3

Cardiovascular Disorders

Cardiovascular disease is the number one cause of death in the United States, killing more than 1,000 patients every day. Recent advances in our understanding of the pathogenesis of some of these disorders, as well as new therapeutic techniques, have greatly improved our ability to treat these patients.

■ I. ISCHEMIC HEART DISEASE

I. Unstable Angina Pectoris

A. Definition. Angina pectoris is chest discomfort that occurs when myocardial oxygen demand exceeds supply. Unstable angina is the manifestation of coronary artery disease that falls somewhere between angina pectoris and myocardial infarction. It is characterized by:

1. Recent onset of ischemic chest pain
2. Increase of severity, duration, or frequency or chronic anginal chest pain, or
3. Angina pain that occurs at rest

The term *acute coronary syndrome* is used to describe the spectrum of acute unstable manifestations of coronary disease including unstable angina and myocardial infarction (Prinzmetal angina is also included here).

B. Risk Factors

1. Increased age
2. Male gender
3. Family history
4. Smoking history
5. Hypertension
6. Diabetes mellitus
7. Dyslipidemia

C. Pathophysiology. Coronary artery atherosclerosis most commonly underlies unstable angina. Unstable atheromatous plaque with the development

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of thrombus is thought to cause the transformation of a stable angina picture into that of unstable angina. Up to 7–9% of hospitalized patients with unstable angina will develop myocardial infarction (MI). Coronary artery spasm, hemorrhage, and increased platelet aggregation also play a role in this syndrome.

D. Clinical Presentation. Substernal pain (pressure, heaviness, tightness, and/or burning) that is new in onset, prolonged, or occurring at rest is common. Shortness of breath, diaphoresis, nausea, and pain in the left arm may be present. On occasion, back and jaw pains are the cardinal features. However, many patients may have unusual symptoms, such as abdominal or back pain.

E. Differential Diagnosis
   1. Acute MI
   2. Acute aortic dissection
   3. Pulmonary disorders including pulmonary embolism, pleurisy, pneumothorax, and pneumonia
   4. Peptic ulcer disease, pancreatitis, esophageal reflux and spasm, cholecystitis, and biliary colic
   5. Musculoskeletal conditions, chest wall pain, costochondritis
   6. Herpes zoster

F. Diagnostic Studies
   1. Diagnosis is made primarily by history.
      Physical examination is usually unraveling. However, one should look for evidence of dyslipidemia, hypertension, and congestive heart failure (CHF) as well as the presence of murmurs.
   2. Electrocardiograms (ECGs) during episodes of pain may show tangent repolarization abnormalities. Normal tracing may be present also. Chest radiographs should be obtained and may show evidence of cardiomegaly and/or pulmonary edema.

G. Treatment. A patient with unstable angina should be placed on bed rest in the intensive care unit (ICU).
   1. Pharmacotherapy
      a. Nitrates
         This class of agents causes relaxation of vascular muscle and venodilatation. Diastolic ventricular wall tension is reduced by decreased venous return following administration of these agents, thus decreasing myocardial oxygen consumption. Therapy may be started with sublingual nitroglycerin, 0.4 mg (1/150) q5 min × 3. Topical therapy with 2% nitroglycerin ointment ($^1/_2$–2 in.) q6 h may also be instituted. Recurrent bouts of pain should prompt institution of intravenous nitroglycerin beginning at 10 μg/min and titrated upward to the desired effect (absence of pain, systolic blood pressure no less than 90–100 mmHg). These agents may cause headache and are commonly associated to tachyphylaxis.
      b. Beta-adrenergic Blocking Agents
         These agents reduce myocardial oxygen demand by decreasing heart rate, blood pressure, and contractility. They also decrease the systemic vascular resistance and cardiac output. Patients with bradycardia of <50 beats per minute, systolic blood pressure <100 mmHg, chest x-ray evidence of pulmonary edema, second- or third-degree atrioventricular (A-V) block, or a PR interval ≥0.24 s, ejection fraction below

