FIFTH EDITION

Hollier's Clinical Guidelines in Primary Care

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Contents

How to Use This Book

Hollier's Clinical Guidelines in Primary Care, Fifth Edition, is designed to be a resource for students and practicing NPs. Whether you are in clinical rotations, studying for the certification exam, or seeing patients, this book provides the evidence-based information you need—in a format that makes it easy to find and easy to understand.

ORGANIZATION

Hollier's Clinical Guidelines in Primary Care is organized into chapters that represent the key body systems and the most common diagnoses seen in primary care. Topics in the section for each diagnosis are then presented in a standard order that corresponds to a typical office visit and integrates evidence-based practice and clinical guidelines into the NP standards of practice process of care. The standard process of care includes the domains of assessment, diagnosis, planning and implementation, and evaluation. That process is guided by the scientific basis of the disease which is essential for clinical decision making in managing patient care and fostering professional practice.

Following are the domains, the components included in that domain, and the corresponding section headings in each chapter:

1. **Assessment** is the process of conducting a comprehensive and relevant assessment based on the pathophysiology and etiology of the disease process. This domain includes obtaining a comprehensive, relevant health, social, and medical history; performing a thorough physical examination; performing or ordering preventive and diagnostic procedures; and identifying health risk factors.
    - Section headings: Description, Etiology, Incidence, Risk Factors, Assessment, and Diagnostic Studies
2. **Diagnosis** involves the application of diagnostic reasoning to formulate differential diagnoses and to arrive at an initial diagnosis. This domain includes synthesizing and analyzing the collected data from assessment and diagnostic studies to develop and prioritize the differential diagnoses and establish a primary diagnosis.
    - Section heading: Differential Diagnoses (overlapping with assessment and diagnostic studies)
3. **Planning/implementation** is the development of an evidence-based, individualized plan of care that includes therapeutic interventions, patient education, counseling, and health promotion.
    - Section headings: Nonpharmacologic Management (including prevention), Pharmacologic Management, and Consultation/Referral
4. **Evaluation** is the systematic process of determining the effectiveness of the plan of care and documenting its outcomes. The NP will reassess and modify the plan of care as necessary to achieve desired outcomes and goals.
    - Section headings: Follow-Up, Expected Course, and Possible Complications

GUIDANCE FOR FACULTY AND NP STUDENTS

Hollier's Clinical Guidelines in Primary Care contains information for each diagnosis and supports the integration of population-focused didactic and clinical courses with continuous progression throughout the program. The NP curriculum requires that students successfully complete the three advanced practice registered nurse (APRN) core courses (advanced health assessment, advanced physiology/pathophysiology, and advanced pharmacology—courses known as the 3Ps) prior to progressing to the NP population coursework, which focuses on diagnosis and management of health problems. This book encompasses the essence of the 3Ps applied to specific diagnosis; it provides information in a way that facilitates clinical reasoning and critical thinking in the practice setting, thereby reinforcing knowledge gained through didactic learning.

NP students can use this book in the classroom and clinical setting to develop clinical competency in

the effective management of patients consistent with the primary care role, for communicating with the preceptor, and to complete assigned readings or clinical assignments (e.g., case studies, SOAP notes). Students are encouraged to focus on the content related to one or two patients seen in their clinical rotations each day. As an example, consider a patient with suspected pneumonia. A student could follow these four steps:

1. Ask yourself: What is pneumonia? What causes it? Which risk factors might predispose a patient to pneumonia? Which symptoms should I expect?
2. Then consider: If my patient has pneumonia, what should I find during the examination? Which characteristics may indicate a different diagnosis (differential)? Which laboratory or imaging studies should I order to help determine the diagnosis? Is there a gold standard for diagnosis? Which findings would support my diagnosis?
3. Based on the diagnosis, decide: What are the appropriate nonpharmacologic interventions? According to the evidence-based guidelines and/or clinical practice guidelines, which pharmacologic interventions are most appropriate to manage the patient? What kind of patient education is needed about the plan of care? What are the potential side effects of medications? How should the patient take the medication?
4. Then determine: What are the appropriate follow-up needs for this patient? Is a test of cure needed? What is the expected course of the disease process? What are the potential complications? What are the indications for referral? Which symptoms may warrant reporting via phone call or a return visit?

This step-by-step process will help NP students build a deep understanding of each diagnosis and know how to apply that understanding in patient care (and on an exam).

Hollier's Clinical Guidelines in Primary Care is the perfect companion to question banks (when more information is needed to understand a rationale). Students can also use it when studying for a predictor exam or when remediating the results of the 3P or the predictor exam, and preparing for the certification exam.

GUIDANCE FOR PRACTICING NPS AND PRECEPTORS

This book is a valuable evidence-based reference for the clinical setting. The concise presentation of the content provides the information most relevant to primary care. The outline follows the standards of care process as described earlier. To use this text in the clinical setting, locate the most common assessment findings associated with the diagnosis, then read down the page to identify appropriate diagnostic tests, differential diagnoses, and evidence-based interventions to formulate an individualized plan of care for the patient. Detailed pharmacology tables are included, along with comments that are specific to the drug class and to individual drugs in that class. Complete the plan of care by reviewing the follow-up recommendations, expected course, and potential complications of the disease process, and by identifying the indications for referral.

