Cardiovascular Concepts

A journey of a thousand miles begins with a single step.

—Lao Tzu

CARDIOVASCULAR TEST BLUEPRINT

Cardiovascular 17% of total test **26 questions**

→ Acute coronary syndrome (e.g., NSTEMI, STEMI, and unstable angina)
→ Acute peripheral vascular insufficiency (e.g., arterial/venous occlusion, carotid artery stenosis, endarterectomy, fem-pop bypass)
→ Acute pulmonary edema
→ Aortic aneurysm
→ Aortic dissection
→ Aortic rupture
→ Cardiac surgery (e.g., CABG, valve replacement or repair)
→ Cardiac tamponade
→ Cardiac trauma
→ Cardiac/vascular catheterization
→ Cardiogenic shock

→ Cardiomyopathies (e.g., dilated, hypertrophic, idiopathic, restrictive)
→ Dysrhythmias
→ Heart failure
→ Hypertensive crisis
→ Myocardial conduction system abnormalities (e.g., prolonged QT interval, Wolff-Parkinson-White)
→ Papillary muscle rupture
→ Structural heart defects (e.g., acquired and congenital, including valvular disease)
→ TAVR

CARDIOVASCULAR TESTABLE NURSING ACTIONS

- □ Apply leads for cardiac monitoring
- □ Identify, interpret, and monitor cardiac rhythms
- □ Recognize indications for, and manage, patients requiring:
  - 12-lead ECG
  - Arterial catheter
  - Cardiac catheterization
  - Cardioversion
  - Central venous pressure monitoring
  - Defibrillation
  - IABP
  - Invasive hemodynamic monitoring*
  - Pacing: epicardial, transcutaneous, transvenous
  - Pericardiocentesis

- QT interval monitoring
- ST segment monitoring

□ Manage patients requiring:
- Endovascular stenting
- PCI

*Since hemodynamic monitoring also includes respiratory concepts and multisystem concepts, this book places the chapter on hemodynamic concepts after the chapters on respiratory concepts and multisystem concepts.

Cardiovascular (CV) questions outnumber all of the other clinical topics included on the Adult CCRN exam, so plan on spending the most time studying CV. The 26-question total for CV includes hemodynamic monitoring; therefore, plan to spend about 26 hours reviewing the CV and Hemodynamics chapters of this book and practicing test questions related to CV and hemodynamics.

Cardiovascular Assessment

Select assessment concepts are included on the Adult CCRN exam. These concepts may be covered in assessment questions, or they may be incorporated in the cardiovascular disorder questions.

Normal Heart Sounds in Adults

S1

- “Lub”
- Caused by closure of AV (mitral, tricuspid) valves
- Loudest at the **apex** of the heart (midclavicular, 5th intercostal space)
- Marks the end of diastole, the beginning of **systole**

S2

- “Dub”
- Caused by closure of semilunar (aortic, pulmonic) valves
- Loudest at the **base** of the heart (right sternal border, 2nd intercostal space)
- Marks the end of systole, the beginning of **diastole**
- S2 splits on inspiration; wide, fixed splitting of S2 is caused by right bundle branch block (RBBB)
- S2 is **louder with a pulmonary embolism**

➤ Each of the 4 valves has an auscultatory point on the chest wall. You need to know these points (Figure 3-1).
➤ The base of the heart is the aortic area, where S2 (“dub”) is loudest. Anatomically, it is at the 2nd intercostal space (ICS), right sternal border.
➤ The apex of the heart is the mitral area, where S1 (“lub”) is loudest. Anatomically, it is at the 5th ICS, midclavicular.

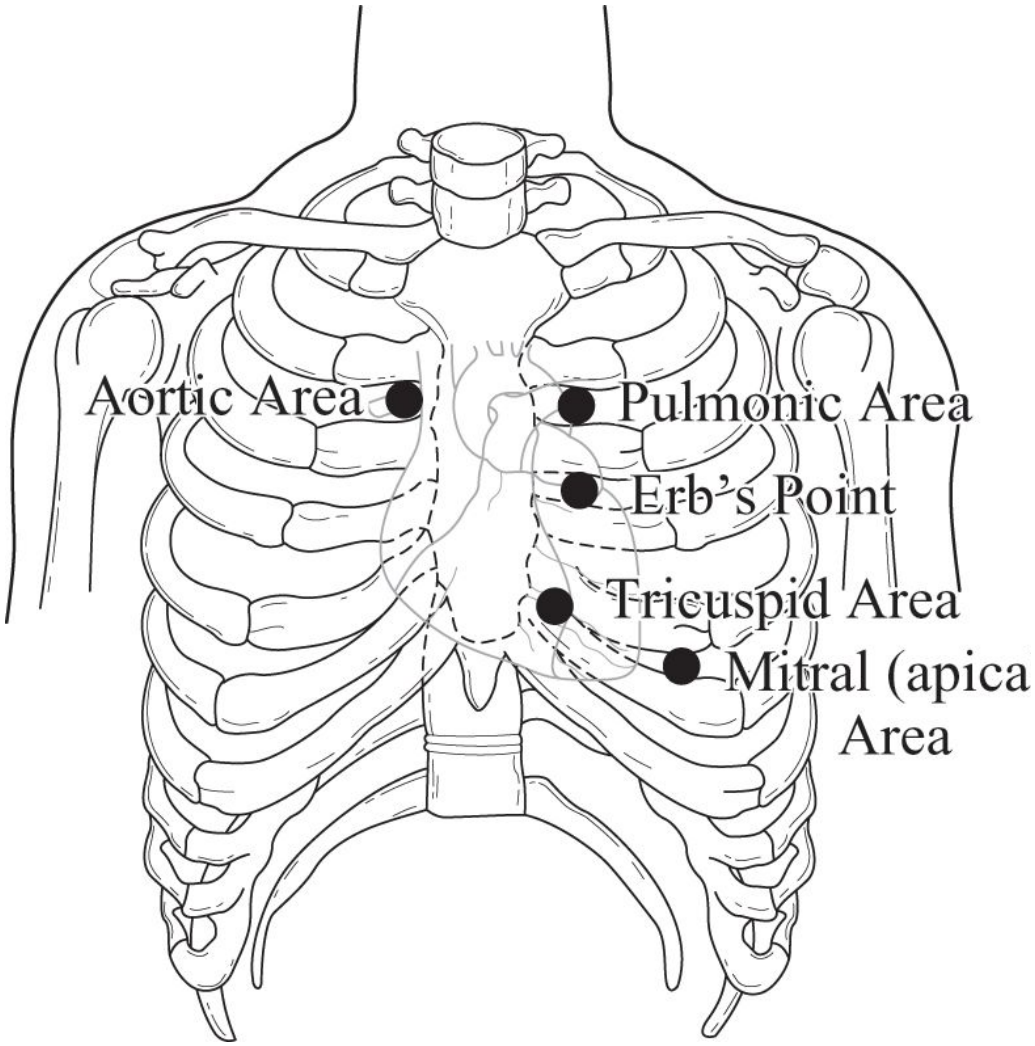


Figure 3-1. Heart auscultatory points on the chest wall and associated valves

Abnormal Heart Sounds in Adults

S3

- Caused by a rapid rush of blood into a dilated ventricle
- Occurs early in diastole, right after S2
- Heard best at the apex with the bell of the stethoscope

☆ Associated with heart failure; may occur before crackles

- Ventricular gallop, “Kentucky”

☆ S3 is also caused by:

- Pulmonary hypertension and cor pulmonale
- Mitral, aortic, or tricuspid insufficiency

S4

- Caused by atrial contraction of blood into a noncompliant ventricle
- Occurs right before S1
- Best heard at the apex with the bell of the stethoscope
- Associated with myocardial ischemia, infarction, hypertension, ventricular hypertrophy, and **aortic stenosis**
- Atrial gallop, “Tennessee”

TIP

S4 is not heard in the presence of atrial fibrillation . . . why?

No atrial contraction

Pericardial Friction Rub

- Due to pericarditis, associated with pain on deep inspiration
- May be positional

Murmurs

- Valvular disease; see the Valvular Heart Disease section that follows for a detailed discussion of this topic
- Septal defects (atrial or ventricular)

Blood Pressure and Pulse Pressure

- Pulse pressure:

$$\text{Systolic} - \text{Diastolic} = \text{Pulse Pressure}$$

- Normal pulse pressure is 40–60 mmHg (i.e., 120/80).
- Systolic blood pressure is an indirect measurement of the cardiac output and stroke volume.
  - A decrease in systolic pressure with little change or an increase in diastolic pressure is narrowing of pulse pressure; seen most often with severe hypovolemia or a severe drop in cardiac output (CO), i.e., 100/78.
- Diastolic blood pressure is an indirect measurement of the systemic vascular resistance (SVR).
  - A decrease in diastolic pressure that widens pulse pressure may indicate vasodilation, a drop in SVR; often seen in sepsis, septic shock (i.e., 100/38).
- Diastole is normally one-third longer than systole.

☆ Coronary arteries are perfused during diastole.

Valvular Heart Disease

You will most likely see several questions related to valvular heart disease and/or heart sound assessment. Since there are 4 heart valves and each valve may be diseased with either **stenosis** or **insufficiency**, it would be next to impossible to memorize whether each problem is a systolic or diastolic murmur. If you can picture which valves are open and which are closed during each phase of the cardiac cycle (systole and diastole), you will be able to decide what problem is being described in each question. First, let's review some heart sound basics.

- Normal heart sounds, S1 and S2 in adults, are due to valve closure.
- Valves open and close based on the pressure changes in the chamber above the valve and below the valve. When the pressure in the chamber above a valve is higher than that below the valve, the valve opens. When the pressure drops in the chamber above the valve and the pressure is greater below the valve, the valve closes.
- **Systole:** ejection, high pressure
- **Diastole:** filling, low pressure
- Which is longer, systole or diastole? Diastole is one-third longer than systole, needs time for filling.
- When are coronary arteries perfused, during systole or diastole? Diastole
- Why do the cardiac output and blood pressure drop with extreme tachyarrhythmias? No time for filling, therefore less output.

Causes of Valvular Heart Disease

- Coronary artery disease, ischemia, and acute MI
- Dilated cardiomyopathy
- Degeneration
- Bicuspid aortic valve; genetic
- Rheumatic fever
- Infection
- Connective tissue diseases

Murmurs

- Murmurs of **INSUFFICIENCY** (regurgitation) occur when the valve is **closed**.
  - Acute or chronic
- Murmurs of **STENOSIS** occur when the valve is **open**.
  - Chronic problem, develops over time
  - NOT acute

SYSTOLIC MURMURS (FIGURE 3-2)

Lub . . . shhhb . . . Dub

- Semilunar valves are OPEN during systole.
  - Aortic stenosis
  - Pulmonic stenosis
- AV valves are CLOSED during systole.
  - Mitral insufficiency
    - Will cause large, giant V-waves on the pulmonary artery occlusion pressure tracing if the patient has a pulmonary artery catheter
  - Tricuspid insufficiency
- Ventricular septal defect (VSD), which is most common with an acute MI, may result in a systolic murmur. It is heard at the left sternal border, 5th intercostal space (ICS).

DIASTOLIC MURMURS (FIGURE 3-3)

Lub . . . Dub . . . shhhb

- Semilunar valves are CLOSED during diastole.
  - Aortic insufficiency (AI)
  - Pulmonic insufficiency (PI)
- AV valves are OPEN during diastole.
  - Mitral stenosis is associated with atrial fibrillation due to atrial enlargement that occurs over time.
  - Tricuspid stenosis

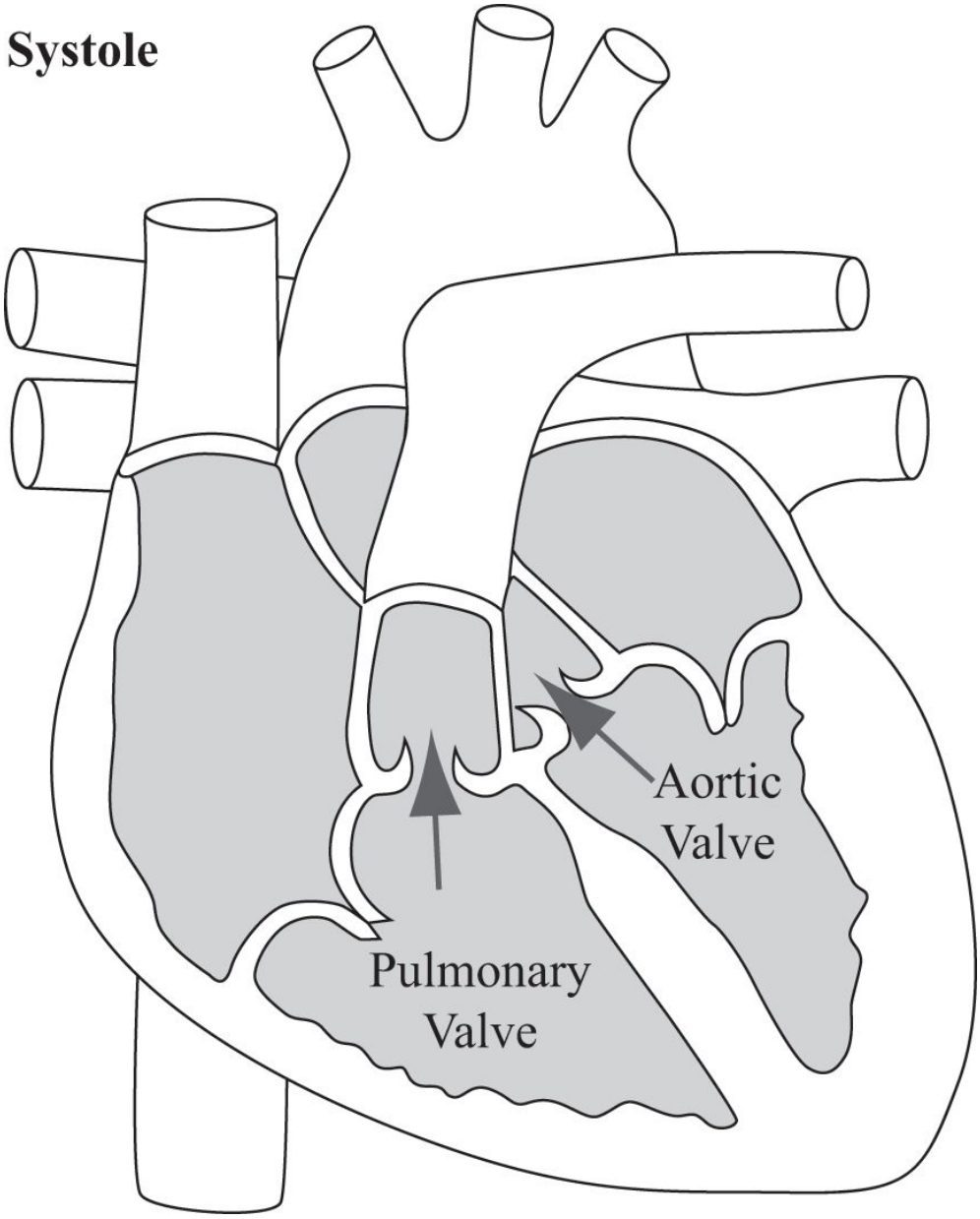


Figure 3-2. Systole, closure of AV valves, S1 “Lub”

→ The semilunar valves (pulmonic, aortic) are OPEN during systole.

