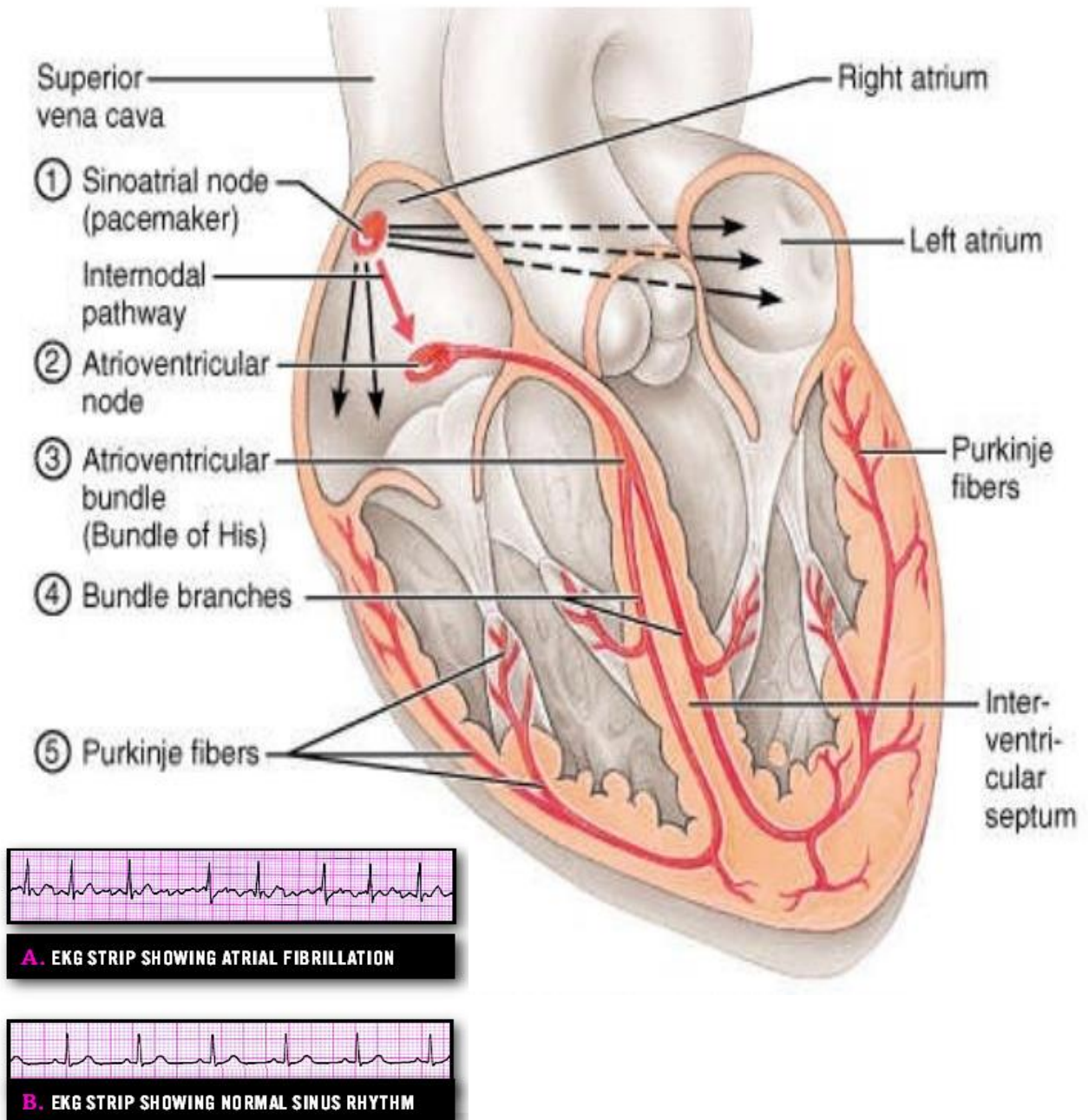


Nursing Management of Patients with Dysrhythmias and Conduction Problems

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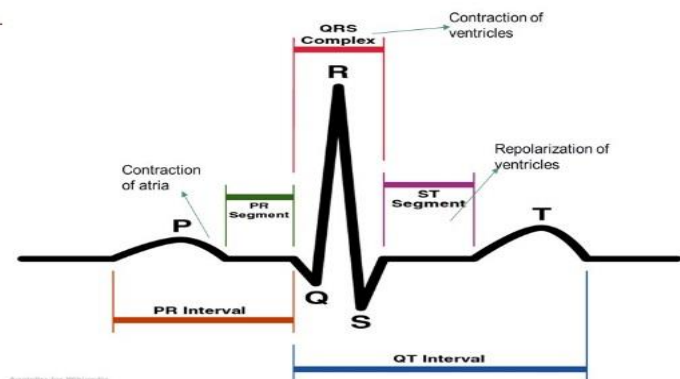
Conductive system of the heart

1. **Sinus node "SA" node:** also called sinoatrial node, located in the right atrium. It is concerned with the generation of rhythmical impulse; it is the pacemaker of the heart that initiates each heart beat. This automatic nature of the heart beat is referred to as automaticity.
2. **Internodal pathways** conduct the impulse generated in SA node to the AV node.
3. **The AV node** (atrioventricular node), located near the right AV valve at the lower end of the interatrial septum, in the posterior septal wall of the right atrium. At which impulse from the atria is delayed before passing into the ventricles.
4. **The AV bundle** (bundle of His) conducts the impulse from the atria into ventricles.
5. **The left and right bundles of Purkinje fibers**, which conduct the cardiac impulse to all parts of the ventricles. The Purkinje fibers distribute the electrical excitation to the myocytes of the ventricles.

The rhythmic sequence of contractions is coordinated by the sinoatrial (SA) and atrioventricular (AV) nodes. The sinoatrial node, often known as the cardiac pacemaker, is

located in the upper wall of the right atrium and is responsible for the wave of electrical stimulation that initiates atrial contraction. Once the wave reaches the AV node, situated in the lower right atrium, it is delayed there before being conducted through the bundles of His and back up the Purkinje fibers, leading to a contraction of the ventricles. The delay at the AV node allows enough time for all of the blood in the atria to fill their respective ventricles. In the event of severe pathology, the AV node can also act as a pacemaker; this is usually not the case because their rate of spontaneous firing is considerably lower than

ECG (EKG) showing wave segments



that of the pacemaker cells in the SA node and hence is overridden.