Clinical preceptors are vital partners in APRN education. While the student has the primary responsibility of meeting the learning needs and objectives of the clinical experience, the preceptor is critical to guiding the student through the effective management of patients and incorporating evidence-based best practices to ensure safe and effective patient outcomes. Both preceptors and students can use these clinical practice guidelines as an effective resource to help meet those objectives.

CHAPTER 1

Cardiovascular Disorders

ABDOMINAL AORTIC ANEURYSM

DESCRIPTION

Abdominal aortic aneurysm (AAA) is a dilation of the abdominal aorta. The pathology of AAA is characterized by chronic aortic wall inflammation, destruction of the elastic media, and depletion of the vascular smooth muscle cells. Aneurysm development involves a complex remodeling process involving connective tissue proteins and the synthesis and degradation of the proteins. The abdominal aorta begins below the diaphragm and extends to the level of the aortic bifurcation. Normal abdominal aorta size is 2.5 to 3 cm. Abdominal aortic diameter ≥3 cm or 50% increase in diameter compared with the adjacent normal structure constitutes an AAA. Most aneurysms are slow growing, do not cause symptoms, and are found incidentally. The risk of rupture is proportional to the size of the aneurysm. Treatment is recommended when an AAA grows to more than 5.5 cm in diameter.

ETIOLOGY

- Multifactorial; commonly involves a weakening of the arterial wall by:
  - Atherosclerosis
  - Trauma
  - Postsurgical anastomotic disruption
  - Vasculitis
  - Sepsis or infective endocarditis—thought to play a role but is uncommon
  - Tobacco use

INCIDENCE

- Lifetime risk is 8.2% in men and 10.5% in current smokers.
- At least 10–25% of patients with AAA have a family member with the same condition.
- 15,000 deaths/year are attributed to AAAs, and it is the 10th leading cause of death in men older than 55 years.

RISK FACTORS

- Older age, ≥65 years
- Male sex
- Smoking history
- Positive family history
- Known atherosclerosis
- Hypertension
- Hyperlipidemia
- White race
- Inherited vascular connective tissue disorder (i.e., Loeys-Sietz syndrome, vascular Ehlers-Danlos syndrome, or Marfan syndrome)

ASSESSMENT

- Usually asymptomatic, but may be nonspecific as a result if compression of adjacent structures.
  - As AAA expands, pain, which is steady, deep, and visceral, may be felt in the lumbosacral region.

- Physical exam has only moderate sensitivity for detecting AAA.
- Abdominal aorta begins at the diaphragm or 12th thoracic vertebrae and lies in the retroperitoneum slightly left of the vertebral column; a pulsatile mass may or may not be felt in the upper abdominal quadrants; systolic bruit may be heard over pulsatile mass.
- Palpation does not precipitate rupture, and a thorough physical exam should be completed.
- Physical exam should include assessment of femoral and popliteal arteries.

Older age, male sex, and smoking are independent, strong risk factors for the development of AAA.

DIFFERENTIAL DIAGNOSES

- Nephrolithiasis
- Myocardial infraction
- Gastric ulcer perforation
- Pancreatitis
- Diverticulitis
- Bowel obstruction
- Appendicitis

DIAGNOSTIC STUDIES

- Ultrasonography is used for screening and initial confirmation of an aneurysm; it can provide accurate measurement of initial size and be used for serial follow-up. (Sensitivity 94–100%; Specificity 98–100%)
- Computed tomography angiography (CTA) with three-dimensional imaging is performed prior to aneurysm repair surgery to show dilation of the aorta in relationship to major branch vessels, the degree of calcification, the presence of mural thrombus, and other information prior to repair.
- AAAs measuring >5.0 cm in women and >5.5 cm in men should be considered for elective repair in otherwise healthy patients.
- The following table is a guide on who to screen, when to screen, and how to screen for AAA.

Recommendations for One-Time Ultrasound Screening

Age/Sex	Risk Factors	Level of Evidence
Male 65-75 years	Ever smoked or family history	Recommended (Grade B, Level 1)
	Never smoked	No recommendation for or against; shared decision making (Grade C)
Female 65-75	Never smoked; no family history	No recommendation for screening (Grade D)
	Ever smoked or positive family history	Insufficient evidence; shared decision making (Grade I)

PLANNING/IMPLEMENTATION

- The goal of AAA management is to prevent rupture while minimizing surgical risk.
- Encourage smoking cessation.
- Control risk factors such as hypertension, hyperlipidemia, diabetes, and appropriately manage coronary artery disease (CAD)/atherosclerosis.
- Encourage AAA screening of first-degree relatives who are 50 years and older.

NONPHARMACOLOGIC MANAGEMENT

- Physical activity to stay fit, but avoid activities that involve heavy lifting or straining.
- Open surgical repair or endovascular AAA repair (EVAR) with stent graft performed by vascular surgeon; EVAR is the preferred method of repair.

