CERTIFICATION REVIEW: Cardiovascular Part 2

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Content Description

This session reviews the pathophysiology, presentation, diagnosis, and collaborative care of chronic and acute decompensated heart failure. It also discusses cardiomyopathy, valvular diseases and cardiac inflammatory disease as they relate to heart failure. Finally, it will look at dysrhythmias and the care of the patient with a temporary pacemaker. These topics will be discussed with an emphasis on possible questions that may be asked on these subjects in the CCRN, PCCN, and CMC examinations. There will be time allotted for sample questions.

Learning Objectives
At the end of this session, the participant will be able to:

1. Describe the clinical presentation, diagnosis and collaborative management of the patient with chronic and acute decompensated heart failure.

2. Describe the presentations and collaborative care of cardiomyopathy, valvular disease, and cardiac inflammatory disease.

3. Discuss the identification of and interventions for patients experiencing cardiac dysrhythmia and those requiring a temporary pacemaker

REFERENCES

NOTE: Please refer to outline for references pertaining to this session.
Certification Review: Cardiovascular Part 2

Approximately 20% of the CCRN exam, 36% of the PCCN exam and 43% of the CMC exam will focus on cardiovascular disease.

<table>
<thead>
<tr>
<th>CCRN, PCCN and CMC</th>
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<th>CCRN and CMC only</th>
<th>PCCN and CMC only</th>
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<tbody>
<tr>
<td>Acute Coronary Syndrome</td>
<td>Interventional cardiology</td>
<td>Cardiac surgery</td>
<td>Acute peripheral vascular insufficiency/ peripheral vascular surgery</td>
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<tr>
<td>☑ Heart Failure</td>
<td>☑ Acute pulmonary edema</td>
<td>Hypertensive crisis</td>
<td>Ruptured or dissecting aneurysm</td>
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<td>☑ Dysrhythmias</td>
<td>☑ Conduction defects</td>
<td>Cardiac trauma</td>
<td>Acute inflammatory disease</td>
</tr>
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<td>☑ Cardiomyopathies</td>
<td>☑ Structural heart defects</td>
<td>Cardiogenic shock</td>
<td>Cardiac tamponade</td>
</tr>
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<td>Hypovolemic shock (in multisystem on PCCN; discussed here)</td>
<td></td>
<td>Hypovolemic shock (in multisystem on PCCN; discussed here)</td>
<td>Pulmonary hypertension (in pulmonary on PCCN. Discussed in pulmonary session)</td>
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Note for PCCN candidates: This presentation includes discussions of pulmonary artery catheter measurements, administration of vasoactive medications, and advanced mechanical devices such as intra-aortic balloon pump and ventricular assist devices. These topics will not be tested in the PCCN exam.

I. Heart failure
   A. Definitions
      1. ACC/AHA Practice Guidelines
         A complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood.
      2. The inability of the heart to maintain a continuous flow of blood commensurate with the metabolic needs of tissues and organs, or to do so only by means of increased filling pressures.
   B. Determination of severity of heart failure

   New York Heart Association Functional Classification System
<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No limitations</td>
</tr>
<tr>
<td>Minimal</td>
<td>Normal daily activity does not initiate symptoms of fatigue, dyspnea, palpitations or angina</td>
</tr>
</tbody>
</table>
New York Heart Association Functional Classification System

Class II  Slightly limited physical activity
Mild     Comfortable at rest
         Normal daily activities initiate onset of symptoms
Class III Markedly limited physical activity
Moderate Comfortable at rest
         Less than normal activity initiates symptoms
Class IV  Any type of activity initiates symptoms
Severe   Symptoms may occur at rest

C.  Cardinal symptoms of heart failure
    Dyspnea
    Fatigue
    Fluid retention

D.  Causes
    Most common causes
        CAD
        Hypertension
        Dilated cardiomyopathy
    Other causes
        Valvular disease
        Diabetes mellitus
        Hyperlipidemia
        Smoking
        PVD
        RHD
        Mediastinal irradiation
        Sleep apnea
        Illicit drug use