→ The AV valves (tricuspid, mitral) are CLOSED during systole.

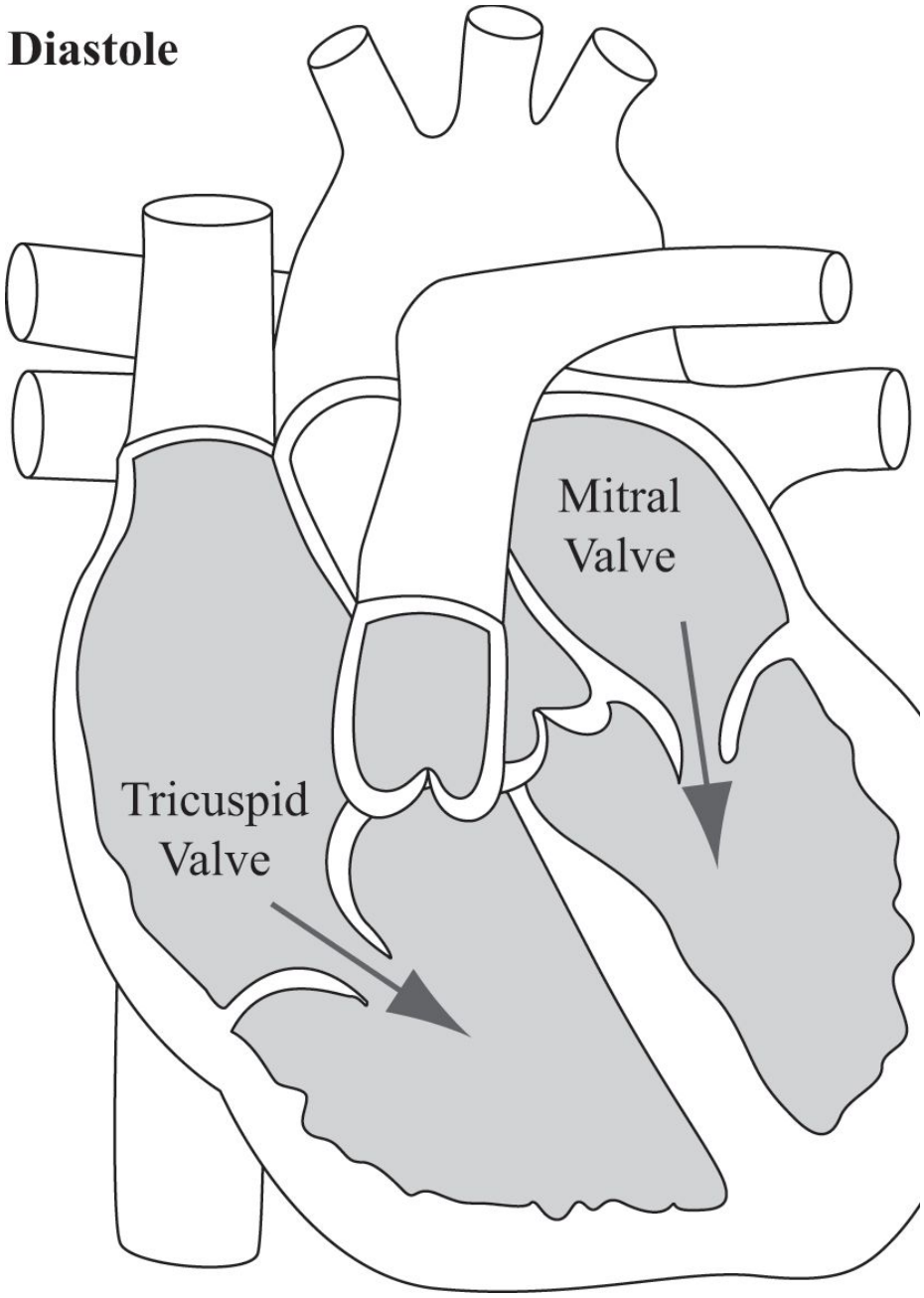


Figure 3-3. Diastole, closure of semilunar valves, S2 "Dub"

→ The semilunar valves (pulmonic, aortic) are CLOSED during diastole.
→ The AV valves (tricuspid, mitral) are OPEN during diastole.

☆ MURMUR REVIEW

- **Mitral insufficiency** occurs when the mitral valve is closed (murmur occurs). When is the mitral valve closed?
  ➤ Systole
- **Mitral stenosis** occurs when the mitral valve is open (murmur occurs). When is the mitral valve open?
  ➤ Diastole
- **Aortic insufficiency** occurs when the aortic valve is closed. When is the aortic valve closed?
  ➤ Diastole
- **Aortic stenosis** occurs when the aortic valve is open. When is the aortic valve open?
  ➤ Systole

Note: If you can picture what the aortic valve is doing, the pulmonic valve is doing the same (opening or closing). If you can picture what the mitral valve is doing, the tricuspid valve is doing the same (opening or closing).

Does a murmur due to a VSD occur during systole or diastole?

➤ During ejection or systole

MURMURS ASSOCIATED WITH AN ACUTE MI

- The mitral valve is attached to the left ventricular wall by the papillary muscles and the chordae tendineae. Myocardial ischemia or infarction can affect mitral valve function and lead to acute mitral valve regurgitation.
- Papillary muscle **dysfunction** (Grade I or II), loudest at the apex
- Papillary muscle **rupture** (Grade V or VI), loudest at the apex . . . surgical emergency!
- Ventricular septal defect
  - **Sternal border**, 5th ICS

Acute Coronary Syndrome (ACS)

☆ Several questions on the exam always cover this topic. The questions may describe ECG findings and a clinical picture and then ask about what type of ACS the patient has. Alternatively, the questions may tell you what type of ACS the patient has (e.g., anterior MI) and expect you to know what the typical clinical picture would be for this type of MI. You do not need to have taken a 12-lead ECG class to do well on these types of questions. However, you DO need to understand which leads are associated with which wall of the heart. If you master the following content, you should do well!

Table 3-1. Risk Factors for Coronary Artery Disease

Category	Risk Factors
Non-modifiable risk factors	Age, sex, family history, genetics
Modifiable risk factors	Smoking, atherogenic diet, alcohol intake, physical activity, dyslipidemias, hypertension, obesity, diabetes, metabolic syndrome

Spectrum of Ischemic Heart Disease

- Asymptomatic coronary artery disease (CAD)
- Stable angina, chest pain with activity, predictable, lesions are usually fixed and calcified

Acute Coronary Syndrome

- Due to platelet-mediated thrombosis
- May result in sudden cardiac death
- Types include:
  1. **UNSTABLE ANGINA:** Chest pain at rest, unpredictable, may be relieved with nitroglycerin, troponin negative, ST depression, or T-wave inversion on the ECG
  2. **NON-ST ELEVATION MYOCARDIAL INFARCTION (NSTEMI):** Troponin positive, ST depression, T-wave inversion on the ECG, unrelenting chest pain
  3. **ST ELEVATION MYOCARDIAL INFARCTION (STEMI):** Troponin positive, ST elevation in 2 or more contiguous leads, unrelenting chest pain

NOTE

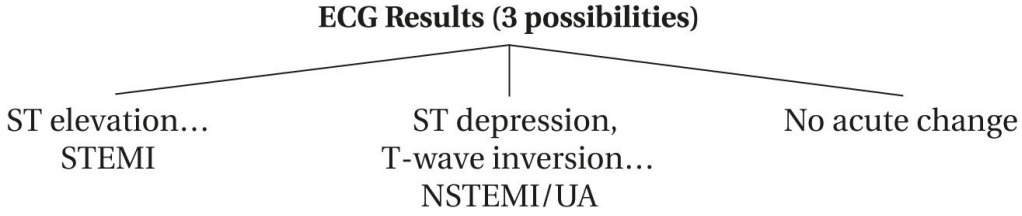
Patients may not have chest pain with an MI, especially women, those with diabetes, and those older than 75. Nausea, SOB, extreme fatigue, syncope, acute delirium, or falling may be signs of ACS in these populations.

☆ Variant or Prinzmetal's angina

- A type of unstable angina associated with transient ST segment elevation
- Due to coronary artery spasm with or without atherosclerotic lesions
- Occurs at rest, may be cyclic (same time each day)
- May be precipitated by nicotine, ETOH, cocaine ingestion
- Troponin negative
- Nitroglycerin (NTG) administration results in relief of chest pain, STs return to normal

Management of Acute Chest Pain

- Stat ECG, done and read within 10 minutes
  - Allows categorization to STEMI or NSTEMI/unstable angina
  - Allows risk stratification to high, medium, low



- Aspirin
  - Give **ASAP**; is chewed; improves morbidity/mortality
- ANTICOAGULANT: heparin or enoxaparin
- Antiplatelet agents
  - Clopidogrel (Plavix)
  - Abciximab (Reopro)
  - Eptifibatide (Integrilin)
  - Tirofiban (Aggrastat)
- Beta blocker
  - Unless ACS is due to **cocaine**
  - Use cardioselective such as metoprolol (Lopressor); do not use non-cardioselective such as propranolol (Inderal).
  - Contraindications include hypotension, bradycardia, use of phosphodiesterase-inhibitor drugs such as sildenafil (Viagra).
- Treat pain
  - Nitroglycerin
  - Morphine
- History, risk factor assessment
  - Lab assessment
  - Cardiac biomarkers, lipid profile, CBC, electrolytes, BUN, creatinine, magnesium, PT, PTT

☆ **ECG lead changes and location of coronary artery disease**

- Changes in II, III, aVF → right coronary artery (RCA), inferior LV

- Changes in V1, V2, V3, V4 → left anterior descending (LAD), anterior LV
- Changes in V5, V6, I, aVL → circumflex, lateral LV
- Changes in V5, V6 → low lateral LV
- Changes in I, aVL → high lateral LV
- Changes in V1, V2 → RCA, posterior LV
- Changes in V3R, V4R → RCA, right ventricular (RV) infarct

Differentiation of the Types of Acute MI

☆ **Inferior MI**

- Associated with right coronary artery (RCA) occlusion
- ST elevation in II, III, and aVF (Figure 3-4)

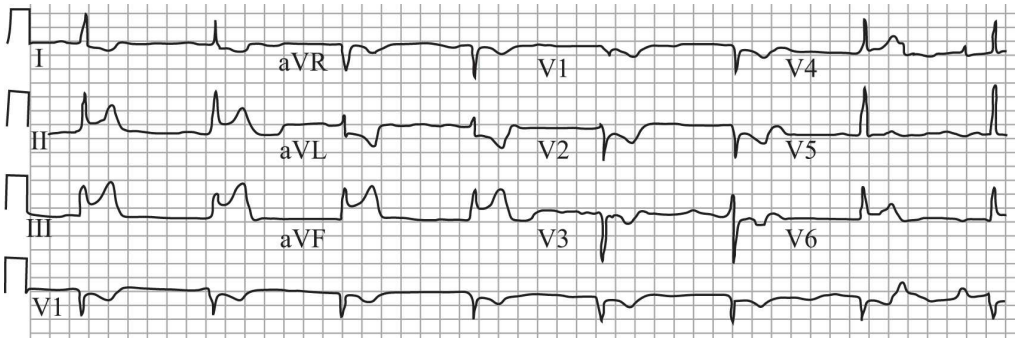


Figure 3-4. Acute inferior wall STEMI

- Reciprocal changes in lateral wall (I, aVL)
- Associated with AV conduction disturbances: 2nd-degree Type I AV block, 3rd-degree AV block, sick sinus syndrome (SSS), and sinus bradycardia
- Development of systolic murmur: mitral valve regurgitation (MVR) secondary to papillary muscle rupture (posterior papillary muscle has only one source of blood supply, the RCA).
- Tachycardia is associated with an inferior MI → higher mortality.
- Also associated with right ventricular (RV) infarct and posterior MI
- Use beta blockers and NTG with CAUTION.

☆ **Right ventricular (RV) infarct**

- The right coronary artery, which supplies the inferior wall of the left ventricle, also supplies the right ventricle; therefore, about 30% of inferior wall MI patients also have a right ventricular (RV) infarct.
- Size of the infarct will determine symptoms.
- A right-sided ECG (Figure 3-5) may demonstrate the ST changes.