Table 3.1. Commonly Used Beta-Blockers in Unstable Angina

Drug	*Acute intravenous dose*	*Oral dose*
Atenolol	5 mg over 5 min, repeat × 1 after 10 min	50 mg q12 h or 100 mg q24 h
Metoprolol	5 mg q5 min × 3 doses	50 mg q6 h; after 48 h, 100 mg q12 h
Labetalol	20–80 mg bolus 2 mg/min infusion, titrate to effect	100 mg bid
Nadolol		40–80 mg qid
Propanolol	0.5–3 mg slow IV bolus, repeat as necessary	40–800 mg/d given bid to qid

Table 3.2. Oral Calcium-Channel Antagonists Used in Unstable Angina

Agent	*Oral dose*
Verapamil	240–480 mg/day
Diltiazem	180–360 mg/day

25%, and those with bronchospastic lung disease should not receive these agents. Intravenous and oral beta-blocker dosage schedules are depicted in Table 3.1.

c. Calcium-Channel Antagonists

These agents decrease myocardial ischemia by coronary and peripheral vasodilatation, negative effects on contractility and also heart rate. They are not first-line agents but may be used in refractory cases.

Oral doses of representative calcium channel antagonists are noted in Table 3.2. Because of their negative inotropic effect, these agents should be administered in patients with congestive heart failure very cautiously.

d. Aspirin

Aspirin has been shown to decrease the rate of myocardial infarction and coronary death in patients with unstable angina. Various dosing regimens ranging from 81 to 325 mg qd have been advocated. Some studies have demonstrated a 50% reduction in cardiovascular death or nonfatal MI.

e. Anticoagulants

Intravenous heparin has been a useful adjunct in unstable angina, demonstrating reduced incidence of MI and refractory angina in some studies. Intermediate- and high-risk presentations are usually treated with heparin. Doses of 60 IU/kg intravenous (IV) bolus followed by 12 IU/kg/h infusion are recommended. Enoxaparin or dalteparin are low molecular weight heparins that may be superior to unfractionated heparin dosed at 1 mg/kg q12 h. Fondaparinux, a synthetic heparin

pentasaccharide is used in some patients. If an invasive strategy is planned, a direct thrombin inhibitor such as bivalirudin can also be used.

f. Thrombolytic Therapy
Despite the benefits in acute myocardial infarction, thrombolytic therapy has not been shown to improve outcome in patients with unstable angina.

g. Glycoprotein IIb/IIIa Receptor Inhibitors
These agents block the receptors that lead to platelet aggregation. Two relatively short-acting (4–8 h) agents (eptifibatide, tirofiban) and one longer acting antibody (abciximab) are available. These agents are beneficial during percutaneous coronary interventions, and the shorter acting agents are approved for use in non-Q MI and unstable angina being medically managed. Decreased combined endpoints of death, infarction, and urgent intervention have been reported. These agents should be administered to high-risk patients (ST depression 1 mm, persistent or recurrent symptoms, widespread electrocardiographic [ECG] abnormalities, depressed left ventricular [LV] function, positive cardiac markers).
(1). Eptifibatide, 180 μg/kg IV bolus followed by a 2-μg/kg/min infusion. A double-bolus regimen has been shown to improve platelet inhibition in some clinical studies (180 mg/kg × 2, 10 min apart).
(2). Tirofiban, 0.4 μg/kg/min × 30 min followed by 0.1 μg/min infusion.
(3). Abciximab, 0.25 mg/kg IV bolus followed by 0.125 μg/kg/min infusion.

h. Clopidogrel—Given (75–300 mg loading) in combination with aspirin lead to benefit.

i. Morphine—Intravenous morphine sulfate (2–4 mg initially) should be given for the relief of chest pain and anxiety.

j. Statins—Many authors recommend early administration of these agents (i.e., atorvastatin 80 mg/day).

k. ACE inhibitors and Angiotensin II receptor blockers are utilized also in patients with concomitant diabetes, heart failure, and a documented ejection fraction <40%.

2. Nonpharmacologic Therapy
Every patient with angina must be placed on supplemental oxygen. Persistent chest pain despite maximal therapy with nitrates, beta-blockers, aspirin, heparin, etc., may require early cardiac catheterization, with the view toward potential mechanical intervention (percutaneous transluminal coronary angioplasty (PTCA) or coronary bypass surgery). Intraaortic balloon pump (IABP) insertion should be performed with the goal of stabilizing the patient when needed. The IABP relieves pain and may provide relative stability for evaluation before intervention.