E.  Physiology of HF
    Effects of HF on cardiac output
        Heart rate
        Preload
        Afterload
        Contractility
        Components of stroke volume
    Neurohormonal mechanisms in heart failure
    Sympathetic nervous stimulation
    Renin-angiotensin-aldosterone system activity
    Meant to be compensatory
    Results in:
        Structural changes
            Chamber dilation or hypertrophy (cardiac remodeling)
            Damaged myocytes
            More spherical shape
Decreased functioning

F. Diagnostic tests

Diagnosis made mainly through history and physical findings
Chest X-Ray: Enlarged cardiac silhouette; Kerley B lines, pleural effusion
ECG: Q waves, IVCD, atrial fibrillation, ventricular ectopy, atrial enlargement, ventricular hypertrophy
Both nonspecific.
Two-dimensional echocardiogram with Doppler flow studies: abnormal wall motion, chamber dilation, hypertrophy, valve dysfunction, decreased ejection fraction. Most useful test

G. Labs

CBC, electrolytes, including Ca++, Mg+ and Phosphorus, BUN, creatinine, liver enzymes, thyroid testing
B-type natriuretic peptide (BNP) levels
Elevated in proportion to severity of HF - ≥100 pg/ml
Lower levels rule out HF as a cause of dyspnea - ≤ 80 pg/ml
Levels correlate with NYHA classifications of heart failure

H. Treatment

Drug therapy

Diuretics

Loop – Furosmide (Lasix), bumetanide (Bumex)
Monitor K+, Mg+, BUN, Cr, hypotension, I&O, daily weights
Administer IV push slowly – 20 mg/min to avoid ototoxicity
Potassium-sparing – spironolactone (Aldactone)
Use in combination with loop diuretics; watch for hyperkalemia
Contraindicated in patients with SPB < 90 mmHg, cardiogenic shock
Thiazide – Metolazone (Zarolyolyn)
Enhance diuresis when used with furosemide or bumetanide
ACE inhibitors - captopril (Capoten), enalapril (Vasotec)
Inhibits RAA system; monitor K+, BUN, creatinine, hypotension, dry cough, angioedema
ARBs - losartan (Cozaar), valsartan (Diovan)
Not as effective as ACEI, less risk of cough, angioedema. Monitor as with ACEI
Beta-blockers - bisoprolol (Zebeta), carvedilol (Coreg), and metoprolol (Lopressor) only βB recommended in AHA/ACC guidelines
Titrate slowly; contraindicated in hypotension, bradycardia, 2\textsuperscript{nd} & 3\textsuperscript{rd} degree AVB, asthma

**Digitalis glycosides**
Positive inotrope, increases cardiac contractility; may also down-regulate sympathetic activity. Indicated for patients who remain symptomatic or who are being titrated on 𝛽B. Monitor dysrhythmia, esp. heart block, N/V, visual disturbances, dig toxicity.

I. **Management of Patient with HF**
Control of hypertension, diabetes, hyperlipidemia
Smoking cessation; restrict/eliminate alcohol use
Healthy diet/low sodium diet, 2-4 gm
Fluid restriction, 64 oz/day; daily weight
   Report weight gain of 3-4 lb over several days.
Flu and pneumonia vaccination
Supervised exercise

J. **Acute decompensated heart failure (ADHF)**
1. **Clinical presentation**
   **Left heart failure**
   **Right heart failure**

   Dyspnea
   Tachycardia
   S\textsubscript{3}
   Crackles
   Frothy sputum
   Oliguria
   Decreased PaO\textsubscript{2}, SaO\textsubscript{2}
   PAOP > 20 mmHg
   CO < 4L/Min
   SVR > 1200 dynes/cm/sec\textsuperscript{-5}

