

DYSRHYTHMIAS (INCLUDING DIGITALIS TOXICITY)

A cardiac dysrhythmia is any disturbance in the normal rhythm of the electrical excitation of the heart. It can be the result of a primary cardiac disorder, a response to a systemic condition, the result of electrolyte imbalance or drug toxicity. Dysrhythmias vary in severity and in their effects on cardiac function, which are partially influenced by the site of origin (ventricular or supraventricular).

CARE SETTING

Generally, minor dysrhythmias are monitored and treated in the community setting; however, potential life-threatening situations (including heart rates above 150 beats/min) usually require a short inpatient stay.

RELATED CONCERNS

Angina
Heart failure: chronic
Myocardial infarction
Psychosocial aspects of care

Patient Assessment Database

ACTIVITY/REST

May report: Generalized weakness and exertional fatigue
May exhibit: Changes in heart rate/BP with activity/exercise

CIRCULATION

May report: History of previous/acute MI (90%–95% experience dysrhythmias), cardiac surgery, cardiomyopathy, rheumatic/HF, valvular heart disease, long-standing hypertension, use of pacemaker
Pulse: Fast, slow, or irregular; palpitations, skipped beats
May exhibit: BP changes (hypertension or hypotension) during episodes of dysrhythmia
Pulses may be irregular, e.g., skipped beats; pulsus alternans (regular strong beat/weak beat); bigeminal pulse (irregular strong beat/weak beat)
Pulse deficit (difference between apical pulse and radial pulse)
Heart sounds: irregular rhythm, extra sounds, dropped beats
Skin color and moisture changes, e.g., pallor, cyanosis, diaphoresis (heart failure, shock)
Edema dependent, generalized, JVD (in presence of heart failure)
Urine output decreased if cardiac output is severely diminished

EGO INTEGRITY

May report: Feeling nervous (certain tachydysrhythmias), sense of impending doom
Stressors related to current medical problems
May exhibit: Anxiety, fear, withdrawal, anger, irritability, crying

FOOD/FLUID

May report: Loss of appetite, anorexia
Food intolerance (with certain medications)
Nausea/vomiting
Changes in weight
May exhibit: Weight gain or loss
Edema
Changes in skin moisture/turgor
Respiratory crackles

NEUROSENSORY

May report: Dizzy spells, fainting, headaches

May exhibit: Mental status/sensorium changes, e.g., disorientation, confusion, loss of memory; changes in usual speech pattern/consciousness, stupor, coma
Behavioral changes, e.g., combativeness, lethargy, hallucinations
Pupil changes (equality and reaction to light)
Loss of deep tendon reflexes with life-threatening dysrhythmias (ventricular tachycardia, severe bradycardia)

PAIN/DISCOMFORT

May report: Chest pain, mild to severe, which may or may not be relieved by antianginal medication
May exhibit: Distraction behaviors, e.g., restlessness

RESPIRATION

May report: Chronic lung disease
History of or current tobacco use
Shortness of breath
Coughing (with/without sputum production)
May exhibit: Changes in respiratory rate/depth during dysrhythmia episode
Breath sounds: Adventitious sounds (crackles, rhonchi, wheezing) may be present, indicating respiratory complications, such as left-sided heart failure (pulmonary edema) or pulmonary thromboembolic phenomena
Hemoptysis

SAFETY

May exhibit: Fever
Skin: Rashes (medication reaction)
Loss of muscle tone/strength

TEACHING/LEARNING

May report: Familial risk factors, e.g., heart disease, stroke
Use/misuse of prescribed medications, such as heart medications (e.g., digitalis), anticoagulants (e.g., warfarin [Coumadin]), benzodiazepines (e.g., diazepam [Valium]), tricyclic antidepressants (e.g., amitriptyline [Elavil]), or antipsychotic agents (e.g., fluphenazine [Prolixin], chlorpromazine [Thorazine]), or OTC medications (e.g., cough syrup and analgesics containing ASA)
Stimulant abuse, including caffeine/nicotine
Lack of understanding about disease process/therapeutic regimen
Evidence of failure to improve, e.g., recurrent/intractable dysrhythmias that are life-threatening
Discharge plan considerations: **DRG projected mean length of inpatient stay: 3.9 days**
Alteration of medication use/therapy
Refer to section at end of plan for postdischarge considerations.

