Cardiology

Self Learning

Package

Module 3: Cardiac Arrhythmias: Mechanisms of Arrhythmias, Atrial, Ventricular, Conduction and ST Changes.

CONTENT

Module 3: Cardiac Arrhythmias: Mechanisums of Arrhythmias – Atrial, Ventricular, Conduction and ST Changes
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<u>INTRODUCTION</u>

Welcome to the ECG self learning package module 3. Patients presenting with the symptom of palpitation form a large proportion of admissions into the departments of cardiology. In the great majority of cases the correct diagnosis can be determined at the initial consultation after careful analysis of the history, findings at clinical examination, and 12-lead ECG. This package will provide an overview of most cardiac arrhythmias and the mechanism that causes them.

This package will provide information on in regards to the different arrhythmias and the abnormalities that causes them such as electrical instability in the myocardial cell membrane, the ability of cardiac cells contract twice, although they only have been activated once and the propagation through issues rather than abnormalities in physiology of individual cells.

The goal of this module is to review:

- Arrhythmia etiology
- Arrhythmia pathophysiology
- The different mechanisms of Arrhythmias
- Defining the different types of arrhythmias.

Learning outcomes for this module are:

- To identify the different clinical manifestations of arrhythmias.
- To state what is define as arrhythmia mechanisms.
- To state what is define as normal automaticity
- To state what is define as abnormal impulse imitation.
- To state what is define as triggered rhythms.
- To state what is define as abnormal impulse conduction
- To identify the arrhythmias in relation to each mechanisms.
- To diagnose arrhythmias.

HOW TO USE THIS SELF LEARNING PACKAGE

Follow the step outline to complete the Self Learning Package.

1) Read the two journal articles provided.

2) Read the information provided in the Self Leaning Package regarding arrhythmias.

3) Complete Question at the end of the package.

4) Diagnosing rhythms Strip provided at the end of the package.

5) Complete the Evaluation form

6) Once the above are completed return to your Clinical Nurse Educator or Clinical Nurse Specialist for marking.

Following the completion of this module, you will receive **10 hours** professional development hours, which will be credited to you education data base (CDHB).

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Too fast, too slow, too ugly:
Dysrhythmias that every

Learn how to recognize an abnormal cardiac rhythm and intervene appropriately. By AnneMarie Palatnik, RN, APN-BC, MSN

CE
2.5
ANCC
CONTACT HOURS

CONTINUOUS CARDIAC MONITORING is used in many different clinical settings, so you need a basic knowledge of cardiac rhythms no matter where you work. In this article, I'll review normal cardiac anatomy and electrophysiology, then describe cardiac rhythms that are too fast or too slow, or just plain ugly. I'll also discuss treatments for each significant dysrhythmia.

When caring for a patient with a dysrhythmia, remember to always treat the patient, not the monitor. Assess and support the patient's airway, breathing, and circulation, and obtain vital signs including pulse oximetry. Administer supplemental oxygen as indicated, ensure patent I.V. access, obtain a 12-lead ECG, and notify the patient's healthcare provider.

Power and the pump

As you know, the atria act as blood reservoirs; about 70% of total cardiac output (CO) flows passively from the atria to the ventricles. The remaining 30% passes to the ventricles when the atria contract, known as *atrial kick*. In some atrial dysrhythmias, atri-

al kick is lost, which decreases CO and can cause hypotension. In addition, the blood that pools in the atria can form thrombi and travel to other parts of the body, including the brain, where a thrombus can cause a stroke.

The ventricles are two separate pumps. The right ventricle pumps blood into the pulmonary circulation for oxygenation in the lungs, the left ventricle pumps the oxygenated blood into the systemic circulation for travel to the rest of the body.

The cardiac conduction system provides the power for the pumps. Electrical impulses generated by the exchange of ions—primarily potassium, sodium, chloride, and calcium—cause depolarization (the electrical event) and myocardial contraction or systole (the mechanical event). In repolarization, which corresponds to diastole, the resting phase of the cardiac cycle, ions are again exchanged, "resetting" the heart electrically so it's ready for the next depolarization and contraction.

Specialized myocardial cells called *pacemaker cells* spontaneously generate electrical

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nurse should recognize

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impulses. Electrical conducting cells in the heart then pass these impulses to the next cell. As the cardiac cells conduct the electrical impulses, filaments in the cells shorten, causing mechanical contraction.

The pathway that the electrical impulse travels is called the heart's conduction system. (See *It's electric! Understanding cardiac conduction.*) Normally, the impulse is initiated in the sinoatrial (SA) node, the heart's natural pacemaker. The SA node is located at the junction of the right atrium and superior vena cava, just above the tricuspid valve, and normally generates 60 to 100 impulses/minute. Numerous other potential pacemakers can be found along the conduction system, and any one of these can take over as the heart's pacemaker if the SA node fails to fire, or if it fires but the electrical impulses are too slow or blocked. For example, if the SA node fails to fire, the atrioventricular (AV) node can kick in and become the heart's pacemaker. However, the AV node generates only 40 to 60 impulses/minute. If both the SA and AV nodes fail to fire, or if the impulses are too slow or are blocked, the Purkinje fibers in the ventricles will kick in as the heart's pacemaker, but at a much slower rate of (20 to 40 beats/minute).

A change in heart rate, whether too fast or too slow, can decrease the patient's CO, which is determined by multiplying heart rate by stroke volume (the amount of blood pumped out of the left ventricle with each beat). For example, a heart rate greater than 100 beats/minute shortens diastolic

When a cardiac rate, rhythm, or both deviate from normal, your job is to determine the significance of this deviation.

filling time and can decrease CO in some patients.

Doing the wave
The heart's electrical activity can be represented graphically on an ECG (see illustration below). As you know, one cardiac cycle normally consists of a P wave, a QRS complex, and a T wave, and the intervals and segments between these waves. Let's take a closer look:
• The *P wave*, the first wave, represents atrial depolarization (the electrical impulse as it travels through the atria). The P wave should be positive in leads II, III, and aVF; positive, isoelectric, or biphasic in leads I and aVL; inverted in lead aVR; and biphasic in lead V1. You should see one P wave before every QRS complex.
• The *QRS complex* represents ventricular depolarization. The normal QRS duration is between 0.06 and 0.10 second.

• The *T wave*, which immediately follows the QRS complex, represents ventricular repolarization.
• The *PR interval*, measured from the beginning of the P wave to the beginning of the QRS complex, represents the time the impulse takes to travel from the SA node to the ventricles. A normal PR interval is 0.12 to 0.20 second.
• The *ST segment* is the straight line between the end of the QRS complex and the beginning of the T wave. This segment represents the time from the end of ventricular depolarization to the beginning of ventricular repolarization.
• The *QT interval*, which includes the QRS complex, the ST segment, and the T wave, represents the time from the beginning of ventricular depolarization to the end of ventricular repolarization. Because the QT interval varies with heart rate, a corrected QT interval (QTc) is calculated by dividing the QT interval by the square root of the R-R interval. Normal QTc intervals are less than 0.43 second in men and less than 0.45 second in women.
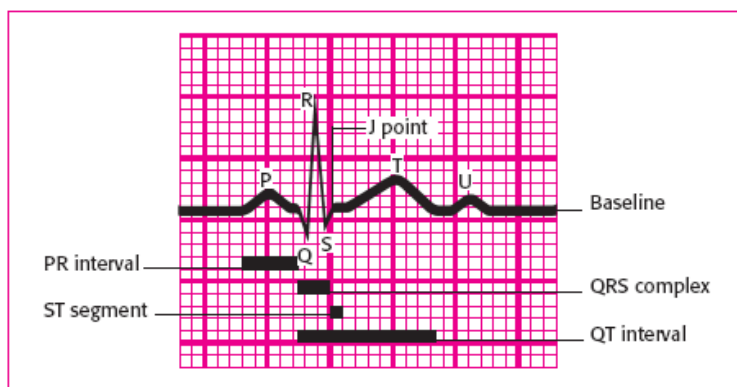
The grid on the ECG paper lets you measure waveform durations, intervals, and the height and depth of a wave. On the horizontal axis, which represents time, one small box equals 0.04 second and a large one (consisting of five small boxes) equals 0.20 second. The vertical axis measures amplitude or voltage. Each small box represents 1 mm (0.1 millivolt); each large box represents 5 mm (0.5 millivolt).

Analyzing the ECG rhythm
Your frame of reference is normal sinus rhythm: a heart rate between 60 and 100 beats/minute with regular atrial and ventricular rhythms and P waves that are uniform, round and upright in most leads, and occur before each identical QRS complex. The PR interval and QRS duration will be normal.

Follow these steps when analyzing a rhythm strip:
• *Determine the rate.* You can make a quick estimation of the ventricular rate by counting the number of QRS complexes in a 6-second section and multiplying that number by 10. Count the number of P waves in a 6-second section

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and multiply that number by 10 to determine the atrial rate.

A more accurate way to determine rate is to count the number of large boxes between QRS complexes for the ventricular rate (or the number of large boxes between P waves for the atrial rate) and divide 300 by this number. This method is especially helpful when you need to estimate the rate in short bursts of dysrhythmias.

• *Determine if the rhythm is regular or irregular.* Measure the distance from one R wave to the next R wave (R-R interval). If the R-R intervals are consistent, the ventricular rhythm is regular. Next, measure the distance from one P wave to the next P wave (P-P interval). If the P-P intervals are consistent, the atrial rhythm is regular.

• *Evaluate the P waves.* Does one P wave appear before every QRS complex? Do all the P waves look normal (round and upright) and the same? If the P waves look abnormal, they could be originating from somewhere in the atria other than the SA node. For example, if P waves are inverted, hidden in the QRS complex, or occur after the QRS complex, the impulse is most likely originating in the AV junction. An absence of P waves means atrial kick has been lost.

• *Evaluate the QRS complexes.* Do they all look the same? Do they have a typical configuration and normal duration? Does a QRS complex follow each P wave?

• *Look at the relationship between the P waves and the QRS complexes.* Does one P wave appear before every QRS, and are all of the PR intervals the same?

When a cardiac rate, rhythm, or both deviate from normal, your job is to determine the significance of this deviation. Compare the abnormal rate or rhythm to normal sinus rhythm—is it too fast, too slow, or too ugly?

## Too fast: Supraventricular tachycardia

Any resting heart rate greater than 100 beats/minute is too fast. But how fast is too fast? Normally, a rate between 100 and 150 beats/minute is a response to increased metabolic demand. For ex-

It's electric! Understanding cardiac conduction

Normal cardiac impulses originate in the sinoatrial (SA) node and travel through interatrial pathways to the atrioventricular (AV) node, the gatekeeper between the atria and the ventricles. The AV node instantaneously holds onto the impulse and allows the ventricles to fill with blood. The impulse then travels from the AV node through the bundle of His, branching off to the right and left bundle branches and the Purkinje fibers through to the myocardium, where mechanical contraction takes place.
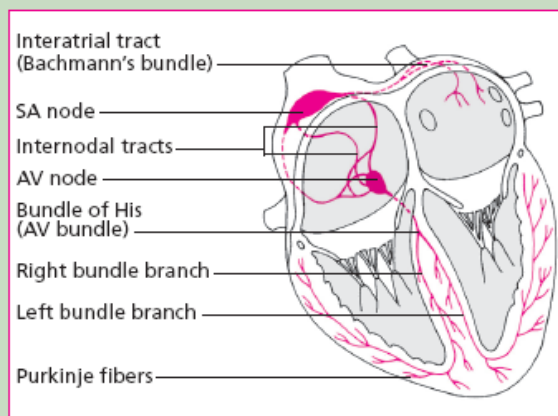
Normal sinus rhythm
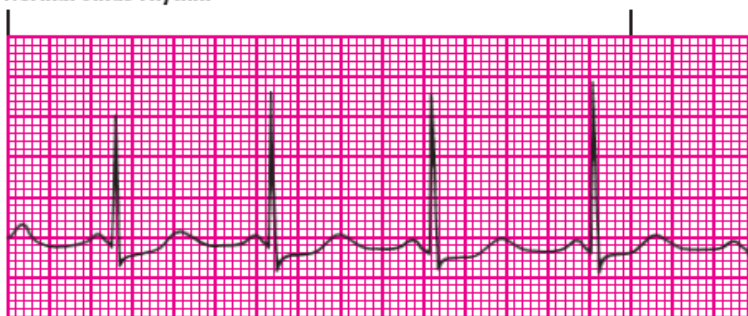
ample, when a patient gets out bed for the first time postoperatively, you can expect that his heart rate might go above 100 beats/minute, especially if he's not taking medications that slow the heart rate, such as beta-blockers. Once the patient is resting back in bed, his heart rate should return to normal.

Supraventricular tachycardia (SVT) is defined as a regular rhythm that originates above the ventricles and has a rate greater than 150 beats/minute. If your patient's heart rate is above 100 beats/ minute, you need to determine if the impulse is coming from above the ventricles (supraventricular) or from the ventricles themselves. Here's how to tell:

If the origin of the impulse is supraventricular, conduction through the ventricles will be normal. On the ECG, you'll see a normal QRS with a normal duration. You may or may not see P waves. Because the heart is contracting so quickly, ventricular filling time decreases, and your patient will begin to exhibit signs and symptoms of decreased CO, such as confusion; diaphoresis; dyspnea; hypotension; near-syncope; syncope; pale, cool extremities; and oliguria. If he's hemodynamically stable (that is, he has no serious signs and symptoms related to the dysrhythmia), have him perform a simple vagal maneuver such as coughing, which

may break the dysrhythmia. If vagal maneuvers don't work, the prescriber may order adenosine, which usually terminates SVT. Because of its very short half-life, give adenosine by rapid I.V. push. Adverse reactions include transient flushing, chest discomfort, brief periods of asystole or bradycardia, and ventricular ectopy. Place the patient supine before administering the drug and record a rhythm strip during administration.

If the SVT doesn't convert with I.V. adenosine administered according to advanced cardiac life support (ACLS) guidelines, the rapid rate may be controlled with I.V. diltiazem or a beta-adrenergic blocker such as metoprolol. If the patient experiences serious signs and symptoms related to the tachycardia (150 beats/minute or greater) associated with a pulse, prepare to assist with immediate synchronized electrical cardioversion.

Too fast and too ugly: Uncontrolled AF and VT

A fast, ugly irregular rhythm is most likely to be *uncontrolled atrial fibrillation* (AF) in which the impulse is generated by multiple sites within the atria. In this case, you won't see discrete P waves on the ECG. Remember that this rhythm isn't always fast, just irregular with no identifiable P waves and a generally normal QRS complex.

Based on current evidence-based guidelines, anticoagulation should always be considered for a patient with AF, but is beyond the scope of this article. Rapid AF is often treated with diltiazem to control the ventricular rate. Diltiazem slows conduction through the AV node, slows the ventricular response, and gives the SA node a chance to resume its role as the normal pacemaker.

Remember that diltiazem is a calcium channel blocker that causes peripheral vasodilation, so it can worsen the hypotension that may be associated with AF. Monitor the patient's BP closely and intervene as indicated. If the patient is hemodynamically unstable with serious signs and symptoms related to the rapid ventricular rate (greater than 150 beats/minute), he'll most likely need synchronized electrical cardioversion.

Atrial flutter is sometimes confused with AF. In both rhythms, P waves are lost. However, in atrial flutter you'll see sawtooth-like flutter waves at regular intervals. The rhythm may be regular or irregular, and the rate may be controlled at less than 100 beats/minute or uncontrolled at more than 100 beats/minute. This causes the atria to quiver and effective atrial contraction, as well as atrial kick, is lost. Because of this loss of atrial kick, CO is reduced by about 30%, increasing the patient's risk of thrombus formation. Treatment strategies are similar to those for AF.

Ventricular tachycardia (VT), another rhythm that's too fast and too ugly, occurs when the ventricles take over as the heart's pacemaker. The most common cause of VT is coronary artery disease. Other causes of VT include serum electrolyte imbalances, myocardial ischemia and infarction, and hypoxemia. You can easily spot rhythms originating in the ventricles by their wide (greater than 0.12 second) and bizarre QRS complexes on the ECG. Typically, VT has a rate of at least 150 beats/minute and is regular. P waves usually are absent; if they
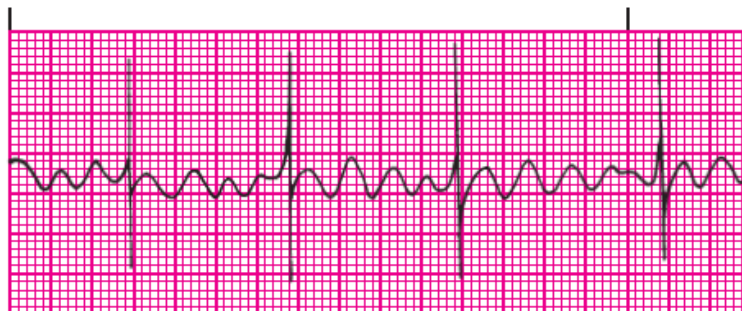
SVT
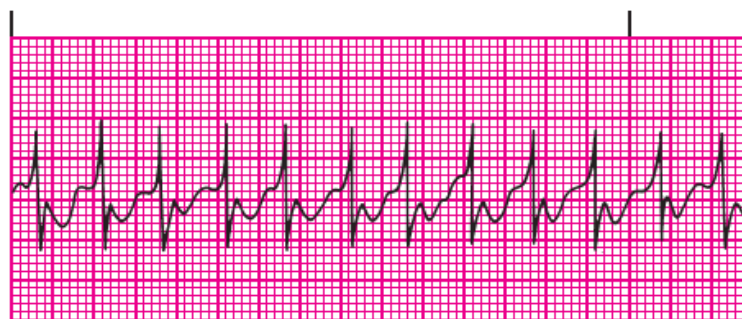
Uncontrolled AF

Atrial flutter

occur, they'll have no consistent relationship to the QRS complexes. This is called AV dissociation. Some patients with VT have a palpable pulse and may be completely asymptomatic, at least initially; other patients are unresponsive, apneic, and pulseless, requiring CPR and ACLS.

Patient management depends on the patient's clinical status. If the patient has pulseless VT, treat the rhythm as you would ventricular fibrillation (VF). Call a code, start CPR, defibrillate the patient as quickly as possible, and provide other interventions according to ACLS guidelines. If the patient is hemodynamically unstable but has a pulse, perform immediate synchronized electrical cardioversion. If the patient is hemodynamically stable, administer amiodarone as prescribed and prepare for elective synchronized cardioversion according to ACLS guidelines.
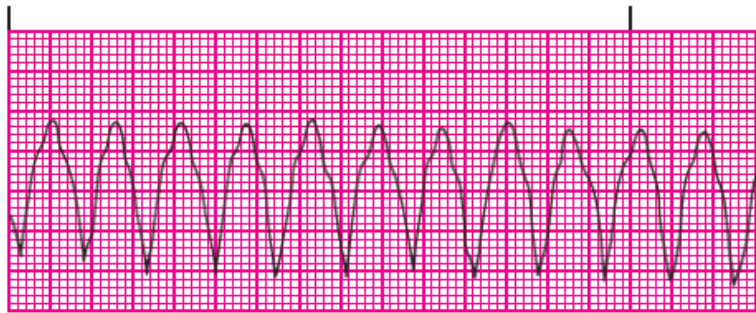
VT

Sinus bradycardia

Too slow: Sinus bradycardia

The most common slow rhythm is *sinus bradycardia,* which is a sinus rhythm with a heart rate of less than 60 beats/minute. Causes include increased vagal tone associated with myocardial infarction, adverse drug reactions, electrolyte imbalances, hypoxemia, hypoglycemia, hypothyroidism, and increased intracranial pressure.

A low heart rate generally means a low CO, but the significance of sinus bradycardia depends on the patient's clinical status. For example, if his daytime heart rate of 64 beats/minute drops to 56 beats/minute when he's sleeping, he isn't likely to be symptomatic with this bradycardia or require treatment. However, if a patient arrives in the ED after a syncopal episode with heart rate of 40 beats/minute and other signs and symptoms of poor perfusion, he requires immediate treatment following ACLS guidelines. Signs and symptoms of poor perfusion include acute change in mental status, hypotension, ongoing chest discomfort, nearsyncope, and syncope.

For symptomatic bradycardia, the drug of choice is I.V. atropine, administered while the patient is being pre-

pared for transcutaneous pacing. Atropine makes the SA node fire faster and speeds impulse conduction through the AV node.

Because atropine increases myocardial oxygen demand, it should be used cautiously in patients with myocardial ischemia. An epinephrine or dopamine infusion can be considered while waiting for transcutaneous pacing or if transcutaneous pacing is ineffective.

Keep in mind that patients who've had a heart transplant won't respond to atropine, because the transplanted heart lacks vagal innervation. For them, transcutaneous pacing is the treatment of choice.

Transcutaneous pacing is also a first-line intervention for symptomatic bradycardia, and should be started immediately for patients who are unstable, particularly those with high-degree heart block (Mobitz type II second-degree or third-degree). Some limitations apply. Transcutaneous pacing can be painful for the patient and may not produce effective mechanical capture.

Noninvasive transcutaneous pacing can be performed at the bedside, and should be started immediately if the

patient doesn't respond to atropine, if atropine is unlikely to be effective, or if the patient is severely symptomatic.

After pacing has been established, verify mechanical capture and reassess the patient's condition. Administer analgesia and sedation as ordered, and consult the cardiologist. If mechanical capture is inconsistent, prepare the patient for transvenous pacing.

Too slow and too ugly: Pauses, junctional, and idioventricular rhythms

A rhythm with *pauses* (missing P, QRS, and T waves on ECG) is too slow and too ugly. Again, the significance of this dysrhythmia depends on the patient's clinical status. Does he have signs or symptoms of poor perfusion caused by the bradycardia? How long are the pauses and how often do they occur? Possible causes and patient management are the same as for bradycardia.

Junctional escape rhythm occurs when the SA node fails to fire, the electrical impulse from the SA node is slower than that of the AV node, or the impulse from the SA node is blocked. The AV junction takes over as pacemaker, but because the AV junction
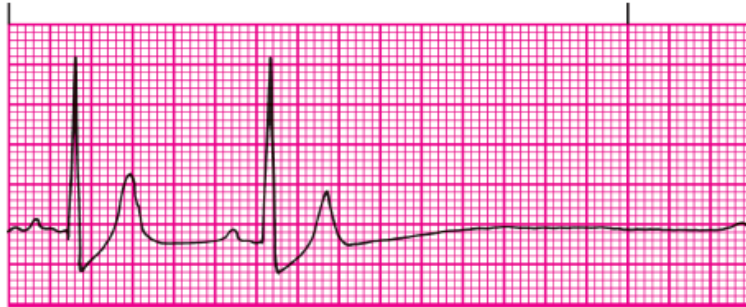
generates only 40 to 60 impulses/minute, junctional rhythms may be too slow. They're also too ugly: Because the impulse is initiated in the AV junction, it can travel retrograde through the atria, resulting in a P wave that's inverted, hidden, or occurs after the QRS complex. The QRS complex will usually appear normal because the impulse travels normally through the ventricles. Management of junctional escape rhythm is the same as for bradycardia.
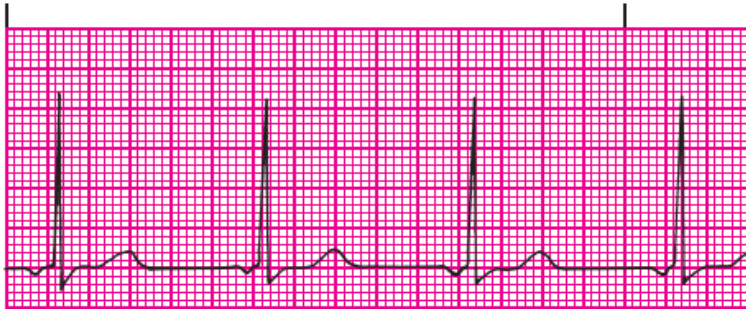
Idioventricular rhythm with a rate below 50 beats/minute is also too slow and too ugly. This rhythm occurs when the SA and AV nodes fail to fire, or the impulses are blocked, leaving the cells of the His-Purkinje system in the ventricles to generate their own impulses. However, the ventricles can generate a rate of only 20 to 40 beats/minute. The resulting rhythm is usually regular, but you'll see wide and bizarre QRS complexes and no P waves because the atria aren't depolarized. T waves are generally inverted because of abnormal ventricular repolarization. CO falls because of the low heart rate and loss of atrial kick. Causes, clinical manifestations, and patient management are the same as for bradycardia.
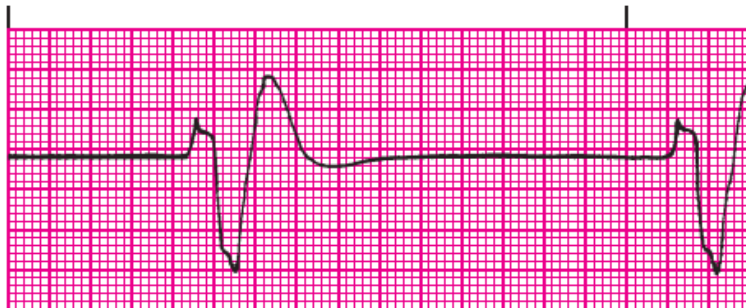
Too ugly: AV dissociation, VF, asystole

AV dissociation, also known as complete heart block or third-degree AV block, is a potentially life-threatening rhythm. Causes of this dysrhythmia are the same as for bradycardia. You'll see varied PR intervals in this dysrhythmia, and no relationship between the P waves and QRS complexes. In third-degree AV block, the SA node is usually working fine, so all of the P waves look the same and occur at regular intervals. But the block means these impulses aren't conducted to the ventricles. The ventricles generate their own impulses, resulting in wide and bizarre QRS complexes that occur at regular intervals, but have no consistent relationship to the P waves.

A patient with third-degree AV block will have loss of atrial kick and a decrease in ventricular rate. Depending on the ventricular rate, the patient can be significantly symptomatic.

Treatment, which is the same as for bradycardia, depends on the patient's clinical condition.

Because of their threat to life, the two ugliest rhythms are VF and asystole. VF is the result of many ventricular ectopic foci firing at once, resulting in an irregular, chaotic twitching of the ventricles without effective ventricular contractions. The patient will be apneic and pulseless. On ECG, you'll see a totally irregular, chaotic rhythm of fine

Pauses

Junctional escape rhythm

Idioventricular rhythm

Third-degree AV block

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or coarse fibrillatory waves—you won't be able to discern P waves, QRS complexes, or T waves.

Call a code, start CPR, defibrillate the patient as soon as possible, and follow ACLS guidelines to improve his chances of survival.

Asystole is a flat or nearly flat line, indicating no cardiac electrical activity and therefore, no mechanical activity. Restoring a cardiac rhythm is very difficult once a patient is in asystole. Patient management includes high-quality CPR and other interventions following ACLS guidelines.

Toward a beautiful ending

If your patient has an abnormal cardiac rhythm, recognizing it as too fast, too slow, or too ugly can help you quickly anticipate the appropriate interventions and intervene to give your patient the best chance at recovery. ✧

Coarse fibrillatory waves in VF

Asystole

RESOURCES

American Heart Association. 2005 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation.* 2005;112(24, suppl 1): IV1-IV203.
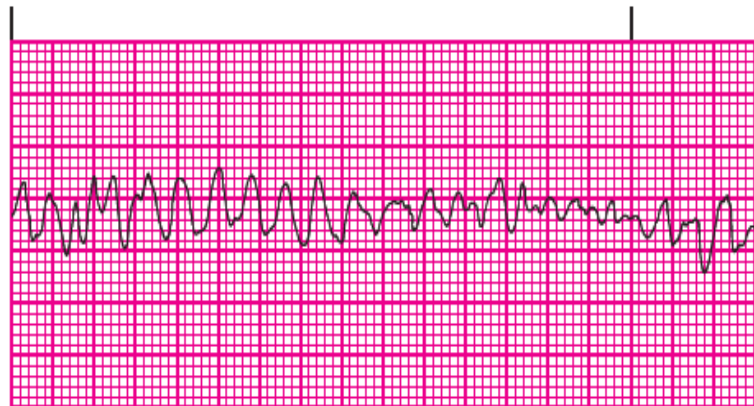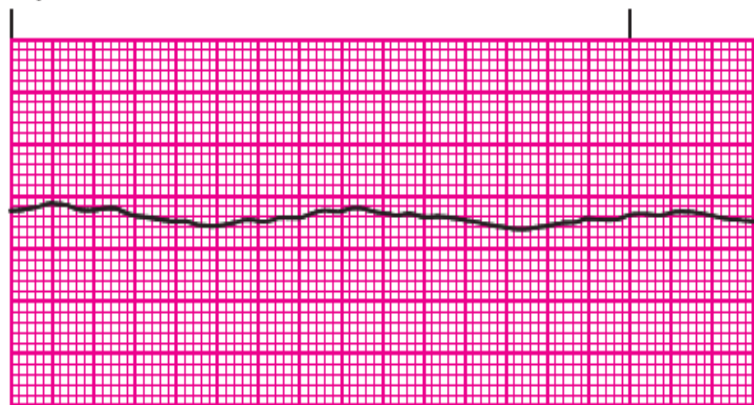
Thaler MS. *The Only EKG Book You'll Ever Need.* 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2007.

Wagner GS. *Marriott's Practical Electrocardiography.* 11th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2007.

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Recognizing ventricular arrhythmias and preventing sudden cardiac death
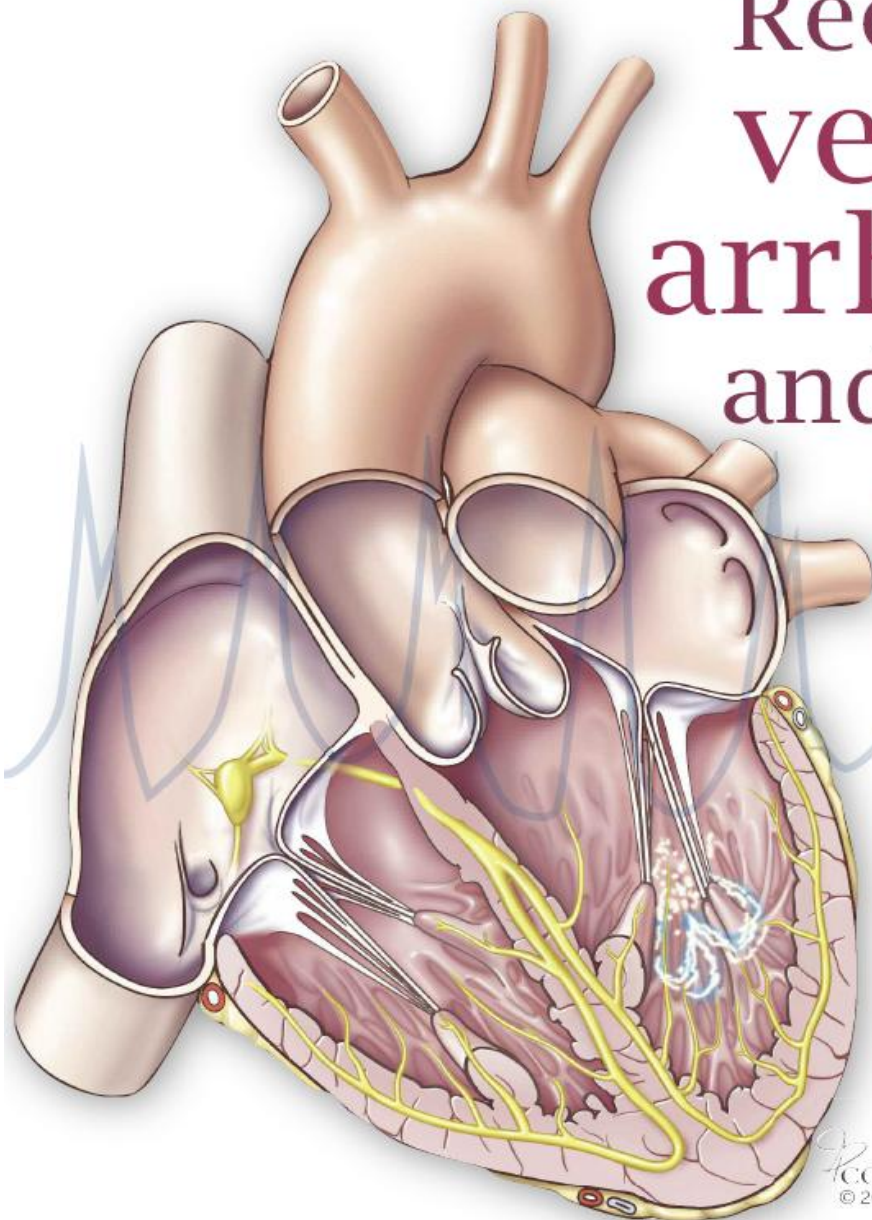
Be prepared to stop these dangerous arrhythmias.

By Rose M. Coughlin, MSN, RN, ACNS-BC

SUDDEN CARDIAC DEATH kills 350,000 to 400,000 Americans a year. That accounts for half of all deaths from coronary artery disease (CAD), the leading cause of death in the United States.

Frequently, a dangerous arrhythmia, such as ventricular tachycardia (VT) or ventricular fibrillation (VF), precedes sudden cardiac death. In fact, VT precedes sudden cardiac death in more than 70% of patients.

If you can detect VT and VF early on and provide prompt cardiopulmonary resuscitation (CPR) and defibrillation, your chances of preventing sudden cardiac death are good.

CE
1.8 contact hours

LEARNING OBJECTIVES

1. Identify the causes of ventricular arrhythmias.

2. Relate the heart's electrical conduction to components of the normal electrocardiogram.

3. Differentiate the types of ventricular tachycardia (VT).

4. Describe the management of ventricular arrhythmias.

Heart's electrical system

Before discussing ventricular arrhythmias, let's review how the heart's electrical conduction system works. The system's main function is to transmit electrical impulses from the sinoatrial (SA) node to the atria and ventricles, causing them to contract. Located high in the wall of the right atrium, the SA node is the heart's natural pacemaker. It normally fires 60 to 100 times per minute, and each impulse results in one heartbeat. (See *The path of cardiac conduction.*)

The electrical impulse passes through both atria to the atrioven-

tricular (AV) node at the junction of the atria and ventricles. If the SA node stops firing, the AV node initiates the impulses, but the rate drops to 40 to 60 times per minute.

From the AV node, the impulse travels through the bundle of His and down the right and left bundle branches into the Purkinje network of the ventricles, depolarizing the ventricular muscle. If the SA and AV nodes fail, the ventricles initiate the impulses but can only manage 20 to 40 impulses per minute.

An electrocardiogram (ECG) reflects the complete wave of depolarization as it travels from the SA node to the Purkinje network.

Reading an electrocardiogram

The key components of an ECG are the P wave, PR interval, QRS complex, and T wave, as shown.
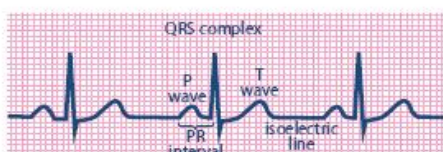
Normal electrocardiogram

The P wave represents atrial depolarization and is the first deflection from the isoelectric line. The P wave should appear rounded and uniform.

The PR interval represents the time needed for an impulse to travel through the atria and pause at the AV node. This pause allows the ventricles to fill with blood before contracting. The PR interval extends from the beginning of the P wave to the beginning of the QRS complex and normally is 0.12 to 0.20 second. Remember that each small square on the ECG paper represents 0.04 second. Five small squares make up a large block of 0.20 second, and 30 large blocks equal 6 seconds.

The QRS complex represents ventricular depolarization. The Q

The path of cardiac conduction

Normally, an electrical impulse starts at the sinoatrial node, spreads through the atria, reaches the atrioventricular node, and then spreads through the ventricles via the His-Purkinje network.
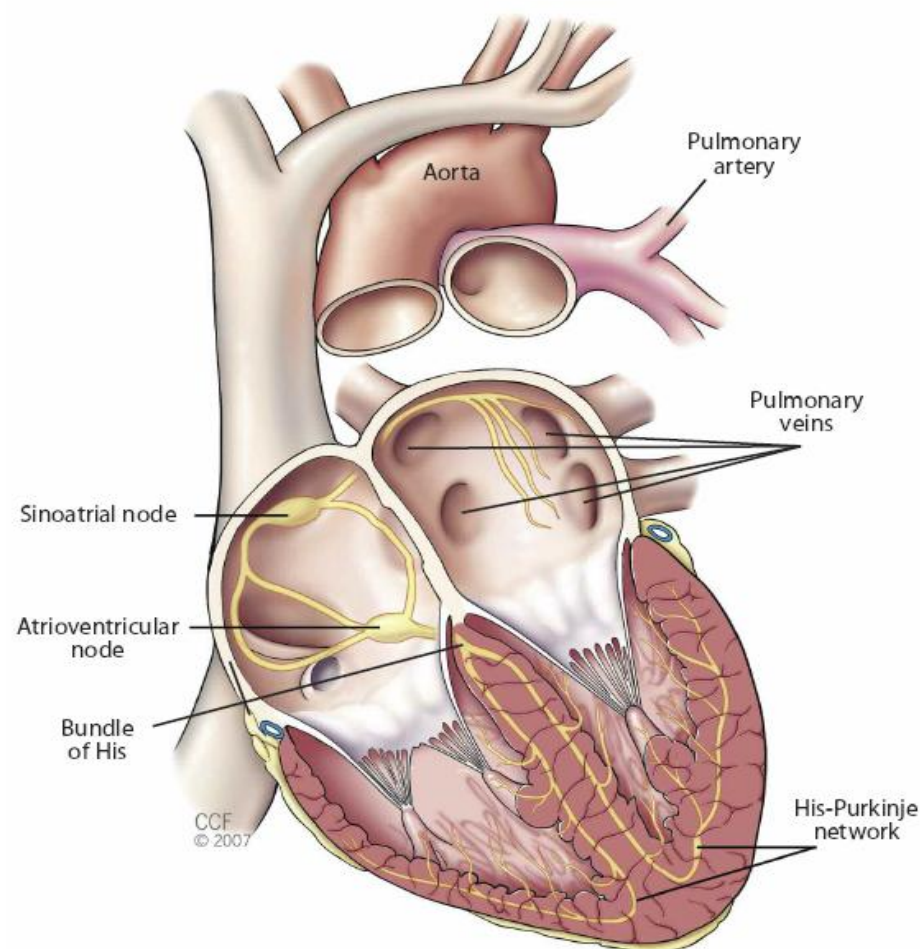
Courtesy of The Cleveland Clinic, Cleveland, Ohio

wave is the first negative deflection; the R wave is the first positive deflection; and the S wave is the negative deflection after the R wave. All three aren't present in every ECG lead. The normal QRS width is 0.06 to 0.12 second.

The T wave represents ventricular repolarization. It follows the QRS complex and is normally in the same direction. The T wave is usually rounded and slightly asymmetric.

The isoelectric line represents absence of electrical activity.

When a ventricular pacemaker takes control

With VT, the myocardium becomes extremely irritable, and a ventricu-

lar pacemaker in the bundle branches, Purkinje network, or ventricular myocardium takes control of the conduction system. The impulses override the higher pacemaker sites.

When impulses originate in the ventricles, the electrical current goes backwards through the ventricles, greatly reducing the heart's efficiency. Because the ventricles are the lowest sites in the conduction system, there are no fail-safe mechanisms if the heart rate drops too low.

Causes of ventricular arrhythmias

The most common cause of VT is

CAD. Other cardiac causes include myocardial infarction (MI), cardiomyopathy, and valvular heart disease. Patients are at higher risk for ventricular arrhythmias after having an MI because of a significant reduction in left ventricular systolic function.

VT may result from metabolic abnormalities, such as acidosis, hypoxemia, hyperkalemia, hypokalemia, and hypomagnesemia. And VT may be caused by certain drugs, such as caffeine, cocaine, alcohol, digoxin, theophylline, antipsychotics, tricyclic antidepressants, and antiarrhythmics with proarrhythmic potential such as flecainide, dofetilide, sotalol, and quinidine.

Tips for differentiating SVT with aberrancy from VT

Ventricular rhythm is usually regular in both supraventricular tachycardia (SVT) with aberrancy and ventricular tachycardia (VT), but these other characteristics may help you distinguish these two arrhythmias.

- VT is four times more common than SVT with aberrancy.
- VT is more common in patients who have a history of myocardial infarction or heart failure.
- Circulatory collapse is more common with VT than with SVT, although patients can maintain a normal blood pressure with VT.
- Atrioventricular dissociation, which can appear as independent P waves marching through the QRS complexes, strongly suggests VT.
- If the QRS complex is more than 0.14 second with right bundle-branch block or more than 0.16 second with left bundle-branch block, VT is more likely.
- If the wide QRS-complex tachycardia has a triphasic pattern in lead V_1, SVT with aberrancy is likely.
- If an upright QRS complex has a taller-left-peak pattern in lead V_1, the diagnosis is likely VT.
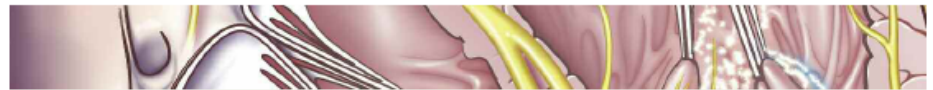
Recognizing ventricular arrhythmias

With VT, an ectopic pacemaker in the ventricles initiates a heart rate between 110 and 250 beats per minute (bpm). On the ECG, the QRS complexes are abnormally wide and bizarre and more than 0.12 second.

The forms of VT are classified by the configurations of the QRS complex. In monomorphic VT, the QRS complexes are the same or almost the same shape, size, and direction, as shown.

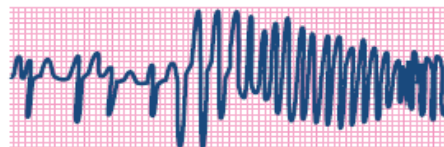Monomorphic ventricular tachycardia

In polymorphic VT, the QRS complexes markedly differ in shape, size, and direction from beat to beat. Torsades de pointes (meaning *twisting around a point*) is a form of polymorphic VT, characterized by QRS complexes that gradually change back and forth from one shape, size, and direction to another over a series of beats, as shown.

Torsades de pointes

VF, a medical emergency, is an erratic, disorganized firing of impulses from the ventricles. The ventricles quiver and are unable to contract or pump blood to the body. VF may be coarse or fine, as shown.

Ventricular fibrillation

Understanding sustained and nonsustained ventricular tachycardia
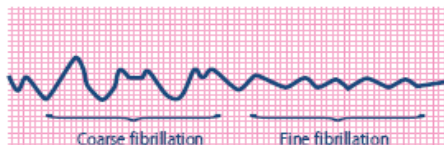
VT may be sustained or nonsustained. Sustained VT lasts longer than 30 seconds and may require termination because the rapid rate doesn't allow the heart to fill with blood, causing hemodynamic compromise. Nonsustained VT consists of three or more beats at a rate of at least 120 bpm lasting less than 30 seconds and terminating spontaneously without causing hemodynamic compromise.

Nonsustained VT is common, and usually patients don't have symptoms. Some patients do develop palpitations, syncope, or lightheadedness. Patients with nonsustained VT may have structural heart disease, such as CAD, dilated cardiomyopathy, or valvular heart disease and should have further evaluation.

The risk of sudden cardiac death in patients with preserved left ventricular function is doubled when nonsustained VT occurs more than 1 week after an MI. The risk of death is the greatest in the first 6 months after an MI and persists for up to 2 years. The risk of death is five times greater in patients with left ventricular dysfunction, defined as an ejection fraction of less than 40%.

The initial treatment of nonsustained VT includes correcting electrolyte imbalances, removing exacerbating factors (such as hypoxia, dehydration, and drugs), and adjusting the patient's beta-blocker

dosage. Antiarrhythmic drug thera-py such as amiodarone, sotalol, or dofetilide are commonly used in patients with implantable cardio-verter defibrillators (ICDs) to de-crease the incidence of ventricu-lar tachycardia (VT) and shock therapy.

Randomized clinical trials con-sistently show that ICD therapy is superior to antiarrhythmic drug therapy in primary and secondary prevention of sudden cardiac death in high risk populations.

According to the American Col-lege of Cardiology/American Heart Association/Heart Rhythm Society 2008 Guidelines for de-vice-based therapy of cardiac rhythm abnormalities, Class I rec-ommendations for ICD are:
- Ejection fraction (EF) ≤ 35% due to prior MI who are at least 40 days post-MI and are New York Heart Association Functional Class (NYHA-FC) II or III
- Left ventricular dysfunction due to prior MI who are at least 40 days post-MI, have a left ventricu-lar ejection fraction (LVEF) ≤ 30%, and are NYHA-FC I
- Survivors of cardiac arrest due to VF or hemodynamically unstable sustained VT after evaluation to define the cause and to exclude reversible causes.

Finding the cause of wide QRS-complex tachycardia

A wide QRS-complex tachycardia has a QRS complex of more than 0.12 second and a ventricular rate of more than 110 bpm. Ventricu-lar conduction is abnormally slow either because the arrhythmia originates in the ventricles out-side of the normal conduction system (ventricular tachycardia) or because there are abnormali-ties in the His-Purkinje system (supraventricular tachycardia with aberrancy).

A supraventricular tachycardia (SVT) originates above the ventri-cles, in the atria, or in the bundle

Diagnostic ECG features of VT, VF, and SVT

Arrhythmia	Heart rate (beats per minute)	Rhythm	P waves	PR interval	QRS complexes
Ventricular tachycardia	110 to 250	Usually regular but can be slightly irregular	• Absent or dissociated from QRS complexes	None	• Wide, bizarre, > 0.12 second
Ventricular fibrillation	Not discernible	Irregular, chaotic	• None	None	• No recog-nizable complexes or low-amplitude complexes
Supra-ventricular tachycardia	160 to 240	Regular	• Usually not visible; often buried in the QRS complex	Usually not discernible	• Usually narrow (< 0.12 second) • May be wide if aberrant conduction occurs • All QRS complexes look alike

of His. If the His-Pukinje system is normal, the QRS complex will be normal, so it's easy to tell the difference between SVT and VT. However, an abnormal system produces aberrancy and creates a wide QRS complex. When this oc-curs, distinguishing VT from SVT can be tricky. When a differential diagnosis can't be made, treatment proceeds as if the patient has VT. The reason? Treating a patient with VT as if he has SVT can lead to hemodynamic instability.

To identify the arrhythmia caus-ing wide QRS-complex tachycar-dia, consider such factors as the patient's age, cardiac history, and physical examination findings. (See Tips for differentiating SVT with aberrancy from VT.) Always report the rhythm to the cardiolo-gist as soon as possible because he has the expertise to identify and manage the rhythm.

A medical history of angina, MI, coronary artery bypass graft-ing, valvular heart disease, or heart failure strongly suggests VT.

The presence or absence of he-modynamic instability doesn't sug-gest the rhythm diagnosis; howev-er, if a patient is hemodynamically unstable, the rhythm should be treated as VT.

The main tool for identifying ar-rhythmias is the 12-lead ECG. (See Diagnostic ECG features of VT, VF, and SVT.) Atrioventricular dissoci-ation, fusion, and capture beats suggest, but don't confirm, VT. The following rhythm strips show the characteristics of SVT and VT. The characteristics of SVT with aberrancy include a rapid, regular heart rate; P waves that are usual-ly not visible because they are buried in the QRS complex, a PR interval that isn't discernible, and QRS complexes that look alike and are usually wide (greater than 0.12 second). Note that the onset of wide QRS-complex tachycardia in lead V_1 shows a triphasic pat-tern, which suggests a diagnosis of SVT with aberrant ventricular con-duction or right bundle-branch block.

Wide QRS-complex supraventricular
tachycardia with aberrancy

The characteristics of VT also include a rapid, regular heart rate, but the P waves are absent or dissociated from the QRS complexes, so there is no PR interval, and QRS complexes are wide and bizarre (more than 0.12 second). The upright QRS complex with a taller-left-peak pattern in lead V_1 indicates a diagnosis of VT.
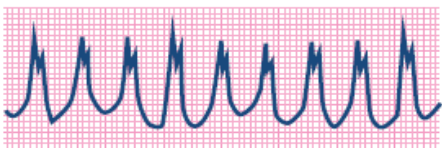
Wide QRS-complex
ventricular tachycardia

Treating ventricular arrhythmias

To treat pulseless arrest from VF or pulseless VT, use the 2010 American Heart Association (AHA) Guidelines for CPR and ECC. (See "Highlights of the 2010 American Heart Association Guidelines for CPR AND ECC" available for download at http://static.heart.org/eccguidelines/guidelines-highlights.html). Initiate CPR immediately and defibrillation as soon as a defibrillator is available. Note that a rhythm check is done only after 5 cycles or 2 minutes of CPR. The rationale is to minimize interruptions in chest compressions and the time between compressions and shock delivery.

After an I.V. line is established, a vasopressor will be given. The preferred antiarrhythmic is amio-

darone. Don't forget to investigate the cause of the arrhythmia. For example, draw blood for arterial blood gas analysis and other blood tests to check for hypoxia, acidosis, and hypoglycemia.

Teaching the patient

After the ventricular arrhythmia has been corrected, assess the patient for risk factors for CAD. Carefully review the patient's past medical history as well as current medications. Obtaining a 12-lead ECG and baseline blood tests can help reveal risk factors.

People who have experienced a sudden cardiac death event fear a recurrence, especially as the day of discharge approaches. To allay fears, use patient-centered care delivery. Provide emotional support and patient teaching that includes family members.

Patient and family teaching should include these topics, as appropriate:
- pathophysiology of CAD and the risk of sudden cardiac death
- CAD risk factor modification
- ICD use and follow-up care
- management of drug therapy
- prescribed activity levels/cardiac rehabilitation (as prescribed by physician)
- importance of follow-up appointments
- use of 911.

Most important, make sure the family knows how to perform CPR and use an automated external defibrillator.

Your role

Preventing sudden cardiac death starts with recognizing the dangerous ventricular arrhythmia that precedes it. The next step is providing prompt CPR and defibrillation, following the

current ACLS guidelines. After these life-saving interventions, your role in preventing sudden cardiac death continues as you identify the patient's risk factors and teach the patient and his family how to stay alive. ★

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OVERVIEW OF ARRHYTHMIAS

An arrhythmia is an irregularity of the heartbeat that can cause the heart too beat too fast (tachycardia), too slow (bradycardia), or create an irregular rhythm. Most arrhythmias are harmless, but some can be serious or even life threatening. Life threatening arrhythmias that is not controlled, can affect the heart ability to pump enough blood to the body. This can cause a lack of blood flow that can damage vital organs, such as the brain, heart. The speed and rhythm of the heart beat is controlled by an internal electrical system that generates the electrical pulse through the heart's conduction system, causing the heart to contract and pump blood. This process repeats with each new heartbeat

Each electrical impulse begins in a group of cells called the sinus node, or sinoatrial (SA) node. It travels through special pathways to the right and left atria. This causes the atria to contract and pump blood into the heart's two lower chambers, the ventricles. The electrical signal then moves down to a group of cells called the atrioventricular (AV) node, located between the atria and the ventricles. At this point, the signal slows down just a little, allowing the ventricles time to finish filling with blood. The electrical signal then leaves the AV node and travels along a pathway called the bundle of His.

This pathway divides into a right bundle branch and a left bundle branch. The signal goes down these branches to the ventricles, causing them to contract and pump blood out to the lungs and the rest of the body. The ventricles then relax, and the heartbeat process starts all over again in the SA node. The problem is if there is any delay or blocks at any part of this process, an arrhythmia can develop.

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Orthodromic atrioventricular reentry tachycardia via left lateral pathway	Atrioventricular nodal reentry tachycardia	Focal atrial tachycardia
Junctional ectopic tachycardia	Permanent junctional reciprocating tachycardia	Atrial flutter

This happens when the special nerve cells that produces the electrical signal don't work properly or when the electrical signal doesn't travel normally through the heart. An arrhythmia also can occur when another part of the heart starts to produce electrical signals, adding to the signals from the special nerve cells and disrupting the normal heartbeat.

Stress, smoking, heavy alcohol use, heavy exercise, use of certain drugs (such as cocaine or amphetamines), use of certain prescription or over-the-counter medicines, and too much caffeine or nicotine can lead to arrhythmia in some people. A heart attack or an underlying condition that damages the heart's electrical system also can cause an arrhythmia.

These conditions include high blood pressure (hypertension), coronary artery disease, heart failure, overactive or underactive thyroid gland (too much or too little thyroid hormone produced), and rheumatic heart disease. For some arrhythmias, such as Wolff-Parkinson-White syndrome, the underlying heart defect that causes the arrhythmia is present at birth (congenital). Sometimes, the cause of an arrhythmia can't be found.

Millions of people worldwide have arrhythmias. Atrial fibrillation is the most common type of cardiac arrhythmia found in adults today, affecting over 5.6 million patients in the United

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States and is expected to affect 15.9 million patients by 2050. Because of heart disease and other health problems that can lead to arrhythmias, adults older than 60 are affected by some of the more serious arrhythmias. Where other arrhythmia happen more often in children and young adults, such as paroxysmal supraventricular tachycardia (a fast heart rate that begins and ends suddenly), including Wolff-Parkinson-White syndrome. Some arrhythmias have no signs or symptoms, but when present the most common signs or symptoms a person will experience are:

➢ Palpitations (a feeling that your heart has skipped a beat or is beating too hard)

➢ A slow heartbeat

➢ An irregular heartbeat

➢ Feeling of pauses between heartbeats

More serious signs and symptoms include:

➢ Anxiety

➢ Weakness

➢ Dizziness and light-headedness

➢ Fainting or nearly fainting

➢ Sweating

➢ Shortness of breath

➢ Chest pain

Arrhythmias are more common in people who have a disease or condition that weakens the heart, such as:

➢ Heart attack

➢ Heart failure or cardiomyopathy, which weakens the heart and changes the way electrical signals move around the heart

➢ Heart tissue that is too thick or stiff or that hasn't formed normally

➢ Leaking or narrowed heart valves, which make the heart work too hard and can lead to heart failure

➢ Congenital problems (problems that are present at birth) with the heart's structure or function

Other conditions also can increase the chances of arrhythmia, such as:

➢ High blood pressure

- ➢ Infections that damage the heart muscle or the sac around the heart
- ➢ Diabetes, which increases the risk of high blood pressure and coronary artery disease
- ➢ Sleep apnoea (when breathing becomes shallow or stops during sleep), which can stress the heart because it doesn't get enough oxygen
- ➢ Overactive or underactive thyroid gland (too much or too little thyroid hormone in the body)

In addition to certain diseases and conditions, several other risk factors increase a person's chance of having an arrhythmia. Heart surgery, certain drugs (such as cocaine or amphetamines), or an imbalance of chemicals or other substances (such as potassium) in the bloodstream can increase a person's chance of having an arrhythmia.

ETIOLOGY

Cardiac Arrhythmias occurs when the heart beats improperly, as a result of incorrect impulse generation, (at the Sino-Atrial (SA) node) or impulse conduction. The heart organ broadly differentiates into two types of cell, pacemaker cells, and non-pacemaker cells. Pacemaker cells beat with their own independent rhythm, in synchrony with their neighbours (a heart attack results when these cells get out of synch with each other). The non-pacemaker cells require stimulus to beat, this stimulus comes from the pacemaker cells. Incorrect impulse generation is the fault of the pacemaker cells, which are beating either too slow, too fast, or not totally in rhythm. Impulse conduction problems are caused because the nervous signal from the pacemaker cells (located primarily at the SA node) fails to reach the non-pacemaker cells correctly. This problem falls into two further categories, a nodal block whereby the signal from the SA node fails to reach the AV node, and a re-entry pathway.

A re-entry pathway occurs when a section of nervous tissue (which conducts the impulse) is damaged in some manner (e.g. Physical trauma, Cardiac infarction (Heart attack)). Part of the tissue only conducts the impulse in a single direction, in the example shown left this is in the opposite direction to the genuine impulse direction. This will set up a loop in the nervous tissue, as the impulse keeps going round and round the junction, stimulating the non-pacemaker cells to contract, and ultimately disturbing the rate of heartbeat. Regardless

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of the specific arrhythmia, the pathogenesis of arrhythmias falls into one of three basic mechanisms. These include enhanced or suppressed automaticity, triggered activity, or re-entry. Automaticity is a natural property of all myocytes. Ischemia, scarring, electrolyte disturbances, medications, advancing age, and other factors may suppress or enhance automaticity in various areas. Suppression of automaticity of the sinoatrial node can result in sinus node dysfunction and sick sinus syndrome.

Sick sinus syndrome is still the most common indication for permanent pacemaker implantation. In contrast to suppressed automaticity, enhanced automaticity can result in multiple arrhythmias, both atrial and ventricular. Triggered activity occurs when early afterdepolarizations and delayed afterdepolarizations initiate spontaneous multiple depolarisations precipitating ventricular arrhythmias. Examples of this include torsades de pointes and ventricular arrhythmias due to digitalis toxicity. Finally, probably the most common mechanism of arrhythmogenesis results from re-entry. Requisites for re-entry include bi-directional conduction and uni-directional block. "Micro-" level re-entry occurs with VT from conduction around the scar of myocardial infarction and "macro-" level re-entry occurs via conduction through manifest (Wolff-Parkinson-White syndrome[2]WPW) or concealed accessory pathways.

ELECTROLYTE IMBALANCE

Hypercalcemia and hypocalcaemia (high and low calcium levels, respectively) may cause heart block and cardiac arrest. Hypernatremia (high sodium level) may result in an erratic heart rate as sodium and calcium ions compete with one another to influence the heart. Imbalances in potassium and magnesium, however, are the usual culprits of cardiac arrhythmia when an electrolyte imbalance occurs. Hyperaemia (high potassium levels) initially causes tachycardia and then bradycardia as the heart fatigues in response to the high sustained heart rate and weak cardiac contraction. Hypokalemia (low potassium levels) results in bradycardia and a slow, weak pulse.

Hypomagnesaemia (high magnesium) causes premature ventricular contractions (PVCs), which may be noticed only if there are several PVCs in a row. Hypomagnesaemia (low magnesium) may result in PVCs, atrial fibrillation (sustained heart rate faster than 140) or

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ventricular fibrillation (a lethal and very high sustained heart rate, over 220. In summary, arrhythmias may be caused by many different factors, including: (1) Coronary artery disease; (2) Electrolyte imbalances in your blood (such as sodium or potassium); (3) Changes in your heart muscle; (4) Injury from a heart attack; (5) Healing process after heart surgery. But also remember this, Irregular heart rhythms can also occur in "normal, healthy" hearts.

CLINICAL MANIFESTATIONS

The symptoms of cardiac arrhythmia are not specifically life-threatening, unless left untreated, cardiac arrhythmia can lead to more fatal forms of rhythm disturbance, e.g.; premature ventricular depolarization may lead to ventricular fibrillation (resulting in a heart attack). The signs and symptoms of cardiac arrhythmias can range from completely asymptomatic to loss of consciousness or sudden cardiac death. In general, more severe

Sinus Rhythms
NSR
Sinus Bradycardia
Sinus Tachycardia
Sinus Arrhythmia
Sinus Pause/Arrest

Atrial Rhythms
PACs
WAP / MAT
Atrial Tachycardia
Atrial Flutter
Atrial Fibrillation

AV Blocks
First Degree
Second Degree (Type I/II)
High Grade (Advanced)
Third Degree (Complete)

Junctional Rhythms
PJCs
Junctional Escape Beats
Junctional Rhythm
Accelerated Junctional Rhythm
Junctional Tachycardia

Ventricular Rhythms
PVCs
Ventricular Escape Beats
Idioventricular Rhythm
Accelerated Ventricular Rhythm
Ventricular Tachycardia
Ventricular Fibrillation

symptoms are more likely to occur in the presence of structural heart disease. For example, sustained monomorphic VT, particularly in a normal heart, may be hemodynamically tolerated without syncope.

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In contrast, even non-sustained VT may be poorly tolerated and cause marked symptoms in patients with severe LV dysfunction. Complaints such as light-headedness, dizziness, quivering, shortness of breath, chest discomfort, heart fluttering or pounding, and forceful or painful extra beats are commonly reported with a variety of arrhythmias. Frequently patients notice their arrhythmia only after checking peripheral pulses. Certain symptoms raise the index of suspicion and can give clues to the type of arrhythmia. The presence of sustained regular palpitations or heart racing in young patients without any evidence of structural heart disease suggests the presence of an SVT due to atrioventricular nodal re-entry, or SVT due to an accessory pathway.

Such tachycardias may frequently be accompanied by chest discomfort, diaphoresis, neck fullness, or a vasovagal type of response with syncope, diaphoresis, and nausea. It has been shown that the hemodynamic consequences of SVT and VT can have an autonomic basis, recruiting vasodepressor reflexes similar to that observed in neurocardiogenic syncope. Isolated or occasional premature beats suggest PACs or PVCs and are benign in the absence of structural heart disease.

Syncope in the setting of noxious stimuli such as pain, prolonged standing, and venepuncture, particularly when preceded by vagal-type symptoms (diaphoresis, nausea, vomiting), suggests neurocardiogenic (vasovagal) syncope. An arrhythmia can be silent and not cause any symptoms. A doctor can detect an irregular heartbeat during a physical exam by taking your pulse or through an electrocardiogram (ECG).

MECHANISMS OF ARRHYTHMIA

Cardiac arrhythmias results from abnormal impulses initiation, abnormal impulse conduction or both mechanisms together. Abnormal impulse initiation includes enhanced normal automaticity, abnormal automaticity and triggered activity resulting from afterdepolarization. Whereas, abnormal impulse conduction includes conduction block and reentry.

Although all these mechanisms have been shown to cause arrhythmias, it is not possible to prove which mechanism is responsible for a particular arrhythmia. However it is possible to

postulate the mechanism of many clinical arrhythmias based on their characteristics and behaviour and to list rhythms most consistent with known electrophysiologic mechanisms.

NORMAL AUTOMATICITY

Normal automaticity involves the slow, progressive depolarization of the membrane potential (spontaneous diastolic depolarization or phase four depolarization) until a threshold potential is reached, at which point an action potential is initiated. Although automaticity is an intrinsic property of all myocardial cells, the occurrence of spontaneous activity is prevented by the natural hierarchy of pacemaker function.

The spontaneous discharge rate of the sinoatrial (SA) nodal complex exceeds that of all other subsidiary or latent pacemakers. As a result, the impulse initiated by the SA node depresses the activity of subsidiary pacemaker sites, before they can spontaneously depolarize to threshold. However, slowly depolarizing and previously suppressed pacemakers in the atrium, AV node, or ventricle can become active and assume pacemaker control of the cardiac rhythm if the SA node pacemaker becomes slow or unable to generate an impulse or if impulses generated by the SA node are unable to activate the surrounding atrial myocardium. The emergence of subsidiary or latent pacemakers under such circumstances is an appropriate fail-safe mechanism which assures that ventricular activation is maintained.

ABNORMAL IMPULSE INITIATION

Is due to enhanced normal automaticity, abnormal automaticity or triggered rhythms - afterdepolarization.

Enhanced normal automaticity: refers to the accelerated generation of an action potential by either normal pacemaker tissue (enhanced normal automaticity) or by abnormal tissue within the myocardium (abnormal automaticity). The discharge rate of normal or abnormal pacemakers may be accelerated by drugs, various forms of cardiac disease, reduction in extracellular potassium, or alterations of autonomic nervous system tone. Enhanced normal automaticity accounts for the occurrence of sinus tachycardia, while abnormal automaticity

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may result in various atrial or ventricular arrhythmias, for example, an accelerated idioventricular rhythm.

Abnormal automaticity: refers to the development of a site of depolarisation in non-pacemaker tissue usually in Purkinje fibres or myocardial cells. This occurs because of the inability of these cells to maintain a constant resting potential, but to decline gradually to reach a threshold potential; e.g. this may occur if the cells are hypoxic, as maintenance of the resting membrane potential is energy dependent as the rate of depolarisation is increased by catecholamine stimulation. In cases, where a person collapses on exertion with aortic stenosis, the compensatory left ventricular hypertrophy causes cell hypoxia. This is cause because when the heart rate increases (less diastolic coronary flow) and the sudden exercise catecholamines are released. Together they act as a potent stimulus for the development of ectopic (i.e. ventricular premature complexes and tachycardia).

Arrhythmias arising from the sinus node

Because the sinus node is the dominant pacemaker of the heart, alterations in its rate may lead to arrhythmias. Sinus tachycardia results when the sinus node fires at rates in excess of 100 beats per minute. Conversely, sinus bradycardia occurs when the sinus node fires at less than 60 beats per minute. Note that either of these two conditions may be either normal or abnormal. Sinus tachycardia can be the appropriate response to external factors such as exercise, fever, or hypotension. In well-conditioned athletes, sinus bradycardia can be the normal response to exercise-increased parasympathetic stimulation.

In the average person, however, sinus bradycardia more often reflects an abnormality of the sinus node and escape pacemakers due to disease, abnormalities in parasympathetic stimulation, or outside factors such as drugs.

Arrhythmias arising from ectopic pacemakers

Arrhythmias due to automaticity also result when the site of dominant pace making shifts from the sinus node to any ectopic (nonsinus) pacemaker. Sinus node dominance can be compromised in several ways: sinus node suppression of subsidiary pacemakers may be reduced, inhibitory influences between nonpacemaker and pacemaker cells may be removed, or secondary pacemakers may be enhanced so that they fire faster than the sinus node.

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TRIGGERED RHYTHMS

Triggered rhythms are caused by afterdepolarizations which are oscillations in the membrane potential following an action potential. There are two types of afterdepolarizations. One occurs early during the repolarisation of the membrane, an early afterdepolarization, and the other occurs after the repolarisation is complete or nearly complete, a delayed afterdepolarization. When either type is large enough to reach threshold, the resulting action potential is called a triggered action potential. Triggered activity, therefore, differs from automaticity in that at least one action potential (the trigger) comes before the train of pulses. Automatic rhythms can arise spontaneously following long periods lacking in electrical activity; whereas, triggered rhythms cannot arise spontaneously. Triggered activity will cause arrhythmias when impulse initiation shifts from the sinus node to the triggered focus. For this, the rate of triggered impulses must be faster than the rate of the sinus node, an event that may be brought about when the sinus node has been slowed or inhibited, when it has been blocked, or when the triggered focus is intrinsically faster.

Early afterdepolarizations:

Early afterdepolarizations (EADs) occur during repolarisation of an action potential initiated from a normal membrane potential. They appear as sudden positive changes in membrane potential; instead of following the normal course of repolarisation, the membrane suddenly shifts toward depolarization. This shift can result from any factor that decreases outward current (carried by K^+) or increases the inward current (carried by Na^+ or Ca^{2+}).

Arrhythmias caused by early afterdepolarizations (EAD)

Torsade de pointes is a ventricular tachycardia characterized by alternating positive and negative QRS polarity and changing amplitude in an undulating pattern over 5 to 20 beats. Because experimental arrhythmias that resemble torsade de pointes are induced by agents known to induce early afterdepolarizations, it has been suggested that naturally occurring torsade de pointes may sometimes be caused by EADs. Antiarrhythmic drugs which prolong the duration of Purkinje fibre action potentials such as sotalol and quinidine can cause EADs

and triggered activity. Both drugs block the repolarising K$^+$ current, and the arrhythmias associated with their use may also result from EADs.

As mentioned above, slowing the heart rate facilitates the appearance of early afterdepolarizations; therefore, it seems likely that some tachycardias that follow bradycardia are the result of EAD triggered activity. It has also been suggested that tachycardias in patients with long QT interval syndrome (in which there may be long action potential duration) may be triggered.

Delayed afterdepolarizations

Delayed afterdepolarizations (DADs) are small transient depolarisations (about 10 mV) that occur shortly *after* the maximal diastolic voltage has been achieved during repolarisation of an action potential (Figure 3.7, solid line). Under normal conditions, cells in the coronary sinus and in the AV valves can present DADs, but under abnormal conditions, many cell types can generate them.

Arrhythmias caused by delayed afterdepolarizations

Delayed afterdepolarizations can reach threshold to cause triggered action potentials especially if the rate of stimulation is fast enough. Digitalis induced ventricular arrhythmias can be initiated by pacing at rapid rates, and as toxicity increases, the DAD train increases. Introduction of catecholamines into the canine coronary sinus causes atrial tachycardia with the characteristics of triggered activity. Some naturally occurring atrial tachycardias induced by the sympathetic nervous system, therefore, are probably caused by DADs. Ventricular muscle and Purkinje fibres can also develop DADs in the presence of catecholamines, and sympathetic stimulation may cause some of the ventricular arrhythmias that accompany exercise, ischemia, or infarction.

ABNORMAL IMPULSE CONDUCTION: is due to conduction block or reentry.

Conduction block

Block of cardiac impulses can occur under several different scenarios. An impulse may arrive at tissue that is unexcitable because the tissue is still refractory after a recent depolarization or because the tissue is abnormally depolarized. Block may occur because the strength of

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the propagating wave front is insufficient despite the fact that tissue is fully excitable (decremental conduction and block). It may also occur because tissue is intrinsically unable to conduct (scar tissue from prior infarct or surgical incision).

If block does occur, arrhythmias may arise in several different ways. Normally, if the sinus impulse fails to propagate to right atrium (sinus node exit block or sinoatrial block), an ectopic pacemaker will take control of the heart. If the AV conduction system is blocked so that the ventricles are not stimulated at an appropriate rate, an ectopic pacemaker distal to that block may assume control of pace making. Under some conditions, however, an escape pacemaker may not arise quickly enough or at a clinically significant rate. A period of asystole (an absence of electrical or mechanical activity), marked bradycardia or both may appear. Block may also appear in one of the bundle branches.

The blocks discussed above prevent conduction of impulses in both directions. A special form of block, unidirectional block, will be discussed in detail in the next section.

Reentry

Normally, the action potential from the sinus node dies out after orderly depolarization of the atria, AV conduction system, and the ventricles. The impulse does not usually conduct backwards because the tissue just stimulated is refractory and, therefore, unable to generate a second action potential. As a result, the normal heart must wait for new sinus pulse for each subsequent heart beat.

Reentry occurs when the action potential does not die out but continues to propagate and reactivate the heart. Almost all clinically important tachyarrhythmias are due to reentry. Reentry can occur almost anywhere in the heart and can assume many sizes and shapes.

<u>Rhythms</u>

Sinus Rhythm:

A normal heart rhythm Normal sinus rhythm) will have a heart rate between 50 and 100 beats per minute and a normal impulse formation from the SA node (P wave). In the absence of any abnormalities, a completely normal rhythm will also have a normal PR interval (interval from the beginning of the P wave to the beginning of the QRS of .12-.20 seconds), a normal QRS width (time it takes for the ventricles to contract of .04-.10 seconds), a normal QT interval (interval from the beginning of the QRS to the beginning of the T wave of .30-.46 seconds). Also, all the waveforms must be of a normal shape with no ST changes.

ARRHYTHMS

Sinus Dysrhythmia - Variable heart rate, also known as sinus arrhythmia

Sinus dysrhythmia can be a normal rhythm, but rather than having a steady rate, the rate varies by more than 10 beats per minute. In the above example, the heart rate varies from 75-94 beats per minute. All other measurements and aspects of the rhythm would be consistent with what is described under Normal Heart Rhythm.

Sinus dysrhythmia is divided into two categories, respiratory (related to breathing) and no respiratory. Respiratory sinus dysrhythmia is present when there is a gradual rate increase with inspiration (breathing in) and a gradual decrease with expiration (breathing out). No respiratory sinus dysrhythmia is present when there is an irregularity not related to the respiratory cycle. Both types may occur in healthy or diseased hearts.

Sinus dysrhythmia is very common and usually normal in children and young adults with rate variations up to 30 beats in one minute not uncommon. Sinus dysrhythmias in the older population can frequently be associated with lung disease.

ATRIAL ARRHYTHMIAS:

Sinus Bradycardia

Sinus bradycardia is a rhythm which is formed by the electrical impulse originating in the SA node in the normal manner. However, the rate is slower than normal <60bpm (normal heart rate range is 60-100 bpm). This rhythm is commonly seen in healthy adults, particularly in well conditioned athletes, and they will not generally experience any symptoms. For patients where this is not their normal rhythm, they may experience dizziness or light-headedness. In rare cases, may feel faint or may pass out if the change to this rate from a faster rate is abrupt.

This rhythm is generally of no consequence. However, in some cases, it may be due to medications the patient is taking, which would suggest the need to review and possibly modify those medications.

Sinus Tachycardia
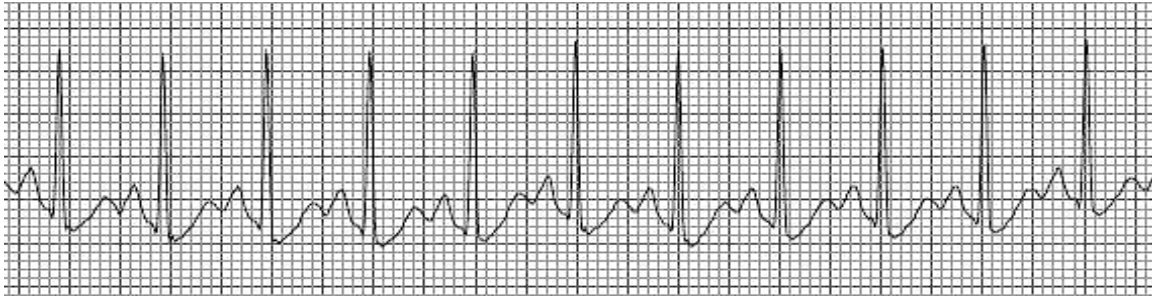
Sinus tachycardia (often referred to as tachy) is a rhythm which is formed by the electrical impulse originating in the SA node in the normal manner. However, the rate is faster than normal, > 100bpm (normal heart rate range is 60-100 bpm). With sinus tachycardia, it is also not unusual for both the PR and QT intervals to decrease in length with increased rates.

This is a common rhythm which in response to any of a variety of stimuli is completely normal. The following are situations in which sinus tachycardia is normal;

➢ Response to exercise (even running up a flight of stairs)
➢ Emotional upset or anxiety
➢ When running a fever
➢ When a patient is pregnant
➢ When ingesting caffeine or alcohol
➢ Smoking

There are also situations when the rhythm is in response to an abnormal physiologic condition such as the following;

➢ Hypotension/hypertension
➢ Congestive heart failure
➢ Shock
➢ In the presence of heart disease
➢ During a heart attack

A patient's normal maximum heart rate decreases with age. At age 16, maximum rates under exertion of 220 beats per minute would not be unusual. However, by age 50, that same person's maximum rate under exertion may decrease to 160 beats per minute.

Patients experiencing sinus tachycardia may complain of rapid rates, palpitations, dizziness, light-headedness or any of a variety of related symptoms. Generally, no treatment is necessary in the presence of sinus tachycardia. However, if the sinus tachycardia is an

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inappropriate response (for example, patient is sitting still and is in sinus tachycardia), medications may be indicated.

Sinus Pauses / Asystole

Sinus pause and sinus block are slight variations of the same rhythm, both of which may lead to an asystole which is an absence of electrical activity in the heart. However, there are other causes of asystole as well. First we discuss sinus pause, and further down the page, asystole.

Sinus Pause/Sinus Block
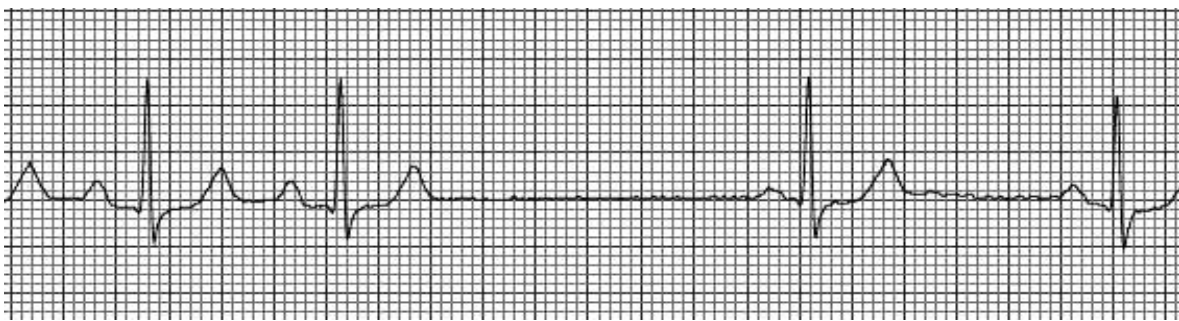
Sinus pause describes a condition where the SA node fails to generate an electrical impulse for what is generally a brief period of time. In the above example, the initial rate is 88 beats per minute (the first two beats are normal), then there is a 1.8 second sinus pause before the heart resumes, initially at a somewhat slower rate of 52 beats per minute. A related rhythm is SA block which is often hard to distinguish from a sinus pause. In SA block, the SA node creates an impulse, but it is blocked from leaving the SA node. The differences are beyond the scope of this discussion.

Patients who have sinus pauses may complain of missed or skipped beats, flutters, palpitations, hard beats or may feel faint, dizzy or lightheaded or experience a syncopal episode (passing out). Frequent pauses would heighten these symptoms. This is a result of patients actually missing or dropping beats. Obviously, if the heart misses a beat, blood does

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not flow during that time period resulting in a lack of oxygen or perfusion throughout the body.

Treatment and prognosis depend on the cause and cardiac status of the patient. This condition may be drug induced or it may be a result of cardiac disease. Treatment may involve the use of medications or the use of a temporary or permanent pacemaker.

Asystole
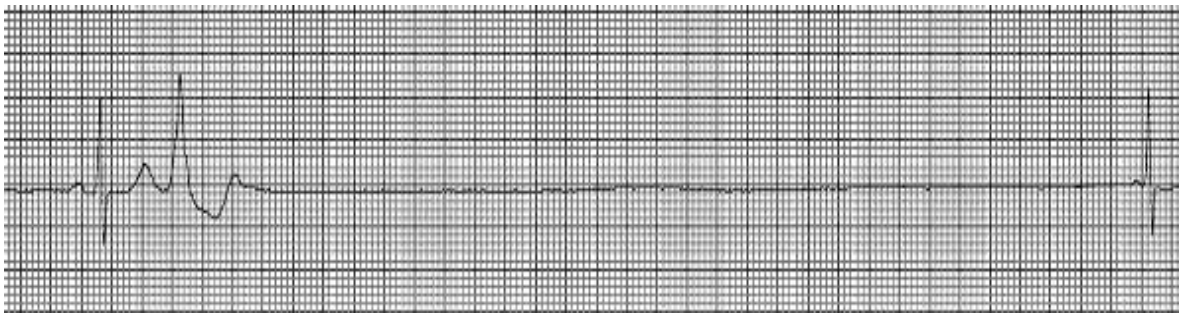
The above is an example of a 6.3 second asystole, caused by sinus arrest with no backup pacemakers taking over. The first beat is normal, the second beat is a PVC, then there is 6.3 seconds of no electrical activity in the heart, followed by a relatively normal looking beat at the far right side of the strip (although technically it is a junctional escape beat, e.g. the impulse originated in the AV junction, not the SA node). Although the above example shows no electrical activity during the 6.3 second asystole, you may also have a similar asystole where there are P waves throughout the asystole but no QRS complex as a result of complete AV block with a failure of the backup pacemaker (all cells in the heart can act as backup pacemakers to the SA node). The result for the patient is the same; severe light-headedness, dizziness, near syncope or a syncopal or passing out episode. Depending on the cause, medications or a permanent pacemaker is indicated.

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Atrial Ectopic Rhythm

An atrial ectopic rhythm is a rhythm where the impulse formation in the atrium is coming from the wrong place. (Ectopic means out of place or from the wrong place.) In the example above, the first P wave is upright, the second one upright and slightly smaller, and the third smaller yet with the 4th and 5th ones barely visible. This suggests that the first P wave may have come from the SA node, but that the following ones come from different locations in the atria, thus causing their shape and the PR interval to change. Atrial ectopic rhythms may also have rate changes along with the changes described above in the P waves and PR intervals. The heart rate in the above example is 63 beats per minute with the PR interval varying from .16 seconds to .06 seconds.

Atrial ectopic rhythms are most commonly found in younger patients and are generally benign. Patients will frequently describe their symptoms as a change in rate.

Atrial Fibrillation / Flutter

Atrial Fibrillation

Atrial fibrillation is characterized by a total disorganization of atrial activity without effective atrial contractions. The atrial rate is generally very fast (300-600 beats per minute), but not all impulses are conducted to the ventricles (they are blocked by the AV node). It is often described as an irregular, irregular (yes, two irregulars) rhythm in that generally, no two consecutive beats are at the same rate. The baseline or space between heart beats often has an irregular appearance to it which is actually the rapid, irregular contractions of the atria.

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Atrial fibrillation is relatively common, occurring in up to .5% of the population or 1 out of every 200 people.

Controlled Atrial Fibrillation
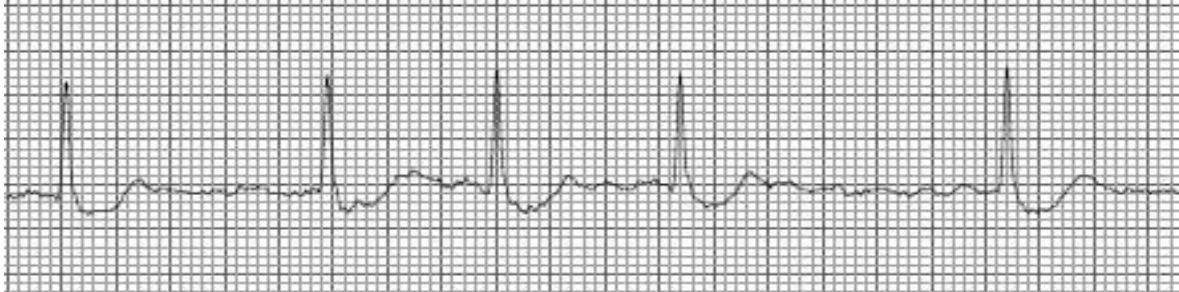
The above example shows a well controlled atrial fibrillation with the rate varying from 50-95 beats per minute with an effective average (usually referred to as MVR or minute ventricular rate) of 75 beats per minute. This example and the one below it also show modest ST depression of approximately 2 mm.

Slow Atrial Fibrillation
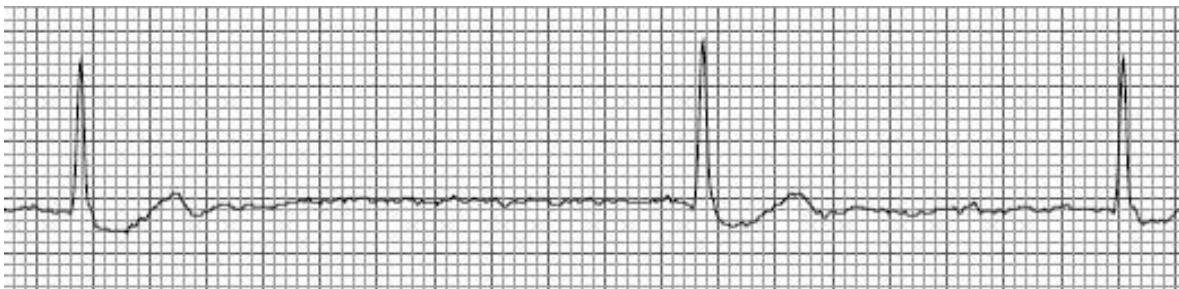
The example immediately above is the same patient, but now the rate is varying from 26-45 beats per minute with an effective average (also referred to as minute ventricular rate or MVR) of 38 beats per minute.

Rapid Atrial Fibrillation

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The above example shows a different patient with a rate varying from 112-250 beats per minute with an effective average rate of 160 beats per minute and some ST depression. Atrial fibrillation may be either paroxysmal (of sudden onset) and last from a few minutes to many hours, or chronic, when the patient is persistently in atrial fibrillation. Chronic atrial fibrillation is almost always associated with underlying heart disease.

Patients will describe this rhythm variously as palpitations, flutters, rapid rate or irregular rate. A number of treatment options exist. If the patient is in an intermittent atrial fibrillation and if it is rapid and causing symptoms, medications may be used to try to convert the rhythm to normal sinus rhythm or a cardioversion (electric shock to the heart, applied under sedation) may be used. If the rate is slower, the doctor may wait to see if it converts back to a normal sinus rhythm on its own.

For patients with chronic atrial fibrillation, the goal is to control the rate so the effective average is between 50 and 100 beats per minute. This is accomplished through the use of various medications. The first example shows a properly controlled atrial fibrillation. The second example shows a rate which is too slow which was corrected by revising the patient's medication. Patients with chronic atrial fibrillation are also generally required to take a blood thinner to reduce the risk of clots occurring in the atrium which could cause a stroke.

Newer procedures called ablations are also available to correct atrial fibrillation. In some cases, pacemakers may also be indicated if the physician decides to block the impulses from the atrium from reaching the ventricles if the effective rate is too fast and other options have proven ineffective.

Atrial Flutter

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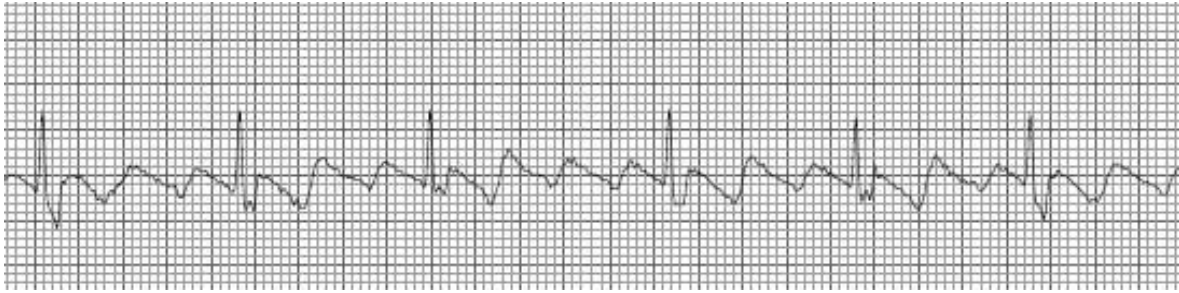
Atrial flutter is a rhythm closely related to atrial fibrillation. Patients may often have both rhythms at different times or may have a rhythm called fibrillation/flutter when the rhythm has characteristics of both or switches back and forth between them. The distinguishing characteristic of atrial flutter is that the P waves are replaced by what are called F waves.

The atrial rate in atrial flutter is regular, but fast, ranging from 150-400 beats per minute. As in atrial fibrillation, only some of the impulses reach the ventricles as the rest are blocked by the AV node. In the above example, variously either 1 out of every 3 or 1 out of every 4 Flutter waves reach and cause the ventricles to contract. In the above example, the atrial rate is approximately 215 beats per minute and the ventricular rate varies from 56-72 beats per minute. Treatment options for atrial flutter are essentially the same as for atrial fibrillation.

Junctional Rhythm
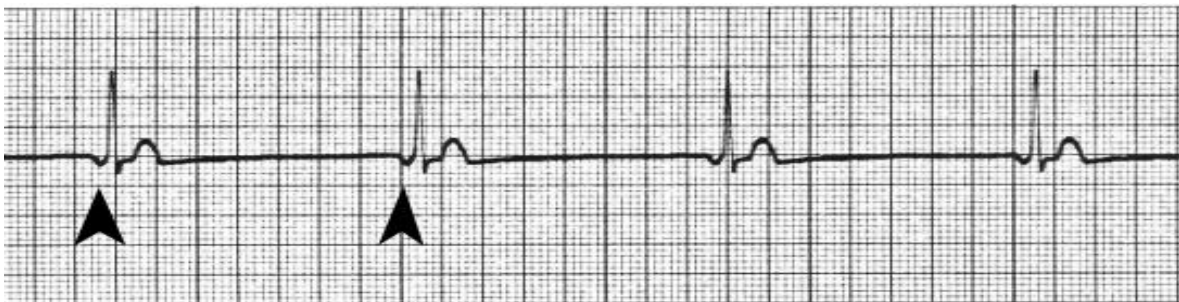
Junctional rhythms occur when the AV node takes over as the primary pacemaker site in the heart either because the SA node has failed or the AV node is going faster and over takes the SA node. Junctional rhythms are classified as follows:

➢ Juntional Rhythm, rate is 40-60 beats per minute

➢ Accelerated Junctional Rhythm, rate is 60-100 beats per minute

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> ➢ Junctional Tachycardia, rate is greater than 100 beats per minute

When a junctional rhythm (rate 40-60 beats per minute) occurs, it is actually a backup pacemaker and is a design feature of the heart. Without a backup, when the SA node fails, the heart may stop. Depending on the heart rate, patients with junctional rhythms may complain of slow rates, light-headedness or dizziness if the rate is slow or rapid rates if the rate is fast. Treatment is directed at first determining the underlying cause and then taking appropriate action.

In the above example, the PR interval is very short (.06 seconds v a normal of 0.12-.20 seconds) and the P wave is inverted (it is actually merging into the front of the QRS complex). The first two P waves are shown by the arrows. The presence of inverted P waves indicates that the primary pacemaker has shifted to the AV node (in this case probably high in the AV node). The heart rate is 41 beats per minute.

PACs (Premature Atrial Contractions)

Premature atrial contractions (PACs) are beats which are initiated in the atria or upper chambers of the heart, prematurely, which cause the SA node (the natural pacemaker of the heart) to be interrupted. The terms SVEs (supraventricular extrasytoles) and PJCs (premature junctional contractions) are also often used when describing these beats but the distinctions are beyond the scope of this discussion.

PACs are one of the two most common heart rhythm abnormalities observed, the other being PVCs (premature ventricular contractions). They are frequently benign and require no treatment. However, in some cases they may be so frequent (over 15-20/minute) that they may cause the heart to beat inefficiently enough to cause symptoms which may need to be addressed. Occasionally, patients who have PACs may also have atrial fibrillation at other times.

PACs may occur singly, in pairs, in short runs or every other beat (bigeminy) and also may be aberrant or non-conducted. Examples of all of these are shown below. Patients who have these types of rhythm abnormalities may often refer to them as palpitations, skipped beats,

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hard beats, irregular beats, missing beats or extra beats. They may also complain of feeling dizzy or lightheaded or experience chest pain. Some patients may have no symptoms at all.

Just to reiterate, PACs are premature beats or beats occurring earlier than they should. Many patients describe them as skipped beats, because when they check their pulse, they don't feel anything for a moment. However, your heart is not actually skipping or missing a beat. What is happening is that when a beat occurs prematurely, the normal volume of blood has not yet returned to your heart from the previous beat. So, even though your heart contracts, not enough blood has returned from the previous beat for it to pump the normal amount of blood. Because of reduced blood being pumped, it may feel like you have skipped a beat, but you have not, although the beat was certainly not as effective as a normal beat.

Patients frequently experience more of these palpitations at night or when they are relaxing. This is because when the natural pacemaker of the heart (the SA node) slows down, as it frequently will when you are relaxed, these ectopic (out of the wrong place) foci (point of origins) do not get reset soon enough to stop them.

Single PACs
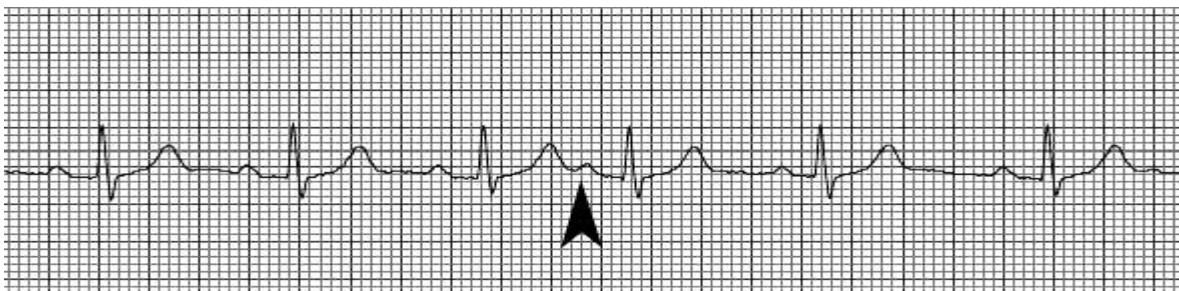
The above is an example of a single, isolated PAC. The heart rate in this example is 79 beats per minute. The first three beats are normal, but the fourth beat occurs early. You can see the P wave right after the T wave of the previous beat. This beat occurs at a rate of 106 beats per minute. We know it is a PAC (instead of a PVC) because a P wave immediately

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precedes it and the QRS is a normal shape which means the beat originated in the upper chambers (atria) of the heart.

Pairs and brief runs of PACs

The above is an example where the underlying rhythm is Normal Sinus Rhythm at 75 beats per minute. In addition, it contains a PAC pair, a single PAC and a 3 beat run of PACs. The first beat is normal, and then we have the PAC pair, then a normal beat, then a single PAC, then 2 normal beats, then a 3 beat run of PACs followed by a normal beat. If a run of PACs contains more than 3 beats it is called a PSVT or PAT.

Bigeminal PACs
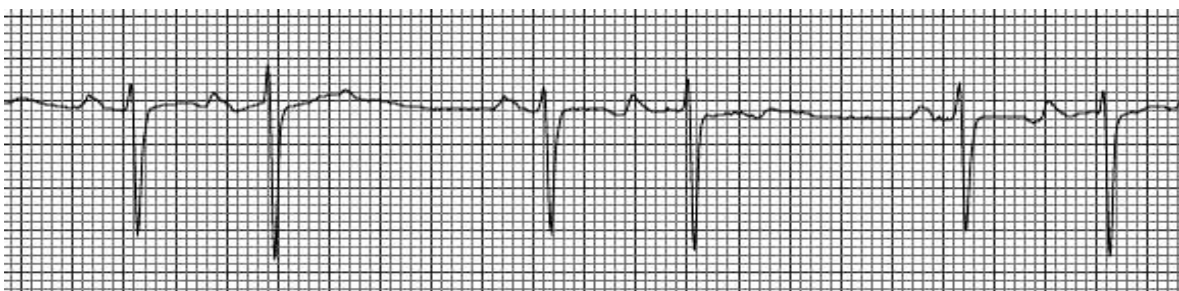
The above is an example of bigeminal PACs. Bigeminal PACs are PACs which occur every other beat. Patients with bigeminal PACs that continue for a while (over 30 seconds), may feel lightheaded or faint. This is because as explained above, the premature beats are not pumping blood very effectively and the body is not receiving enough oxygen.

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Aberrantly conducted PACs

The above example is a variation of a single PAC (the third beat) called an aberrant PAC. In this case, the beat does not look the same as the normal beats (which is usually the case with PACs), but through a number of clues, we know it is coming from above the ventricles. The reason it looks different is that the part of the conduction system that carries the impulse from the atria to the ventricles (the bundle branches) has not yet fully recovered from the previous beat (because this beat occurred early), causing the beat to be slowed down while travelling through one of the bundle branches. This slowing of the signal is what causes the shape to change since one of the ventricles begins to contract before the other ventricle.

Non-conducted PACs

The above example is a nonconducted PAC. The first three beats are normal, but if you look closely at the end of the T wave after the third beat (pointed out by the arrow), you will see a little notch not present in the other T waves. This is actually a P wave, but it does not get conducted to the ventricles, which in effect creates a skipped beat, since the ventricles do

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not contract. The atria contract, causing a P wave, but the ventricles does not. This is a nonconducted PAC. In this case it has likely occurred because the P wave came so early that the rest of the conduction system (the AV node and the bundle branches) did not have time to recover from the previous beat and could not transmit the impulse.

PSVT / PAT / SVT
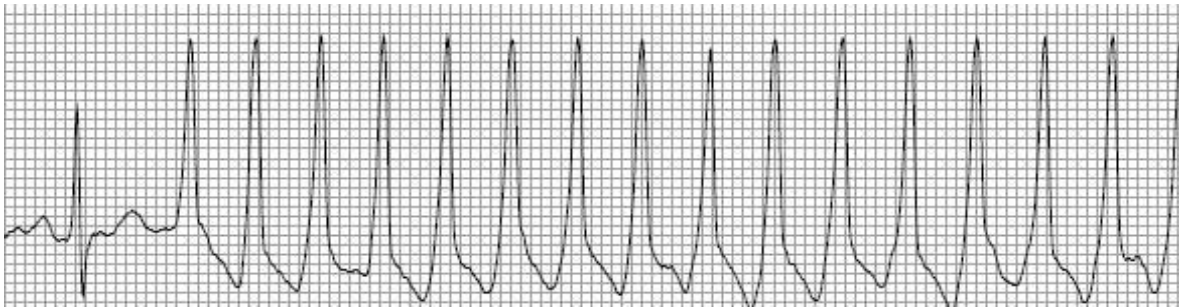
PSVT stands for paroxysmal (which means sudden onset), supraventricular (coming from above the ventricles) tachycardia (rate greater than 100); PAT stands for paroxysmal atrial (originating in the atria) tachycardia; SVT stands for supraventricular tachycardia. The only difference between PSVT and SVT is that the onset of the PSVT can be seen as in the example above. In PATs, the origin of the rapid beats is clearly in the atria whereas in PSVTs and SVTs, a strict determination cannot be made. The duration of these rhythms can vary greatly from a minimum of 4 beats (the minimum number of consecutive beats for it to be classified a SVT) to many hours in duration.

The above rhythm would be described as a PSVT since no P waves can be seen. The first beat is a normal beat with the second beat being the beginning of the PSVT at a rate of 205-225 beats per minute. Patients may describe their symptoms as rapid beats, palpitations or flutters and patients may experience light-headedness, dizziness, SOB, chest pain or anxiety.

These rhythms are not generally life threatening, although emergency medical attention may be required depending on the specific circumstances. Emergency treatment can be as simple as a doctor supervised patient manoeuvre to stop the rhythm, the administration of medications or cardioversion.

If episodes are short and infrequent, no treatment may be necessary. If patients have significant or frequent symptoms, the use of medications or an ablation may be indicated.

CONDUCTION DISTURBANCE

Bundle Branch Block

In a normal heart beat, the impulse travels from the SA node, through the atria, the AV node (where AV blocks occur as described above) and then through the Bundle of His, the bundle branches and the Purkinje fibres to the ventricles. There are two main bundle branches, the right and left, with a further subdivision of the left bundle branch into two minor branches. The determination that a bundle branch block (often referred to as BBB) exists is made by reviewing a patient's EKG. In the presence of a bundle branch block, the QRS is wider than its normal value of .04-.10 seconds and will typically be .12-.16 seconds.

In bundle branch block, only one of the ventricles is directly caused to contract by the impulse from the atria. The other ventricle is actually caused to contract by the impulse travelling through the ventricles heart tissue itself. Since these results in one ventricle contracting before the other, the QRS width is increased. Both right and left bundle branch blocks can occur (with additional classifications due to the fact that there are two minor branches of the left bundle). These are referred to as right and left bundle branch blocks or RBBB and LBBB.
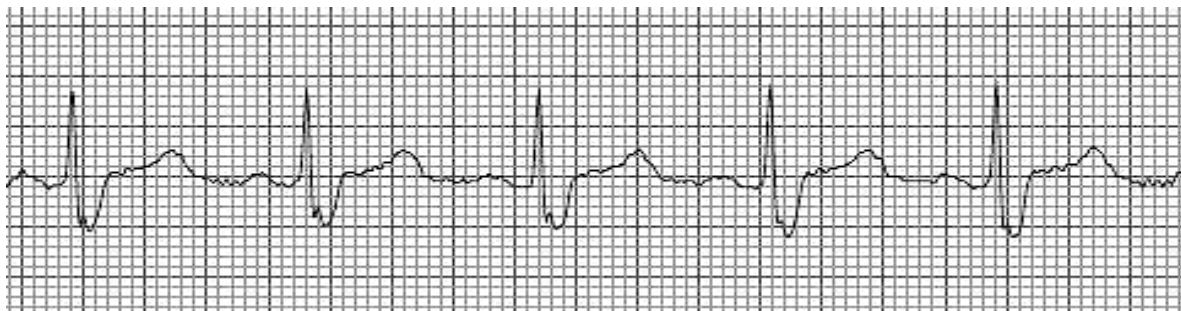
In the example above, the rate is 78 beats per minute, the PR interval is .18 seconds (normal) and the QRS width is .14 seconds (abnormal). The significance of a bundle branch block varies greatly. Some people can be born with them, while in others; they can develop slowly as people get older. When a complete bundle branch block does not exist, but some degradation has occurred, the QRS may vary from .10 to .11 seconds. This is often referred to as an IVCD (intraventricular conduction defect). In other people, the BBB may be a new

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finding which suggests that some underlying heart disease may exist or the patient may have had some type of recent cardiac event.

In many cases of bundle branch block no treatment is required. In other cases, the bundle branch block may indicate the existence of some underlying heart disease and that underlying heart disease may need to be treated.

First Degree AV Block

First degree AV block is simply an increase in the time it takes for the impulse from the atrium to reach the ventricles. In a normal heart rhythm, the PR interval is in the range of .12 to .20 seconds. In first degree AV block, that interval will exceed .20 seconds and can be as long as .50 seconds in extreme examples. The cause for this delay lies in the AV node. The AV node is suppose to cause a certain amount of delay in the impulse reaching the ventricles to allow for the ventricles to fill with blood, but in first degree AV block, this delay is increased.
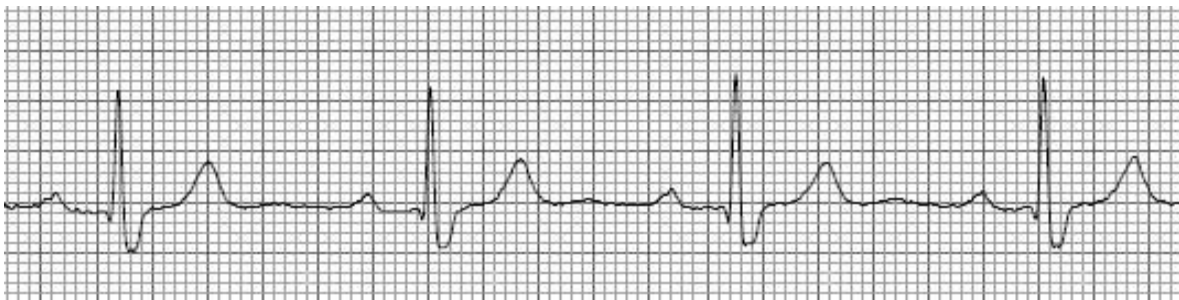
In the above example, the heart rate is 55 beats per minute and the PR interval is .27 seconds. (In addition, the QRS is .13 seconds which means this patient also has a bundle branch block, which is described below.) It is not uncommon to have an AV block and a bundle branch block in the same patient. First degree AV block is the most common conduction disturbance. It may occur in healthy as well as diseased hearts. It also can vary with heart rate. As the rate decreases, the PR interval can get longer and as the rate increases, it can get shorter. First degree AV block is not uncommon in well conditioned athletes with slow resting heart rates. It is also quite common in elderly patients without heart disease. Occasionally, it can be a result of medications. Patients will not generally have any symptoms.

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Second Degree AV Block

There are two different forms of second degree heart block called Mobitz type I (Wenckebach after the person who first described it) and Mobitz type II. In both forms of second degree AV block, the electrical impulse is delayed by the AV node and occasionally, the impulse is blocked completely with the result being the ventricles do not contract, and therefore no blood is pumped through the body. The difference between the two forms of block is that in Mobitz type I, the PR interval progressively lengthens until an impulse is blocked. In Mobitz type II, the PR interval is prolonged but constant with an occasional impulse being blocked. Of the two forms, Mobitz type II is generally considered more serious because it is more likely to progress to high degree or 3rd (complete) degree blocks which are described below. Patients with second degree block will frequently complain of palpitations or skipped beats or may feel lightheaded or dizzy. They may occasionally experience near syncope or syncope (passing out).
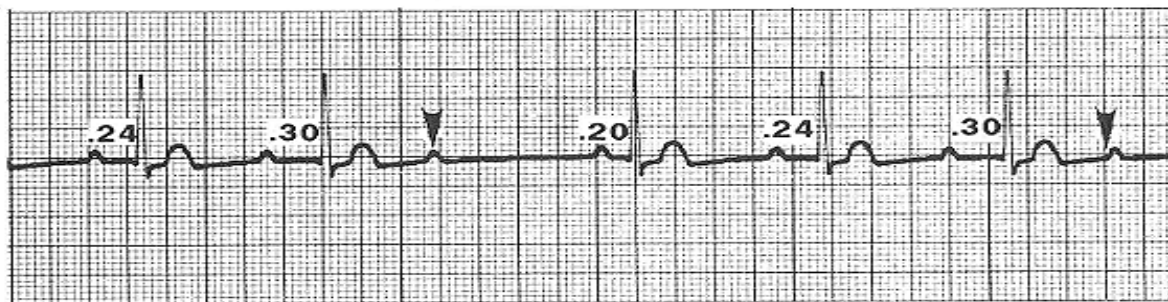
The above is an example of Mobitz type I. The distinguishing characteristic is that the PR interval progressively lengthens from .20 to .30 seconds until one of the P waves is not conducted to the ventricles. This causes the heart to in effect, skip or drop a beat. The arrows point to the dropped beats when the P waves were not conducted to the ventricles. Treatment is often not indicated with Mobitz type I.

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The above is an example of Mobitz type II. The distinguishing characteristic is that the PR interval for conducted beats is .18 seconds, but some of the P waves are not conducted to the ventricles. In the above example, the arrows point to the P waves that were not conducted to the ventricles. Treatment is generally indicated with this rhythm and may consist of medications or an implantable pacemaker

Third, High or Complete AV Block

In high degree AV block, two out of every three or more impulses from the atria are blocked by the AV node and fail to reach the ventricles. In third (complete) AV block, all the impulses from the atria are blocked by the AV node. This results in the heart rate being much slower than normal. In certain cases if the subsidiary (backup) pacemakers in the ventricles are absent, the result can be that the ventricles may fail to contract and the heart may stop. These abnormalities may be caused by drug toxicity in which case, modifying the patient's medications may correct the problem. In other cases, a permanent pacemaker may be required.
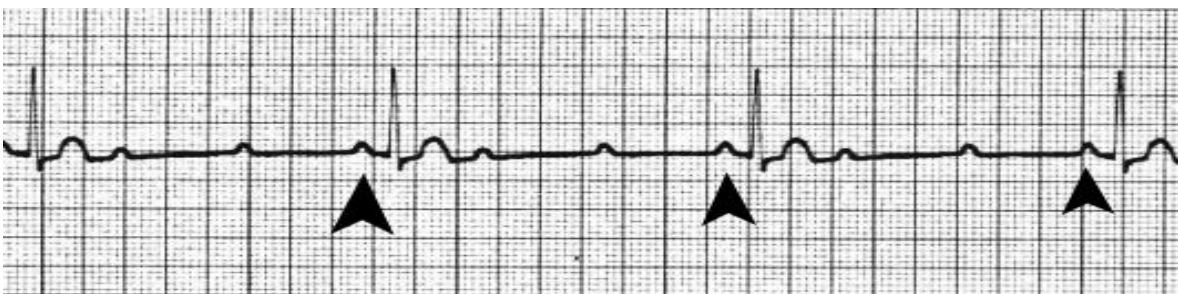
The above is an example of High Degree AV block. The distinguishing characteristic is that two out of every three P waves are blocked from reaching the ventricles, resulting in an effective heart rate of 34 beats per minute. The arrows point to the P waves that are conducted to the ventricles. The other P waves are blocked by the AV node.

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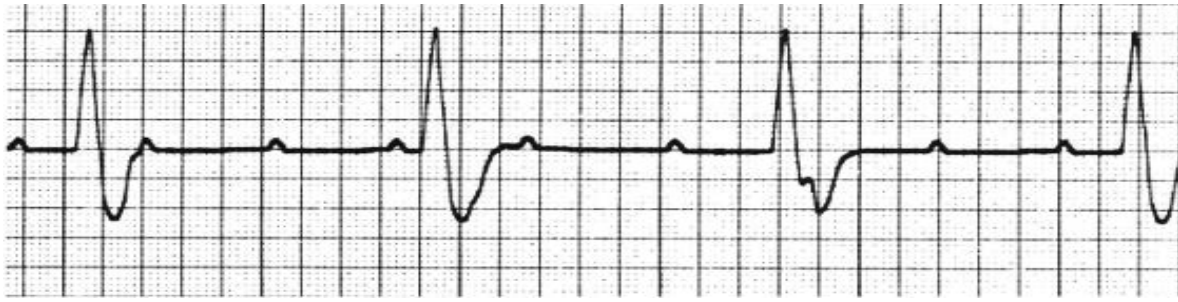
The above is an example of complete heart block. This rhythm is an idioventricular rhythm. The distinguishing characteristic is that no P waves from the atria are conducted to the ventricles. A careful examination will show that the PR interval is random as a result of there being no relationship between the P wave and the QRS complex. This is also referred to as AV dissociation. The QRS complex in this rhythm actually originates in the ventricles (we know this because there is no relationship between the P wave and the QRS and because the QRS complex is wide, nearly .36 seconds) and the effective heart rate is 33 beats per minute.

VENTRICULAR ARRHYTHMIAS

PVCs (Premature Ventricular Contractions)

Premature ventricular contractions (PVCs) are beats which are initiated in the ventricles or lower chambers of the heart, prematurely. As opposed to PACs, when the SA node (the natural pacemaker of the heart) gets interrupted, PVCs do not interrupt the SA node. However, with PVC the ventricles contract, which normally causes the impulse from the atria to be blocked from reaching the ventricles.

PVCs are one of the two most common heart rhythm abnormalities, the other being PACs (premature atrial contractions). They are frequently benign and require no treatment. However, in some cases they may be so frequent (over 15-20/minute) that they may cause the heart to beat inefficiently enough to cause symptoms which may need to be addressed.

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PVCs may occur singly or in pairs (generally referred to as couplets), every other beat (bigeminy) or interpolated and may also be described as multiform. Examples of all of these are shown below. (Three or more consecutive PVCs are technically referred to as ventricular tachycardia.) Patients who have these types of rhythm abnormalities may often refer to them as palpitations, skipped beats, hard beats, irregular beats, missing beats or extra beats. They may also complain of feeling dizzy or lightheaded or experience chest pain. Some patients may have no symptoms at all.

Just to reiterate, PVCs are premature beats or beats occurring earlier than they should. Many patients describe them as skipped beats, because when they check their pulse, they don't feel anything for a moment. However, your heart is not actually skipping or missing a beat. What is happening is that when a beat occurs prematurely, the normal volume of blood has not yet returned from your body from the previous beat. So, even though your heart contracts, not enough blood has returned from the previous beat for it to pump the normal amount of blood. Because of reduced blood being pumped, it may feel like you have skipped a beat, but you have not, although the beat was certainly not as effective as a normal beat.

Patients frequently experience more of these palpitations at night or when they are relaxing. This is because when the natural pacemaker of the heart (the SA node) slows down as it frequently will when you are relaxed, these ectopic (out of the wrong place) foci (point of origins) do not get reset soon enough to stop them.

Single PVCs

The above is an example of two isolated PVCs. The heart rate in this example is 70 beats per minute. The first beat, third, fourth and fifth beats are normal, the second and sixth beats

are PVCs. There are a number of identifying characteristics which allow us to identify them as PVCs. First, the QRS complex is wide, causing the beat to look strange. (These beats are sometimes referred to euphemistically as FLBs or funny looking beats.) Second, there is a delay after the PVC called a compensatory pause, while the ventricles are waiting for an impulse to reach them from the atria. Third, we can actually see that the atria have contracted and that the SA node has not been reset. If you look carefully in the portion of the QRS complex closest to the bottom of the strip, you will see the waveform rise briefly (arrow), and then go down again before rising again. This is actually the P wave, which are the atria contracting. However, this impulse is blocked because the ventricles have just contracted. The next P wave is then conducted normally to the ventricles.

Bigeminal PVCs
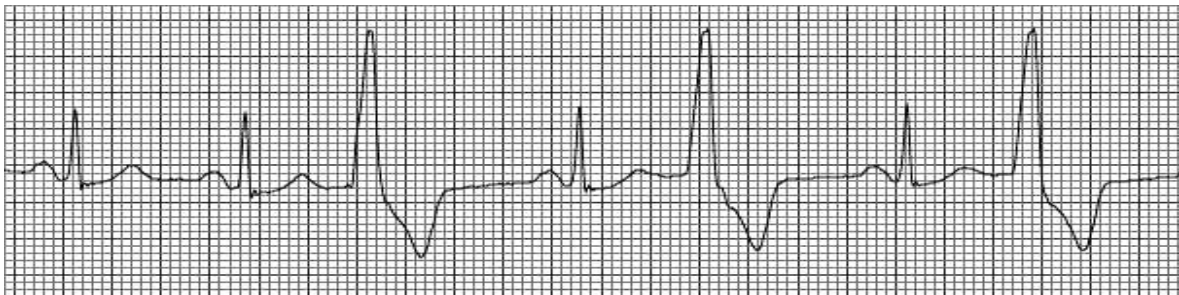
The above is an example of three bigeminal PVCs. The heart rate in this example is 83 beats per minute. The first, second, fourth and sixth beats are normal and the third, fifth and seventh are PVCs. PVCs which occur every other beat are called bigeminal PVCs. There may be just a few of these or they may continue for some time.

Multiform Multiform PVCs
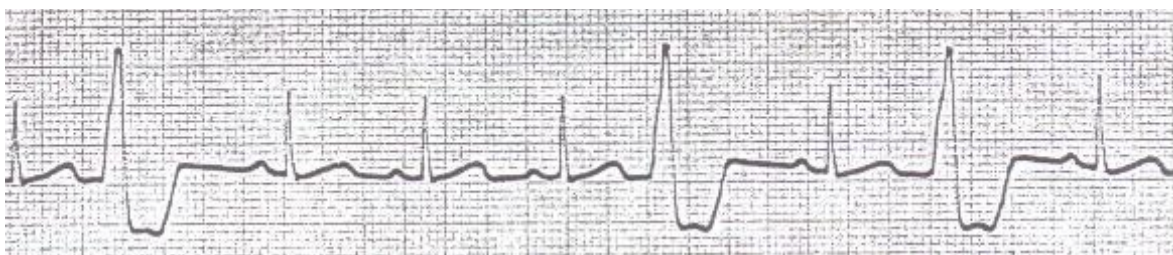
The above is an example of multiform PVCs (sometimes incorrectly referred to as multifocal PVCs). The heart rate in this example is 63 beats per minute. The first, third and fifth beats

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are normal beats (although the observant reader will note that this patient has a bundle branch block) and the second and fourth beats are PVCs, although they are different shapes, thus the term multiform. Generally, they are a different shape because they are originating in two different areas of the ventricles.

Ventricular Couplets
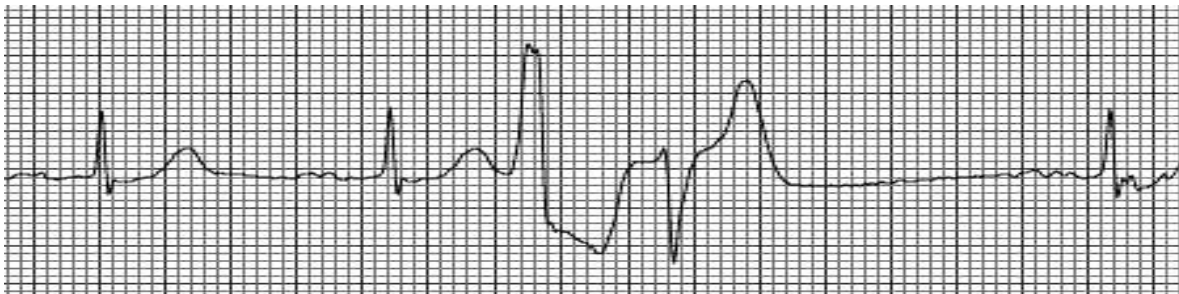
The above is an example of a ventricular couplet or two consecutive PVCs. The heart rate in this example is 68 beats per minute. In this case they are also multiform PVCs. The first, second and fifth beats are normal and the third and fourth beats are PVCs.

Interpolated PVCs

The above is an example of interpolated PVC. The heart rate in this example is 68 beats per minute. The first, second, fourth, fifth and sixth beats are normal and the third beat is a PVC. An interpolated PVC is one which is "sandwiched" between two normal beats and therefore does not have a compensatory pause after the PVC. This is because the P wave (which is

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hidden in the QRS complex) is still conducted to the ventricles and causes them to contract, although there may be a slight delay.

Ventricular Fibrillation
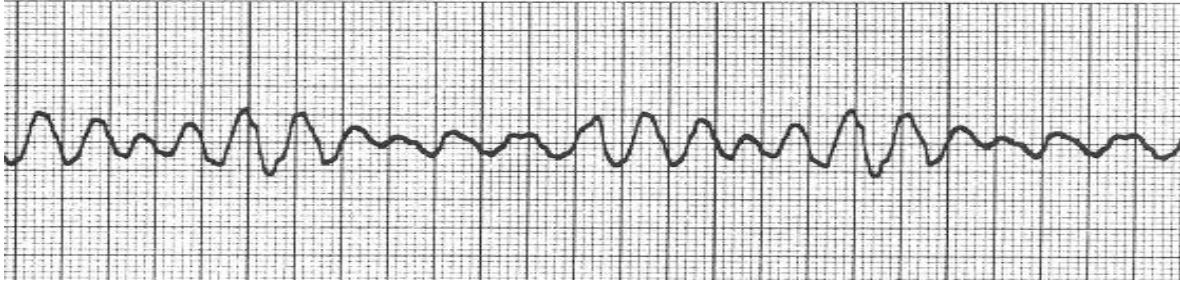
Ventricular fibrillation (much different that atrial fibrillation) is an ominous rhythm in which there is no clear distinction between the various EKG complexes (P wave, QRS, T wave) and there are no effective cardiac contractions. The result is no cardiac output (blood flow) and no blood pressure. Immediate emergency medical attention is required.

Ventricular Standstill

Ventricular standstill is the absence of any ventricular activity for more than a few seconds. There may be atrial activity as evidenced by P waves in which case complete heart block is blocking all impulses from reaching the ventricles and the backup or subsidiary pacemaker has failed, or there may be an absence of atrial and ventricular activity. This is an emergency and requires immediate attention as this rhythm is fatal if a normal rhythm is not restored.

Treatment requires an evaluation of the underlying cause. Medications or a permanent pacemaker may be required.

Ventricular Tachycardia

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Ventricular tachycardia (V-tach) is a rhythm with a rapid recurrence of premature ventricular contractions with no normal beats in between. If this rhythm persists, emergency medical attention is generally required. In the example above, the rate is 190 – 210 beats per minute.

If this rhythm is persistent and reoccurring cannot be corrected through the use of medication or an ablation, the implantation of an AICD (automatic implantable cardiac defibrillator) may be required.

ST CHANGES

ST changes generally refer to an elevation or depression in the ST segment of the EKG complex. Both ST segment elevation and depression must be evaluated in conjunction with any patient symptoms with any treatment being directed at fixing the underlying problem.

ST Depression

The above is technically a sinus tachycardia with a rate of 107 beats per minute with ST depression of approximately 3 mm (3 small boxes). The arrow is pointing at the ST segment

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of the EKG. If you look at Normal Heart Rhythm, you will note that the ST segment is either elevated or depressed which is referred to as being isoelectric.

A thorough evaluation of ST changes requires the use of a 12 lead EKG. ST segment depression can suggest the presence of ischemia, or lack of blood flow to the heart muscle itself. Patients may complain of chest tightness, pressure or pain and/or light-headedness or dizziness in these situations.

ST Elevation
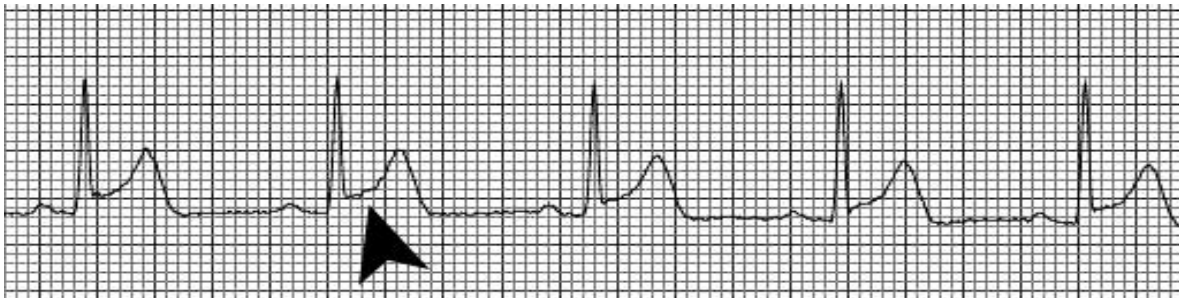
The above is a normal sinus rhythm with a rate of 68 beats per minute with ST elevation of 2 mm (2 small boxes). The arrow is pointing at the ST segment of the EKG. ST elevation in certain leads can be normal, so a thorough evaluation would require a 12 lead EKG and perhaps additional tests if the patient is symptomatic. Symptoms may include chest tightness, pressure or pain and/or light-headedness or dizziness. ST segment elevation can suggest the presence of ischemia, or lack of blood flow to the heart muscle itself.

Notes: _____
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Questions:

1. Cardiac Arrhythmias occur as a result of_____

_____

_____

_____

2. Complete the following sentence;

   ➢ 'Pacemaker cells beat with _____, _____
      _____neighbours'.

   ➢ 'The non-pacemaker cells _____pacemaker
      cells'.

3. Incorrect impulse generation is the fault of; the_____

   _____

   _____

4. Impulse conduction problems are caused; _____

   _____

   _____

5. This problem falls into two further categories, _____

   _____

   _____

6. Usual culprits of cardiac arrhythmia when an electrolyte imbalance occurs: _____

   _____

   _____

7. Hyperkalemia (high potassium levels) can cause; _____

   _____

   _____

8. In relation to the mechanisms of arrhythmias, cardiac arrhythmias result from; _____

   _____

   _____

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9. Abnormal impulse initiation includes; _____

_____

_____

10. Abnormal impulse conduction includes; _____

_____

11. Enhanced normal automaticity refers to: _____

_____

_____

12. Abnormal automaticity refers to: _____

_____

_____

13. Triggered rhythms are caused by: _____

_____

_____

14. Abnormal impulse conduction is due to _____

_____

_____

15. Blocking of cardiac impulses can occur because: _____

_____

16. Reentry occurs when: _____

_____

_____

Notes: _____

_____

_____

_____

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RHYTHM INTERPRETATION

1.

Ventricular rate/rhythm _____

Atrial rate/rhythm _____

P wave _____

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P-R Interval _____

QRS _____

QRS Complex _____

QRS Duration _____

S-T Segment _____

T wave _____

Q-T Interval _____

Diagnose _____

2.

Ventricular rate/rhythm _____

Atrial rate/rhythm _____

P wave _____

P-R Interval _____

QRS _____

QRS Complex _____

QRS Duration _____

S-T Segment _____

T wave _____

Q-T Interval _____

Diagnose _____

3.

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Ventricular rate/rhythm

Atrial rate/rhythm

P wave

P-R Interval

QRS

QRS Complex

QRS Duration

S-T Segment

T wave

Q-T Interval

Diagnose

4.

This rhythm strip is from an 86-year-old woman who experienced a cardiopulmonary arrest. The initial rhythm was asystole. The following rhythm resulted after IV administration of epinephrine and atropine.

Ventricular rate/rhythm

Atrial rate/rhythm

P wave

P-R Interval

QRS

QRS Complex

QRS Duration

S-T Segment

_____

T wave

_____

Q-T Interval

_____

Diagnose

_____

5.

This rhythm strip is from a 69-year-old man complaining of shortness of breath. Lung sounds reveal bilateral rattles. Blood pressure: 160/58.

Ventricular rate/rhythm _____

Atrial rate/rhythm _____

P wave _____

P-R Interval _____

QRS _____

QRS Complex _____

QRS Duration _____

S-T Segment _____

T wave _____

Q-T Interval _____

Diagnose _____

6.

This rhythm strip is from a 52-year-old man found unresponsive and pulseless.

Ventricular rate/rhythm _____

Atrial rate/rhythm   _____

P wave   _____

P-R Interval   _____

QRS   _____

QRS Complex   _____

QRS Duration   _____

S-T Segment   _____

T wave   _____

Q-T Interval   _____

Diagnose   _____

7.

These rhythm strips are from a 78-year-old man complaining of shortness of breath. He has a history of COPD, coronary artery disease, and hypertension.

Ventricular rate/rhythm _____

Atrial rate/rhythm   _____

P wave   _____

P-R Interval   _____

QRS   _____

QRS Complex   _____

QRS Duration   _____

S-T Segment   _____

T wave   _____

Q-T Interval   _____

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Diagnose _____

8.

    This rhythm strip is from a 97-year-old woman after a fall.

Ventricular rate/rhythm _____

Atrial rate/rhythm _____

P wave _____

P-R Interval _____

QRS _____

QRS Complex _____

QRS Duration _____

S-T Segment _____

T wave _____

Q-T Interval _____

Diagnose _____

9.

    This rhythm strip is from a 70-year-old man complaining of a sharp pain across his shoulders. Blood pressure: 218/86.

Ventricular rate/rhythm _____

Atrial rate/rhythm _____

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P wave _____

P-R Interval _____

QRS _____

QRS Complex _____

QRS Duration _____

S-T Segment _____

T wave _____

Q-T Interval _____

Diagnose _____

10.

This rhythm strip is from a 76-year-old man complaining of indigestion.

Ventricular rate/rhythm _____

Atrial rate/rhythm _____

P wave _____

P-R Interval _____

QRS _____

QRS Complex _____

QRS Duration _____

S-T Segment _____

T wave _____

Q-T Interval _____

Diagnose _____

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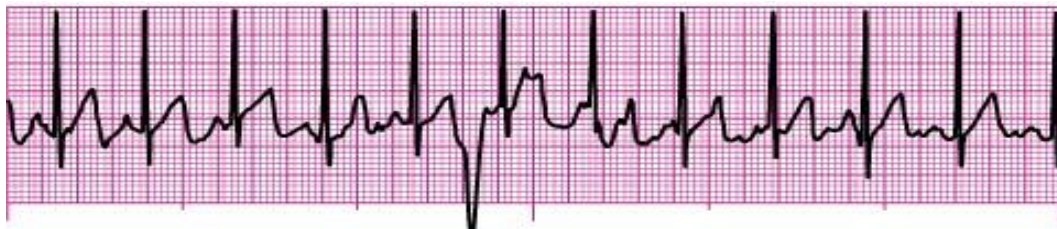2011   Page 65

11.

This rhythm strip is from a 78-year-old man complaining of palpitations. Note the point at which the patient was defibrillated.

Ventricular rate/rhythm _____

Atrial rate/rhythm _____

P wave _____

P-R Interval _____

QRS _____

QRS Complex _____

QRS Duration _____

S-T Segment _____

T wave _____

Q-T Interval _____

Diagnose _____

12.

This rhythm strip is from a 3-month-old infant who had an altered level of responsiveness. She was limp with a respiratory rate of 4 breaths/min. Blood sugar was 174.

Ventricular rate/rhythm _____

Atrial rate/rhythm _____

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P wave _____

P-R Interval _____

QRS _____

QRS Complex _____

QRS Duration _____

S-T Segment _____

T wave _____

Q-T Interval _____

Diagnose _____

13.

This rhythm strip is from an 83-year-old man complaining of chest pain. He had a new pacemaker implanted 5 days ago. His blood pressure is 148/60.

Ventricular rate/rhythm _____

Atrial rate/rhythm _____

P wave _____

P-R Interval _____

QRS _____

QRS Complex _____

QRS Duration _____

S-T Segment _____

T wave _____

Q-T Interval _____

Diagnose _____

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14.

This rhythm strip is from a 52-year-old man with substernal chest pain. He has a history of COPD and mitral valve regurgitation. Blood pressure: 140/78.

Ventricular rate/rhythm _____

Atrial rate/rhythm _____

P wave _____

P-R Interval _____

QRS _____

QRS Complex _____

QRS Duration _____

S-T Segment _____

T wave _____

Q-T Interval _____

Diagnose _____

15

This rhythm strip is from a 77-year-old woman with a congested cough.

Ventricular rate/rhythm _____

Atrial rate/rhythm _____

P wave _____

P-R Interval _____

QRS   _____

QRS Complex   _____

QRS Duration   _____

S-T Segment   _____

T wave   _____

Q-T Interval   _____

Diagnose   _____

16.

This rhythm strip is from a 66-year-old man complaining of chest pain. Blood pressure: 170/96.

Ventricular rate/rhythm _____

Atrial rate/rhythm   _____

P wave   _____

P-R Interval   _____

QRS   _____

QRS Complex   _____

QRS Duration   _____

S-T Segment   _____

T wave   _____

Q-T Interval   _____

Diagnose   _____

17.

This rhythm strip is from a 43-year-old woman complaining of palpitations.

Ventricular rate/rhythm _____

Atrial rate/rhythm _____

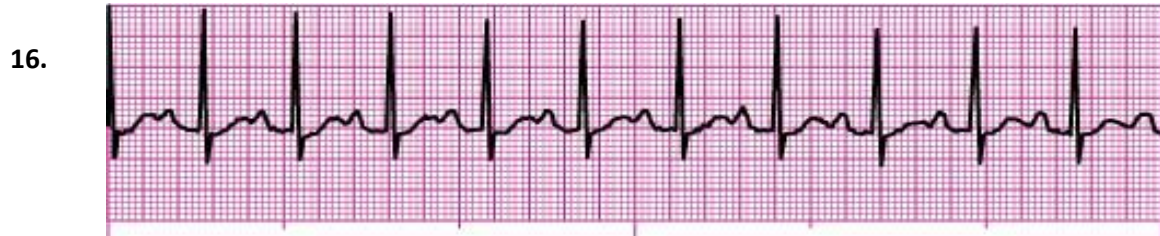P wave _____

P-R Interval _____

QRS _____

QRS Complex _____

QRS Duration _____

S-T Segment _____

T wave _____

Q-T Interval _____

Diagnose _____

18.
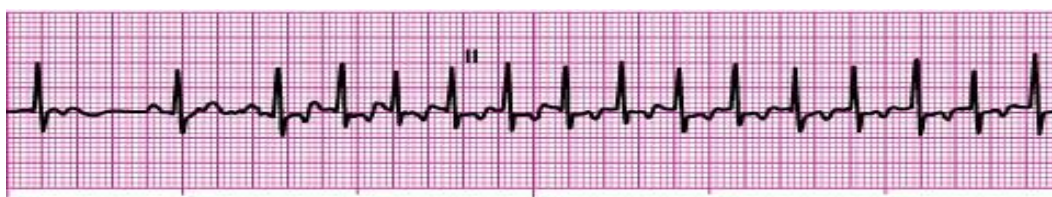
A 43-year-old woman is complaining of palpitations. The patient has a history of SVT and states she cannot tolerate adenosine. The following rhythm is observed on the cardiac monitor after diltiazem administration

Ventricular rate/rhythm _____

Atrial rate/rhythm _____

P wave _____

P-R Interval _____

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QRS   _____

QRS Complex   _____

QRS Duration   _____

S-T Segment   _____

T wave   _____

Q-T Interval   _____

Diagnose   _____

19.

This rhythm strip is from a 73-year-old man complaining of chest pain. History of hypertension and lung disease. Medications include aspirin, albuterol, and Lotensin.

Ventricular rate/rhythm _____

Atrial rate/rhythm   _____

P wave   _____

P-R Interval   _____

QRS   _____

QRS Complex   _____

QRS Duration   _____

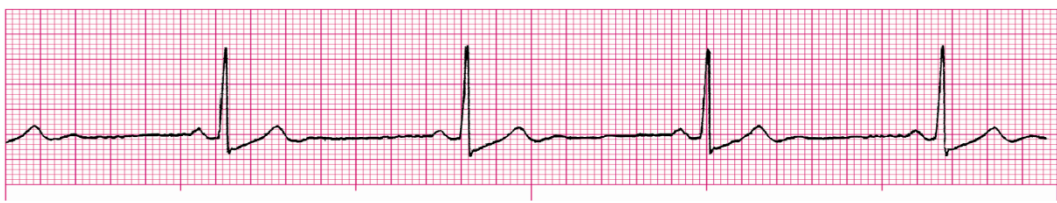S-T Segment   _____

T wave   _____

Q-T Interval   _____

Diagnose   _____

20.

This rhythm strip is from a 51-year-old man complaining of dull chest pain that began about 2 hours ago. He rates his discomfort as a 6 on a scale of 1 to 10. BP: 70/48, R: 24. His skin is cool, pale, and diaphoretic.

Ventricular rate/rhythm

Atrial rate/rhythm

P wave

P-R Interval

QRS

QRS Complex

QRS Duration

S-T Segment

T wave

Q-T Interval

Diagnose

Notes

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EVALUATION FORM

Topic: Module 3: Cardiac Arrhythmias: Mechanisms of Arrhythmias – Atrial, Ventricular, Conduction and ST Changes.

We want to ensure that the training/education you have received is effective and relevant. We would be grateful if you would complete this evaluation. Please circle the most appropriate rating.

(The response range from 1 for limited use, to 5 for very useful)

Circle your choice

1. **Please rate the overall value of the Self Learning Package**

Module 3: Cardiac Arrhythmias: Mechanisums of Arrhythmias – Atrial, Ventricular, Conduction and ST Changes
Developed by Tony Curran (Clinical Nurse Educator) and Gill Sheppard (Clinical Nurse Specialist) Cardiology, June 2011
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			1	**2**	**3**	**4**	**5**

Comments:

2. **Please rate how relevant the information was to your practice**

			1	**2**	**3**	**4**	**5**

Comments:

3. Please rate the presentation of the Self Learning Package

			1	**2**	**3**	**4**	**5**

Comments:

Please add any further comments you consider may improve the package.