   Diagnostics
   CXR, ECG, BNP

2. **Drug therapy in ADHF**
Diuretics, ACEI/ARB as with chronic HF
Decrease dose of beta-blocker
   May not be able to discontinue
Vasodilators: Nitroglycerin, nitroprusside (Nipride)
   Decrease preload and afterload
   May increase RA activity
Use with ACEI or ARB to prevent
Decrease myocardial oxygen consumption
Do not cause dysrhythmias.
Nitroglycerin
Venous dilator and preload reducer. Decreases coronary vascular resistance. Afterload reducer at doses >30 mcg/min;. May cause headache and abdominal pain
Nitroprusside
Venous and arterial dilator at all doses. Effective in acute mitral or aortic regurgitation. May cause coronary steal – vasodilation in nonischemic areas that can shunt blood from areas of ischemia. Should have arterial line and/or PA catheter. Monitor for cyanide toxicity.
Morphine
If given, should be after nitroglycerin treatment. No evidence of efficacy; second-line treatment
Neurohormonal: nesiritide (Natrecor)
Reduces preload and afterload
Improves CO/CI
Diuretic and natriuretic
Decreased levels of aldosterone and norepinephrine
Fast onset of action
Decreases PAOP within 15 minutes
No atrial or ventricular arrhythmias as with positive inotropes
Does not require ICU admission, arterial line, PA catheter.
Does not appear to increase mortality.
Major side effect is hypotension.
Turn off or decrease rate of infusion. Reposition patient on side, administer fluids. Restart nesiritide at rate 30% lower when BP stable
Recent concern that it may cause decreased renal function
Inotropes: Dobutamine (Dobutrex), milrinone (Primacor)
Increase CO by increasing cardiac contractility
Increases heart rate
Increases myocardial oxygen demand
Associated with increased mortality
Contraindicated in hypertrophic cardiomyopathy, aortic stenosis
Should only be used in shock or poor perfusion
Milrinone does not compete with beta blockade

3. Oxygen
CPAP or BiPAP
Noninvasive increase in intrathoracic positive pressure decreases preload
Mechanical ventilation if NIPPV is not effective and in cardiogenic shock

4. Other treatment modalities

ICD
Sudden death often cause of mortality in HF. Implantation of ICD may decrease mortality. Increase in hospital admission for HF, possibly due to pacemaker function of IVCD causing ventricular dyssynchrony. Used in conjunction with ventricular antidysrhythmics such as Amiodarone

Cardiac resynchronization
IVCD results in dyssynchronous ventricular contractions. Worsens systolic HF, interferes with diastolic function. May worsen MR.
Biventricular pacemaker synchronized to patient’s sinus rhythm and programmed to stimulate the right ventricle with a conventional lead and the left ventricle through a specially designed coronary sinus lead.
Biventricular pacing and ICD capabilities in most cases.
Improves NYHA class, exercise tolerance, LVEF, and quality of life

Ultrafiltration
Removal of fluid via ultrafiltration mode of CRRT
Administered over 8 hours
Maximum fluid removal 500 ml/hr
Well tolerated

5. Nursing interventions
Patient assessment Q 1-2 hours:
VS, LOC
Head-to-toe assessments
Neuro: decreased mentation can indicate poor perfusion to brain
Cardiac: Heart sounds: S3, gallop, murmur, distant
Peripheral pulses, edema
Skin temperature, color, moisture
Nailbeds: color, capillary refill
Hemodynamic parameters: CO, PAP, PAOP, SVR
Baseline, after initiation of therapy to determine effectiveness
Pulmonary: Crackles, wheezes, tachypnea, frothy sputum
GI: N/V, appetite, bowel sounds
GU: urine output: oliguria, anuria. Appearance: concentrated, dilute
Daily weights, I&O
Monitor electrolytes
Assess effectiveness of interventions
Patient/family education
  Medication
  Tests
  Therapies
  ICU routine
Discharge teaching
  Activity level
  Diet, including sodium and fluid restriction
  Discharge medications
  Weight monitoring

Certification Questions

1. A patient is admitted with complaints of chest pain accompanied by nausea and vomiting. The skin is cool and clammy. The following are noted:
   VS:  BP 140/90; HR 120; RR 26  CO 3.5  L/min
   CVP 10 mmHg    CI 2.1  L/min/M³
   PAOP 20 mmHg    Presence of S3
   12 lead ECG shows acute changes in V2, V3, and V4
For this patient, the goals of therapy would be to:
   A. Decrease preload and afterload 
   B. Increase preload and afterload 
   C. Increase contractility and preload
   D. Decrease contractility and afterload

2. A patient with acute decompensated heart failure is receiving a continuous infusion of nesiritide (Natrecor) at 0.1 mcg/kg/min. Currently, the patient has the following vital signs: HR 116 bpm, sinus tachycardia with premature atrial contractions, BP 78/48 mmHg, RR 28/min, and SpO₂ 90% on 40% BiPAP mask. Immediate interventions by the nurse would include which of the following?
   A. Discontinue the nesiritide (Natrecor) infusion
   B. Place the patient in a supine position
   C. Administer a 250 ml normal saline fluid bolus
   D. Continue the nesiritide (Natrecor) infusion and administer 40 mg furosemide (Lasix) intravenously

3. After implantation of a biventricular pacemaker for end-stage heart failure, ECG signs that the pacemaker is not functioning properly would include:
A. A-V interval less than 0.20 sec  
B. Widening of the QRS  
C. T-wave inversion  
D. More than one P wave for each QRS

4. A Patient admitted with shortness of breath demonstrates the following findings: temperature 36.8oC, HR 120/min sinus tachycardia, BP 130/76 mmHg, RR 36/min with SpO2 91%. Breath sounds reveal inspiratory crackles and rhonchi in all lung fields, The chest X-ray report states that there are Kerley B lines, enlargement of the peribronchial hilar spaces, and enlarged cardiac silhouette. These findings are consistent with which of the following?  
A. Pericardial tamponade  
B. Pulmonary edema  
C. Pneumonia  
D. Acute inferior wall MI with right ventricular failure

5. When performing patient teaching with a heart failure patient, the nurse should instruct the patient to immediately contact the HCP for which of the following?  
A. Weight gain greater than 2 kg in 24 hours  
B. Development of cough  
C. Leg edema  
D. Increased fatigue and exercise intolerance for 24 hours

II. Cardiomyopathy  
A. A Comparison of Three Types of Cardiomyopathy

<table>
<thead>
<tr>
<th>Normal</th>
<th>Dilated</th>
<th>Hypertrophic</th>
<th>Restrictive</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="image1" alt="Heart Normal" /></td>
<td><img src="image2" alt="Heart Dilated" /></td>
<td><img src="image3" alt="Heart Hypertrophic" /></td>
<td><img src="image4" alt="Heart Restrictive" /></td>
</tr>
<tr>
<td>Pathophysiology</td>
<td>Most common; all four chambers dilated; ♦ cardiac contractility; ⊤ risk of thrombus formation</td>
<td>Hypertrophy of ventricular septum and free wall Obstruction of outflow tract through aortic valve</td>
<td>Abnormal diastolic filling D/T rigid ventricular walls; contractility mostly unimpaired</td>
</tr>
<tr>
<td>Clinical Manifestations</td>
<td>S/S right and left heart failure, chest pain,</td>
<td>Dyspnea, palpitations, fatigue, mitral and aortic murmur, syncope,</td>
<td>Chest pain, dyspnea, fatigue, S/S RHF, JVD, peripheral edema,</td>
</tr>
</tbody>
</table>
Normal | Dilated | Hypertrophic | Restrictive
--- | --- | --- | ---
palpitations, fatigue, syncope | murmur of mitral regurgitation, aortic stenosis | hepatomegaly

### Hemodynamic Findings

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<tr>
<td>CO normal</td>
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<td>CO normal</td>
</tr>
<tr>
<td>CVP, PAP, PAOP, SVR</td>
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### Pharmacology

<table>
<thead>
<tr>
<th>Normal</th>
<th>Dilated</th>
<th>Hypertrophic</th>
<th>Restrictive</th>
</tr>
</thead>
<tbody>
<tr>
<td>HF medications, anticoagulants</td>
<td>HF medications, antidysrythmics, anticoagulants. No inotropes</td>
<td>HF medications, antidysrythmics.</td>
<td></td>
</tr>
<tr>
<td>Caution with nitrates, diuretics, preload lowering drugs</td>
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<td></td>
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### Collaborative care

- All: activity restriction, sodium and fluid restriction, oxygen, VS, ECG and hemodynamic monitoring, I&O, daily weights, systems assessments, space activities, promote rest. ICD, IABP, heart transplant.
- Teaching: Diet, medications, activity, support groups, coping strategies, CPR for family.

### III. Valvular Heart Disease

#### A. Description

- caused either by stenotic or incompetent valves; usually affects the mitral or aortic valve
  - **Stenotic**
    - Valve opening progressively decreases in size; forward flow of blood is restricted. Affected chamber becomes hypertrophied
  - **Incompetent**
    - Also known as insufficiency or regurgitation
    - Valve does not completely close; blood backflows into chamber, resulting in volume overload and dilated chamber
    - Causes specific murmur, either systolic or diastolic

#### B. Etiology

- Rheumatic heart disease most common cause
- Infection, MI, systemic disease
- Aortic and mitral valves most commonly affected

#### C. Location and timing of murmurs with clinical manifestations
### Valvular dysfunction

<table>
<thead>
<tr>
<th>Location and timing of murmur</th>
<th>Clinical Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aortic insufficiency</strong></td>
<td>Dyspnea, chest pain, palpitations, S/S LHF and RHF, widened pulse pressure, ▼ CO ▲ CVP, PAP, PAOP</td>
</tr>
<tr>
<td><strong>Aortic stenosis</strong></td>
<td>Dyspnea, chest pain, palpitations, fatigue, S/S LHF and RHF, syncope, narrowed pulse pressure, AF, AV block, LBBB, ▲ CO ▼ CVP, PAP, PAOP, SVR</td>
</tr>
<tr>
<td><strong>Mitral insufficiency</strong></td>
<td>Dyspnea, fatigue, pulmonary HTN, pulmonary edema, S/S RHF, AF, ▼ CO ▲ CVP, PAP, PAOP</td>
</tr>
<tr>
<td><strong>Mitral stenosis</strong></td>
<td>Dyspnea, S/S RHF, fatigue, palpitations, pulmonary HTN, pulmonary and systemic edema, AF, ▼ CO ▲ CVP, PAP, ▼ PAOP w/ large a waves</td>
</tr>
</tbody>
</table>

#### D. Medical and surgical management

**Medical**
- Activity restriction, sodium and fluid restriction, oxygen
- Management of heart failure; inotropes, diuretics, ACE inhibitors
- Monitor for complications: dysrhythmias, emboli

**Surgical**
- Valvuloplasty - repair of valve
- Commissurotomy - surgical separation of valve leaflets
- Valve replacement
  - Homograft - valve from human cadaver; lasts 5-8 years
  - Heterograft - valve from animal, lasts 5-8 years
  - Artificial graft - lasts 10-15 years; requires long term anticoagulation

**Nursing care post-op similar to CABG**

#### IV. Inflammatory disease

**A. Endocarditis**

**Description**
- Infection of microorganisms circulating in the bloodstream
Bacteria carried through system and deposited onto endocardial surfaces, especially valve leaflets

B. Infectious process
   Bacteria grow on leaflets – Vegetation
   Lesions or vegetations form on valves
   Valves have irregular or “cauliflower” appearance
   Leaflets damaged and dysfunctional
   Life-threatening
   May require valve surgery
   Can grow to involve the:
      Cordae tendinae
      Papillary muscles
      Conduction system

C. Causative organisms
   Streptococcus (most common)
   Staphylococcus
   Gram negative bacilli (E. coli, Klebsiella)
   Fungi (Candida, Histoplasma)

D. Affected valves
   Mitral most commonly affected
   Aortic second most affected

E. Populations at risk
   Rarely occurs in people with normal hearts
   IV drug abuse
   Risk increased with preexisting cardiac conditions
      Prosthetic valves
      History of previous endocarditis
      Damaged or abnormal heart valves due to:
      Rheumatic fever
      Congenital heart disease
      Congenital valve defects

F. Presenting symptoms
   Dependent on:
   Valve involved
   Organism
   Duration of time
   Extent of vegetative growth
   Fever
   Fatigue
   Chills
   Headache
   Night sweats
   Musculoskeletal complaints
Cough         New murmur
Weight loss   Heart failure
General malaise Positive blood cultures
Weakness      Anemia

G. Treatment
Blood cultures to identify organism
Administration of antibiotics or antifungals
Oxygen if indicated
HF treatment if present
May require valve replacement surgery

V. Dysrhythmias
A. Defined
Any cardiac rhythm other than sinus rhythm at a normal rate
Due to MI, ischemia, hypoxemia, electrolyte imbalance, acid-base imbalance
B. Clinical Manifestations
Anxiety, weakness, dizziness, syncope, chest pain, dyspnea, change in level of consciousness, palpitations, S3 heart sound, crackles.
Signs and symptoms of decreased cardiac output:
   Tachycardia, hypotension, tachypnea, cool, clammy skin, oliguria/anuria, restlessness, confusion.
C. Collaborative Management
Monitor cardiac rhythm for dysrhythmias
Obtain 12-lead ECG with onset of dysrhythmia
Administer oxygen if indicated
Maintain patent IV access
Treat etiology, follow ACLS algorithms - http://www.acls.net
Antidysrhythmic therapy
   Asystole, PEA: Transcutaneous pacemaker (TCP), epinephrine, atropine
   Pulseless VT, VF: Shock, epinephrine, vasopressin in place of one dose of epi
   Sinus tach, SVT, AF, atrial flutter: adenosine, amiodarone
   Sinus brady, Wenckebach, 3rd degree AVB: atropine, TCP
   VT with pulse: amiodarone
Defibrillation or cardioversion
Provide emotional support to patient and family
Patient/family education
D. Pacemaker therapy for dysrhythmias
1. Indications: 2nd degree AVB, type II, 3rd degree AVB, atrial fibrillation with
slow ventricular response; symptomatic bradycardias

2. Types of temporary pacemakers
   Transcutaneous (TCP)
   Pacing through chest; electrodes on skin surface
   Usually used in emergent situations
   Not as effective as other forms of temporary pacing
   Transvenous
   Pacing electrode advanced through central access into RV
   Epicardial
   Pacing electrodes sewn to epicardium during cardiac surgery

3. Most frequently used modes of temporary pacing and pacing codes
   Three categories, each represented by a letter.
   First letter refers to chamber that is paced
   Second letter represents chamber being sensed
   Third letter is chamber being triggered and/or inhibited in response to the sensing
   Five categories for permanent pacemakers
   Ventricular pacing – VVI
   Ventricle paced, sensed and inhibited
   One pacemaker spike, just before QRS; appears wide
   May not see with every beat; depends on rate. If rate if faster that set rate, pacemaker will not fire.

   ![Dual chamber pacing](image)

   pacing - DDD
   Atrium and ventricle paced, sensed and triggered or inhibited in response to sensing
   Two pacemaker spikes, one before P wave, one before QRS
   May see any combination, depending on rate, conduction
   No dual chamber with TCP
TCP

Does not use letter system; only two types of settings
Demand (Synchronous)
   Paces only when patient’s HR falls below set rate
   Appearance similar to VVI
Fixed (Asynchronous)
   Unable to obtain capture
   Pacer unable to sense intrinsic activity
   Artifact prevents sensing
   Danger of pacemaker spike falling on T wave and causing ventricular tachycardia or ventricular fibrillation

4. Pacemaker settings
   Rate control
      Regulates impulses per minute; usually set between 60-80 bpm.
      AV pacemaker, rate controls both
      Higher if being used for overdrive suppression of tachyarrhythmias.
      Ordered by physician
   Output dial
      Regulates amount of electrical current delivered to initiate depolarization and contraction
      Measured in milliamperes (mA).
   Threshold (capture)
      Point at which depolarization occurs
      Pacemaker spike followed by P wave (atrial pacing) or QRS (ventricular pacing)
      Separate output dials for atrial and ventricular pacing.
   Sensitivity control
      Regulates ability of the pacemaker to detect the heart’s intrinsic electrical activity.
      Measured in millivolts (mV)
      Pacemaker has a sense indicator, usually a light, which will indicate each time the pacemaker senses electrical activity.
      To increase sensitivity, the dial is turned down
      No sensitivity on TCP.
   AV interval control
      Used in AV pacing.
      Regulates the time interval between the atrial and ventricular pacing stimuli
      Sets PR interval; usually equal to PR of .20.
5. Initiating pacing
   Set heart rate and AV control per physician order
   Determine threshold
   Set mA 2-3 times higher than threshold to ensure capture
   TCP may increase mA by 10% after capture obtained
   Set sensitivity control so that pacemaker senses the heart’s intrinsic
electrical activity (R wave)
   Ensure that sensitivity not so low that it also senses lower
amplitude electrical signals such as the T wave
   Will consider it to be an R wave and double-count rate
   Will not pace when HR is below set rate
   No sensitivity on TCP

TCP Preparation
   Explain to patient and family
   TCP will be uncomfortable
   May require analgesia
   Thoroughly wash and dry skin
   Use skin preparation
   Pad placement
   Anterior-posterior preferred
   Anterior-anterior may need to be used

6. Nursing care
   Report settings as part of shift change report
   Check connections
   Temporary/epicardial
   Wear gloves when handling pacing wires to prevent microshock
   and VF
   When not in use, cover leads with gauze and secure to
   patient with tape
   Secure temporary pacemaker to patient’s waist with
   strap/telemetry pouch.
   Suspend from IV pole with twill tape if patient is on
   bedrest.
   Have extra batteries and pacemaker on hand.
   Perform site care, inspect site to prevent/identify infection

TCP
   Assess integrity of TCP pads
   Assess skin integrity
   Be sure TCP is plugged in

7. Pacemaker malfunction
Failure to capture
Pacemaker spikes not followed by complex

All: May be due to mA too low, low battery
Transvenous/epicardial: fibrin at tip of lead, fractured lead wire, movement away from ventricular wall
Increase mA, replace battery, notify physician

Failure to sense
Pacemaker does not sense patient’s intrinsic rhythm; paces inappropriately
Pacemaker spikes throughout strip, do not correlate with complexes

Transvenous/epicardial: Due to sensitivity set to low
Correct by increasing sensitivity
To increase, turn sensitivity down
TCP: No sensitivity setting
Change to fixed mode setting

Certification Questions
1. Which of the following medications is administered to prevent sudden death associated with dilated cardiomyopathy?
   A. Warfarin (Coumadin) to prevent clot formation
   B. Calcium channel blockers to control tachycardia
   C. Nitrates to improve coronary artery perfusion
   D. Digoxin to reduce atrial dysrhythmias and improve contractility

2. Chest pain associated with aortic stenosis can be caused by:
A. Decreased stroke volume  
B. Disproportionate oxygen supply versus demand  
C. Prolapsed valve leaflets  
D. Decreased contractility

3. Which of the following medications may worsen symptoms of heart failure associated with hypertrophic cardiomyopathy?  
   A. Calcium channel blockers  
   B. Beta-blockers  
   C. Nitroglycerin  
   D. Amiodarone

4. Which of the following medication regimens would be most appropriate to relieve chest pain in a patient with a diagnosis of myocarditis?  
   A. Nitroglycerin 1/150 grains sublingual  
   B. Furosemide 40 mg IV  
   C. Ibuprofen 800 mg PO  
   D. Morphine sulfate 2 mg IV

5. In a coronary care unit in which medications are not permitted to be left at the bedside and the crash cart with monitor/transcutaneous pacemaker/defibrillator is outside the central nurses’ station, an intubated patient on mechanical ventilation develops third-degree AV block at a rate of 35 beats/min with signs of poor tissue perfusion. The most appropriate initial intervention for the nurse assigned to this patient would be to  
   A. Initiate transcutaneous pacing  
   B. Administer atropine 0.5 mg IV  
   C. Initiate an infusion of epinephrine at 5 mcg/min  
   D. Initiate an infusion of dopamine at 5 mcg/kg/min

6. The nurse should perform which of the following interventions for a patient with chest pain, hypotension and tachycardia at a rate of 180 bpm?  
   A. Administer amiodarone 150 mg IV over 10 minutes  
   B. Administer adenosine 6 mg rapid IV push  
   C. Perform synchronized cardioversion  
   D. Defibrillate with 300 joules

7. A patient who suffered complete heart block after an anterior wall infarction receives a temporary pacemaker. The following rhythm develops:
The nurse would respond by initiating which pacemaker action:
   A. Changing the pacing mode from paced to fixed (asynchronous).
   B. Increasing the output
   C. Decreasing the output
   D. Increasing the paced rate