Every cardiac cycle produces ECG waves designated as P, Q, R, S and T. They represent potentials between rested and depolarized or depolarized and repolarized

parts of whole heart. Amplitude and duration of these waves correspond to electrical power fluctuation in entire heart.

After producing impulse in SA-node depolarization begins at first in cells of right atrium and ascend part of P wave is recorded. When depolarization spreads into left atrium, the ECG line returns to baseline level. Delay of depolarization in AV-node recorded as PQ-interval in baseline. Then impulse spreads into middle part of septum and heart apex. This event recorded as descend part of Q wave. In next depolarization of right ventricle wall ECG line deflexed upward and formation of R wave begins. When impulse spreads into left ventricle wall, the ECG line returned in contrary side towards the lowest point of S wave. Depolarization of ventricles basis afterwards caused formation of S wave, which continues to baseline.

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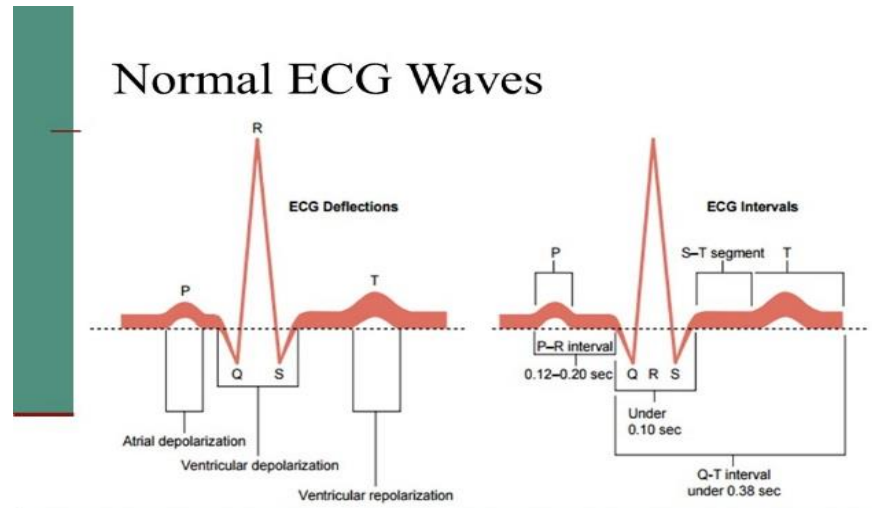


Figure 2: EKG Waves and intervals

continues to baseline.

P wave: The P wave represents the wave of depolarization that spreads from the SA node throughout the atria, and is usually 0.08 to 0.1 seconds (80-100 ms) in duration.

P-R interval: The period of time from the onset of the P wave to the beginning of the QRS complex, normally ranges from 0.12 to 0.20

seconds in duration. This interval represents the time between the onset of atrial depolarization and the onset of ventricular depolarization. If the P-R interval is >0.2 sec, there is an AV conduction block, which is also termed a first-degree heart block if the impulse is still able to be conducted into the ventricles.

QRS complex: The QRS complex represents ventricular depolarization. Ventricular rate can be calculated by determining the time interval between QRS complexes. The duration of the QRS complex is normally 0.06 to 0.1 seconds. This relatively short duration indicates that ventricular depolarization normally occurs very rapidly. If the QRS complex is prolonged (>0.1 sec), conduction is impaired within the ventricles. This can occur with bundle branch blocks.

ST segment: The isoelectric period (ST segment) following the QRS is the time at which the entire ventricle is depolarized and roughly corresponds to the plateau phase of the ventricular action potential. The ST segment is important in the diagnosis of ventricular ischemia or hypoxia because under those conditions, the ST segment

can become either depressed or elevated.

T wave: The T wave represents ventricular repolarization and is longer in duration than depolarization (i.e., conduction of the repolarization wave is slower than the wave of depolarization). Sometimes a small positive U wave may be seen following the T wave. This wave represents the last remnants of ventricular repolarization. Inverted or prominent U waves indicates underlying pathology or conditions affecting repolarization.

Q-T interval: The Q-T interval represents the time for both ventricular depolarization and repolarization to occur, and therefore roughly estimates the duration of an average ventricular action potential. This interval can range from 0.2 to 0.4 seconds depending upon heart rate. Normal corrected Q-Tc intervals are less than 0.44 seconds.

Interpreting Dysrhythmia

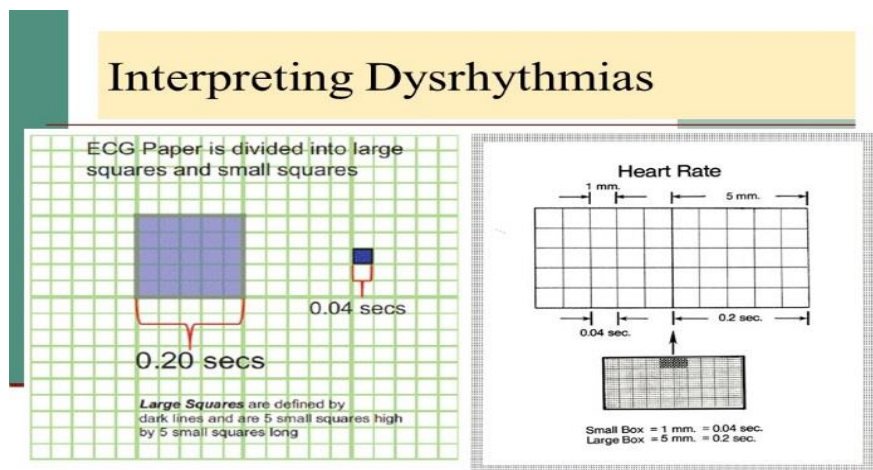


Figure 3: Interpreting cardiac rhythms

P WAVES

- Does a P wave precede every QRS?
- Do the P waves all look the same?
- Are the P waves regular?