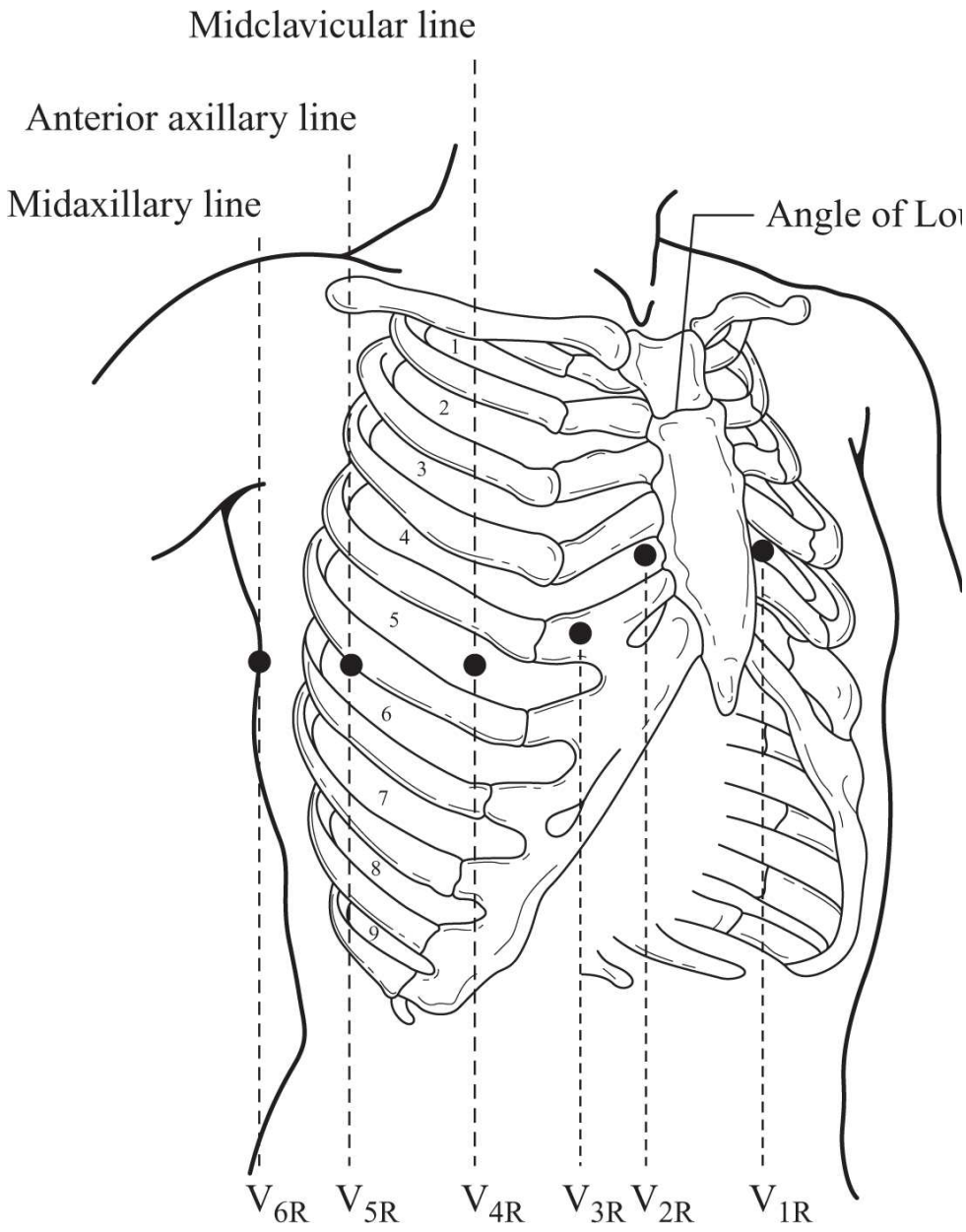


Figure 3-5. Lead placement for right-sided ECG to assess for RV infarct

- Signs/symptoms
  - JVD at 45°, high CVP, hypotension, usually clear lungs, bradyarrhythmias
  - ECG with ST elevation in **V4R** (Figure 3-6)

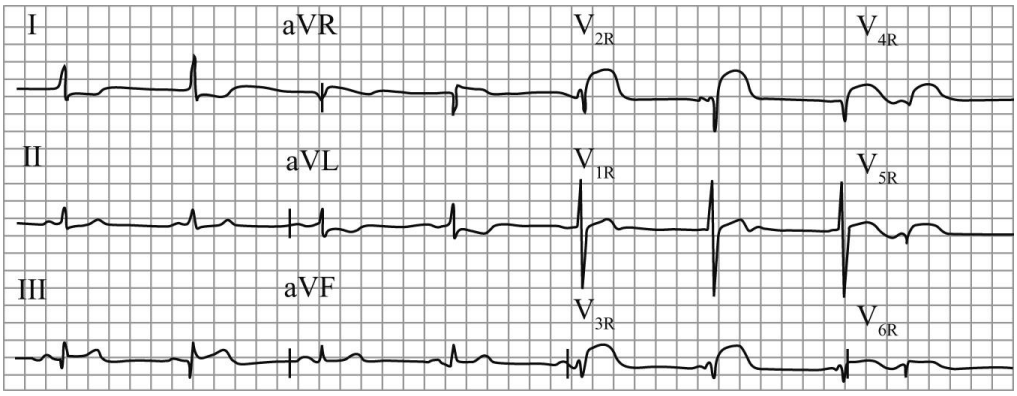


Figure 3-6. Right-sided ECG results with evidence of RV infarct

- Treatment
  - — Fluids
  - — Positive inotropes
- Avoid
  - — Preload reducers → nitrates, diuretics
  - — Caution with beta blockers, often cannot give initially due to hypotension

☆ **Anterior MI**

- Associated with left anterior descending (LAD) occlusion
- ST elevation in V1–V4: precordial leads, V leads (Figure 3-7)

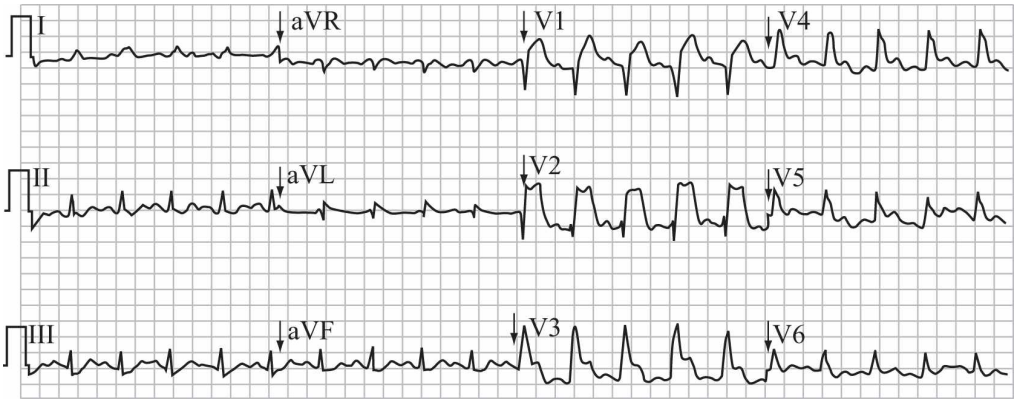


Figure 3-7. Anterior STEMI

- Reciprocal changes (ST depression) in inferior wall (II, III, aVF)
- May develop 2nd-degree Type II AV block or RBBB (the LAD supplies the common bundle of His) → **ominous** sign
- Development of systolic murmur: possible ventricular septal defect
- Higher mortality than an inferior MI: **HEART FAILURE**

- **Lateral MI**
  - ST elevation in V5, V6 (low lateral)
  - ST elevation in I, aVL (high lateral)
  - Generally involves left circumflex artery

Treatment of STEMI

- Determine onset of infarct, if symptoms < 12 hours → **REPERFUSION**
  - Percutaneous coronary intervention (PCI)—standard is door-to-balloon within 90 minutes
  - Fibrinolytic drug therapy—standard is door-to-drug within 30 minutes
- Eligibility criteria
  - ST elevation in 2 or more contiguous leads **or** new onset left bundle branch block (LBBB)
  - Onset of chest pain < 12 hours
  - Chest pain of 30 minutes in duration
  - Chest pain unresponsive to sublingual (SL) nitroglycerin (NTG)

PATIENT CARE FOLLOWING REPERFUSION FOR STEMI

PCI (90 minutes, door-to-balloon inflation in coronary artery at point of lesion)

- Monitor for signs of reocclusion: chest pain, ST elevation → contact physician.
- Monitor for vasovagal reaction during sheath removal → give fluids, atropine.
  - Hypotension < 90 systolic with or without bradycardia, absence of compensatory tachycardia
  - Associated symptoms of pallor, nausea, yawning, diaphoresis
- Monitor for bleeding: sheath site.
  - Immediately apply manual pressure 2 fingerbreadths above the puncture site.
  - Continue manual pressure for a minimum of 20 min (30 min if still on GP IIb/IIIa inhibitors) to achieve hemostasis.
- Monitor for bleeding: retroperitoneal → fluids, blood products.
  - Sudden hypotension
  - Severe low back pain
- Monitor for vascular complications → pulse assessments.

Fibrinolytic Therapy (30 minutes door-to-drug administration)

- Absolute contraindications
  - Any prior intracranial hemorrhage
  - Known structural cerebral vascular lesion (e.g., arteriovenous malformation)

- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 months EXCEPT acute ischemic stroke within 3 hours
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed-head or facial trauma within 3 months

☆ **Evidence of reperfusion**

- Chest pain relief: due to fibrinolysis of clot
- Resolution of ST segment deviations: due to return of blood flow
- Marked elevation of troponin/CK-MB: due to myocardial “stunning” when vessel opens
- Reperfusion arrhythmias (VT, VF, accelerated idioventricular rhythm [AIVR]): due to myocardial “stunning” when vessel opens

- Nursing management
  - Assess for major and minor bleeding.
    - Major bleed, change in LOC, brain bleed
  - Institute bleeding precautions.
  - Assess for reperfusion (see above).
  - Assess for reocclusion as evidenced by recurring chest pain, ST deviation.

Treatment of NSTEMI

- **No** emergent reperfusion
- Same meds as STEMI
- If high risk score **or** continued chest pain, signs of instability, start GP IIb/IIIa inhibitors (Integrilin, Reopro) and prepare for diagnostic cardiac catheterization within 24 hours.

Complications of an Acute MI

ARRHYTHMIAS—MOST COMMON!

- Ventricular tachycardia or ventricular fibrillation
  - Defibrillate VF
  - Drug therapy for stable, sustained VT and to prevent recurrent VF
  - Synchronized cardioversion for unstable, sustained VT
- Bradycardia, heart blocks, sick sinus syndrome (SSS)
- Atrial fibrillation
  - Increases risk of mortality 10–15%, even when returned to NSR
- Heart failure
- Cardiogenic shock
- Reinfarction
- Thromboembolic events
- Pericarditis
- Ventricular aneurysm
- Ventricular septal defect
- Papillary muscle rupture
- Cardiac wall rupture

Interventional Cardiology

Cardiac Catheterization Lab Procedures

- Diagnostic cardiac catheterization
- Percutaneous Coronary Interventions (PCI)
  - Intracoronary stenting: most common PCI procedure
  - Balloon angioplasty without stent (PTCA): seldom used, high reocclusion rates
- Percutaneous balloon valvotomy
- Pacemaker implantation
- Electrophysiology (EP) studies
  - Implantable cardioverter defibrillator (ICD)
  - Cardiac ablation therapy

Goal of PCI with Stent

- Restoration of blood flow distal to a coronary artery lesion with partial or total occlusion

Complications of PCI

- In-hospital death is rare, ~ 1.8%.
- In-hospital MI ~ 0.4%
- Coronary artery perforation
- Distal coronary artery embolization
- Intramural hematoma
- Failure of stent deployment

☆ **Stent thrombosis is most likely to be on the exam.**

- Most incidents occur acutely (within 24 hours of stent placement) or subacutely (within the first 30 days).

- Stroke or TIA: greater risk if with aortic stenosis
- Arrhythmias
- Renal failure

☆ **Retroperitoneal bleed is most likely to be on the exam.**

Patient Care During Sheath Removal

- Record baseline peripheral pulses and vitals.
- Provide comfort (i.e., morphine 2–4 mg IV) before removal.
- Monitor BP q 5–10 min during sheath removal.
- Monitor for **vasovagal response** (hypotension < 90 systolic with or without bradycardia, absence of compensatory tachycardia and associated symptoms of pallor, nausea, yawning, diaphoresis).
- Vasovagal management
  - Hold nitrates
  - Atropine 0.5 mg IV (even in absence of bradycardia if other signs occur)
  - IV bolus of 250 mL 0.9 NS if patient is not immediately responsive to atropine
  - Assess for anxiety/pain as contributing factors.
- Achieve hemostasis.
  - Manual pressure for 20 to 30 minutes
  - Mechanical clamp compression using FemoStop or C-clamp
  - Closure device

Manage Complications of Cardiac Catheterization Procedures

- Monitor for signs of coronary artery reocclusion (if PCI): chest pain, ST elevation → contact physician.
- Monitor for vasovagal reaction during sheath removal → give fluids, atropine.
- Monitor for bleeding: sheath site.
  - Immediately apply manual pressure 2 fingerbreadths above the puncture site.
  - Continue manual pressure for a minimum of 20 min (30 min if still on GP IIb/IIIa inhibitors) to achieve hemostasis.
- Monitor for bleeding: retroperitoneal → fluids, blood products.
  - Sudden hypotension
  - Severe low back pain
- Monitor for vascular complications: pulse assessments.
- Monitor for hematoma at sheath insertion site: assess sheath insertion site for swelling.

Hypertensive Crisis/Emergency

One question may focus on this topic. The question will usually be related to how the topic differs from hypertensive urgency or what drugs are used for treatment.

- Hypertensive **emergency or CRISIS** is elevated BP with evidence of end organ damage (brain, heart, kidney, retina) that can be related to acute hypertension → needs critical care admission.
- Hypertensive **urgency** is elevated BP without evidence of acute end organ damage → usually no need for critical care admission.
- Treatment of hypertensive crisis or emergency → emergent lowering of BP needed.
  - **Nitroprusside (Nipride)**
    - Preload **and** afterload reducer
    - Assess for cyanide toxicity secondary to drug metabolite (Thiocyanate): mental status change (restlessness, lethargy), tachycardia, seizure, a need for ↑ in dose, unexplained metabolic acidosis, especially in those with renal impairment or when a drug is used > 24 hrs.
  - **Labetalol (Normodyne, Trandate)**
    - Intermittent IV doses preferred to a continuous infusion, due to the possibility of continuing the drug beyond the maximum dose of 300 mg.
    - Duration of effect persists 4–6 hrs after IV dose is discontinued.

- Greatest risk is STROKE.

Acute Peripheral Vascular Insufficiency

One question about this topic may appear on the exam. That question generally covers **acute** issues that are related to peripheral arterial disease (PAD) or acute symptomatic carotid artery disease.