DIAGNOSTIC STUDIES

ECG: Reveals type/source of dysrhythmia and effects of electrolyte imbalances and cardiac medications. Demonstrates patterns of ischemic injury and conduction aberrance. *Note:* Exercise ECG can reveal dysrhythmias occurring only when patient is not at rest (can be diagnostic for cardiac cause of syncope).

Extended or event monitoring (e.g., Holter monitor): Extended ECG tracing (24 hr to weeks) may be desired to determine which dysrhythmias may be causing specific symptoms when patient is active (home/work) or at rest. May also be used to evaluate pacemaker function, antidysrhythmia drug effect, or effectiveness of cardiac rehabilitation.

Signal-averaged ECG (SAE): May be used to screen high-risk patients (especially post-MI or unexplained syncope) for ventricular dysrhythmias, presence of delayed conduction, and late potentials (as occurs with sustained ventricular tachycardia).

Chest x-ray: May show enlarged cardiac shadow due to ventricular or valvular dysfunction.

Myocardial imaging scans: May demonstrate ischemic/damaged myocardial areas that could impede normal conduction or impair wall motion and pumping capabilities.

Electrophysiological (EP) studies: Provides cardiac mapping of entire conduction system to evaluate normal and abnormal pathways of electrical conduction. Used to diagnose dysrhythmias and evaluate effectiveness of medication or pacemaker therapies.

Electrolytes: Elevated or decreased levels of potassium, calcium, and magnesium can cause dysrhythmias.

Drug screen: May reveal toxicity of cardiac drugs, presence of street drugs, or suggest interaction of drugs, e.g., digitalis and quinidine.

Thyroid studies: Elevated or depressed serum thyroid levels can cause/aggravate dysrhythmias.

ESR: Elevation may indicate acute/active inflammatory process, e.g., endocarditis, as a precipitating factor for dysrhythmias.

ABGs/pulse oximetry: Hypoxemia can cause/exacerbate dysrhythmias.

NURSING PRIORITIES

1. Prevent/treat life-threatening dysrhythmias.
2. Support patient/SO in dealing with anxiety/fear of potentially life-threatening situation.
3. Assist in identification of cause/precipitating factors.
4. Review information regarding condition/prognosis/treatment regimen.

DISCHARGE GOALS

1. Free of life-threatening dysrhythmias and complications of impaired cardiac output/tissue perfusion.
2. Anxiety reduced/managed.
3. Disease process, therapy needs, and prevention of complications understood.
4. Plan in place to meet needs after discharge.

NURSING DIAGNOSIS: Cardiac Output, risk for decreased

Risk factors may include

Altered electrical conduction
Reduced myocardial contractility

Possibly evidenced by

[Not applicable; presence of signs and symptoms establishes an *actual* diagnosis.]

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Cardiac Pump Effectiveness (NOC)

Maintain/achieve adequate cardiac output as evidenced by BP/pulse within normal range, adequate urinary output, palpable pulses of equal quality, usual level of mentation.

Display reduced frequency/absence of dysrhythmia(s).