II. Myocardial Infarction

A. Definition. Myocardial infarction (MI) is necrosis of the cardiac muscle resulting from insufficient supply of oxygenated blood.

1. Q-Wave MI
Q-wave MI presents with ST-segment elevation and the subsequent development of pathologic Q waves in the ECG.

2. Nonacute MI
   More than 50% of acute MIs in the United States do not present with ST-segment elevation but rather have nonspecific ECG changes or even normal ECGs.

B. Pathophysiology
1. MI is nearly universally the result of coronary artery atherosclerosis.
2. Atherosclerotic lesions reduce and limit the flow through coronary arteries, resulting in ischemic myocardial cells.
3. The formation of thrombi plays a significant role in acute MI and almost all ST-segment elevation infarcts will have an occlusive thrombus in the infarct-related artery if examined early enough in the course of the MI.
4. Occlusion of the right coronary artery (RCA) generally results in inferior/posterior MI.
5. Occlusion of the left anterior descending artery (LAD) generally leads to anterior infarctions, while blockage of the left circumflex artery (LCA) results in lateral and/or inferior/posterior MI.
6. Spasm of the coronary arteries may also play a role in MI. As many as 2% of all MI patients, a significantly higher percentage of those patients who are less than 35 years of age, will have angiographically normal coronary arteries, and presumably spasm is a significant pathophysiologic event.

C. Risk Factors. Risk factors for coronary artery disease including MI are age, male gender, family history, smoking, hypertension, elevated cholesterol, and diabetes mellitus. Cocaine use is a significant risk factor for MI.

D. Clinical Presentation
1. Patients present with chest pain (typically substernal) lasting 30 min or longer, which is unrelieved by rest or nitroglycerin, and pain that may radiate from left or right arm into the jaw. The pain is typically nonpleuritic and may be associated with dyspnea, diaphoresis, nausea, or vomiting.
2. As many as 25% of all MIs are painless.
3. "Burning" discomfort is as predictive of acute MI as pressure-type discomfort.

E. Physical Findings
1. Skin may be cool. Diaphoresis may be evident.
2. Heart may demonstrate an apical systolic murmur, mitral regurgitation secondary to papillary muscle dysfunction. S3 or S4 gallop sounds may be present.
3. Advanced signs of congestive heart failure (CHF) with pulmonary edema may be present with rales auscultated in lung fields.
4. In many instances the physical examination will not reveal specific abnormalities.

F. Diagnostic Studies
1. The diagnosis of MI must be presumptive, based on history, physical examination, and ECG.
2. Electrocardiogram (see Table 3.3)
   a. Q-Wave MI: The classic description of the evolution of Q-wave MI includes the following:
      (1). ST-segment elevation is indicative of an area of injury.
      (2). T-wave inversion, a sign of ischemia.

Table 3.3. ECG Localization of Infarcts

Infarct location	*ECG abnormality*
Anterior	V_1–V_4
Anteroseptal	V_1–V_2
Anterolateral	I, aVL, V_4, V_5, V_6
Lateral	I and AVL
Inferior	II, III, aVF
Posterior	R > S in V_1

(3). Q waves indicate areas of infarction. Development of Q waves may be early or may not occur for several days during the evolution of a MI.

b. Non Q-wave infarction: ST-segment depression and T-wave inversion may be seen.

3. Enzyme Studies
Necrotic heart muscle cells release enzymes into the bloodstream. Classically, creatine kinase (CK or CPK) has been used in laboratory diagnosis of MI.
   a. CK becomes elevated within 24 h. CK is also present in the skeletal muscle and brain and thus may be released in other clinical conditions. To increase specificity, the assay of the MB isoenzyme of CK is used. This enzyme is found primarily in the myocardium.
   b. Cardiac-specific Troponin I and Troponin T are regulatory components of the contractile apparatus of the heart. These proteins are very specific for myocardial injury and are released into the blood in the hours following myocardial infarction. They remain elevated for several days and are thus quite useful in patients presenting late after infarct. Troponin elevation, however, can occur in the absence of MI in patients with renal dysfunction.
   c. CK-MB Subforms: The MB isoenzyme of CK exists in only one form in the myocardial cell. After it is released into the blood stream, enzymatically mediated cleavage of a terminal lysine residue occurs, creating two subforms of CK-MB. The ratio of the freshly released CK-MB to the old cleaved CK-MB is a very sensitive and specific early marker of myocardial injury.
4. Nuclear Medicine Techniques
Thallium 201 is taken up by perfused viable cardiac myocytes and may indicate areas of infarction by the presence of "cold spots". Unfortunately, this technique may not distinguish between acute MI and previous scar. Technetium 99 (Tc^{99}) results in "hot spots" as the tracer accumulates in damaged myocardial cells.
5. Other diagnostic studies in patients with suspected MI that should be obtained include blood counts, electrolytes, glucose, blood urea nitrogen (BUN) and creatinine, and lipid profile.

