CHAPTER 5: Cardiac System

Cardiovascular disease is the leading cause of death in the United States. This chapter covers cardiac anatomy, coronary artery disease, acute coronary syndromes, heart failure, shock states, hypertension, and basic EKG interpretation. A solid understanding of cardiac pathophysiology is essential for nursing practice in any setting.

Heart Anatomy and Cardiac Cycle

Heart Chambers and Valves

Right Atrium (RA): Receives deoxygenated blood from SVC and IVC
Tricuspid Valve: Between RA and RV; prevents backflow
Right Ventricle (RV): Pumps deoxygenated blood to lungs via pulmonary artery
Pulmonic Valve: Between RV and pulmonary artery

Left Atrium (LA): Receives oxygenated blood from pulmonary veins
Mitral (Bicuspid) Valve: Between LA and LV; most common valve affected by disease
Left Ventricle (LV): Pumps oxygenated blood to body via aorta (most muscular chamber)
Aortic Valve: Between LV and aorta

Blood Flow Path: RA → TV → RV → PV → Lungs → LA → MV → LV → AV → Aorta → Body

Coronary Circulation: The heart muscle itself is supplied by the coronary arteries. **Right Coronary Artery (RCA)** supplies the RV and inferior LV. **Left Coronary Artery (LCA)** splits into LAD (left anterior descending) which supplies anterior LV and septum, and Circumflex which supplies lateral LV. Coronary arteries fill during diastole when the aortic valve is closed.

Coronary Artery Disease (CAD)

CAD is the narrowing of coronary arteries by atherosclerotic plaque. Over years, cholesterol and other substances accumulate in the intima, gradually narrowing the artery lumen. When a blockage becomes severe (>70% luminal narrowing), the artery cannot dilate enough to provide adequate blood flow during stress or increased demand, causing ischemia (insufficient blood supply).

Risk Factors for CAD

- **Non-Modifiable:** Age, sex, family history
- **Modifiable:** Hypertension, hyperlipidemia (high cholesterol), diabetes mellitus, smoking, obesity, sedentary lifestyle, stress, excessive alcohol

Angina Pectoris

Stable Angina

Stable angina occurs when demand exceeds supply. **Predictable pattern:** Occurs with exertion or stress and resolves with rest or nitroglycerin. Same location and character each time. **Pathophysiology:** Narrowed coronary artery allows adequate blood flow at rest but cannot accommodate increased demand. **S/S:** Crushing chest pain, substernal, may radiate to jaw/shoulder/left arm, associated with dyspnea, diaphoresis, nausea. **Treatment:** Stop activity, sit down, take sublingual nitroglycerin (should relieve pain in 5 minutes), rest.

Unstable Angina

MEDICAL EMERGENCY. Unstable angina indicates a plaque rupture with partial occlusion and/or vasospasm. **Unpredictable pattern:** Occurs at rest, increases in frequency/severity, or does not respond to NTG. Indicates acute coronary syndrome. **Troponin and CK-MB may be normal** (distinguishes from MI), but troponin can be slightly elevated. **Treatment:** Immediate hospitalization, antiplatelet therapy (aspirin, clopidogrel/Plavix), anticoagulation (heparin), Beta-blockers, consider cardiac catheterization for revascularization.

Prinzmetal Angina (Vasospastic)

Caused by coronary artery vasospasm (not atherosclerotic plaque). Often occurs at rest, particularly early morning. EKG may show ST elevation during pain. **Treatment:** Calcium channel blockers (diltiazem, verapamil) as first-line because they prevent vasospasm.

Myocardial Infarction (MI) / Acute Coronary Syndrome (ACS)

MI occurs when a coronary artery is completely blocked, usually by plaque rupture with thrombosis. The myocardium supplied by that artery is deprived of oxygen and begins to die (infarct). This is a life-threatening emergency requiring immediate intervention. MI is classified as STEMI or NSTEMI based on EKG findings.