CONSULTATION/REFERRAL

- Immediate transfer to emergency department if suspected AAA rupture (patient complains of severe abdominal or back pain, has hypotension or shock and tachycardia)
- Consult vascular surgeon for repair when AAA detected and ultrasonography performed and results available
- Referral to smoking cessation specialist to assist patient with smoking cessation (if available)

FOLLOW-UP

- AAA measuring 3.0–3.9 cm should have surveillance ultrasound every 3 years to assess for change.
- AAA measuring 4.0–4.9 cm in men and 4.0–4.4 cm in women should have surveillance ultrasound annually to assess for change.
- In men with AAA ≥5 cm and women with AAA ≥4.5 cm should have surveillance ultrasound every 6 months to assess for change.
- After EVAR, baseline surveillance imaging with computed tomography is recommended at 1 month postoperatively and then ultrasound at 12 months and then annually.

EXPECTED COURSE

- Mortality rate from ruptured AAA estimated to be 80–90% with most patients never reaching the hospital; for those who present to the hospital, the mortality rate for open surgical repair is 50%.
- 30-day mortality rate for open surgical repair is 3–5%; for EVAR, 1–2%.
- Patients with AAA not yet in need of intervention should continue to manage their blood pressure, manage their smoking status, remain physically active, and continue with surveillance monitoring for AAA increase in size.

POSSIBLE COMPLICATIONS

- Surgical complications: groin hematoma, arterial thrombosis, iliac artery rupture, thromboemboli
- EVAR complications: endoleak (persistent filling of the aneurysm sac); severe graft kinking, graft migration, graft thrombosis, renal dysfunction

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ABNORMAL HEART RHYTHMS (Arrhythmias)

DESCRIPTION

Abnormal heart rhythms are electrical abnormalities of the cardiac conduction system. These may occur in the presence or absence of structural heart defects or cardiac disease and vary in severity from benign to life-threatening. Abnormal heart rhythms are the result of abnormal impulse formation or conduction from functional components of the cardiac conduction system—either the impulse-generating tissue (sinoatrial [SA] and atrioventricular [AV] nodes) or the impulse-propagating tissue (His–Purkinge system).

The abnormal impulses or conduction can be categorized into one or more of three mechanisms: abnormal automaticity, triggered activity, or reentry. Tachycardia is defined as a heart rate >100 beats per minute (bpm). Bradycardia is defined as a heart rate <60 bpm.

Common tachyarrhythmias

- Atrial fibrillation (AF): See separate chapter discussing this diagnosis.
- Atrial flutter: The atrial rate ranges from 250 to 350 bpm, producing a sawtooth appearance of the P waves. The atrial rate of 300 bpm usually has a 2:1 conduction to the ventricle, producing a ventricular rate of 150 bpm.
- Supraventricular tachycardia (SVT): SVT is an umbrella term used to describe tachycardias (atrial and/or ventricular rates >100 bpm at rest). Tissue from the bundle of His and above is generally involved in the conduction of the arrhythmia. This does not include atrial fibrillation.
- Premature atrial complex (PAC): Ectopic or irregular heartbeat that occurs prior to the next sinus beat; initiated in the left or right atrium and not by the SA node; PAC is a normal-looking complex except it is premature.
- Ventricular tachycardia (VT): 3 or more consecutive ventricular ectopic beats with a heart rate >100 bpm. Can be sustained (>30 seconds) or nonsustained (<30 seconds). QRS can be monomorphic (QRS complexes are the same) or polymorphic (QRS shape changes from beat to beat).
- Ventricular fibrillation (VF): Rapid, disorganized electrical activity within the ventricle with no discrete QRS complexes; the heart is asystole and there is no pulse.

Common bradyarrhythmias

- Sinus bradycardia: 1:1 relationship between each P wave and QRS complex; SA node fires at a rate ≤60 bpm or less.
- First-degree AV block: PR intervals are equal and longer than 0.20 seconds, but every P wave is conducted to the ventricle.
- Second-degree AV block Type I: Progressive prolongation of the PR interval until a P wave is dropped or not conducted; also called Wenckebach block.
- Second-degree AV block Type II: Constant PR interval until a P wave is not conducted to the ventricle and the QRS complex is skipped or missing; less common but more severe to progress to complete heart block.
- Third-degree AV block: None of the P waves are conducted to the ventricle; P waves have no relationship to the QRS complexes. Also called complete heart block; requires immediate intervention.

ETIOLOGY AND RISK FACTORS

- Structural heart defects
- Cardiac disease (atherosclerosis, ischemia, or infarction)
- Underlying pulmonary disease (chronic obstructive pulmonary disease [COPD], obstructive sleep apnea)
- Congenital or genetic disorder
- Myocarditis
- Rheumatic fever
- Viral infection
- Hypothyroidism
- Advanced liver disease
- Hypothermia
- Medication induced (calcium-channel blockers, beta blockers, or digoxin)

INCIDENCE

- 50% of all people with or without heart disease experience premature ventricular contractions (PVCs) at some point in their life.
- Half of patients with out-of-hospital cardiac arrest present with VF as the first identified rhythm.
- Sudden cardiac death (SCD) accounts for approximately 50% of all cardiovascular deaths, with at least 25% being the first symptomatic cardiac event.