Peripheral Arterial Disease (PAD)

- Signs and symptoms (the 6 “Ps”)
  - **P**ain (activity, rest)
  - **P**allor
  - **P**ulse absent or diminished
  - **P**aresthesia
  - **P**aralysis
  - **P**oikilothermia: loss of hair on toes or lower legs; glossy, thin, cool, dry skin (chronic sign of PAD)
  - Additionally, cool to touch, minimal edema
- Ankle-Brachial Index (ABI)
  - Test to assess for PAD
  - Used to assess the adequacy of lower extremity perfusion
  - Normal is > 0.90
    - Divide the ankle pressure by the brachial pressure on the same side
  - **You only need to remember what a normal ABI is for the exam.**
- Additional diagnostic testing
  - Doppler ultrasound
  - Arteriography
- Patient care management for PAD
  - Embolectomy, bypass graft, angioplasty

☆ Bed in reverse Trendelenburg

☆ **Do NOT elevate the affected extremity**—will decrease perfusion

- Medications
  - Thrombolytics (tPA)

- Anticoagulants (heparin)
- Antiplatelet agents (ASA, clopidogrel)
- Vasodilators

Acute Symptomatic Carotid Artery Disease

- Signs and symptoms
  - Transient ischemic attack (TIA)
  - Monocular visual disturbances
  - Aphasia
  - Stroke
- Diagnostic testing may include one of the following:
  - Angiography (gold standard): risk of stroke during exam
  - Carotid duplex ultrasound
  - Computed tomography angiography (CTA)
  - Magnetic resonance angiography (MRA)
- Treatment
  - Carotid endarterectomy (CEA)
  - Carotid stenting
  - Aspirin
  - Statin therapy
- Post-procedure monitoring
  - Frequent neurological and motor checks
  - Close blood pressure and heart rate monitoring: patient may experience labile BP and/or bradyarrhythmia with hypertension, hypotension, or bradycardia.
  - Monitor for bleeding.
  - Patient may develop hypoperfusion syndrome with the signs and symptoms of a headache ipsilateral to the revascularized carotid artery, focal motor seizures, and/or an intracerebral hemorrhage.

Arrhythmia Interpretation, Arrhythmia Emergencies, and Pacemaker Therapy

Test candidates are expected to have mastered arrhythmia interpretation and have an understanding of ACLS principles, so look these over. An exam question may describe the arrhythmia **or** provide an arrhythmia strip for interpretation, but the question will usually be related to patient management.

☆ **For exam questions that include the development of an arrhythmia, pay close attention to the patient's clinical response to the arrhythmia. Is the patient stable or unstable? If the clinical description is NOT provided, consider this fact and do NOT assume the patient response!**

Conduction Abnormalities

- **Wolff-Parkinson-White (WPW) syndrome** is a genetic conduction abnormality in which an abnormal conduction pathway exists that allows a reentrant tachycardia pathway to bypass the normal AV node conduction pathway, resulting in supraventricular tachycardia. WPW is primarily seen in those younger than 30-years-old.
  - When in sinus rhythm, the ECG demonstrates a short PR interval and the presence of a delta wave (seen as a slow rise of the initial upstroke of the QRS).
  - WPW results in a supraventricular tachycardia (SVT) when the abnormal pathway takes over, but it may also present as **pre-excited atrial fibrillation** (irregular rhythm, rates of 150 beats/minute or greater, and a wide QRS).
  - The signs and symptoms during the SVT include palpitations, dizziness, chest pain, shortness of breath, and syncope.
  - Treatment of WPW
    - Radiofrequency ablation to eliminate the reentrant pathway
    - If unstable SVT is present, perform synchronized cardioversion **or** administer adenosine.
    - If pre-excited atrial fibrillation (AF) is present, administer beta blockers, amiodarone, or procainamide IV.
      - Do NOT give adenosine, digoxin, or calcium channel blockers for pre-excited AF; these agents may enhance antegrade conduction through the abnormal pathway by

increasing the refractory period in the AV node, **resulting in ventricular fibrillation**.

☆ **Prolongation of the QT interval** (causes, treatment) is often addressed on the Adult CCRN exam. QT prolongation may lead to torsades de pointes. Causes of QT prolongation include:

- Drugs—amiodarone, quinidine, haloperidol, procainamide
- Electrolyte problems—hypokalemia, hypocalcemia, hypomagnesemia

- Treatment for torsades VT—magnesium

Pacemaker Therapy

For pacemaker therapy, which includes some knowledge of implantable cardioverter-defibrillator (ICD) devices, review the following information.

Pacemaker Code

$$\mathrm{A} = \text{atria} \qquad \mathrm{V} = \text{ventricle} \qquad \mathrm{D} = \text{dual (both)}$$

- First initial = chamber **paced**; this was “invented” first
- Second initial = chamber **sensed**; this function came along second
- Third initial = response to sensing; last to be developed
  - I = inhibits (pacer detects intrinsic cardiac activity and withholds its pacing stimuli) . . . demand
  - D = inhibits **and** triggers (pacer detects intrinsic cardiac activity and fires a pacing stimulus in response)
  - O = none

On the Adult CCRN exam, you may see a question that asks how a “VVI” or a “DDD” pacemaker works. If you remember the code, you should be able to figure out the answer.

Sample questions:

a. Which pacemaker paces both the atria and the ventricles, senses both the atria and the ventricles, and can inhibit and

trigger in response to sensing?

b. Which pacemaker paces the ventricle, senses the ventricle, and inhibits pacing in response to sensing?

Answers: a. DDD b. VVI

Review the 3 basic pacer malfunctions:

1. Failure to pace (no spike at all when expected)
2. Failure to capture (spikes without a QRS for ventricular pacing)
3. Failure to sense (pacing in native beats)

Implantable Cardioverter-Defibrillator (ICD)

ICDs can provide “tiered” therapy:

- Programmed to **shock** (defibrillate or synchronized cardioversion)
- Programmed to **burst pace** (sense tachyarrhythmia, provide a series of beats faster than the tachyarrhythmia, and then suddenly stop [with the hope of the recovery of the SA node])
- Programmed to provide pacing for **bradyarrhythmias**

If the ICD does not correct the sudden death arrhythmia, shock as usual; do not place shocking pads directly over the ICD.

- The patient will require special education related to the device and emotional support, since many patients experience a FEAR

of being shocked.

Heart Failure

Heart failure (HF) is a broad topic. For the exam, if you focus on the following, you will be ready!

Heart failure may be acute, chronic, acute exacerbation of chronic HF, systolic or diastolic HF, or right-sided or left-sided HF. The most extreme HF occurs when all compensatory mechanisms have failed, and the result is cardiogenic shock. An understanding of these concepts and the management of each, as well as an understanding of heart failure classifications, is needed in order to successfully answer the Adult CCRN exam questions on this topic.

- **Heart failure** is a clinical syndrome that is characterized by signs and symptoms associated with high intracardiac pressures and decreased cardiac output.
- **Acute decompensated heart failure** is the abrupt onset of symptoms that are severe enough to merit hospitalization.
  - ~ 75% of patients with acute decompensated HF have a history of chronic heart failure.
- Heart failure with **systolic dysfunction** (left ventricular systolic dysfunction—LVSD): ejection fraction (EF) is 40% or less, problem with ejection
- Heart failure with **diastolic dysfunction**: EF is > 50%, problem with filling, ejection is OK

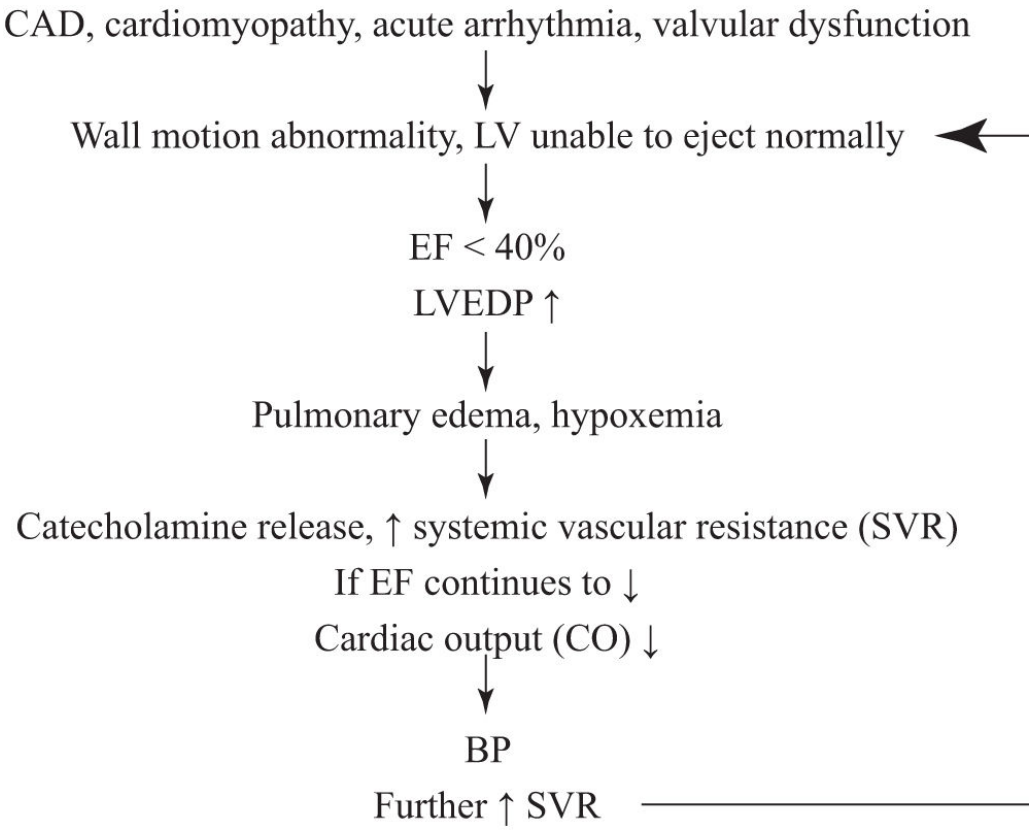
TIP

When studying, compare and contrast systolic and diastolic heart failure and differentiate the clinical signs of left-sided and right-sided heart failure.

What Is BNP?

- B-type natriuretic peptide (BNP) is released by the ventricle when the ventricle is under wall stress in attempts to dilate and decrease ventricular pressure.
- BNP elevates when the left ventricle is under stress (left ventricular failure) or, to a lesser degree, BNP elevates when the right ventricle is under stress (pulmonary hypertension, pulmonary embolism).

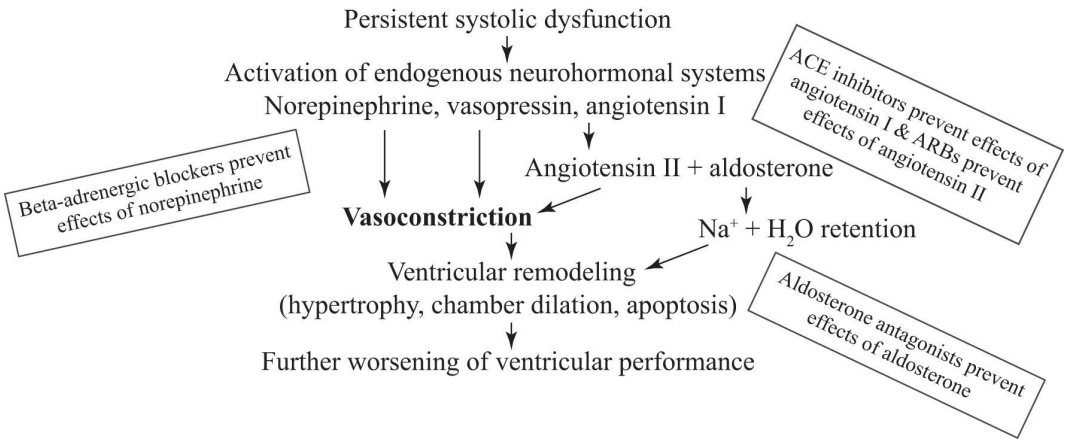
Pathophysiology of Acute Decompensated Systolic Dysfunction



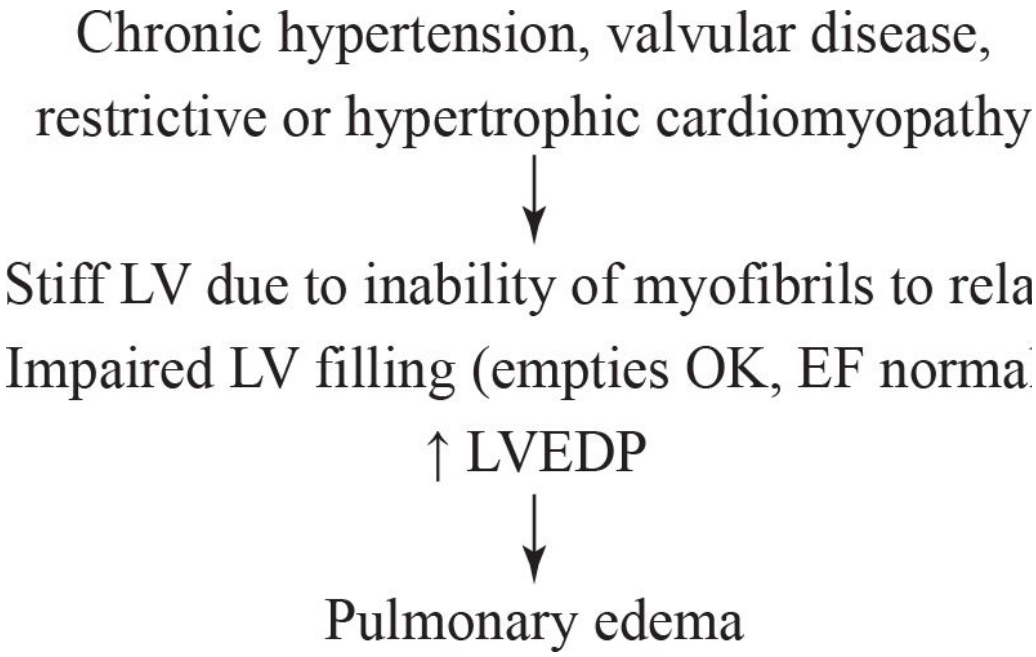
In the figure above: CAD = coronary artery disease; LV = left ventricle; EF = ejection fraction; LVEDP = left ventricular end-diastolic pressure

- When systolic dysfunction is prolonged and becomes chronic, compensatory **hormones** lead to ventricular remodeling over time.
- Drugs are used to decrease neurohormonal effects.