PR INTERVAL

- Is the PR interval normal length?

QRS WAVES

- Do the QRS's all look the same?
- Are the QRS's regular?
- What is the ventricular rate?
- Is the QRS normal length?

ST SEGMENT

- Where is the ST segment?
- Are there any extra waves? If yes, where in the cycle?

Cardiac Arrhythmia (Dysrhythmias)

Disorders of electrical impulse formation or conduction (or both) within heart. Can cause disturbances of Rate, Rhythm or both. Potentially can alter blood flow and cause hemodynamic changes. Diagnosed by analysis of electrographic waveform.

Normal sinus rhythm

Normal sinus rhythm (NSR) is the characteristic rhythm of the healthy human heart. NSR is the rhythm that originates from the sinus node. The rate in NSR is generally regular but will vary depending on autonomic inputs into the sinus node. There can be an irregularity in the sinus rate and, when this occurs, it is termed "**sinus arrhythmia**". A sinus rhythm faster than the normal range is called a sinus tachycardia, while a slower rate is called a sinus bradycardia.

Causes of Cardiac Arrhythmia

Sinus Bradycardia: sinus rhythm with HR less than 60/min arising from the SA node.

Impulses follow the normal pathway through the conduction system. P wave and QRS complexes normal duration and pattern. May occur as a result of increased vagal stimulation and as a normal variation in athletes and

healthy young adults. May be as a result of certain medical conditions such as: anorexia nervosa, atherosclerotic heart disease, hypopituitary states, hypothermia,

increased intracranial pressure, and myocardial infarction. Certain medicines such as: certain antihypertensive drugs, beta blockers, calcium channel blockers, central nervous system

Normal Sinus Rhythm

- Rhythm: Regular
- Rate: 60 - 100
- P wave: Upright & uniform
- PR interval: 0.12 – 0.20 second
- QRS: 0.06 – 0.12 second
- P to QRS ratio = 1:1

(CNS) depressants and digoxin can cause sinus bradycardia.

Symptoms of bradycardia: symptoms are related to decrease in cardiac output. They include; Chest pressure and pain, Dyspnea, Hypotension, Dizziness, Seizures, Syncope.

Management: only if patient is symptomatic- aimed at increasing the heart rate and cardiac output. Medications that may be used include atropine and isoproterenol. Pacemaker- pacing is used if patient is hemodynamically compromised, suppression of the parasympathetic nervous system, stimulation of the sympathetic nervous system.

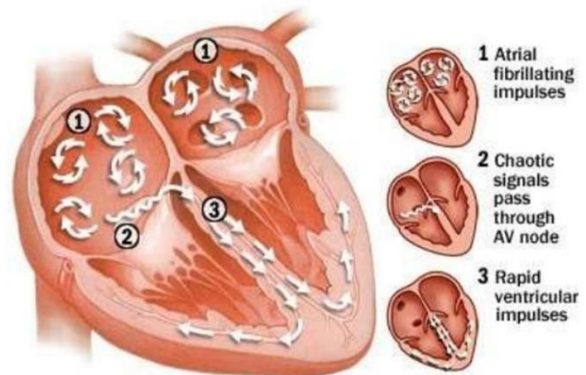
Sinus Tachycardia: sinus tachycardia being defined as a sinus rhythm with a rate exceeding 100 beats per minute. Heart rate of 100-160/ min, May occur as a normal response to sympathetic nervous system stimulation, and any condition that produces an increase in metabolic rate. Sinus tachycardia can result from caffeine use, smoking / nicotine, certain medical conditions such as anemia, hemorrhage, fever, hypotension, pain, shock, and myocardial damage, and medications like central nervous system stimulants.

Symptoms of tachycardia: primary symptoms are related to decreased cardiac output, they include chest pressure and pain, dyspnea, a characteristic “fluttering” in the chest, dizziness, and syncope.

Management of tachycardia: find and treat the cause of the tachycardia. Medications such as calcium channel blockers, digoxin, beta blockers, antianxiety agents, adenosine and carotid massage.

ATRIAL DYSRHYTHMIAS

Atrial Rhythms

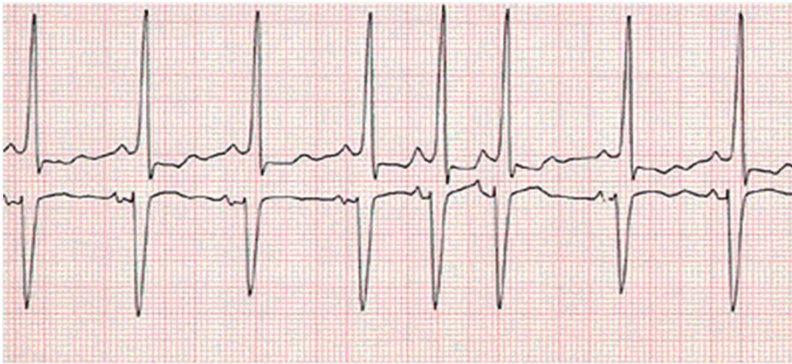


Electrical impulses that originate from the atrium (not from the SA node).

Atrial rhythms originate in the atria rather than in the SA node. The P wave will be positive, but its shape can be different than a normal sinus rhythm because the electrical impulse follows a different path to the AV (atrioventricular) node.

Atrial dysrhythmias include wandering atrial pacemaker, premature atrial contractions, paroxysmal atrial tachycardia, atrial flutter, and atrial fibrillation.

Premature atrial contractions (PAC): also known as atrial premature complexes are the most common type of arrhythmias. PACs occur due to the premature discharge of an electrical impulse in an irritable area of the atria, causing a premature contraction. A PAC



is premature, because they occur earlier than the next regular beat should have occurred. There are abnormally shaped P waves, QRS complex is not affected. Most common symptom is palpitations often reported as "missing" or "skipping" of the heartbeat.