Signs and Symptoms of MI

- **Chest Pain:** Crushing, squeezing, oppressive quality; substernal; may radiate to left arm, jaw, or back
- **Duration:** Lasts >20 minutes (distinguishes from stable angina)
- **Associated Symptoms:** Shortness of breath, diaphoresis (sweating), nausea/vomiting, dizziness
- **Women:** May present with atypical symptoms: fatigue, dyspnea, nausea (without chest pain), jaw pain
- **Anxiety:** Sense of doom or impending death
- **Silent MI:** Some patients (especially diabetics) may have no symptoms or atypical symptoms

Diagnostic Tests

Troponin I and T: Most specific markers for myocardial necrosis; rise 3-4 hours after MI onset, peak at 24-48 hours, remain elevated 7-14 days. Even small elevations indicate myocardial injury. **CK-MB:** Older marker, rises 3-12 hours, peaks at 24 hours, returns to normal by 48-72 hours. Useful for detecting reinfarction. **Myoglobin:** Earliest marker (1-2 hours) but not cardiac specific. **EKG:** ST elevation in STEMI, ST depression or T wave changes in NSTEMI. Location indicates affected artery. **Echocardiography:** Shows wall motion abnormalities, ventricular dysfunction, complications (septal rupture, papillary muscle rupture).

STEMI vs NSTEMI

STEMI vs NSTEMI Comparison

Feature	STEMI	NSTEMI
EKG	ST elevation	ST depression or T wave changes
Troponin	Elevated	Usually normal initially
Artery	Complete occlusion	Partial occlusion or recanalization
Treatment	PCI <90 min or fibrinolytics	Medical management, may need PCI

Mnemonic: MONA - Initial Treatment for ACS

M - Morphine (for pain and anxiety)
O - Oxygen (if SaO2 <90%)
N - Nitroglycerin (sublingual, reduces preload)
A - Aspirin 325mg (antiplatelet, chewed for faster absorption)

Definitive Treatment

STEMI: Goal is to open the occluded artery within 90 minutes. **PCI (Percutaneous Coronary Intervention)** is preferred: catheterization, angiography to locate blockage, balloon angioplasty to dilate, and stent placement to keep artery open. If PCI unavailable: **Fibrinolytic therapy** (thrombolytics like alteplase/tPA, streptokinase) to dissolve clot within 12 hours of symptom onset. **NSTEMI:** Antiplatelet (aspirin, clopidogrel/Plavix), anticoagulation (heparin), beta-blockers, ACE inhibitors, statins. PCI may be done if high-risk features or recurrent ischemia.

Clinical Pearl

Time is Muscle: The longer an artery is occluded, the more myocardium dies. Every minute counts in STEMI. The goal of <90 minutes from door-to-balloon time for PCI is critical. If presented with chest pain at home, call 911 immediately; pre-hospital EKG and notification of catheterization lab can reduce door-to-balloon time significantly.

Heart Failure (HF)

Heart failure occurs when the heart cannot pump enough blood to meet the body's oxygen demands. It may be systolic (pump dysfunction) or diastolic (filling dysfunction). HF is classified as left-sided, right-sided, or biventricular. **New York Heart Association (NYHA) Classification:** Class I (no symptoms with activity), II (symptoms with exertion), III (symptoms at rest), IV (symptoms at rest).

Left-Sided Heart Failure (Most Common)

LV cannot pump blood forward effectively. Blood backs up into the lungs causing pulmonary congestion. **S/S:** Dyspnea (worsens with exertion), orthopnea (shortness of breath when lying flat), paroxysmal nocturnal dyspnea (PND - wakes at night gasping), crackles on lung auscultation (fine crackles that don't clear with cough), pink frothy sputum (severe pulmonary edema), fatigue from decreased cardiac output.

Right-Sided Heart Failure

RV cannot pump blood forward effectively. Blood backs up into the systemic circulation. **S/S:** Jugular venous distention (JVD), peripheral edema (dependent areas, pitting), hepatomegaly (enlarged liver), hepatic congestion with right upper quadrant pain, ascites (fluid in peritoneal cavity), weight gain from fluid retention, decreased appetite from hepatic congestion.