Participate in activities that reduce myocardial workload.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Hemodynamic Regulation (NIC)</p> <p>Independent</p> <p>Palpate pulses (radial, carotid, femoral, dorsalis pedis), noting rate, regularity, amplitude (full/thready), and symmetry. Document presence of pulsus alternans, bigeminal pulse, or pulse deficit.</p> <p>Auscultate heart sounds, noting rate, rhythm, presence of extra heartbeats, dropped beats.</p> <p>Monitor vital signs. Assess adequacy of cardiac output/tissue perfusion, noting significant variations in BP/pulse rate equality, respirations, changes in skin color/temperature, level of consciousness/sensorium, and urine output during episodes of dysrhythmias.</p> <p>Determine type of dysrhythmia and document with rhythm strip (if cardiac/telemetry monitoring is available):</p> <p style="padding-left: 40px;">Sinus tachycardia;</p> <p style="padding-left: 40px;">Sinus bradycardia;</p> <p style="padding-left: 40px;">Atrial dysrhythmias, e.g., PACs, atrial flutter, atrial fibrillation (AF), atrial supraventricular tachycardias) (i.e., PAT, MAT, SVT);</p>	<p>Differences in equality, rate, and regularity of pulses are indicative of the effect of altered cardiac output on systemic/peripheral circulation.</p> <p>Specific dysrhythmias are more clearly detected audibly than by palpation. Hearing extra heartbeats or dropped beats helps identify dysrhythmias in the unmonitored patient.</p> <p>Although not all dysrhythmias are life-threatening, immediate treatment may be required to terminate dysrhythmia in the presence of alterations in cardiac output and tissue perfusion.</p> <p>Useful in determining need for/type of intervention required.</p> <p>Tachycardia can occur in response to stress, pain, fever, infection, coronary artery blockage, valvular dysfunction, hypovolemia, hypoxia, or as a result of decreased vagal tone or of increased sympathetic nervous system activity associated with the release of catecholamines. Although it generally does not require treatment, persistent tachycardia may worsen underlying pathology in patients with ischemic heart disease because of shortened diastolic filling time and increased oxygen demands. These patients may require medications.</p> <p>Bradycardia is common in patients with acute MI (especially anterior and inferior) and is the result of excessive parasympathetic activity, blocks in conduction to the SA or AV nodes, or loss of automaticity of the heart muscle. Patients with severe heart disease may not be able to compensate for a slow rate by increasing stroke volume. Therefore, decreased cardiac output, HF, and potentially lethal ventricular dysrhythmias may occur.</p> <p>PACs can occur as a response to ischemia and are normally harmless but can precede or precipitate atrial fibrillation. Acute and chronic atrial flutter and/or fibrillation (the most common dysrhythmia) can occur with coronary artery or valvular disease and may or may not be pathological. Rapid atrial flutter/fibrillation reduces cardiac output as a result of incomplete ventricular filling (shortened cardiac cycle) and increased oxygen demand.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Hemodynamic Regulation (NIC)</p> <p>Independent</p> <p>Ventricular dysrhythmias, e.g., premature ventricular contractions/ventricular premature beats (PVCs/VPBs), ventricular tachycardia (VT), ventricular flutter/ fibrillation (VF);</p> <p>Heart blocks.</p> <p>Provide calm/quiet environment. Review reasons for limitation of activities during acute phase.</p> <p>Demonstrate/encourage use of stress management behaviors, e.g., relaxation techniques, guided imagery, slow/deep breathing.</p> <p>Investigate reports of chest pain, documenting location, duration, intensity (0–10 scale), and relieving/aggravating factors. Note nonverbal pain cues, e.g., facial grimacing, crying, changes in BP/heart rate.</p> <p>Be prepared to initiate cardiopulmonary resuscitation(CPR) as indicated.