Causes of PACs: Stress, Stimulants (Caffeine, Tobacco, Alcohol), Hypertension, Valve disorder, previous myocardial infarct, abnormal blood levels of magnesium and/or potassium,

Digitalis toxicity. In the majority of cases, PACs occur in normal healthy individuals without any evidence of heart disease. The great majority of PACs are completely benign and require little if any treatment at all.

Management: In rare cases, PACs may be the only sign of underlying heart conditions and these should be ruled out with appropriate evaluations. However, PACs may change into atrial flutter, atrial fibrillation, or

supraventricular tachycardia. Treatment directed toward cause treatment not necessary if less than 6 per minute, decrease caffeine consumption, decrease stress, antianxiety agents, beta blockers and calcium channel blockers may be administered if symptoms are severe enough.

Paroxysmal atrial tachycardia (PAT): Caused by an irritable area of tissue in the atria that dominates the sinoatrial node and takes over as the pacemaker. Usually preceded by premature atrial contractions. Begin and end abruptly. The rapid rate prevents adequate ventricular filling.

Symptoms of PAT include Rapid pulse rate, sudden onset of palpitations, dyspnea, dizziness, lightheadedness, fatigue, or chest

pressure, syncope- With rapid rate and severe hypotension.

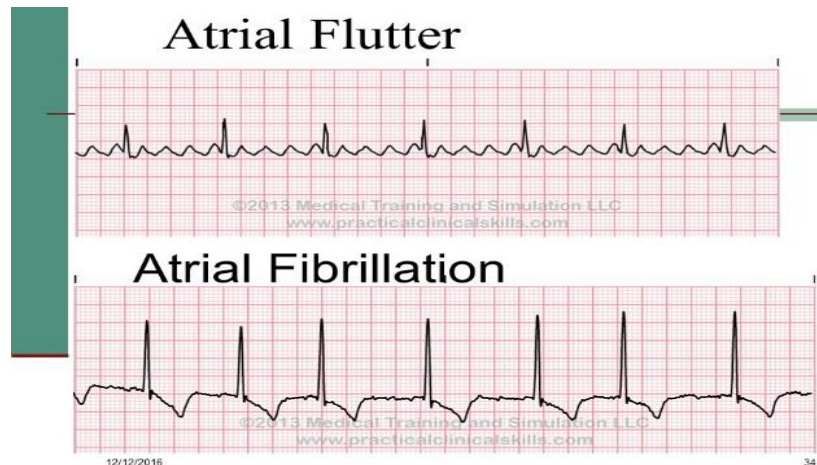
Treatment: The primary treatment atrial tachycardia is rate control using carotid sinus pressure, vagal nerve stimulation, and medications such as diltiazem, verapamil,

Digoxin, propranolol, procainamide, quinidine, vasopressor.

Patient education: Minor lifestyle changes such as stopping the use of caffeine, alcohol, and OTC cold medications may help minimize symptoms. Encourage patient to keep a journal of when, where, and what circumstances surround their palpitations, including lightheadedness, nausea, sweating, chest pain, or shortness of breath. Teach patient how to check their pulse.

Atrial flutter: atrial ectopic pacer fires at a rate of 250-400/ min. Occurs in a variety of heart diseases- rheumatic, coronary, hypertensive, also cardiomyopathy, hypoxia, heart failure, Patient may be asymptomatic or have palpitations. Management- digitalis, beta blockers, calcium channel blockers, may use cardioversion

Atrial fibrillation: Irregular and rapid atrial contraction, resulting in a quivering of the atria rather than contract at a rate greater than 400/min. Ventricular rate depends on the



number of impulses conducted through the AV node.

Signs and symptoms of A fib include; palpitations and sometimes weakness, vague chest discomfort, activity intolerance, dyspnea, and lightheadedness. Atrial thrombi often form, causing a significant risk of embolic stroke. Diagnosis by ECG-absence of P waves.

Treatment involves rate control with drugs, prevention of thromboembolism with anticoagulation, and sometimes conversion to sinus rhythm by drugs or cardioversion. Drugs commonly used to control ventricular rate in patients with atrial fibrillation Calcium channel blockers [Diltiazem (Cardizem), Verapamil (Calan, Isoptin)], Beta blockers [(Esmolol (Brevibloc), Propranolol (Inderal)], Digoxin

(Lanoxin). Cardioversion - Immediate
immediate synchronized cardioversion is recommended in hemodynamically unstable patients (patients with hypotension, altered mental status, or decreasing level of consciousness). Catheter or surgical ablation may also be used as a method of treatment.

NURSING INTERVENTIONS for patient with atrial arrhythmia:

Nursing interventions depend upon the type of treatment modality. Patient on Pharmacological therapy need frequent monitoring of EKG rhythm strips, heart sounds, and apical pulse rate. Be aware of side effects of these various medications and monitor for any potential problems. Since these medications affect the cardiovascular system, they may cause hemodynamic changes, such as hypotension, dizziness, and syncope, so safety measures are important. Patient's response to the medication, system assessment—cardiac, respiratory, neurological—vital signs, and lab values should constantly be monitored. If the patient is taking anticoagulants, then lab values (activated partial thromboplastin time (aPTT) for heparin, prothrombin time (PT), international normalized ration (INR) for warfarin) should be monitored as well as signs of bleeding and vital signs.

Prior to cardioversion sedation medication and/or analgesics should be given for client comfort. The nurse would administer and monitor the effects of analgesia.

Medications such as midazolam (Versed), lorazepam (Ativan), or propofol (Diprivan) may be used for client sedation. When these medications are administered, synchronized cardioversion is considered to be a conscious sedation procedure. Nurses should check with their institution for policies and procedures related to nursing responsibilities when conscious sedation is used. Vital signs should be monitored both prior to and after the procedure.

Assess the monitor for a change to normal sinus rhythm. Inform the physician of any rhythm that occurs post-cardioversion. After the procedure, monitor the chest wall for burns, and medicate for pain as ordered. A patient may have a sore chest after synchronized cardioversion, so providing comfort measures, such as repositioning, mild analgesic, and warm compresses to the chest, may assist in easing the discomfort. Burns on the chest wall also may occur as a result of synchronized cardioversion, so always assess the chest wall after the procedure.

Monitoring after a catheter ablation involves monitoring vital signs, heart sounds,

and apical pulse, as well as the femoral catheter-insertion site. Pressure should be maintained at this site. The femoral site should be monitored further for active signs of bleeding as well as a hematoma formation. The foot also should be assessed for capillary refill, pedal pulses, color, and movement. Decreased foot perfusion is manifested by a pallor, blue color, absent or decreased capillary refill, pain, and difficulty moving the toes. If the patient is being monitored, then closely assess the EKG for changes in rhythm. Continually assess the patient's level of consciousness as well. Monitor complete blood count (CBC) and electrolytes. Assist with activities of daily living (ADLs) and ambulation as needed.

PATIENT EDUCATION

Education for the patient with a diagnosis of atrial fibrillation is vital. Discuss the side effects of each medication. If they are prescribed anticoagulants for the first time, they need to know signs and symptoms of bleeding, and to make sure they do not miss their follow-up appointments with their healthcare providers. Lab work will be drawn, and their anticoagulant medication dosage may be based on those results. Caution patients against beginning any type of herbal medications, and before taking any over-the-counter medications

they should check with their healthcare provider. Not using aspirin or aspirin-containing products is imperative. Alcohol and tobacco intake should cease.

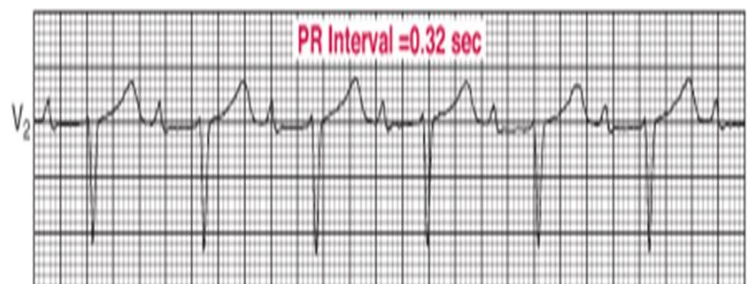
Supraventricular Tachycardia

Supraventricular tachycardia is a series of rapid heartbeats that begin in or involve the upper chambers (atria) of the heart. SVT can cause the heart to beat very rapidly or erratically. Symptoms begin and end suddenly. QRS complexes are typically narrow, rapid, and regular. Treatment such as Vagotonic maneuvers (eg, Valsalva maneuver) sometimes help. Adenosine is the first choice, and if ineffective, verapamil or diltiazem are alternatives.

Avoid AV nodal blockers for wide complex tachycardia; use synchronized cardioversion or procainamide or amiodarone.

Atrioventricular Heart Blocks

Atrioventricular (AV) block is partial or complete interruption of impulse

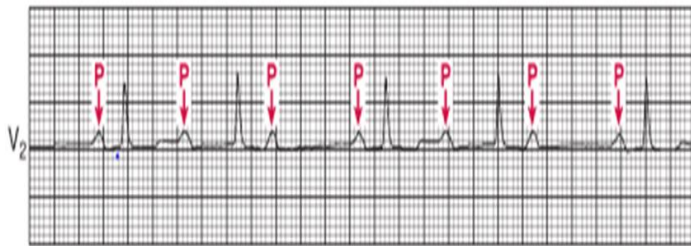


transmission from the atria to the ventricles. The most common cause is idiopathic fibrosis and sclerosis of the conduction system. Diagnosis is by ECG. Symptoms and treatment depend on degree of block, but treatment, when necessary, usually involves pacing.

First degree AV Block: All normal P waves are followed by QRS complexes, but the PR interval is longer than normal (> 0.20 sec)

Second Degree AV block: Some normal P waves are followed by QRS complexes, but some are not. Three types exist.

In **Mobitz type I 2nd-degree AV block**,



the PR interval progressively lengthens with each beat until the atrial impulse is not conducted and the QRS complex is dropped (Wenckebach phenomenon).

In **Mobitz type II 2nd-degree AV block**, the PR

interval remains constant. Beats are intermittently nonconducted and QRS complexes dropped, usually in a repeating cycle of every 3rd (3:1 block) or 4th (4:1 block) P wave.



Second-degree AV block (high grade):

In high-grade 2nd-degree AV block, every 2nd (or more) P wave is blocked. Unlike 3rd degree heart block there is still some relationship between the P waves and the QRS complexes.

Third-degree AV block or complete heart block: Heart block is complete.

There is no electrical communication between the atria and ventricles and no relationship between P waves and QRS



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complexes (AV dissociation). Cardiac function is maintained by an escape junctional or ventricular pacemaker.

The distinction between Mobitz type I and Mobitz type II block is difficult to make because 2 P waves are never conducted in a row. Risk of complete AV block is difficult to predict, and a pacemaker is indicated. Patients with any form of 2nd-degree AV block and a structural heart disorder should be considered candidates for permanent pacing unless there is a transient or reversible cause.

Treatment for AV

block: Most patients require a pacemaker. Stop any medication causing the AV block.

Case study 1

Ms. Vera Johnson 25

Rate-atrial rate is usually normal; ventricular rate is usually less than 70/bpm. The atrial rate is always faster than the ventricular rate. P wave- normal with constant P-P intervals, but not "married" to the QRS complexes. QRS - may be normal or widened depending on where the escape pacemaker is located in the conduction system. Conduction-atrial and ventricular activities are unrelated due to the complete blocking of the atrial impulses to the ventricles. The rhythm is irregular.

Bundle Branch Block: Abnormal conduction through the bundle branches will cause a depolarization delay through the ventricular muscle, this delay shows as a widening of the QRS complex. Right Bundle Branch Block (RBBB) indicates problems in the right side of the heart. Left Bundle Branch Block (LBBB) is an indication of heart disease.

years of age, is a female patient who received a permanent atrioventricular pacemaker for the diagnosis of sick sinus rhythm, a disorder that leads to periods of tachycardia and periods of extreme bradycardia or sinus arrest. The nurse received the end-of-shift report and arrives at Ms. White's room where she assesses the patient's incision dressing on the upper left chest and it is dry. The patient's left arm is edematous and ecchymotic and twice the size of the other arm. The patient states that her left arm feels numb and tingling. The distal pulses are present and at baseline. None of the findings were noted in the end-of-shift report.

a. **What nursing management should the nurse provide immediately?**

The patient is bleeding from the surgical site and the blood is accumulating into the third space and into the tissue of the left arm. The nurse needs to call the cardiologist immediately and remove the dressing to further inspect the site and place a pressure dressing as

ordered on the site. The arm needs to be elevated on several pillows so that it is above the level of the heart. This will promote venous return. The nurse needs to place ice on the incisional site. The nurse will need to perform vital signs using the right arm for blood pressure and continue to assess the patient's left arm using neurovascular checks every 15 minutes. The cardiologist will want to assess the patient further.

b. Explain the general care of the patient after receiving an implanted pacemaker.

- Minimize the movement of the affected arm for at least 24 hours or as directed by the physician. This helps prevent dislodgement of the pacemaker leads.
- Assess the chart to ensure that the date of insertion, model, type of pacemaker, and settings are recorded. There should also be a wallet card with the same information that will be given to the patient during patient teaching.
- Instruct the patient to avoid lifting anything over 5 pounds of weight until cleared by the physician, usually in 6 weeks.
- The patient is continuously monitored with the ECG while hospitalized to detect any problems with the pacemaker. The nurse observes for the following and notifies the physician of their development:
 - Failure to pace: This may be due to dislodgement of pacer wires or inappropriate sensing.
 - Failure to capture: The pacemaker spike is not followed by either atrial or ventricular depolarization. The output of the pacemaker may be inadequate or the pacer wires may be dislodged.
 - Improper sensing: The pacemaker fires without regard to the underlying heart rhythm. This will lead to the risk of dysrhythmias, such as ventricular tachycardia, and a decrease in the cardiac output. The pacemaker needs adjustments.
- Observe the incision site for bleeding, hematoma, or infection.
- A chest x-ray is taken before discharge to assess for the proper location of the pacer leads and if there are any complications, such as a pleural effusion or pneumothorax.
- Observe for potential complications including:
 - Myocardial perforation: Observe for hiccups that are persistent because this is a sign that the lead is near the diaphragm after the lead perforated the ventricle.
 - Cardiac tamponade: Observe for hypotension, distant heart sounds, and jugular vein distention.

- Pneumothorax or hemothorax: Observe for decreased lung sounds on one side, and possibly jugular vein distention, and development of shortness of breath.
- Skin breakdown: Observe for wound dehiscence or thinning of skin around the pacemaker pocket.
- Emboli: Patient develops symptoms of stroke, pulmonary embolus, or heart attack.
- Endocarditis: Observe for development of heart murmur, fatigue, joint pain, fever, and chest pain.
- Instruct the patient and family:
 - Use a model or picture and discuss where the generator and leads are placed and how the pacemaker works and the rate it is set. The pacemaker battery will need to be replaced, usually in 6 to 12 years, and is done outpatient.
 - Demonstrate how to take a pulse and have the patient/family return the demonstration. Instruct to take the pulse before rising from the bed and report if the pulse is five or more beats lower than the preset rate for the pacemaker.
 - Instruct the patient to report dizziness, syncope, chest pain, generalized weakness, or palpitations to the physician at once.
 - Instruct the patient on incisional care and the signs and symptoms of infection to report to the physician.
 - Demonstrate arm exercises to begin on postoperative day 2 and explain the progression of the exercises used to regain full mobility of the arm and shoulder.
 - Instruct the patient to avoid heavy lifting over 5 pounds for 6 weeks or until cleared by the physician and also to avoid vacuuming or pushing a lawn mower during this time.
 - Instruct the patient to carry the pacemaker identification wallet card at all times. It is best to get a MedicAlert bracelet as well. The patient needs to inform all health care providers that she has a pacemaker.
 - Instruct the patient to avoid strong electromagnetic fields, such as high power lines or power stations, strong magnets, or an MRI.
 - Instruct the patient about resuming sexual activity, but avoid positions that increase the pressure on the site where the pacemaker is placed.
 - Instruct the patient to follow up with the physician as directed. The patient will have periodic telephone

pacemaker checks where the patient will use a special transmitter that will send the ECG recording across the telephone to the clinic. The pacemaker clinic assesses how well the pacemaker is functioning. (Either the nurse or someone from the pacemaker clinic will show the patient the transmitter device and how it works and send the device home with the patient.)