Mnemonic: Left vs Right Heart Failure

Left = Lung symptoms
Remember: Lungs are on the left side
Dyspnea, crackles, pink frothy sputum

Right = Rest symptoms
Remember: Rest of the body
Peripheral edema, JVD, hepatomegaly

Heart Failure Treatment

First-Line Medications: ACE Inhibitors (enalapril, lisinopril) - reduce afterload and cardiac remodeling; **Beta-Blockers** (carvedilol, metoprolol) - reduce heart rate and contractility workload; **Diuretics** (furosemide/Lasix) - reduce fluid overload. **Additional: ARBs** (valsartan) if ACE-I not tolerated; **Aldosterone antagonists** (spironolactone); **Digoxin** (positive inotrope, slows AV conduction) for symptomatic systolic HF; **Vasodilators** like nitrates. **Nursing interventions:** Daily weights (report gain >2-3 lbs), sodium restriction (<2 g/day), fluid restriction (1-1.5 L/day in advanced HF), monitor lung sounds, elevate head of bed, oxygen if needed, teach about medication adherence.

Digoxin (Digitalis): Important Cardiac Glycoside

Action: Positive inotrope (increases myocardial contractility) and negative chronotrope (slows heart rate by slowing AV conduction). Increases stroke volume and decreases compensation, improving symptoms in HF. **Therapeutic Level:** 0.5-2.0 ng/mL. **Narrow therapeutic window:** Only 0.5-2.0 range; toxicity occurs above 2.0.

Nursing Considerations for Digoxin

- **Check Apical Pulse:** ALWAYS check for 1 full minute before administering. Hold drug if HR <60 bpm (bradycardia develops)
- **Monitor Labs:** Digoxin level, potassium (critical!), magnesium, calcium
- **Hypokalemia Risk:** Increases digoxin toxicity risk significantly. If patient on diuretics (common), monitor K+ closely
- **Drug Interactions:** Many drugs increase digoxin level: quinidine, verapamil, amiodarone. If started on these, reduce digoxin dose
- **Renal Function:** Digoxin is renally excreted; renal failure increases toxicity risk

Digoxin Toxicity Signs and Symptoms

- **Gastrointestinal:** Nausea, vomiting, anorexia (first signs)
- **Cardiac:** Dysrhythmias (most serious; any dysrhythmia in patient on digoxin suggests toxicity), bradycardia
- **Visual:** Visual disturbances: yellow or green halos around lights (pathognomonic)
- **CNS:** Headache, weakness, confusion, fatigue

■ WARNING

Digoxin Toxicity Emergency: If severe dysrhythmias occur, contact provider immediately. Digoxin-specific antibody (Digibind) may be given. Hold digoxin, check potassium (if low, correct it first), monitor EKG. Hypokalemia increases toxicity risk, so it's the #1 risk factor to monitor.

Shock States

Shock is a state of inadequate tissue perfusion and cellular oxygenation. The body attempts to compensate with tachycardia, vasoconstriction, and increased respiratory rate. If untreated, shock progresses to irreversible organ damage and death.

Classification of Shock Types

Type	Cause	Early S/S	Later S/S
Hypovolemic	Blood/fluid loss (hemorrhage, burns, GI losses)	Tachycardia, BP, UO, cool/clammy, thirst	Cyanosis, confusion, weak pulse, death

Cardiogenic	Heart pump failure (MI, HF, dysrhythmias)	Tachycardia, BP, crackles, UO	Pulmonary edema, confusion, organ failure
Septic	Severe infection/sepsis	Warm, flushed, tachycardia, fever	Later cool/clammy, BP, UO, DIC
Anaphylactic	Allergic reaction (drug, food, insect)	Urticaria, angioedema, stridor, wheezing	Respiratory collapse, cardiovascular collapse
Neurogenic	Spinal cord injury	Bradycardia, BP, warm/dry skin (paradox)	Paralysis, respiratory failure

Clinical Pearl

Septic vs Other Shock: Early septic shock is **WARM** and **PINK** (vasodilation despite hypotension). Other shock states are **COLD** and **CLAMMY** (vasoconstriction). This is a key distinguishing feature.

Shock Management

• **Hypovolemic:** IV fluids, blood products, treat source of bleeding, monitor urine output (goal 30-50 mL/hr)

• **Cardiogenic:** Improve cardiac output (inotropes like dobutamine), reduce preload with diuretics, treat underlying cause (MI, dysrhythmia)

• **Septic:** Antibiotics, supportive care, fluid resuscitation, vasopressors (norepinephrine) if hypotensive despite fluids, source control

• **Anaphylactic:** IMMEDIATE epinephrine IM (0.3-0.5 mg of 1:1000), IV access, oxygen, airway protection, antihistamines, steroids

• **Neurogenic:** Supportive care, respiratory support if needed, treat spinal cord injury

Hypertension

Hypertension is defined as persistent elevation of blood pressure. Chronic HTN damages blood vessels and the heart, increasing risk of MI, stroke, and renal disease. The 2017 ACC/AHA guidelines define: **Normal <120/80 mmHg**, **Elevated 120-129 and <80**, **Stage 1: 130-139 or 80-89**, **Stage 2: 140 or 90**.