ASSESSMENT

- During the patient interview, ask about tachyarrhythmias and/or palpitations or if symptoms occur with vagal mechanism (straining with bowel movement, vomiting) for bradyarrhythmia.
- Determine if there is a history of underlying heart disease, a family history of heart disease, or a previous history of rhythm disturbance and its treatment.
- Evaluate for coronary risk factors, and inquire about the use of alcohol, tobacco, caffeine,

sympathomimetics (commonly found in over-the-counter cold medicines or diet aids), and prescription medications (e.g., theophylline or thyroid supplements).
- Ask about the use of street drugs, especially those known to be stimulants, such as cocaine, methamphetamines, synthetic cannabinoids (e.g., spice), and synthetic cathinones (known as bath salts).
- Symptoms include palpitations, lightheadedness, dizziness, syncope, dyspnea, or fatigue.
- Serious arrhythmia can result in hemodynamic decompensation, causing hypotension, chest pain, heart failure symptoms, change in level of consciousness, or SCD.
- Perform a thorough physical examination with close attention to the cardiac system.

Patients with cardiac arrhythmia who are hemodynamically unstable, have acute symptoms or signs of myocardial infarction, or who have ischemia need to be transported to the emergency department immediately.

DIFFERENTIAL DIAGNOSES

Tachyarrhythmias

- Sinus tachycardia
- Atrial tachycardia
- Multifocal atrial tachycardia
- Paroxysmal supraventricular tachycardia
- Atrioventricular nodal reentry tachycardia
- Atrioventricular reentry tachycardia
- Wolff-Parkinson-White pattern
- Atrial flutter
- Atrial fibrillation
- Supraventricular tachycardia with aberrancy
- Ventricular tachycardia
- Torsades de pointes
- Brugada syndrome
- Ventricular fibrillation
- Premature atrial complex
- Premature ventricular complex

Bradyarrhythmias

- Sinus bradycardia
- First-degree block
- Second-degree block; Mobitz type 1 or Mobitz type II
- Third-degree AV block
- Left bundle branch block
- Right bundle branch block
- Hemiblocks

DIAGNOSTIC STUDIES

- Vital signs
- 12-lead electrocardiogram (ECG): priority diagnostic study
- 24- to 48-hour Holter monitor if ECG is nondiagnostic
- Complete blood count (CBC)
- Comprehensive metabolic panel (CMP)
- Thyroid-stimulating hormone (TSH)
- Toxicology screening for street drugs and prescription medications such as digoxin if applicable
- Echocardiogram
- Chest X ray

PLANNING/IMPLEMENTATION

- If patient presents to the clinic and is hemodynamically unstable, establish presence of a pulse, perform a quick assessment to determine hemodynamic stability, and obtain ECG. Call 911 if needed for transport to the emergency department.
- If hemodynamically stable, perform diagnostic work-up and refer to cardiology.

NONPHARMACOLOGIC MANAGEMENT

- Eliminating stimulants or offending agent that can provoke the arrhythmia if possible
- Correction of electrolyte imbalance if necessary
- Synchronized cardioversion if indicated for atrial tachyarrhythmia, patient is hemodynamically unstable, and performed in an emergency care setting
- Defibrillation for life-threatening ventricular arrhythmias, performed in an emergency care setting
- Temporary transcutaneous pacemaker for bradyarrhythmia, performed in an emergency care setting
- Implantation of an internal cardioverter-defibrillator for ventricular arrhythmias by cardiology
- Implantation of a permanent pacemaker for bradyarrhythmias by cardiology

PHARMACOLOGIC MANAGEMENT

- Pharmacologic management initiated by cardiology in the emergency or inpatient setting
- Medications used for tachyarrhythmias: beta blockers, calcium-channel blockers, antiarrhythmic agents such as sodium-channel blockers (lidocaine, propafenone), and potassium-channel blockers (sotalol, amiodarone)

CONSULTATION/REFERRAL

- Immediate referral to the emergency department for serious, life-threatening arrhythmias
- Consultation of cardiology or electrophysiology cardiology for diagnosis and management of arrhythmia

FOLLOW-UP

- Follow-up per cardiology or electrophysiology cardiology depending on the course of treatment
- Primary care provider annually for health maintenance, vaccinations, screening tests, and other care

EXPECTED COURSE

- When arrhythmia is appropriately identified and treated, prognosis is good overall.
- Educating patient on medication use, side effects, and timing of doses is important to ensure adherence.
- Family of patient with abnormal heart rhythm should learn cardiopulmonary resuscitation and develop an emergency plan to be prepared for next arrhythmia event.

POSSIBLE COMPLICATIONS

- Sudden cardiac death due to ventricular arrhythmia, especially in patients with underlying cardiac disease
- Worsening of cardiac ischemia or infarction
- Heart failure
- Lethal proarrhythmias: side effect of some antiarrhythmic drugs; should be monitored

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ACUTE CORONARY SYNDROME

DESCRIPTION

Acute coronary syndrome (ACS) is a spectrum of clinical presentations including ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), unstable angina (UA), and noncardiac chest pain. ACS is associated with a mismatch between myocardial oxygen supply and demand that is often related to coronary artery obstruction. The most common presenting symptom is chest pain. The chest pain could also be described as pressure, tightness, or discomfort in the chest, shoulders, arms, neck, back, upper abdomen, or jaw. Chest pain as well as shortness of breath and fatigue should all be considered anginal equivalents. The resulting myocardial ischemia causes damage

and, in some cases, permanent cell death of the cardiac musculature.

ACS Classifications

UA and NSTEMI are now classified as non-ST elevation acute coronary syndrome (NSTE-ACS).

Classification of ACS is based on ECG changes and the presence or absence of cardiac markers in blood. Distinguishing between NSTEMI and STEMI is useful because the prognosis and treatment for these conditions are different.

- **Unstable angina** (UA): result of acute coronary artery obstruction without myocardial infarction and is defined as:
  - Rest angina that is prolonged (usually more than 20 minutes)
  - New-onset angina that limits physical activity
  - Increasing angina that is more frequent, lasts longer, or occurs with less exertion than previous angina
- **Non-ST-segment elevation MI** (NSTEMI): myocardial necrosis (evidenced by cardiac markers in blood; high-sensitivity cardiac troponin) without acute ST-segment elevation or Q waves. ECG changes such as ST-segment depression, T-wave inversion, or both, may be present.
- **ST-segment elevation MI** (STEMI): myocardial necrosis with ECG changes showing ST-segment elevation not quickly reversed by nitroglycerin, or showing new left bundle branch block. Q waves may be present. Cardiac markers and high-sensitivity cardiac troponin are elevated.

ETIOLOGY

- Coronary thrombosis
- Plaque rupture
- Coronary artery vasospasm
- Noncoronary causes of myocardial oxygen supply–demand mismatch such as hypotension, severe anemia, and severe aortic stenosis

INCIDENCE

- Chest pain accounts for more than 6.5 million emergency department visits per year and more than 4 million outpatient visits per year.
- Median age at time of presentation is 68 years.
- More women than men present with symptoms of ACS, but women may experience symptoms other than chest pain—such as nausea, vomiting, and shortness of breath—and therefore may have delayed diagnosis.

RISK FACTORS

- Family history of premature coronary artery disease (before age 60)
- Hyperlipidemia
- Age (men older than age 40 and postmenopausal women)
- Cigarette smoking
- Hypertension
- Sedentary lifestyle
- Obesity, especially central adiposity
- Diabetes mellitus, metabolic syndrome
- Stressful lifestyle
- Preeclampsia, gestational diabetes, or pregnancy-induced hypertension
- Evidence of subclinical atherosclerosis: coronary calcification, carotid plaque
- Autoimmune collagen vascular disease: lupus, rheumatoid arthritis

ASSESSMENT

- Pain, pressure, squeezing or a burning sensation across the precordium, with possible radiation to neck, shoulder, jaw, back, upper abdomen, or either arm; symptoms last longer than 10 minutes; especially concerning if unrelieved by nitroglycerin
- Escalating severity of angina
- Nausea, vomiting from vagal stimulation
- Diaphoresis from sympathetic discharge
- Severe fatigue, weakness, syncope
- Feeling of impending doom
- Hypertension/hypotension
- Palpitations
- Dyspnea that resolves with pain or rest
- Decreased exercise tolerance
- Pulmonary edema and/or other signs of left-sided heart failure
- Jugular venous distention
- A third heart sound (S_3) and, frequently, a fourth heart sound (S_4)
- A systolic murmur related to dynamic obstruction of the left ventricular outflow tract
- Rales on pulmonary examination (suggestive of left ventricular dysfunction or mitral regurgitation)

In comparison to men, women often receive less timely and appropriate care, despite presenting with chest pain. Common symptoms of ACS in women are severe fatigue, nausea, and pain in the jaw, arm, or back. ACS should be ruled out as a cause of these symptoms in women first.

DIFFERENTIAL DIAGNOSES

- Aortic dissection
- Esophageal spasm, gastroesophageal reflux disease (GERD), gastritis, peptic ulcer
- Cholecystitis, pancreatitis
- Pericarditis, myocarditis
- Costochondritis
- Pulmonary emboli, pneumonia, pleurisy, tension pneumothorax, rib fracture
- Cervical disc disease
- Neuropathic pain, herpes zoster
- Anxiety disorder/panic attack
- Somatization/psychogenic pain disorder

DIAGNOSTIC STUDIES

Electrocardiography (ECG) is the most important diagnostic test for angina. ECG changes during angina episodes may include the following:

- Transient ST-segment elevations
- Dynamic T-wave changes: inversions, normalizations, or hyperacute changes
- ST depressions: junctional, downsloping, or horizontal
- Q waves

- **ECG:** Should be obtained within 10 minutes of arriving to a medical facility, whether in the outpatient setting or the emergency department.
- **Cardiac troponin (cTn I or T):** High-sensitivity cTn is the preferred biomarker to detect or exclude myocardial injury. A cTn concentration >99th percentile upper reference limit, which is assay-dependent, is an indicator of myocardial injury. Creatine kinase-MB (CK-MB) or myoglobin in addition to cTn is not beneficial for evaluation of patients with chest pain.
- **Lab studies:** Prothrombin time (PT), international normalized ratio (INR), CBC, glucose, metabolic and lipid panels, TSH, and as indicated by history
- **Chest X ray:** Identify cardiomegaly, heart failure (HF), and pulmonary diseases that may mimic or exacerbate cardiac disease.
- **Angiography:** Demonstrates narrowed coronary artery by atherosclerotic lesion
- **Echocardiogram:** 2D and M mode
- **Radiology imaging:** Contrast-enhanced chest computed tomography (CT) scan or cardiovascular magnetic resonance imaging (MRI) to differentiate myocardial infarction (MI) from aortic dissection