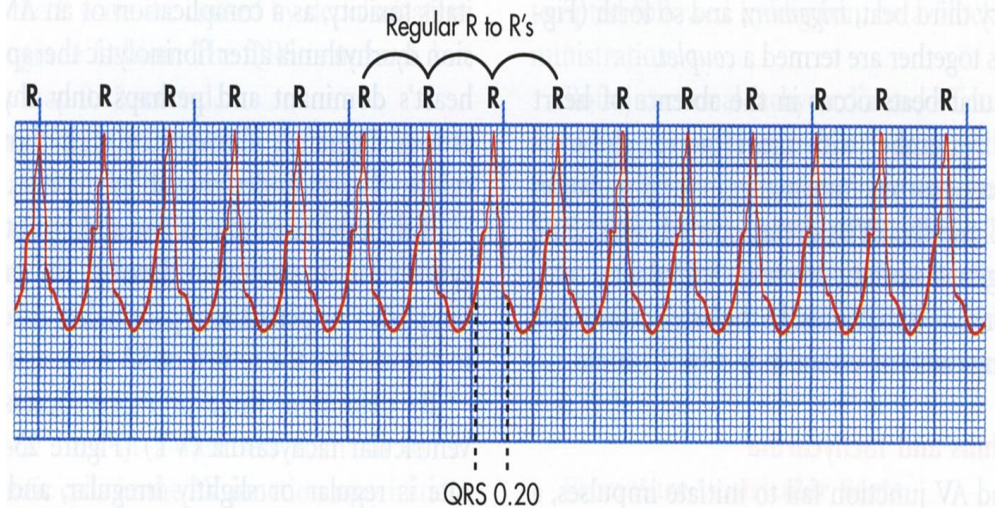
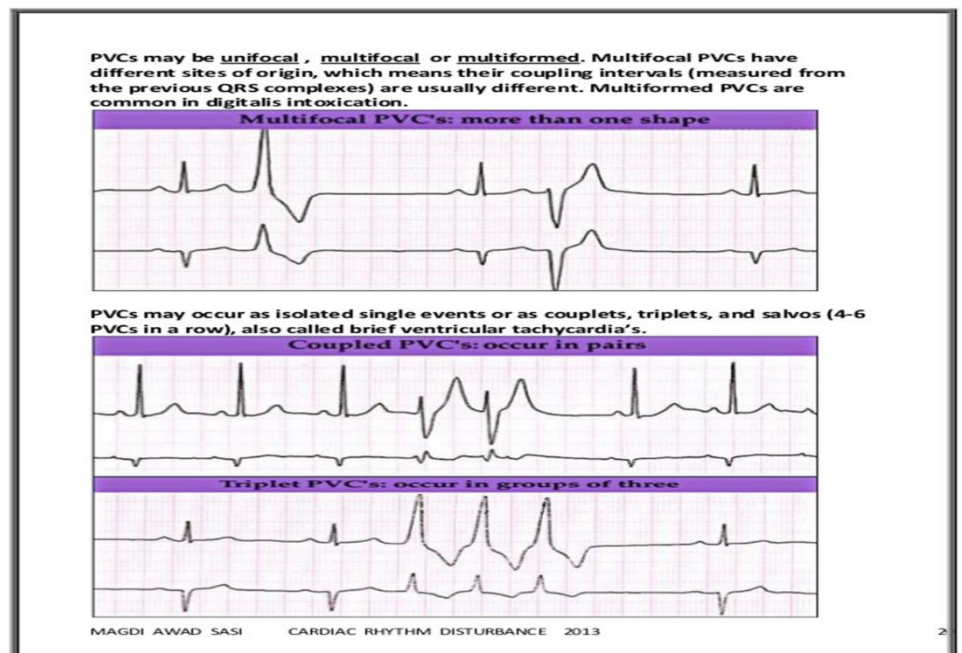
or at predictable intervals (eg, every 3rd [trigeminy] or 2nd [bigeminy] beat). Extremely common in healthy patients and in patients with heart disorder. VPBs may increase with stimulants (e.g. anxiety, stress, alcohol, caffeine, and sympathomimetic drugs), hypoxia, or electrolyte abnormalities. VPB may be asymptomatic or cause palpitations.

VENTRICULAR DYSRHYTHMIAS

Impulse originates in the ventricles. Causes may be drug toxicity, hypoxia, hypothermia, and electrolyte imbalances.

Premature Ventricular Contractions (PVC) or Ventricular Premature Beats (VPB)

Beats originating in the ventricles instead of the sinoatrial node in the atria, causing the ventricles to contract before the atria and resulting in a decrease in the amount of blood pumped to the body. May occur erratically



Diagnosis is by ECG. Treatment is usually not required beyond avoiding obvious triggers.

Ventricular Tachycardia

Defined as three or more premature ventricular contractions in a row. A rapid heartbeat with a rate of more than 100 bpm, usually originating in the ventricles. Symptoms depend on duration and vary from none to palpitations to hemodynamic collapse and death.

Etiology- increased myocardial irritability associated with coronary artery disease, myocardial infarction, electrolyte imbalance, and cardiomyopathy. VT may be monomorphic or polymorphic and nonsustained or sustained.

Monomorphic VT: Single abnormal focus or reentrant pathway and regular, identical-appearing QRS complexes. Polymorphic VT: Several different foci or pathways and irregular, varying QRS complexes.

Nonsustained VT: Lasts < 30 sec

Sustained VT: Lasts \geq 30 sec or is terminated sooner because of hemodynamic collapse.

VT frequently deteriorates to ventricular fibrillation and thus cardiac arrest.

Signs and symptoms include palpitations, dizziness, chest pain, SOB, hypotension.

If rapid or sustained may lose consciousness

Management depends upon severity if stable – continue monitoring, obtain 12 lead electrocardiogram. If patient is unstable-unconscious / without a pulse – treat as ventricular fibrillation with immediate defibrillation. Medications like Lidocaine or procainamide, amiodarone, magnesium sulfate (MgSO₄). If there is a pulse – synchronized cardioversion. If pulseless – defibrillation, CPR must be instituted.

Ventricular Fibrillation

Rapid disorganized contractions of the ventricles resulting in the inability of the heart to pump any blood to the body, which will result in death unless treated immediately.



Sign and symptoms include loss of consciousness, absent pulse, no heart sounds, BP drops to 0/0.

Treatment is with cardiopulmonary resuscitation, including immediate defibrillation.

Asystole



This dysrhythmia occurs when there is a total absence of electrical activity in the heart.

The patient is clinically dead. Sometimes referred to as straight line or flat line. There will be an absence of P waves and QRS complexes.

Case study 2

2. The nurse on the telemetry unit responds to the cardiac monitor alarm on a patient recovering from a myocardial infarction. Upon entering the room, the nurse notes the rhythm on the monitor appears to be ventricular tachycardia.

a. What action should the nurse take first?

Assess the patient for a pulse.

b. The nurse notes the patient has no pulse, and tells someone to get the code cart and another to call the hospital's code team. Upon arrival of the code cart, the nurse prepares the defibrillator for use. At what energy level does the nurse set the biphasic defibrillator for the first shock?

Because the patient is demonstrating pulseless ventricular tachycardia, the patient will initially receive a shock of 150 to 200 joules.

c. After defibrillation, the nurse assesses the patient, finds him pulseless, and initiates cardiopulmonary resuscitation. The code team has arrived, and epinephrine is administered. What is the rationale for this medication in this emergency situation?

Epinephrine or vasopressin may be administered to facilitate subsequent shocks to convert the dysrhythmias. These medications may also increase cerebral and coronary artery blood flow.

d. What is the difference between monophasic and biphasic defibrillators?