Hypertension Management

• **Lifestyle Modifications First:** Weight loss, DASH diet, reduce sodium to <2,300 mg/day, limit alcohol, increase exercise, stress management, smoking cessation

• **Medications if Needed: ACE Inhibitors/ARBs** (first-line for most), **Beta-blockers**, **Calcium Channel Blockers (CCBs)**, **Thiazide Diuretics**. Choice depends on comorbidities (e.g., ACE-I in DM and CKD, beta-blockers after MI)

• **Monitoring:** Home BP monitoring encouraged, follow-up in 1 month if started on meds, goal BP <130/80 for most

EKG Basics

Normal Sinus Rhythm

Characteristics: Regular rhythm at 60-100 bpm, each QRS preceded by P wave, PR interval 0.12-0.20 seconds, QRS duration <0.12 seconds. All P waves and QRS complexes have consistent morphology.

Common Dysrhythmias

- **Atrial Fibrillation (AFib):** Disorganized atrial activity. EKG: no P waves, irregularly irregular rhythm (completely random), sawtooth baseline. Most common dysrhythmia in elderly. Complications: thrombus formation → stroke (give anticoagulation like warfarin or apixaban)
- **Supraventricular Tachycardia (SVT):** Heart rate 150-250 bpm, regular rhythm, narrow QRS. Sudden onset and offset. Treatment: vagal maneuvers, adenosine IV, beta-blockers
- **Ventricular Tachycardia (VTach):** Life-threatening. Wide QRS (>0.12), rate usually 150-250 bpm. Can degenerate to VFib. Treatment: CPR, defibrillation, amiodarone or lidocaine
- **Ventricular Fibrillation (VFib):** Chaotic electrical activity. No organized complexes, no pulse. Medical emergency - organ death occurs in minutes. Treatment: IMMEDIATE defibrillation, CPR, epinephrine, amiodarone

Practice Question

A patient with chest pain and known CAD takes nitroglycerin. The pain does not resolve after 5 minutes. What should the nurse do?

A) Wait 10 more minutes
B) Chew another aspirin tablet
C) Call 911 immediately
D) Have patient rest and try again

Answer: C - Call 911 immediately

Practice Question

A patient on digoxin for heart failure reports seeing yellow halos around lights and develops nausea. What is the priority nursing action?

A) Give antiemetics
B) Notify provider immediately - signs of digoxin toxicity
C) Check apical pulse
D) Increase fluid intake

Answer: B - Notify provider immediately - signs of digoxin toxicity

Practice Question

When assessing a patient in shock, which finding would indicate septic shock rather than other types?

A) Cold, clammy skin
B) Warm, flushed skin with fever
C) Severe bradycardia
D) Pale, cyanotic appearance

Answer: B - Warm, flushed skin with fever

The most common cause of death in cardiac patients after MI is _____ or cardiogenic shock from pump failure.

Answer: Dysrhythmias (or Ventricular fibrillation)

Before administering digoxin, the nurse must check the apical pulse for at least _____ minute(s) and hold the dose if HR is <60 bpm.

Answer: One (or 1 full)

Calcium Channel Blockers (CCBs) - Detailed

• Two distinct types with different effects on heart rate and blood pressure:

Comparison Table

Type	Medications	Primary Effect	Heart Rate	Uses
Dihydropyridines	Nifedipine, Amlodipine, Felodipine	Vasodilation (drop BP)	Usually no effect	Hypertension, Angina
Non-Dihydropyridines	Verapamil (Isoptin), Diltiazem (Cardizem)	Vasodilation + HR reduction	DECREASES	HTN, Angina, Arrhythmias

Mnemonic: CCB Remember

Dipines = Drop pressure only; Verapamil/Diltiazem = Drop rate AND pressure

• CRITICAL NURSING INTERVENTIONS: