (QT prolonging). They are exaggerated by slow rates and low K. Rapid rates, K, and Mg2 will retard or stop these arrhythmias. The arrhythmia mechanism is a net increase in inward plateau current. The representative arrhythmia of EADs is **Torsades**.

Delayed afterdepolarizations (DADs). DADs are oscillations that occur after completion of the action potential (during phase 4). This may be incited by digitalis or catecholamines (drugs that cause increased intracellular calcium). Rapid rates will cause an exaggerated effect. Calcium channel blocking agents are helpful for control or termination. The mechanism for DAD arrhythmias is an overload of intracellular Ca2+. Representative arrhythmias include those of digitalis toxicity, ischemia, and some genetic syndromes (catecholaminergic VT).

The differentiation of triggered activity from reentry may be challenging. Triggered activity is like automaticity in that it has a warm up and cool down period. Reentry, however, is more likely the cause with underlying structural cardiac disease present.

Self-Study Questions

1. Define bradycardias and tachycardias.
2. Define altered automaticity, reentry, and triggered activity.
3. How do the above three mechanisms differ?
4. What arrhythmia types might you see with each arrhythmia mechanism?
5. Contrast early afterdepolarization with delayed afterdepolarization.
6. What are the two mechanisms responsible for bradycardias?

References:

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