Monophasic defibrillators deliver electric current in only one direction and require increased energy levels. Newer biphasic defibrillators deliver the electrical charge to the positive paddle, which then reverses back to the originating paddle. These defibrillators require

lower, possibly nonprogressive energy levels, which potentially produce less myocardial damage.

Case 3

A client is receiving Propranolol (Inderal) for the treatment of arrhythmia, what should the nurse include in the patient's education?

a. **What type of cardiac med is propranolol?**

Propranolol is a beta-blocker.

b. **What are the contraindications for the use of propranolol?**

c. **What are the common side effects for the use of beta blockers (propranolol)?**

- Abrupt discontinuation of medication may cause myocardial infarction, severe hypertension and ventricular arrhythmias because of a potential rebound effect.
- Instruct patient to:
- Never discontinue the medication abruptly and to take the medication exactly as prescribed.

- Take the medication even if feeling well.
- Avoid drinking alcohol. It may increase your blood levels of propranolol.
- Warn patients against interruption or discontinuance of beta-blocker therapy without physician advice
- Take pulse prior to taking the medication. If pulse is irregular, instruct patient to withhold medication and contact the health care provider immediately.
- Instruct patient to change position slowly to avoid dizziness.
- Instruct patients with Type I Diabetes Mellitus:
 - • to check blood sugar regularly.
 - • to report unusually low blood sugar reading to the health care provider.

- Instruct patient to take medication with food to decrease GI upset.

Nursing Alert

When giving an oral antiarrhythmic drug, the nurse withholds the drug and notifies the primary health care provider immediately when the pulse rate is above 120 bpm or below 60 bpm. In some instances, the primary health care provider may establish additional or different guidelines for withholding the drug.

Nursing Alert

When older adults take the antiarrhythmic drugs, they are at greater risk for adverse reactions such as the development of additional arrhythmias or aggravation of existing arrhythmias, hypotension, and congestive heart failure (CHF). A dosage reduction may be indicated. Careful monitoring by the nurse is necessary for early identification and management of adverse reactions. The nurse monitors the intake and output and reports any signs of CHF, such as increase in weight, decrease in urinary output, or shortness of breath.

Important note about Afib.

Atrial fibrillation with a wide QRS complex may indicate Wolff-Parkinson-White (WPW) syndrome; in such cases, use of AV node-blocking drugs may be fatal.

Do not give digoxin or nondihydropyridine calcium channel blockers (e.g. verapamil, diltiazem) to patients with atrial fibrillation and WPW because these drugs may trigger ventricular fibrillation.

Nursing Diagnoses Checklist

- Ineffective Tissue Perfusion: Peripheral related to adverse drug reactions (hypotension)
- Decreased cardiac output related to electrical conduction disturbances, decreased myocardial contractility.
- Decreased Cardiac Output related to adverse drug reactions (drug-induced arrhythmias)
- Activity Intolerance related to weakness and fatigue
- Risk for Injury related to adverse drug reactions (dizziness, light-headedness)

Feature	Description	Duration
RR interval	The interval between an R wave and the next R wave: Normal resting heart rate is between 60 and 100 bpm.	0.6 to 1.2s
P wave	During normal atrial depolarization, the main electrical vector is directed from the SA node towards the AV node, and spreads from the right atrium to the left atrium. This turns into the P wave on the ECG.	80ms
PR interval	The PR interval is measured from the beginning of the P wave to the beginning of the QRS complex. The PR interval reflects the time the electrical impulse takes to travel from the sinus node through the AV node and entering the ventricles. The PR interval is, therefore, a good estimate of AV node function.	120 to 200ms
PR segment	The PR segment connects the P wave and the QRS complex. The impulse vector is from the AV node to the bundle of His to the bundle branches and then to the Purkinje fibers. This electrical activity does not produce a contraction directly and is merely traveling down towards the ventricles, and this shows up flat on the ECG. The PR interval is more clinically relevant.	50 to 120ms
QRS complex	The QRS complex reflects the rapid depolarization of the right and left ventricles. They have a large muscle mass compared to the atria, so the QRS complex usually has a much larger amplitude than the P-wave.	80 to 120ms
J-point	The point at which the QRS complex finishes and the ST segment begins, it is used to measure the degree of ST elevation or depression present.	N/A
ST segment	The ST segment connects the QRS complex and the T wave. The ST segment represents the period when the ventricles are depolarized. It is isoelectric.	80 to 120ms
T wave	The T wave represents the repolarization (or recovery) of the ventricles. The interval from the beginning of the QRS complex to the apex of the T wave is referred to as the absolute refractory period. The last half of the T wave is referred to as the relative refractory period (or vulnerable period).	160ms
ST interval	The ST interval is measured from the J point to the end of the T wave.	320ms
QT interval	The QT interval is measured from the beginning of the QRS complex to the end of the T wave. A prolonged QT interval is a risk factor for ventricular tachyarrhythmias and sudden death. It varies with heart rate and for clinical relevance requires a correction for this, giving the QTc.	Up to 420ms in heart rate of 60 bpm

U wave	The U wave is hypothesized to be caused by the repolarization of the interventricular septum. They normally have a low amplitude, and even more often completely absent. They always follow the T wave and also follow the same direction in amplitude. If they are too prominent, suspect hypokalemia, hypercalcemia or hyperthyroidism usually.	
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ARRHYTHMIA	DESCRIPTION
ATRIAL FLUTTER	Rapid contraction of the atria (up to 300 bpm) at a rate too rapid for the ventricles to pump efficiently
ATRIAL FIBRILLATION	Irregular and rapid atrial contraction, resulting in a quivering of the atria and causing an irregular and inefficient ventricular contraction
PREMATURE VENTRICULAR CONTRACTIONS	Beats originating in the ventricles instead of the sinoatrial node in the atria, causing the ventricles to contract before the atria and resulting in a decrease in the amount of blood pumped to the body
VENTRICULAR TACHYCARDIA	A rapid heartbeat with a rate of more than 100 bpm, usually originating in the ventricles
VENTRICULAR FIBRILLATION	Rapid disorganized contractions of the ventricles resulting in the inability of the heart to pump any blood to the body, which will result in death unless treated immediately

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