</p>	<p>PVCs or VPBs reflect cardiac irritability and are commonly associated with MI, digitalis toxicity, coronary vasospasm, and misplaced temporary pacemaker leads. Frequent, multiple, or multifocal PVCs result in diminished cardiac output and may lead to potentially lethal dysrhythmias, e.g., VT or sudden death/cardiac arrest from ventricular flutter/fibrillation. <i>Note:</i> Intractable ventricular dysrhythmias unresponsive to medication may reflect ventricular aneurysm. Polymorphic VT (torsades de pointes) is recognized by inconsistent shape of QRS complexes and is often drug related, e.g., procainamide (Pronestyl), quinidine (Quinaglute), disopyramide (Norpace), and sotalol (Betapace).</p> <p>Reflect altered transmission of impulses through normal conduction channels (slowed, altered) and may be the result of MI, coronary artery disease with reduced blood supply to sinoatrial (SA) or atrioventricular (AV) nodes, drug toxicity, and sometimes cardiac surgery. Progressing heart block is associated with slowed ventricular rates, decreased cardiac output, and potentially lethal ventricular dysrhythmias or cardiac standstill.</p> <p>Reduces stimulation and release of stress-related catecholamines, which can cause/aggravate dysrhythmias and vasoconstriction, increasing myocardial workload.</p> <p>Promotes patient participation in exerting some sense of control in a stressful situation.</p> <p>Reasons for chest pain are variable and depend on underlying cause. However, chest pain may indicate ischemia due to altered electrical conduction, decreased myocardial perfusion, or increased oxygen need (e.g., impending/evolving MI).</p> <p>Development of life-threatening dysrhythmias requires prompt intervention to prevent ischemic damage/death.</p>
<p>Collaborative</p> <p>Monitor laboratory studies:</p> <p>Electrolytes;</p> <p>Drug levels.</p>	<p>Imbalance of electrolytes, such as potassium, magnesium, and calcium, adversely affects cardiac rhythm and contractility.</p> <p>Reveal therapeutic/toxic level of prescription medications or street drugs that may affect/contribute to presence of dysrhythmias.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Hemodynamic Regulation (NIC)</p> <p>Collaborative</p> <p>Administer supplemental oxygen as indicated.</p> <p>Administer medications as indicated:</p> <p style="padding-left: 40px;">Potassium</p> <p style="padding-left: 40px;">Antidysrhythmics, such as: Class I drugs;</p> <p style="padding-left: 40px;">Class Ia, e.g., disopyramide (Norpace), procainamide (Pronestyl, Procan SR), quinidine (Quinaglute, Cardioquin);</p> <p style="padding-left: 40px;">Class Ib, e.g., lidocaine (Xylocaine), phenytoin (Dilantin), tocainide (Tonocard), mexiletine (Mexitil); moricizine (Ethmozine);</p> <p style="padding-left: 40px;">Class Ic, e.g., flecainide (Tambocor), propafenone (Rhythmol), encainide (Enkaid);</p>	<p>Increases amount of oxygen available for myocardial uptake, which decreases irritability caused by hypoxia.</p> <p>Dysrhythmias are generally treated symptomatically.</p> <p>Correction of hypokalemia may be sufficient to terminate some ventricular dysrhythmias. <i>Note:</i> Potassium imbalance is the number one cause of atrial fibrillation.</p> <p>Class I drugs depress depolarization and alter repolarization, stabilizing the cell. These drugs are divided into groups a, b, and c, based on their unique effects.</p> <p>These drugs increase action potential, duration, and effective refractory period and decrease membrane responsiveness, prolonging both QRS complex and QT interval. Useful for treatment of atrial and ventricular premature beats, repetitive dysrhythmias (e.g., atrial tachycardias and atrial flutter/fibrillation). <i>Note:</i> Myocardial depressant effects may be potentiated when class Ia drugs are used in conjunction with any drugs possessing similar properties.</p> <p>These drugs shorten the duration of the refractory period (QT interval), and their action depends on the tissue affected and the level of extracellular potassium. Drugs of choice for ventricular dysrhythmias, they are also effective for automatic and re-entrant dysrhythmias and digitalis-induced dysrhythmias. <i>Note:</i> These drugs may aggravate myocardial depression.</p> <p>These drugs slow conduction by depressing SA node automaticity and decreasing conduction velocity through the atria, ventricles, and Purkinje fibers. The result is prolongation of the PR interval and lengthening of the QRS complex. They suppress and prevent all types of ventricular dysrhythmias. <i>Note:</i> Flecainide increases risk of drug-induced dysrhythmias post MI. Propafenone can worsen or cause new dysrhythmias, a tendency called the “proarrhythmic effect.” Encainide is available only for patients who demonstrated a good result before the drug was removed from the market.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Hemodynamic Regulation (NIC)</p> <p>Collaborative</p> <p>Class II drugs: e.g., atenolol (Tenormin), propranolol (Inderal), nadolol (Corgard), acebutolol (Sectral), esmolol (Brevibloc), sotalol (Betapace); bisoprolol (Zebeta);</p> <p>Class III drugs: e.g., bretylium tosylate (Bretylol), amiodarone (Cordarone), sotalol (Betapace), ibutilide (Corvert);</p> <p>Class IV drugs: e.g., verapamil (Calan), nifedipine (Procardia), diltiazem (Cardizem);</p> <p>Class V drugs: e.g., atropine sulfate, isoproterenol (Isuprel), cardiac glycosides: digoxin (Lanoxin);</p> <p>Adenosine (Adenocard).</p> <p>Prepare for/assist with elective cardioversion.</p> <p>Assist with insertion/maintain pacemaker function.</p>	<p>Beta-adrenergic blockers have antiadrenergic properties and decrease automaticity. Therefore, they are useful in the treatment of dysrhythmias caused by SA and AV node dysfunction (e.g., SVTs, atrial flutter or fibrillation). <i>Note:</i> These drugs may exacerbate bradycardia and cause myocardial depression, especially when combined with drugs that have similar properties.</p> <p>These drugs prolong the refractory period and action potential duration, consequently prolonging the QT interval. They are used to terminate ventricular fibrillation and other life-threatening ventricular dysrhythmias/sustained ventricular tachyarrhythmias, especially when lidocaine/procainamide are not effective. <i>Note:</i> Sotalol is a nonselective beta-blocker with characteristics of both class II and class III.</p> <p>Calcium antagonists (also called calcium channel blockers) slow conduction time through the AV node (prolonging PR interval) to decrease ventricular response in SVTs, atrial flutter/fibrillation. Calan and Cardizem may be used for bedside conversion of acute atrial fibrillation.</p> <p>Miscellaneous drugs useful in treating bradycardia by increasing SA and AV conduction and enhancing automaticity. Cardiac glycosides may be used alone or in combination with other antidysrhythmic drugs to reduce ventricular rate in presence of uncontrolled/poorly tolerated atrial tachycardias or flutter/fibrillation.</p> <p>First-line treatment for paroxysmal supraventricular tachycardia (PVST). Slows conduction and interrupts reentry pathways in AV node. <i>Note:</i> Contraindicated in patients with second- or third-degree heart block or those with sick sinus syndrome who do not have a functioning pacemaker.</p> <p>May be used in atrial fibrillation or certain unstable dysrhythmias to restore normal heart rate/relieve symptoms of heart failure.</p> <p>Temporary pacing may be necessary to accelerate impulse formation or override tachydysrhythmias and ectopic activity, to maintain cardiovascular function until spontaneous pacing is restored or permanent pacing is initiated.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Hemodynamic Regulation (NIC)</p> <p>Collaborative</p> <p>Insert/maintain IV access.</p> <p>Prepare for invasive diagnostic procedures/surgery as indicated.</p> <p>Prepare for implantation of cardioverter/defibrillator (ICD) when indicated.</p>	<p>Patent access line may be required for administration of emergency drugs.</p> <p>Differential diagnosis of underlying cause may be required to formulate appropriate treatment plan. Resection of ventricular aneurysm may be required to correct intractable ventricular dysrhythmias unresponsive to medical therapy. Surgery, e.g., CABG, may be indicated to enhance circulation to myocardium and conduction system.</p> <p>This device may be surgically implanted in those patients with recurrent, life-threatening ventricular dysrhythmias unresponsive to tailored drug therapy. The latest generation of devices can provide multilevel (“tiered”) therapy, that is, antitachycardia and antibradycardia pacing, cardioversion, or defibrillation, depending on how each device is programmed.</p>

<p>NURSING DIAGNOSIS: Poisoning, risk for digitalis toxicity</p> <p>Risk factors may include Limited range of therapeutic effectiveness, lack of education/proper precautions, reduced vision/cognitive limitations</p> <p>Possibly evidenced by [Not applicable; presence of signs and symptoms establishes an <i>actual</i> diagnosis.]</p> <p>DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:</p> <p>Knowledge: Medication (NOC) Verbalize understanding of individual prescription, how it interacts with other drugs/substances, and importance of maintaining prescribed regimen. Recognize signs of digitalis overdose and developing heart failure, and what to report to physician.</p> <p>Cardiac Pump Effectiveness (NOC) Be free of signs of toxicity; display serum drug level within individually acceptable range.</p>

ACTIONS/INTERVENTIONS	RATIONALE
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<p>Medication Management (NIC)</p> <p>Independent</p> <p>Explain patient's specific type of digitalis preparation and its specific therapeutic use.</p> <p>Instruct patient not to change dose for any reason, not to omit dose (unless instructed to, depending on pulse rate), not to increase dose or take extra doses, and to contact doctor if more than one dose is omitted.</p> <p>Advise patient that digitalis may interact with many other drugs (e.g., barbiturates, neomycin, cholestyramine, quinidine, and antacids) and that physician should be informed that digitalis is taken whenever new medications are prescribed. Advise patient not to use OTC drugs (e.g., laxatives, antidiarrheals, antacids, cold remedies, diuretics, herbals) without first checking with the pharmacist or healthcare provider.</p> <p>Review importance of dietary and supplemental intake of potassium, calcium, and magnesium.</p> <p>Provide information and have the patient/SO verbalize understanding of toxic signs/symptoms to report to the healthcare provider.</p> <p>Discuss necessity of periodic laboratory evaluation: Serum digoxin (Lanoxin) or digitoxin (Crystodigin) level;</p> <p>Electrolytes, BUN, creatinine, liver function studies.</p>	<p>Reduces confusion due to digitalis preparations varying in name (although they may be similar), dosage strength, and onset and duration of action. Up to 15% of all patients receiving digitalis develop toxicity at some time during the course of therapy because of its narrow therapeutic range.</p> <p>Alterations in drug regimen can reduce therapeutic effects, result in toxicity, and cause complications.</p> <p>Knowledge may help prevent dangerous drug interactions.</p> <p>Maintaining electrolytes at normal ranges may prevent or limit development of toxicity and correct many associated dysrhythmias.</p> <p>Nausea, vomiting, diarrhea, unusual drowsiness, confusion, very slow or very fast irregular pulse, thumping in chest, double/blurred vision, yellow/green tint or halos around objects, flickering color forms or dots, altered color perception, and worsening heart failure (e.g., dependent/generalized edema, dyspnea, decreased amount/frequency of voiding) indicate need for prompt evaluation/intervention. Mild symptoms of toxicity may be managed with a brief drug holiday. <i>Note:</i> In severe/refractory heart failure, altered cardiac binding of digitalis may result in toxicity, even with previously appropriate drug doses.</p> <p>Digitalis has a narrow therapeutic serum range, with toxicity occurring at levels that are dependent on individual response. Laboratory levels are evaluated in conjunction with clinical manifestations and ECG to determine individual therapeutic levels/resolution of toxicity.</p> <p>Abnormal levels of potassium, calcium, or magnesium increase the heart's sensitivity to digitalis. Impaired kidney function can cause digoxin (mainly excreted by the kidney) to accumulate to toxic levels. Digitoxin levels (mainly excreted by the bowel) are affected by impaired liver function.</p>
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<p>ACTIONS/INTERVENTIONS</p> <p>Medication Management (NIC)</p>	<p>RATIONALE</p>
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<p>Collaborative</p> <p>Administer medications as appropriate: Other antidysrhythmia medications, e.g., lidocaine (Xylocaine), propranolol (Inderal), and procainamide (Pronestyl) Digoxin immune Fab (Digibind).</p> <p>Prepare patient for transfer to CCU as indicated (e.g., dangerous dysrhythmias, exacerbation of heart failure).</p>	<p>May be necessary to maintain/improve cardiac output in presence of excess effect of digitalis. A digoxin/digitoxin antagonist that increases drug excretion by the kidneys in acute or severe toxicity when standard therapies are unsuccessful.</p> <p>In the presence of digitalis toxicity, patients frequently require intensive monitoring until therapeutic levels have been restored. Because all digitalis preparations have long serum half-lives, stabilization can take several days.</p>
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<p>NURSING DIAGNOSIS: Knowledge, deficient [Learning Need] regarding cause, treatment, self-care, and discharge needs</p> <p>May be related to Lack of information/misunderstanding of medical condition/therapy needs Unfamiliarity with information resources Lack of recall</p> <p>Possibly evidenced by Questions, statement of misconception Failure to improve on previous regimen Development of preventable complications</p> <p>DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:</p> <p>Knowledge: Disease Process (NOC) Verbalize understanding of condition, prognosis, and function of pacemaker (if used). Relate signs of pacemaker failure.</p> <p>Knowledge: Treatment Regimen (NOC) Verbalize understanding of therapeutic regimen. List desired action and possible adverse side effects of medications. Correctly perform necessary procedures and explain reasons for actions.</p>	
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<p>ACTIONS/INTERVENTIONS</p> <p>Teaching: Disease Process (NIC)</p> <p>Independent</p> <p>Assess patient/SO level of knowledge and ability/desire to learn.</p>	<p>RATIONALE</p> <p>Necessary for creation of individual instruction plan. Reinforces expectation that this will be a “learning experience.” Verbalization identifies misunderstandings and allows for clarification.</p>
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<p>ACTIONS/INTERVENTIONS</p> <p>Teaching: Disease Process (NIC)</p>	<p>RATIONALE</p>
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<p>Independent</p> <p>Be alert to signs of avoidance, e.g., changing subject away from information being presented or extremes of behavior (withdrawal/euphoria).</p> <p>Present information in varied learning formats, e.g., programmed books, audiovisual tapes, question-and-answer sessions, group activities.</p> <p>Provide information in written form for patient/SO to take home.</p> <p>Reinforce explanations of risk factors, dietary/activity restrictions, medications, and symptoms requiring immediate medical attention.</p> <p>Encourage identification/reduction of individual risk factors, e.g., smoking and alcohol consumption, obesity.</p> <p>Review normal cardiac function/electrical conduction.</p> <p>Explain/reinforce specific dysrhythmia problem and therapeutic measures to patient/SO.</p> <p>Identify adverse effects/complications of specific dysrhythmias, e.g., fatigue, dependent edema, progressive changes in mentation, vertigo.</p> <p>Instruct and document teaching regarding medications. Include why the drug is needed (desired action), how and when to take the drug, what to do if a dose is forgotten (dosage and usage information), and expected side effects or possible adverse reactions/interactions with other prescribed/OTC drugs or substances (alcohol, tobacco, herbal remedies), as well as what and when to report to the physician.</p>	<p>Natural defense mechanisms, such as anger or denial of significance of situation, can block learning, affecting patient's response and ability to assimilate information. Changing to a less formal/structured style may be more effective until patient/SO is ready to accept/deal with current situation.</p> <p>Multiple learning methods may enhance retention of material.</p> <p>Follow-up reminders may enhance patient's understanding and cooperation with the desired regimen. Written instructions are a helpful resource when patient is not in direct contact with healthcare team.</p> <p>Provides opportunity for patient to retain information and to assume control/participate in rehabilitation program.</p> <p>These behaviors/chemicals have direct adverse effect on cardiovascular function and may impede recovery and increase risk for complications.</p> <p>Provides a knowledge base to understand individual variations and to understand reasons for therapeutic interventions.</p> <p>Ongoing/updated information (e.g., whether the problem is resolving or may require long-term control measures) can decrease anxiety associated with the unknown and prepare patient/SO to make necessary lifestyle adaptations. Educating the SO may be especially important if patient is elderly, visually or hearing impaired, or unable or even unwilling to learn/follow instructions. Repeated explanations may be needed because anxiety and/or bulk of new information can block/limit learning.</p> <p>Dysrhythmias may decrease cardiac output, manifested by symptoms of developing cardiac failure/altered cerebral perfusion. Tachydysrhythmias may also be accompanied by debilitating anxiety/feelings of impending doom.</p> <p>Information necessary for patient to make informed choices and to manage medication regimen. <i>Note:</i> Use of herbal remedies in conjunction with drug regimen may result in adverse effects (e.g., cardiac stimulation, impaired clotting), necessitating evaluation of product for safe use.</p>
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<p>ACTIONS/INTERVENTIONS</p>	<p>RATIONALE</p>
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<p>Teaching: Disease Process (NIC)</p> <p>Independent</p> <p>Encourage development of regular exercise routine, avoiding overexertion. Identify signs/symptoms requiring immediate cessation of activities, e.g., dizziness, lightheadedness, dyspnea, chest pain.</p> <p>Review individual dietary needs/restrictions, e.g., potassium, caffeine.</p> <p>Demonstrate proper pulse-taking technique. Recommend weekly checking of pulse for 1 full minute or daily recording of pulse before medication and during exercise as appropriate. Identify situations requiring immediate medical intervention, e.g., dizziness or irregular heartbeat, fainting, chest pain.</p> <p>Review safety precautions, techniques to evaluate/maintain pacemaker or ICD function, and symptoms requiring medical intervention, e.g., report pulse rate below set limit for demand pacing or less than low-limit rate for rate- adaptive pacers, prolonged hiccups.</p> <p>Recommend wearing medical alert bracelet or necklace and carrying pacemaker ID card.</p> <p>Discuss environmental safety concerns, e.g., microwave ovens and other electrical appliances (including electrical blankets, razors, radio/TV), can be safely operated if they are properly grounded and in good repair. There is no problem with metal detectors, although pacemaker may trigger sensitive detectors. Although cordless phones are safe, cellular phones held directly over pacemaker may cause interference, so it is recommended that patient not carry phone in shirt pocket when phone is on. Additionally, high-voltage areas, magnetic fields, and radiation can interfere with optimal pacemaker function, so patient should avoid high-tension electric wires, arc welding, and large industrial magnets, e.g., demolition sites and MRI.</p>	<p>When dysrhythmias are properly managed, normal activity should not be affected. Exercise program is useful in improving overall cardiovascular well-being.</p> <p>Depending on specific problem, patient may need to increase dietary potassium, such as when potassium-depleting diuretics are used. Caffeine may be limited to prevent cardiac excitation.</p> <p>Continued self-observation/monitoring provides for timely intervention to avoid complications. Medication regimen may be altered or further evaluation may be required when heart rate varies from desired rate or pacemaker's preset rate.</p> <p>Promotes self-care, provides for timely interventions to prevent serious complications. Instructions/concerns depend on function and type of device, as well as patient's condition and presence/absence of family or caregivers.</p> <p>Allows for appropriate evaluation and timely intervention, especially if patient is unable to respond in an emergency situation.</p> <p>Aids in clarifying misconceptions and fears, and encourages patient to be proactive in avoiding potentially harmful situations.</p>
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POTENTIAL CONSIDERATIONS following discharge from care setting (dependent on patient's age, physical condition/presence of complications, personal resources, and life responsibilities)

Activity Intolerance—imbalance between oxygen supply/demand.

Therapeutic Regimen: ineffective management—complexity of therapeutic regimen, decisional conflicts, economic difficulties, inadequate number/types of cues to action.