PLANNING/IMPLEMENTATION

- Decrease coronary artery disease risk factors by managing hypertension, hyperlipidemia, and diabetes mellitus; encourage smoking cessation and increased physical activity.
- Modify coronary artery disease risk factors (pre- and post MI)
  - Consumption of diet low in saturated fat
  - Smoking cessation
  - Regular aerobic exercise (initiate cardiac rehabilitation)
  - Stress reduction, management
- Medication adherence
- Family history is a risk factor but is not modifiable.
- Calculating a risk score can be useful to help focus on lifestyle modifications using the Framingham risk score or other risk score calculators.

NONPHARMACOLOGIC MANAGEMENT

- Immediate referral to nearest emergency department (give aspirin stat, O_2, nitroglycerin, and transport)
- Stabilize patient to provide immediate relief of ischemia and prevent MI and death
- For unstable angina and NSTEMI: angiography within 24–48 hours of hospitalization to identify coronary lesions requiring percutaneous coronary intervention (PCI; stent) or coronary artery bypass graft surgery (CABG); fibrinolysis not helpful
- For STEMI: emergency PCI needed when door-to-balloon-inflation time is <90 minutes; fibrinolysis if timely PCI is not available

PHARMACOLOGIC MANAGEMENT

- Initial inpatient management can include antiplatelet agents, anticoagulants, beta blockers, angiotensin-converting enzyme inhibitors, nitroglycerin, oxygen, and statin therapy

Acute Coronary Syndrome Pharmacologic Management

Nitrates

General Comments:

- **MOA:** Stimulates cGMP production, resulting in vascular smooth muscle relaxation
- Nitrates do not improve mortality but provide symptomatic relief through coronary vasodilation, improved collateral blood flow, decrease in preload, and decrease in afterload
- **Indication:** Treatment and prevention of angina
- **Side effects:** Headaches, flushing, nausea, hypotension
- **Drug/drug:** Contraindicated with concomitant use of phosphodiesterase type 5 inhibitor as it will cause severe hypotension, syncope, or MI
- **Monitor:** Blood pressure during administration
- **Pregnancy:** Should be given only if clearly indicated; limited human subject data

Generic/Brand	Dosage: Adult	Dosage: Pediatric	Comments
Short-Acting Nitrates: **nitroglycerin/**Nitrostat, Nitrolingual, Nitro-Dur, Nitro-Bid *Nitrostat:* *Tabs:* 0.3 mg, 0.4 mg, 0.6 mg *Nitrolingual:* *Spray:* 0.4 mg/spray ***Long-Acting Nitrates:*** *Nitro-Dur:* *Patch:* 0.1 mg/hr, 0.2 mg/hr, 0.3 mg/hr, 0.4 mg/hr, 0.6 mg/hr, 0.8 mg/hr	*Initial:* 1 sublingual at onset; may repeat in 5 min *Max:* 3 sublingual tabs over 15 min **OR** **Spray:** *Initial:* 1–2 sprays at onset; may repeat in 5 min *Max:* 3 sprays over 15 min **Topical Ointment:** *Initial:* ½ inch AM, repeat in 6 hr *Max:* 2 inches AM, repeat in 6 hr **Topical Patches:** *Initial:* 0.2–0.4 mg/hr patch worn daily for up to 12 hr, then removed *Max:* 0.8 mg/hr patch worn daily for up to 12 hr, then removed	Not applicable	• Acute angina pain—unrelieved after 15 minutes, even with nitroglycerin, warrants evaluation in emergency department
isosorbide mononitrate/Imdur *Immediate-release tabs:* 10 mg, 20 mg *Extended-release tabs:* 30 mg, 60 mg, 120 mg	**Immediate Release:** *Initial:* 20 mg BID *Max:* 40 mg/day **Extended Release:** *Initial:* 30–120 mg/day *Max:* 240 mg/day		
ranolazine/Ranexa *ER tabs*: 500 mg, 1000 mg	*Initial:* **500 mg BID** *Max:* **2000 mg/day**	Not applicable	Caution use if CrCl <60 Can prolong QT interval

(*continues*)

Acute Coronary Syndrome Pharmacologic Management *(continued)*

Beta Blockers

General Comments:

- **MOA:** Selectively antagonizes beta-1 adrenergic receptors inhibiting the chronotropic and inotropic effects on the heart which reduces oxygen demand and ventricular wall tension, reduces heart rate and blood pressure.
- End in "lol"
- **Indication:** Prevention of cardiovascular event; post-MI treatment, coronary artery disease, tachycardia; decreases mortality and adverse cardiovascular events
- **Side effects:** May cause hypotension, bradycardia, fatigue, dizziness, possible bronchospasm in patients with asthma
- **Contraindications:** Systolic blood pressure (BP) <90 mm Hg, cardiogenic shock, severe bradycardia, second- or third-degree heart block, asthma, or emphysema that is sensitive to beta blockers, peripheral vascular disease, uncompensated heart failure
- **Drug/drug:** CYP2D6 substrate
- **Monitor:** BP, heart rate (Do not stop treatment abruptly; taper gradually over 1–2 weeks.)
- **Pregnancy:** May restrict intrauterine growth especially in second and third trimesters; no known risk of teratogenicity

Generic/Brand	Dosage: Adult	Dosage: Pediatric	Comments
carvedilol/Coreg *Tabs:* 3.125 mg, 6.25 mg, 12.5 mg, 25 mg *ER caps:* 10 mg, 20 mg, 40 mg, 80 mg	*Initial:* 3.125–6.25 mg BID *Max:* 50 mg daily *ER caps initial:* 20 mg/day *ER caps max:* 80 mg/day	Not applicable	
metoprolol tartrate/Lopressor *Tabs:* 25 mg, 37.5 mg, 50 mg, 75 mg, 100 mg	*Initial:* 25–50 mg BID *Max:* 400 mg/day	Not applicable	
metoprolol succinate/Toprol XL *ER tabs:* 25 mg, 50 mg, 100 mg, 200 mg	*Initial:* 25 mg/day *Max:* 400 mg/day	Not applicable	• Indicated for use in patients with MI and heart failure

Angiotensin-Converting Enzyme (ACE) Inhibitors

General Comments:

- **MOA:** Cause vasodilation by the inhibition of angiotensin-converting enzyme, which blocks conversion of angiotensin I to angiotensin II, leading to increased plasma renin activity and reduced aldosterone secretion
- **Indication:** Treatment of acute MI, hypertension (HTN), systolic heart failure
- End in "pril"
- **Side effects:** Dry cough; hyperkalemia, hypotension, elevated BUN and creatinine; angioedema (rare, more common in Blacks); neutropenia
- **Drug/drug:** Insulin or oral antidiabetic agents may increase hypoglycemia (ACEIs enhance insulin sensitivity).
- **Dosage adjustments:** For CrCl <30 mL/min/1.73 m^2
- **Contraindications:** Use with caution in abnormal renal function; avoid with aortic valve stenosis, hypovolemia; renal artery stenosis
- **Monitor:** Potassium, blood urea nitrogen (BUN)/creatinine
- **Pregnancy:** Contraindicated; fetal toxicity

Angiotensin-Converting Enzyme (ACE) Inhibitors			
Generic/Brand	**Dosage: Adult**	**Dosage: Pediatric**	**Comments**
benazepril/Lotensin *Tabs:* 5 mg, 10 mg, 20 mg, 40 mg	*Initial:* 10 mg/day *Max:* 40 mg/day (1 or 2 divided doses)	Not applicable	
captopril/Capoten *Caps:* 12.5 mg, 25 mg, 50 mg, 100 mg	*Initial:* 6.25 to 12.5 mg TID *Max:* 50 mg TID as tolerated	Not applicable	
lisinopril/Zestril *Tabs:* 2.5 mg, 5 mg, 10 mg, 20 mg, 30 mg, 40 mg	*Initial:* 2.5–10 mg/day *Max:* 40 mg/day	Not applicable	

HMG-CoA Reductase Inhibitors (Statins)

General Comments:

- **MOA:** Inhibits 3-hydroxy-3-methyglutaryl-coenzyme A (HMG-CoA) reductase, inhibiting cholesterol synthesis; lowers total cholesterol, low-density lipoprotein (LDL0, and triglyceride concentrations
- **Indication:** Hyperlipidemia; mixed dyslipidemia, hypertriglyceridemia. Primary prevention of atherosclerotic cardiovascular disease (ASCVD); Secondary prevention in patients with ASCVD
- End in "statin"
- **Side effects:** Myopathy, hepatotoxicity, diabetes mellitus, increased serum transaminases, rhabdomyolysis (rare)
- **Contraindications:** Patients with active liver disease and unexplained elevated liver function tests (LFTs)
- **Drug/drug:** Interactions with CYP3A4 substrate, P-gp substrate, CYP3A4 inhibitor, weak
- **Monitor:** Creatinine (Cr) and LFTs at baseline, then as clinically indicated; creatine kinase (CK) at baseline if myopathy risk, then as clinically indicated
- **Pregnancy:** Contraindicated

Generic/Brand	Dosage: Adult	Dosage: Pediatric	Comments
atorvastatin/Lipitor *Tabs:* 10 mg, 20 mg, 40 mg, 80 mg	*Initial:* 10 mg/day *Max:* 80 mg/day	Not applicable	• No dose adjustment for impaired renal function; monitor in combination with CYP3A4 inhibitors (i.e., ciprofloxacin, clarithromycin, verapamil, etc.)
rosuvastatin/Crestor *Tabs:* 5 mg, 10 mg, 20 mg, 40 mg	*Initial:*10–20 mg/day *Max:* 40 mg/day	Not applicable	• Adjust dose for CrCl <30 mL/min/1.73 m^2: 5–10 mg once daily. Monitor in combination with CYP2C9 drugs (i.e., ketoconazole, metronidazole, sulfamethoxazole-trimethoprim
simvastatin/Zocor *Tabs:* 5 mg, 10 mg, 20 mg, 40 mg, 80 mg	*Initial:* 10–20 mg/day *Max:* 40 mg/day	Not applicable	• No dose adjustment for impaired renal function; max dose 40 mg/day; avoid with gemfibrozil

(*continues*)

Acute Coronary Syndrome Pharmacologic Management *(continued)*

Antiplatelet Agents

General Comments:

- **MOA:** Aspirin—irreversibly inhibits cyclooxygenase-1 and -2 in the prostaglandin synthesis pathway (PGH2); others listed—selectively inhibit P2Y12 component of ADP receptors to prevent platelet aggregation.
- **Indication:** Acute coronary syndrome, primary prevention of CAD, mechanical heart valves, stable angina
- **Contraindications:** Large esophageal varices, stroke within 2 years, history of intracranial hemorrhage, significant thrombocytopenia, end-stage renal disease on hemodialysis, severe hypertension (BP >200/110)
- **Side effects:** Hemorrhage, thrombocytopenia, chronic gastritis, GI Bleeding, ecchymosis, hematuria
- **Drug/drug:** See individual drug comments
- **Monitor:** Initial assessment for bleeding risk, renal function; no routine monitoring required. Normal platelet function returns 7-10 days after discontinuation
- **Pregnancy:** Risk of fetal harm not expected; consider discontinuing 5–7 days prior to labor and delivery

Generic/Brand	Dosage: Adult	Dosage: Pediatric	Comments
acetylsalicylic acid/ aspirin *Tabs:* 81 mg, 325 mg, 500 mg	*Initial:* 162–325 mg once, then 81–325 mg/day	Not applicable	• Consider enteric-coated medication in patients with prior gastric irritation • Monitor for aspirin-induced asthma • Hold 7-10 days prior to surgery
clopidogrel/Plavix *Tabs:* 75 mg, 300 mg	*Initial:* 300 mg once, then 75 mg/day	Not applicable	• CYP2C19 inhibitors may reduce concentrations of active metabolite • CYP2C19 polymorphisms may affect clopidogrel efficacy. Hold 5 days prior to surgery
prasugrel/Effient *Tabs:* 5 mg, 10 mg	*Initial:* 60 mg once, then 10 mg/day	Not applicable	• Reduce maintenance dose to 5 mg in patients <60 kg. • Contraindicated in patients with history of stroke, TIA • Not recommended in patients ≥75 years • Hold 7 days prior to surgery
ticagrelor/Brilinta *Tabs:* 60 mg, 90 mg	*Initial:* 90 mg BID × 12 months, then 60 mg BID	Not applicable	• When used in combination with aspirin, aspirin maintenance dose should not exceed 81 mg • CYP3A4 drug interactions • Monitor closely for dyspnea, bradyarrhythmia (including ventricular pauses) • Hold 3–5 days before surgery

CONSULTATION AND REFERRAL

- Consult cardiology specialist to manage inpatient and outpatient care

FOLLOW-UP

- Per cardiologist
- Encourage participation in cardiac rehab program
- Primary care provider annually for health maintenance, vaccinations, screening tests, and other care

EXPECTED COURSE

- Depends on severity, underlying coronary artery disease, age, response time to emergency facility
- Post–myocardial infarction care

- Dual antiplatelet therapy (aspirin + clopidogrel, prasugrel, or ticagrelor) for at least 12 months if no bleeding issues, or as directed by cardiologist
- Beta blocker therapy for secondary prevention of myocardial infarction or if left ventricular dysfunction
- Assess and treat for anxiety or depression post myocardial infarction
- Resume sexual activity when physically and mentally ready; there is no recommendation related to waiting a certain amount of time
- Wait 2 weeks before resuming air travel
- May receive vaccinations for disease prevention, such as influenza or pneumonia, at any time

POSSIBLE COMPLICATIONS

- Death (may be sudden)
- Pulmonary edema
- Heart failure
- Dysrhythmias
- Left ventricular aneurysm, thrombus
- Deep vein thrombosis (DVT), pulmonary embolism
- Mitral regurgitation
- Ventricular rupture
- Acute mitral regurgitation
- Constipation
- Urinary retention
- Bleeding
- Anxiety, insomnia

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ANGINA, STABLE (Angina Pectoris)

DESCRIPTION

Angina pectoris is defined as substernal chest pain or chest pressure—that is, tightness or discomfort in the chest, shoulders, arms, neck, back, upper abdomen, or jaw—that occurs when myocardial oxygen demand is greater than myocardial oxygen supply, resulting in myocardial ischemia. Other symptoms considered anginal equivalents are shortness of breath and fatigue. Patients with these symptoms should seek treatment immediately. Chest pain is the most common symptom for men and women, and women are more likely to present with accompanying symptoms such as nausea and shortness of breath.

Stable angina is typically provoked by physical exertion or emotional stress, and is usually relatively predictable. It is usually relieved by rest or nitroglycerin.

Unstable angina (UA) is the result of acute coronary artery obstruction without myocardial infarction (MI). It is defined as:

- Rest angina that is prolonged (usually more than 20 minutes)
- New-onset angina that limits physical activity
- Increasing angina that is more frequent, lasts longer, or occurs with less exertion than previous angina