Progressive (Chronic) Systolic Dysfunction
(Compensatory Mechanisms)



Pathophysiology of HF with Diastolic Dysfunction



☆ **Table 3-2. Summary of the Differences Between Systolic and Diastolic Heart Failure**

	Systolic	Diastolic
Primary problem	Ejection problem, dilated chamber • Can fill OK	Filling problem, hypertrophied chamber or septum • Can eject OK
Signs	Dilated left ventricle PMI shifted to left Valvular insufficiency (dilation causes mitral valve insufficiency) EF $\leq$ 40% Pulmonary edema due to poor ventricular emptying S3 heart sound BP is normal or low (usually) BNP is elevated	Normal ventricular size Thick ventricular walls and/or thick septum Normal contractile function Normal EF Pulmonary edema due to high ventricular pressure S4 heart sound with hypertension BP is often high BNP is elevated
Treatment	Beta blockers ACEI/ARB Diuretics Dilators Aldosterone antagonists Positive inotropes	Beta blockers ACEI/ARB Calcium channel blockers Diuretics (low dose) Aldosterone antagonists
Contraindicated	Negative inotropes (calcium channel blockers and, in acute phase, beta blockers)	Positive inotropes Dehydration further worsens filling Tachyarrhythmias decrease filling time and worsen symptoms
Cardiomyopathy Types	Cardiomyopathies that result in systolic HF: • Dilated ○ May result in mitral insufficiency as the left ventricular wall enlarges	Cardiomyopathies that result in diastolic HF: • Idiopathic hypertrophic subaortic stenosis (IHSS) • Hypertrophic cardiomyopathy (HCM) • Restrictive

☆ Chest X-Ray Findings: Systolic vs. Diastolic HF

- Systolic HF may be evidenced by a large, dilated heart **or** by a normal heart size on the chest film.
  - An enlarged heart is often associated with a shift of the point of maximal impulse (PMI) from midclavicular to the **left** (Figure 3-8).
- Diastolic HF generally is evidenced by a normal heart size on the chest film. However, on the 12-lead ECG, there **may** be a left ventricular hypertrophy pattern, especially when the patient has a history of uncontrolled hypertension.

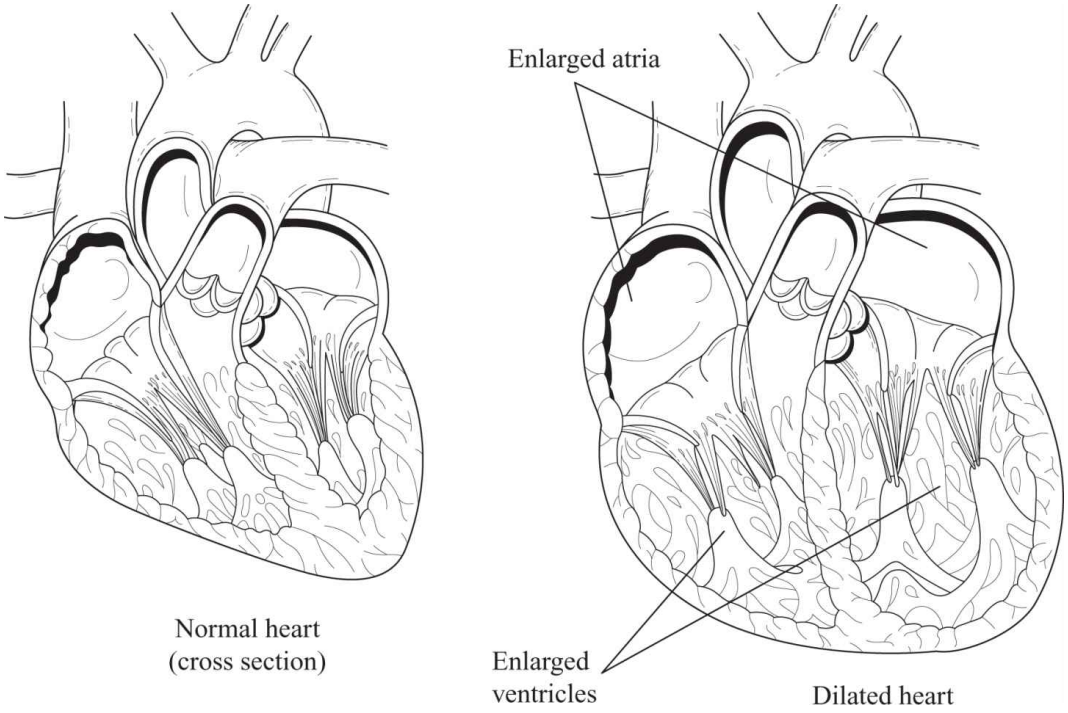


Figure 3-8. Normal and enlarged heart size

Heart failure may also be categorized according to which ventricle is failing, the right or the left (Tables 3-3 and 3-4). The etiologies and treatment are different. The Adult CCRN exam may present a case scenario with background information, signs and symptoms, and a description of whether the HF is right-sided or left-sided. Then, you will be asked to identify the correct treatment.

Table 3-3. Causes of Right-Sided and Left-Sided Heart Failure

Right-Sided	Left-Sided
Acute RV infarct	Coronary artery disease, ischemia
Pulmonary embolism (massive)	Myocardial infarction
Septal defects	Cardiomyopathy
Pulmonary stenosis/insufficiency	Fluid overload
COPD	Chronic, uncontrolled hypertension
Pulmonary hypertension	Aortic stenosis/insufficiency
Left ventricular failure	Mitral stenosis/insufficiency
	Cardiac tamponade

Table 3-4. Signs and Symptoms of Right-Sided and Left-Sided Heart Failure

Right-Sided	Left-Sided
Hepatomegaly	Orthopnea, dyspnea, tachypnea
Splenomegaly	Hypoxemia
Dependent edema	Tachycardia
Venous distention	Crackles
Elevated CVP/JVD	Cough with pink, frothy sputum
Tricuspid insufficiency	Elevated PA diastolic/PAOP
Abdominal pain	Diaphoresis
	Anxiety, confusion

Heart Failure Classifications

The Adult CCRN exam may include a question related to heart failure classifications. There are two types of classifications: the American Heart Association (AHA) stages of heart failure (which are classified according to heart failure progression and recommended therapy for each stage) and the New York Heart Association (NYHA) four functional classes (which are based on the patient's symptoms and do not include suggested treatment).

- The main cause of death from heart failure is the development of **sudden death arrhythmia**. Select patients with NYHA Class II to IV may be candidates for an implantable cardioverter-defibrillator (ICD).

American Heart Association (AHA) Stages of Heart Failure

- **Stage A**—High risk; no evidence of dysfunction
- **Stage B**—Heart disorder or structural defect; no symptoms
- **Stage C**—Heart disorder or structural defect, with symptoms (past or present)
- **Stage D**—End-stage cardiac disease, with symptoms despite maximal therapy (inotropic or mechanical support)

NYHA Heart Failure Classes

- **Class I**—Ordinary activity does not cause symptoms, although **EXTRAORDINARY ACTIVITY** results in heart failure symptoms.
- **Class II**—Comfortable at rest, but **ORDINARY ACTIVITY** results in heart failure symptoms.
- **Class III**—Comfortable at rest, but **MINIMAL ACTIVITY** causes heart failure symptoms.
- **Class IV**—Symptoms of heart failure occur **AT REST**; there is a severe limitation of physical activity.

Cardiomyopathy

If the Adult CCRN exam includes a question on cardiomyopathy, it will most likely be on either **dilated** or **hypertrophic** cardiomyopathy (Table 3-5).

Table 3-5. Differences Between Dilated and Hypertrophic Cardiomyopathy

Dilated	Hypertrophic
SYSTOLIC dysfunction, problem ejecting Classical signs • Thinning, dilation, enlargement of LV chamber • Mitral valve regurgitation (MVR) common due to ventricular dilation Symptoms are similar to that of **SYSTOLIC** heart failure. Treatment • Similar to that for systolic heart failure • Heart failure may progress through stages, classes • May need a ventricular assist device (VAD), heart transplant	**DIASTOLIC** dysfunction, problem filling Classical sign • Increased thickening of the heart muscle and septum inwardly at the expense of the LV chamber Symptoms are similar to that of **DIASTOLIC** heart failure. • Fatigue • Dyspnea • Chest pain • Palpitations • S3, S4 heart sounds • Presyncope or syncope Treatment similar to that for diastolic heart failure. **Increased risk of sudden cardiac death!**

Cardiogenic Shock

When compensatory mechanisms fail to maintain the cardiac output, the most extreme end on the continuum of heart failure occurs—cardiogenic shock (Table 3-6). Cardiogenic shock has several causes. Most commonly, it is due to an extreme drop in stroke volume secondary to systolic dysfunction, which results in:

- Elevated left ventricular preload (PAOP) with associated pulmonary symptoms
- Elevated left ventricular afterload (SVR) due to vasoconstrictive compensatory mechanisms
- A resultant drop in cardiac output to the point where perfusion to organs is no longer adequate

Table 3-6. Clinical Presentation of Cardiogenic Shock

Compensatory Stage	Progressive Stage
Tachycardia Tachypnea Crackles, mild hypoxemia ABG with respiratory alkalosis or early metabolic acidosis Anxiety, irritability Neck vein distention S3 heart sounds (S4 heart sounds if there is also an acute MI) Cool skin Urine output is down **Narrow pulse pressure** BP is maintained or lower than baseline	Hypotension Worsening tachycardia, tachypnea, oliguria Metabolic acidosis Worsening crackles and hypoxemia Skin is clammy, mottled Worsening anxiety or lethargy • At any time, chest pain or arrhythmias may occur

Etiologies of Cardiogenic Shock

- Acute MI
- Chronic heart failure
- Cardiomyopathy
- Dysrhythmias
- Cardiac tamponade
- Papillary muscle rupture
  - Obliterates the mitral valve
  - Life-threatening emergency
  - Requires immediate surgical intervention

Treatment of Cardiogenic Shock

- Identify the cause
- Manage arrhythmias (brady, tachy) that may be contributing to a decrease in cardiac output
- Reperfusion if there is STEMI (percutaneous coronary intervention or fibrinolytic therapy)
- Emergent surgery if cardiogenic shock is due to a mechanical problem—ruptured papillary muscle, VSD
- Mechanical support
- See Table 3-7

Table 3-7. Treatment of Cardiogenic Shock

Enhance Effectiveness of Pump	Decrease Demand on Pump
Positive inotropic support • Norepinephrine (Levophed) • Dopamine 4–10 mcg/kg/min • Dobutamine, milrinone (Primacor) AVOID negative inotropic agents! Vasodilators • May be used in conjunction with intra-aortic balloon pump (IABP) therapy and positive inotropic agents if the patient is in the progressive stage with hypotension	Preload reduction (or optimization) Afterload reduction Optimize oxygenation Mechanical ventilation Treat pain IABP for short term support Ventricular assist device (VAD) may be used for longer periods of time than IABP

Mechanical Circulatory Support

- There are several types of mechanical circulatory support devices available, but intra-aortic balloon pump (IABP) therapy is specifically listed on the test blueprint. You will not be tested on the details of this therapy. However, you need to know that it is used in the management of left ventricular heart failure, cardiogenic shock, and cardiomyopathies, and it is also used for patients who are awaiting a heart transplant.

☆ **Benefits of IABP Therapy**

- You will likely have at least 1 question on this.
- Just remember, the balloon does 2 things: it INFLATES and it DEFLATES (Figure 3-9 and Figure 3-10).

Benefits of INFLATION

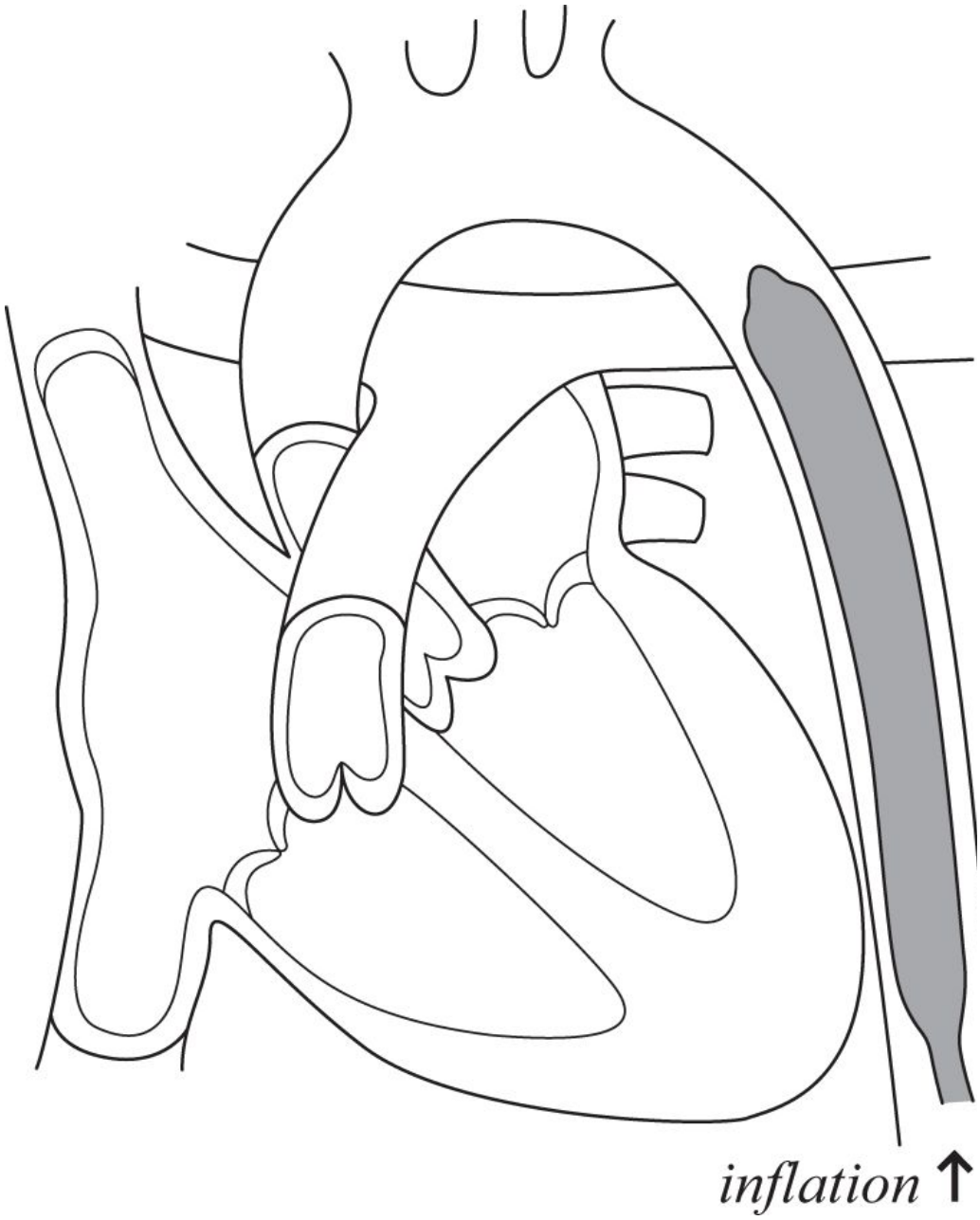


Figure 3-9. Balloon inflation—increases coronary artery perfusion

→ Inflates at dicrotic notch of the arterial waveform, beginning of diastole

Benefits of DEFLATION

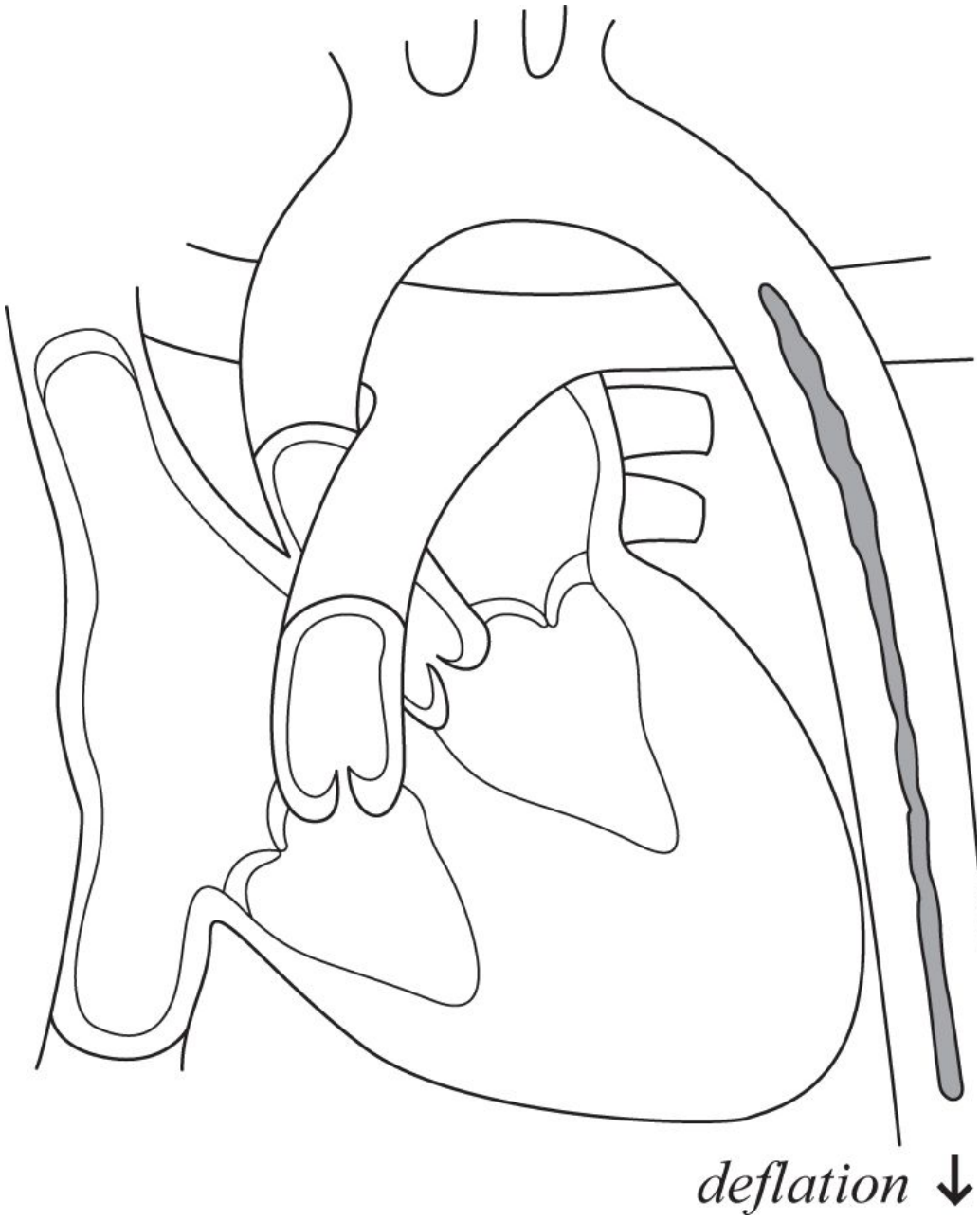


Figure 3-10. Balloon deflation—decreases afterload

→ Deflates right before systole begins; determined by a set TRIGGER for deflation, R-wave of ECG or upstroke of the arterial pressure wave

Cardiac Surgery

NOTE

The exam questions related to heart surgery generally focus on complications and nursing care postoperatively.

Cardiopulmonary Bypass

- Aortic cross-clamping is done, and the heart is stopped during surgery.
- Most common cannulation sites:
  - Aorta
  - Right atrium
- The longer the bypass time, the more bleeding there is and the more complications there may be postoperatively.

Coronary Artery Bypass Graft (CABG) Procedure

- Priming with isotonic crystalloids (hemodilution); enhances oxygenation by improving blood flow
- Hypothermia (28°C–36°C) is induced.
- Anticoagulation with large heparin doses
- Rapid circulatory arrest is achieved during diastole with the infusion of a potassium cardioplegic agent; the cardioplegic agent is reinfused at regular intervals; note that either a warm or a cold cardioplegic agent may be used.

Post-Op Assessment for Complications Related to CABG

- Hemodynamic abnormalities
- Arrhythmias

☆ Tamponade

☆ Pericarditis

- Electrolyte abnormalities
- Hematologic abnormalities, bleeding
- Pulmonary problems (pneumonia, atelectasis, difficulty weaning from mechanical ventilation)
- Pain, anxiety
- Renal failure
- Endocrine problems (issues with glycemic control)
- Gastrointestinal problems (nausea, vomiting, ileus)
- Infections

☆ **Post-op chest tube management**

- Maintain patency.
  - Do not allow dependent loops.
  - Milking or stripping chest tubes is not routinely indicated.
    - If clots appear, gently milk the chest tubes.
- Mediastinal chest tubes remove serosanguinous fluid from the operative site, whereas pleural chest tubes remove air, blood, or serous fluid from the pleural space.
- Keep chest tubes lower than patient's chest.
- Do not clamp the system unless you are changing the drainage system or there is a system disconnect. When the tube is

clamped, the connection to the negative chamber is lost.

- A chest tube output > 100 mL for 2 consecutive hours generally requires intervention.
  - Maintain hemodynamic stability.
  - Correct the volume status.
  - Administer blood products.

Valve Surgery

Table 3-8. Advantages and Disadvantages of Mechanical and Biological Valves

Mechanical Valve	Biological Valve
Advantages: • Relatively easy to insert • Very reliable • Lasts longer than biological valve Disadvantages: • High risk of thrombosis • Permanent anticoagulation therapy	Advantages: • Only short-term anticoagulation is required (but the patient will need long-term antiplatelet [ASA] therapy) Disadvantages: • Wears down, especially in high-pressure systems

Nursing Considerations Post-Valve Repair or Replacement

- Avoid a drop in preload. Most patients who have had valvular stenosis or chronic regurgitation have had elevated end-diastolic volumes. Sudden preload normalization may result in hypotension.
- Anticoagulation will be needed for mechanical valve replacement; biological valve replacement will require antiplatelet therapy (aspirin).
- Anticipate **conduction disturbances** since the mitral, tricuspid, and aortic valves are anatomically close to conduction pathways. Temporary or permanent pacing may be needed.

Transcatheter Aortic Valve Replacement (TAVR)

- TAVR, which was approved in 2011, is a procedure that involves placement of a collapsible prosthetic valve (either bovine or porcine) over the diseased valve (either a native valve or a previously placed artificial valve).
- Access to the aorta is usually achieved percutaneously or through a small incision, avoiding cross-clamping of the aorta and cardiopulmonary bypass.
- Most TAVR procedures are done via the femoral artery and are performed in a cardiac catheterization laboratory that is modified to accommodate TAVR procedures.
- Ideal candidates for TAVR are those with severe aortic valve disease that is classified as **high-risk** for open surgery.
  - Those who are at an intermediate risk for open surgery may qualify for either TAVR or open surgical replacement, depending upon a decision from the interdisciplinary heart valve team based on an evaluation of the patient.
  - Patients who are considered extreme high-risk/inoperable or low-risk for open procedure are NOT candidates for TAVR.
  - **Note:** There are studies looking at TAVR for a patient who is classified as **low-risk** for an open procedure for aortic valve replacement, but as of the date of this publication, a low-risk patient is not considered an ideal candidate for this procedure.
- Complications: vascular complications associated with femoral access (similar to post-PCI, including hematomas, retroperitoneal bleeding, and arterial occlusion); heart block; stroke; acute kidney

injury; and paravalvular regurgitation (associated with a mismatch of the prosthetic valve and the native valve annulus)

- Dual antiplatelet therapy will be required, including aspirin (75–100 mg/day) for life and clopidogrel (75 mg/day) for 3 to 6 months post-procedure.

Cardiac Tamponade

- Etiologies: surgical-related cause (post-op cardiac surgery), medical-related cause (pericarditis, pericardial effusion), trauma
- Signs and symptoms
  - Restlessness and agitation
  - Hypotension
  - ↑ JVD
  - Equalization of CVP, pulmonary artery diastolic pressure, and PAOP
  - Muffled heart sounds
  - Enlarging cardiac silhouette and mediastinum on a chest radiograph

☆ Narrowed pulse pressure (i.e., 82/68)

☆ Pulsus paradoxus: an excessive drop (greater than 12 mmHg) in the SBP during inspiration, which is the result of cardiac muscle restriction caused by the tamponade with inspiration; the intrathoracic pressure increases, and the venous return decreases

— Best seen on an arterial waveform as respiratory variation (Figure 3-11).

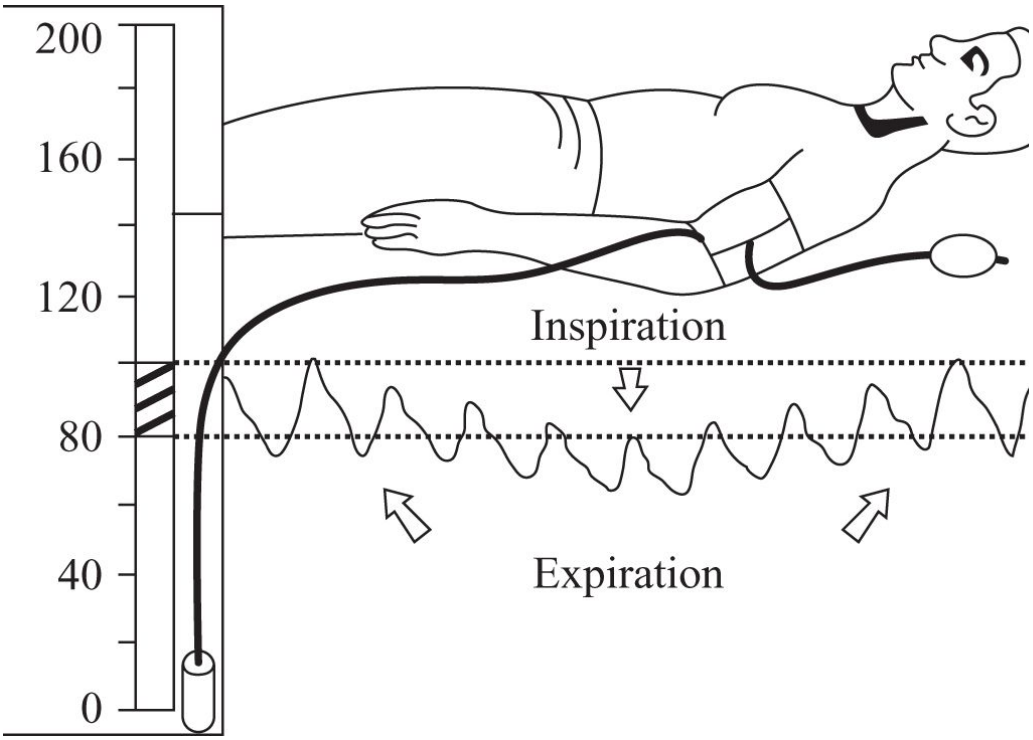


Figure 3-11. Pulsus paradoxus as seen on an arterial waveform

Cardiac Trauma

Which valve is most at risk for a rupture due to trauma?

- **Aortic valve** because it is most anterior in the chest
- A myocardial contusion has signs and symptoms that are similar to that for pericarditis, but there are important differences between these two conditions, as outlined in Table 3-9.

Table 3-9. Pericarditis vs. Myocardial Contusion

	☆ Medical—Pericarditis	☆ Trauma—Myocardial Contusion
Etiology	Trauma (rare) Viral After an MI Post-op cardiac surgery Radiation Idiopathic Dressler's syndrome—immune response after an MI, surgery, or traumatic injury	Trauma • Worse outcome than pericarditis • Broken vessels bleed into heart, similar to an MI • Cardiac dysrhythmias • Death can occur within the first 48 hours
Signs and symptoms	Chest pain Pain worsens with inspiration Dyspnea Low-grade temp ↑ Sed rate ST elevation in all the leads Cardiac tamponade Post-MI, Dressler's syndrome, may last months	Signs of trauma Chest pain Pain worsens with inspiration Dyspnea Low-grade temperature ST elevation in the **area of injury**
Treatment	Symptom relief Analgesics Anti-inflammatory agents NSAIDs Steroid Antibiotics Monitor for worsening symptoms Monitor for constrictive pericarditis Monitor for cardiac tamponade	Monitor for arrhythmias Analgesics as needed

Aneurysms

An aneurysm is a localized, blood-filled outpouching in the wall of an artery. The larger it becomes, the more likely it is to rupture. The Adult CCRN exam may contain a question about abdominal aortic or thoracic aortic aneurysms.

Etiology of Aneurysms

- Arteriosclerosis
- Hypertension
- Smoking
- Obesity
- Bacterial infections
- Congenital anomalies
- Trauma
- Marfan syndrome

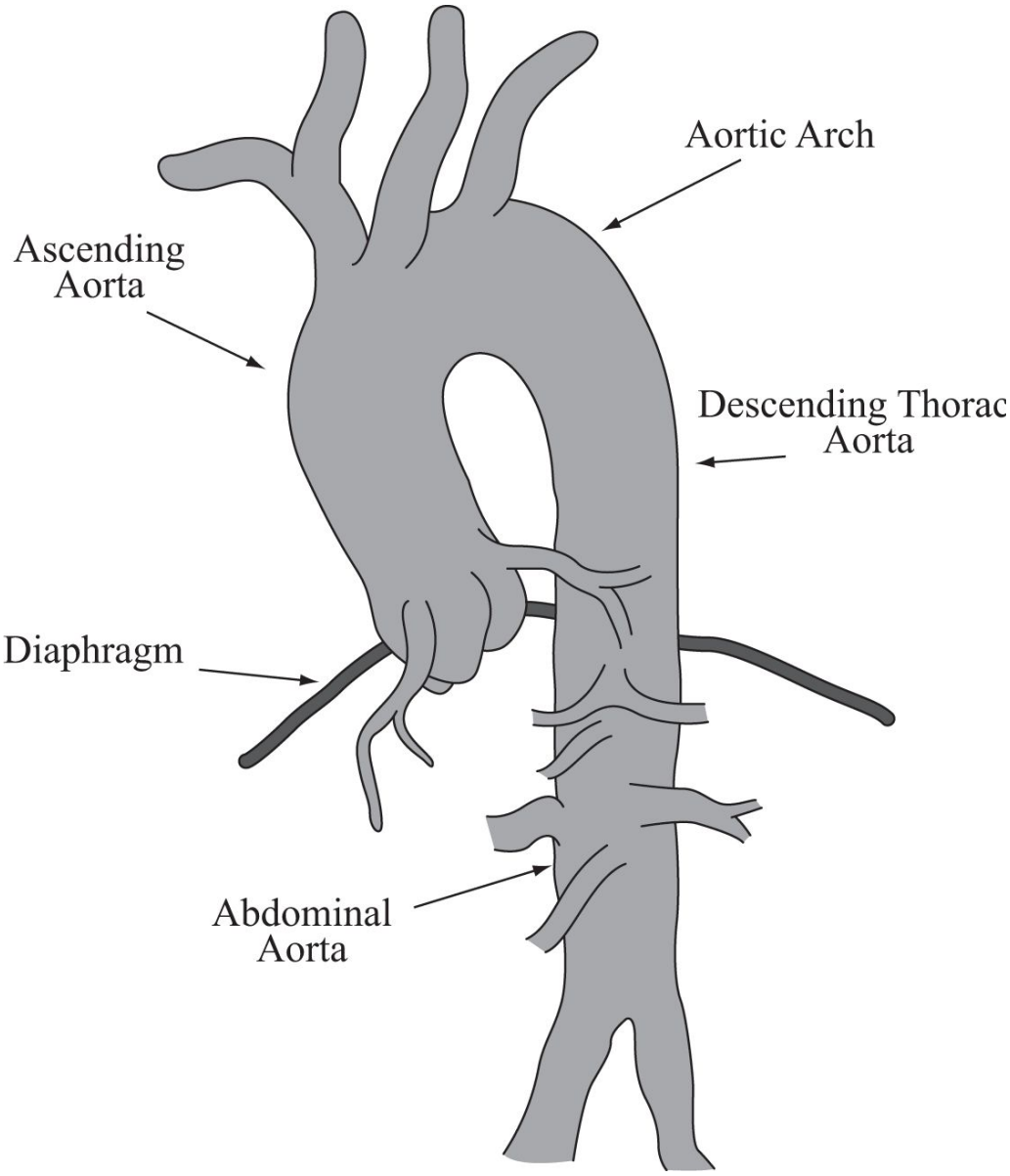


Figure 3-12. Types and locations of cardiovascular-related aneurysms

Abdominal Aortic Aneurysms (75% of all CV-related aneurysms)

- Asymptomatic if small
- Pulsations in abdominal area
- Abdominal or low back pain
- Nausea, vomiting
- Shock

Thoracic Aortic Aneurysms (25% of all CV-related aneurysms)

- Sudden tearing, ripping pain in chest that radiates to the shoulders, neck, and back
- Cough
- Hoarseness
- Dysphagia
- Dyspnea
- Dizziness, difficulty walking and speaking
- Widening of mediastinum on a chest X-ray

Treatment of Aneurysms

- Aneurysms that are < 5 cm in diameter and produce no symptoms
  - Monitor regularly.
    - Ultrasound or CT scan
  - Treat hypertension: drug class of choice is beta blockers, which may slow growth.
  - People with Marfan syndrome are often treated sooner.
- Thoracic aortic aneurysm that is causing symptoms or is > 6 cm
  - Surgical repair
  - Dissection: SURGERY
  - Aggressive treatment of hypertension and heart rate control
    - Labetalol drip

Aortic Dissection

Blood passes through the inner lining and between the layers of the aorta.

- The tear is spiral in nature.
- A dissection can occur suddenly or gradually.
- A dissection occurs in the ascending aorta or in the aortic arch.
- A dissection is life-threatening.
- A dissection requires immediate surgical intervention.

Now that you have reviewed the key cardiovascular concepts, go to the Practice Questions. Answer the questions, and then check your answers. Continue to review the information until you answer 80% of the practice questions correctly.

Practice Questions

1. A 59-year-old male is admitted with complaints of chest pain and dyspnea. ST elevation and T-wave inversion were seen on the ECG in V2, V3, and V4. IV thrombolytic therapy was started in the Emergency Department. Indications of successful reperfusion would include all of the following EXCEPT:

   (A) pain cessation.
   (B) a decrease in CK or troponin.
   (C) reversal of ST segment elevation with a return to baseline.
   (D) short runs of ventricular tachycardia.

2. Which of the following medication orders should the nurse question for the patient described in question 1?

   (A) metoprolol (Lopressor)
   (B) aspirin
   (C) propranolol (Inderal)
   (D) heparin

3. If heart block develops while caring for the patient described in question 1, which of the following types would it most likely be?

   (A) sinoatrial block
   (B) second-degree, Type I
   (C) second-degree, Type II
   (D) third-degree, complete

4. Appropriate drug therapy for dilated cardiomyopathy is aimed toward:

(A) decreasing contractility and decreasing both preload and afterload.
(B) decreasing contractility and increasing both preload and afterload.
(C) increasing contractility and increasing both preload and afterload.
(D) increasing contractility and decreasing both preload and afterload.

5. An 18-year-old is admitted with a history of a syncopal episode at the mall, and he has a history of an eating disorder. The nurse notes a prolonged QT on the 12-lead ECG and anticipates that a reduction in an electrolyte is the cause. Which of the following is least likely to cause this patient's problem?

(A) sodium
(B) magnesium
(C) potassium
(D) calcium

6. On the third day after admission for an acute MI, a 67-year-old male complains of chest pain and develops a fever. The pain is worse with deep inspiration and is relieved when he leans forward. There are nonspecific ST changes in the precordial leads of the ECG. The nurse anticipates that this patient will most likely need treatment for:

(A) a thoracic aneurysm.
(B) Dressler's syndrome.
(C) reinfarction.
(D) pleuritis.

7. A patient is admitted to the CCU after PCI with a stent. A femoral sheath is in place, and the site is dry with no hematoma. He suddenly complains of severe back pain. His neck veins are flat, the head of the bed is at 30 degrees, and his heart sounds are normal. His vital signs are as follows: BP is 78/48, heart rate is 124 beats/minute, and respiratory rate is 26 breaths/minute. What should the nurse suspect?

   (A) cardiac tamponade
   (B) retroperitoneal bleeding
   (C) coronary artery dissection
   (D) acute closure of the stented coronary artery

8. A patient admitted with an NSTEMI develops acute shortness of breath, a recurrence of chest pain, and a loud systolic murmur at the apex of the heart. Which of the following has most likely occurred?

   (A) The patient has developed acute mitral valve regurgitation.
   (B) The patient has developed acute reinfarction.
   (C) The patient has developed acute mitral valve stenosis.
   (D) The patient has developed an acute ventricular septal defect.

9. A patient has just returned from the OR after insertion of a VVI pacemaker. In order to assess the functioning of this pacemaker accurately, the nurse needs to understand that:

(A) both the atrium and ventricle are paced and sensed and may either inhibit or pace in response to sensing.
(B) the ventricle is paced, ventricular activity is sensed, and pacing is inhibited in response to ventricular sensing.
(C) both the atrium and ventricle are paced, but only ventricular pacing can be inhibited by a sensed intrinsic ventricular impulse.
(D) the ventricle is paced, in response to a sensed intrinsic atrial impulse or inhibited by a sensed intrinsic ventricular impulse.

10. A patient complains of sudden dyspnea 5 days S/P an acute MI (ST elevation in II, III, and aVF, with ST depression in I and aVL). The patient is anxious, diaphoretic, and hypotensive. An examination reveals the development of a loud holosystolic murmur at the apex that radiates to the axilla. The patient has crackles throughout but no S3 heart sound at the apex. What is the most likely cause of this patient's deterioration?

    (A) right ventricular failure related to a right ventricular MI
    (B) ventricular septal defect
    (C) left ventricular failure due to extension of the MI
    (D) acute mitral regurgitation due to papillary muscle rupture or dysfunction

11. A patient with a diagnosis of cardiogenic shock now requires high dose dopamine (greater than 10 mcg/kg/min) to maintain her blood pressure, and the cardiologist is planning to start IABP therapy. This therapy will benefit the patient because it will:

(A) increase afterload with balloon inflation and decrease diastolic augmentation with balloon deflation.
(B) decrease afterload with balloon deflation and increase diastolic augmentation with balloon inflation.
(C) decrease afterload with balloon inflation and decrease diastolic augmentation with balloon deflation.
(D) increase afterload with balloon deflation and decrease diastolic augmentation with balloon inflation.

12. Four days after mitral valve replacement, a patient goes into atrial fibrillation with a rapid ventricular response. What should be the nurse's initial action?

(A) Order a 12-lead ECG.
(B) Evaluate the patient for clinical signs of hypoperfusion.
(C) Notify the physician.
(D) Ask the patient to bear down as if having a bowel movement.

13. A patient's 12-lead ECG shows sinus bradycardia at 44 beats/minute and ST segment elevation in leads II, III, and aVF. Which of the following treatments for bradycardia would best resolve the problem for this patient?

(A) temporary transvenous pacing
(B) transcutaneous pacing
(C) percutaneous coronary intervention
(D) administration of atropine

14. Which drug would most likely be given to a patient with hypertrophic cardiomyopathy?

(A) metoprolol
(B) digoxin
(C) dopamine
(D) dobutamine

15. A patient is admitted with ST elevation in V2, V3, and V4. Four days after admission, the patient suddenly developed a holosystolic murmur at the lower left sternal border, chest pain, and hypotension. What complication should the nurse expect?

    (A) papillary muscle rupture
    (B) ventricular septal defect
    (C) acute mitral valve stenosis
    (D) acute reinfarction

16. A postoperative patient on the surgical unit suddenly develops chest pain, extreme weakness, and dyspnea and is found to have ST elevation in II, III, and aVF on the stat ECG. Her BP is 92/62, her heart rate is 58 beats/minute, her respiratory rate is 28 breaths/minute, her lungs are clear, and a heart sound assessment reveals an S4 with no murmurs. In addition to preparing the patient for PCI, which of the following interventions would the nurse anticipate?

    (A) a nitroglycerin drip and the administration of aspirin
    (B) the administration of furosemide (Lasix) and atropine
    (C) transcutaneous pacing and the administration of morphine
    (D) aggressive fluid administration and a right-sided ECG

17. A 52-year-old male presents with complaints of blurred vision and shortness of breath. His BP is 232/136, his heart rate is 102 beats/minute, his respiratory rate is 28 breaths/minute, he has

crackles in the lower lung fields bilaterally, and there are S3 and S4 heart sounds upon auscultation. Which of the following would be indicated for this patient?

(A) a nitroprusside drip and admission to the critical care unit
(B) the administration of digoxin and furosemide
(C) a labetalol drip and admission to a medical unit
(D) the administration of lisinopril and a calcium channel blocker

18. An 80-year-old female presents with a chief complaint of acute shortness of breath. A clinical exam reveals that her BP is 180/102, her heart rate is 105 beats/minute, her respiratory rate is 32 breaths/minute, she has lung crackles bilaterally, her pulse oximetry is 88%, and there are S4 heart sounds upon auscultation. An ECG revealed sinus tachycardia and a left ventricular hypertrophy pattern; a chest radiograph showed a normal heart size and pulmonary vascular congestion; and an echocardiogram showed an EF of 55%. Which of the following should be avoided in this patient's treatment plan?

(A) a calcium channel blocker
(B) digoxin
(C) low dose diuretics
(D) oxygen

19. A patient had a left internal carotid artery stent placed, and the latest assessment reveals that the patient is alert and oriented, has a weakened right hand grasp and a right facial droop, has stable vital signs, and has a dry procedure site without bleeding. The RN should contact the physician because this patient is most likely experiencing which of the following?

(A) a right cerebral hemorrhage
(B) hyperperfusion syndrome
(C) acute cerebrovascular insufficiency
(D) hypovolemia

20. Mrs. Jones has an exacerbation of her heart failure, with signs and symptoms of jugular venous distention (JVD), peripheral edema, and abdominal discomfort. These are clinical signs specific to:

(A) acute left ventricular failure.
(B) chronic right ventricular failure.
(C) acute right ventricular failure.
(D) chronic dehydration.

21. A nurse who is managing a post-op CABG patient assesses a sudden drop in BP, distended neck veins, muffled heart sounds, minimal chest tube output, and a systolic pressure that fluctuates with the breathing pattern. This patient most likely needs:

(A) an emergent return to the OR.
(B) clamping of the chest tube.
(C) a transfusion of PRBCs.
(D) high dose dopamine.

22. Physical assessment findings that are indicative of a significant right ventricular (RV) infarction would include:

(A) bibasilar crackles.
(B) flat neck veins with the patient in Semi-Fowler’s position.
(C) jugular venous distention.
(D) tachypnea and frothy sputum.

23. What pulse change might a nurse expect in the presence of cardiac tamponade?

    (A) pulsus alternans
    (B) pulsus paradoxus
    (C) pulsus magnus
    (D) pulsus bisferiens

24. A patient with mitral regurgitation develops atrial fibrillation with a heart rate of 88 beats/minute and a BP of 118/75. Which of the following may be indicated?

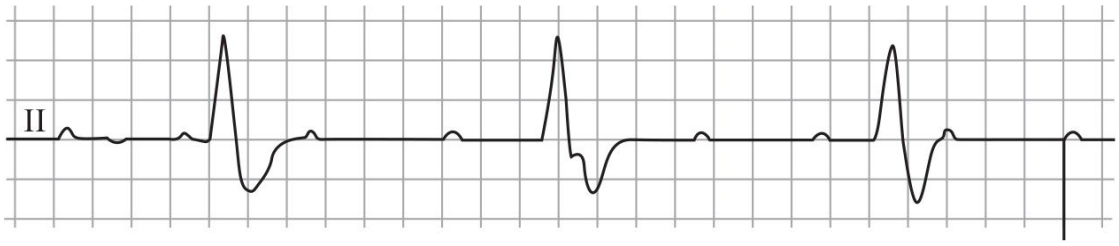
    (A) beta blockers and vasopressors
    (B) cardiac glycosides and calcium channel blockers
    (C) beta blockers and calcium channel blockers
    (D) antiarrhythmics and angiotensin-converting enzyme inhibitors

25. Which of the following are predominant signs of left ventricular systolic dysfunction?

    (A) pedal edema, ascites, hepatomegaly, weight gain, ejection fraction less than 40%
    (B) S4 heart sound, bibasilar crackles, hypertension, ejection fraction greater than 40%
    (C) S3 heart sound, new cough, bibasilar crackles, ejection fraction less than 40%
    (D) hypertension, murmur, chest pain, weight gain, ejection fraction greater than 40%

26. A nurse was preparing a patient with the diagnosis of STEMI for a percutaneous coronary intervention (PCI). The monitor had previously shown a normal sinus rhythm (NSR); the BP had been

128/78; and chest pain had improved from a “9” to a “2” on a 0–10 scale. The monitor alarm then sounded, and the rhythm below was observed by the nurse:



Based on this information, which of the following statements is TRUE?

(A) This change is most commonly seen with an acute inferior MI. Assess the patient. If serious signs and symptoms develop, begin transcutaneous pacing (TCP).
(B) This change is most commonly seen with an acute anterior MI. Assess the patient. If serious signs and symptoms develop, give atropine.
(C) This change is most commonly seen with an acute inferior MI. Assess the patient. If serious signs and symptoms develop, begin dobutamine.
(D) This change is most commonly seen with an anterior MI. Assess the patient. If serious signs and symptoms develop, begin transcutaneous pacing (TCP).

27. A 58-year-old patient developed chest pain that he scored as an “8.” A rapid assessment included profuse diaphoresis, a BP of 78/52, a heart rate of 104 beats/minute, a respiratory rate of 20 breaths/minute, clear lungs, and an SpO_2 of 98%. The patient is currently connected to the bedside monitor with a nasal cannula at 2 L/minute in place and intravenous fluids (0.9 NS) being

administered at a rate of 10 mL/hour. Which of the following sequences of interventions would be the most appropriate for the nurse to follow at this time?

(A) Give a chewable aspirin, do an ECG, and then start a fluid bolus.
(B) Give NTG sublingual, increase the FiO_2, and then give morphine.
(C) Do an ECG, give NTG sublingual, and then give a chewable aspirin.
(D) Start a fluid bolus, give a chewable aspirin, and then do an ECG.

28. The location or type of an acute MI is often associated with specific clinical findings. Which of the following statements related to the location of an MI is TRUE?

(A) An anterior MI is often associated with heart blocks or bradyarrhythmias.
(B) An inferior MI is often associated with right ventricular wall infarction.
(C) A lateral MI is most likely to be associated with a posterior MI.
(D) A posterior MI is most likely to lead to the complication of heart failure.

29. Which of the following statements is accurate regarding heart valves?

(A) The aortic valve is closed during systole.
(B) The mitral valve is closed during systole.
(C) The mitral valve is closed during diastole.
(D) The aortic valve is open during diastole.

30. The following drugs are all considered positive inotropic drugs that primarily affect the beta-1 receptors in the heart EXCEPT:

(A) dopamine drip at 12 mcg/kg/min dose.
(B) dopamine drip at 5 mcg/kg/min dose.
(C) dobutamine drip at 7 mcg/kg/min dose.
(D) milrinone at 7 mcg/kg/min dose.

Answer Key

1. **B**
2. **C**
3. **C**
4. **D**
5. **A**
6. **B**
7. **B**
8. **A**
9. **B**
10. **D**
11. **B**
12. **B**
13. **C**
14. **A**
15. **B**
16. **D**
17. **A**
18. **B**
19. **C**
20. **B**
21. **A**
22. **C**
23. **B**
24. **B**

25. **C**
26. **A**
27. **D**
28. **B**
29. **B**
30. **A**

Answers and Explanations

1. **(B)** Coronary artery reperfusion (due to either PCI or fibrinolysis) results in an **elevation** of, NOT a decrease in, creatine kinase (CK) or troponin. The theory is that the return of blood flow distal to the occlusion can result in a “reperfusion injury” of muscle, elevating cardiac biomarkers. The other 3 choices are indicators of successful reperfusion.
2. **(C)** The patient in this scenario is having an acute anterior wall MI. A beta blocker is beneficial for an acute MI, as these agents decrease the work of the heart and increase the threshold for ventricular fibrillation. Although it is a beta-adrenergic blocker like metoprolol, propranolol is NOT a cardioselective beta blocker. It affects beta receptors in heart muscle AND lung tissue. Therefore, it is more likely to cause bronchoconstriction than a cardioselective beta blocker. The other 3 choices (a cardioselective beta blocker, an antiplatelet, and an anticoagulant) are indicated for an acute MI, such as the one that the patient in this scenario is experiencing.
3. **(C)** The patient is having an acute anterior MI, which is generally due to LAD occlusion. The LAD supplies the bundle of His, so an LAD occlusion could result in a second-degree Type II heart block. If symptomatic bradycardia were to occur, the patient may require transcutaneous pacing. The other 3 types of heart blocks are due to SA node or AV node ischemia, which generally occur with an RCA occlusion (inferior wall MI).

4. **(D)** Dilated cardiomyopathy is likely to result in systolic dysfunction, which decreases contractility, causes compensatory arterial constriction, and results in a higher left ventricular preload. To treat this, therapy is aimed at increasing contractility, decreasing afterload (arterial constriction), and decreasing preload that is too high.
5. **(A)** An abnormal level of sodium does NOT cause QT prolongation. In contrast, a low level of magnesium, potassium, or calcium may cause QT prolongation and may result in torsades de pointes ventricular tachycardia and, if self-limiting, a transient syncopal episode.
6. **(B)** The pain described in the scenario is typical of the pain caused by pericarditis. Dressler's syndrome is the pericarditis that may result after an acute MI.
7. **(B)** Retroperitoneal bleeding may cause signs of hypovolemia and hypovolemic shock, as described in this scenario. It may be a complication of a PCI if the femoral artery is the access site during the procedure. Only this problem results in severe back pain; none of the other 3 choices results in back pain.
8. **(A)** The location of the murmur, at the apex of the heart (midclavicular, 5th ICS), is one clue to this answer. In addition, regurgitation occurs when the valve should be closed, and the mitral valve should be closed during systole. Mitral valve stenosis, choice (C), occurs when the mitral valve is open. Additionally, mitral valve stenosis cannot be acute; it develops gradually.
9. **(B)** The first letter indicates chamber paced (ventricle). The second letter indicates chamber sensed (ventricle). The third

letter indicates the response to sensing (inhibited in response to sensing).

10. **(D)** The scenario describes a patient having an acute inferior wall MI, which is generally due to occlusion of the RCA. The RCA occlusion may result in papillary muscle dysfunction or rupture of the mitral valve because it supplies the area of the left ventricle where this valve is attached. Although RV infarct could result in RCA occlusion, RV infarct does not result in a holosystolic murmur at the apex of the heart or lung crackles.
11. **(B)** Cardiogenic shock results in a decrease in cardiac output with a resultant drop in coronary artery perfusion and compensatory vasoconstriction. The deflation of the balloon that is placed into the descending aorta is beneficial. Deflation decreases afterload and work of the left ventricle. Inflation of the balloon is beneficial because it “boluses” blood into the coronary arteries, increasing perfusion.
12. **(B)** The patient’s response to the arrhythmia will determine whether treatment needs to be emergent and what the treatment will be. Vagal maneuvers (e.g., bearing down) are not known to be effective for atrial fibrillation.
13. **(C)** PCI would address the cause of the problem, not only treat the signs and symptoms. Selection of the other 3 choices presumes that the patient had serious signs and symptoms. Do not read into the questions.
14. **(A)** In hypertrophic cardiomyopathy, there is a problem with filling. A decrease in heart rate provided by a beta blocker such as metoprolol would increase the filling time. Diastolic dysfunction does NOT cause a problem with ejection, and the

EF is normal. The other 3 choices may be indicated for systolic dysfunction.

15. **(B)** This scenario describes an acute anterior STEMI, generally caused by an occlusion of the LAD. This type of MI is most likely to result in a VSD. Additionally, the location of the murmur is important. Mitral valve disease–related problems do NOT cause murmurs to be loudest at the left sternal border, whereas a VSD would result in a murmur at this location.
16. **(D)** This scenario describes a patient having an acute inferior STEMI, generally due to RCA occlusion. An RCA occlusion may result in an RV infarct, which this patient has signs of (hypotension with clear lungs). The definitive treatment is emergent PCI. Fluid administration will help increase coronary artery perfusion by correcting hypotension and ensure adequate RV preload. A right-sided ECG may help confirm the RV infarct. Nitroglycerin, diuretics, and morphine may decrease preload, which would worsen the hypotension.
17. **(A)** This patient has signs of organ dysfunction (heart failure) secondary to extreme hypertension. Therefore, he has a hypertensive crisis or emergency. The BP needs to be emergently decreased. Most often, this treatment is best done in a critical care setting.
18. **(B)** This patient presents with signs of heart failure due to diastolic dysfunction (hypertension, left ventricular hypertrophy, EF > 40%). This patient has a problem with FILLING, not ejection. Digoxin, a positive inotrope, may increase wall stress and worsen filling of the left ventricle.

19. **(C)** Contralateral motor weakness and aphasia may be signs of cerebrovascular insufficiency due to left stent patency issues. A right cerebral hemorrhage (choice (A)) would result in left-sided motor weakness and is not usually associated with post-carotid stent placement. The signs and symptoms of hyperperfusion syndrome (choice (B)) do not include contralateral motor weakness. There were no signs of hypovolemia (choice (D)), such as hypotension or oliguria, described in the question.
20. **(B)** The signs described are those of chronic right-sided heart failure. Acute right ventricular failure may result in JVD, but not peripheral edema or abdominal discomfort (which is due to liver engorgement).
21. **(A)** The signs described in the scenario are those of cardiac tamponade. The treatment for cardiac tamponade for a post-op open heart surgery patient is a return to the OR to drain the pericardial fluid that has accumulated. Development of this problem in other patient populations would necessitate an emergent pericardiocentesis to drain the fluid.
22. **(C)** A right ventricular infarction that is large enough to cause RV failure causes a problem with RV emptying, leading to an elevated right atrial pressure. This causes jugular venous distention. Choices (A) and (D) are signs of left ventricular failure. Choice (B) is a sign of dehydration.
23. **(B)** Pulsus paradoxus is fluctuation of the systolic blood pressure with inspiration and expiration by more than 12 mmHg, best seen when an arterial line is in place. Inspiration increases thoracic pressure. When combined with fluid surrounding the heart in cardiac tamponade, inspiration further

decreases venous return to the heart, leading to a drop in systolic pressure by > 12 mmHg during the inspiratory phase of breathing. Pulsus alternans (choice (A)) is characterized by a change in amplitude of the systolic waveform from beat to beat, usually indicative of severe left ventricular failure. Pulsus magnus (choice (C)) is a bounding pulse. Pulsus bisferiens (choice (D)) is a double pulse and is not covered on the Adult CCRN exam.

24. **(B)** This scenario describes the development of atrial fibrillation with a controlled ventricular response and a stable BP. Even with a normal BP, the development of atrial fibrillation drops the cardiac output by 20% to 25% due to a loss in “atrial kick” provided by a normal sinus rhythm. A cardiac glycoside (such as digoxin) may be beneficial since it is a weak positive inotrope that may compensate for the loss of atrial kick, and calcium channel blockers will keep the heart rate controlled. Pressors are not needed in this case. The use of both beta blockers and calcium channel blockers would decrease the heart rate too much. ACE inhibitors would offer no benefit in this case.
25. **(C)** An S3 heart sound in an adult is indicative of high left ventricular pressure. A new cough and lung crackles are signs of pulmonary edema secondary to elevated left ventricular end-diastolic pressure (PAOP), although these may be signs of other problems, such as pulmonary fibrosis. The EF is less than 40% in systolic heart failure.
26. **(A)** The patient’s clinical status describes a stable BP, but the development of a bradyarrhythmia, specifically a complete heart block with a ventricular rate of 30 beats/minute. This is usually

seen with RCA disease, generally associated with an inferior MI. A patient assessment will determine treatment. Transcutaneous pacing would be an appropriate treatment for an unstable patient with this arrhythmia.

27. **(D)** This clinical description may be that of acute coronary syndrome complicated by hypotension. Addressing the hypotension is a priority since this is further decreasing coronary artery perfusion. A fluid bolus would address the hypotension, and no contraindications seem to be present for a fluid bolus since the lungs are clear. Aspirin is indicated for acute chest pain and could be given while preparing to do the ECG, which is needed to help make the diagnosis.
28. **(B)** Since most inferior MIs are due to RCA occlusion and the RCA also supplies blood to the right ventricular muscle wall, an inferior MI is associated with RV infarct.
29. **(B)** During systole (left ventricular ejection), the aortic valve is open, allowing for ejection. The mitral valve is closed at this time. The mitral valve is open during filling (diastole).
30. **(A)** At high doses (> 10 mcg/kg/min), dopamine stimulates alpha receptors in the arteries and causes vasoconstriction. The other 3 drugs/doses affect mainly the beta-1 receptors in the heart, producing a positive inotropic effect.