G. Treatment of Acute MI. Several goals exist in the management of acute ST-elevation MI (STEMI): Minimizing the amount of infarcted myocardium, optimizing function, and controlling the complications of acute MI.
1. Patients with suspected MI should have continuous ECG monitoring and an IV line established. They should also receive supplemental oxygen to maintain adequate oxygen saturation.

2. If the clinical condition of the patient permits, sublingual nitroglycerin (0.4 mg q5 min × 3) should be given to help differentiate those patients who may be suffering from angina rather than MI.
3. Aspirin (ASA) should be given to *all* patients without contraindications (160–325 mg PO).
4. Thrombolytic Therapy.
   a. Patients with ST-segment elevation without contraindications for thrombolytic therapy and who present within 6–12 h of the onset of their symptoms should be considered for thrombolytic therapy. If a cardiac catheterization laboratory is available on-site, we recommend primary percutaneous coronary intervention (see below) for all patients with an STEMI. It is presumed that the procedure will be done expeditiously (door-to-balloon time less than 60 min) in such hospitals.
   b. Patients presenting perhaps up to 24 h may also be considered for thrombolysis, as some studies have reported improved outcomes (ISIS II trial LATE trial).
   c. Guidelines for Thrombolytic Agent Administration
      (1). Symptoms suggestive of acute MI not resolved with sublingual nitroglycerin, lasting 20 min and <12 h
      (2). ST-segment elevation in two or more contiguous ECG leads or left bundle branch block not known to be old.
      (3). Exclusion Criteria
         (a). Bleeding diathesis
         (b). Active gastric or duodenal ulcers
         (c). Significant surgery within 3 weeks
         (d). Severe trauma within 6 months
         (e). History of cardiovascular accident (CVA) within 1 year or other central nervous system (CNS) processes or hemorrhage with a potential for bleeding
         (f). Severe, poorlycontrolled hypertension (180/110)
         (g). Poor underlying prognosis (i.e., malignancy) where risk/benefit assessment may not favor treatment
      (4). Thrombolytic Agents
         (a). Tissue plasminogen activator (rt-PA, Activase) dosage 100 mg (accelerated dosing improves patency rates without increasing complications). Give 15 mg as an IV bolus followed by 50 mg infused over 30 min and the remaining 35 mg infused over 60 min. Concomitantly administer heparin intravenously (HART trial evidence suggests unacceptable reocclusion rates if rt-PA is given without heparin). ASA should be given as well.
         (b). Streptokinase (Streptase)
         1.5 million units IV over 1 h. Because of the systemic state induced by streptokinase and high levels of fibrin-split products, the need for heparin therapy with streptokinase has been questioned.
         (c). Anistreptolase anisoylated (plasminogen/streptokinase activator complex, Eminase). This drug is given as a bolus of 30 U over 5 min. Like streptokinase, heparin therapy is uncertain.
         (d). Reteplase: This agent is a modified rt-PA. In Gusto III reteplase had similar efficacy to rt-PA. It is given as two IV boluses of

10 U over 2 min, 30 min apart. Heparin and ASA should be used.

(e). Tenecteplase (TNK): This is a modified rt-PA that is given as a single IV bolus (0.5 mg/kg). In ASSENT-II, this drug was equivalent to rt-PA in overall mortality, with fewer rates of some bleeding complications. Heparin and ASA should be used.

5. Percutaneous Transluminal Coronary Angioplasty (PTCA)/Percutaneous Coronary Intervention (PCI)
   PTCA/PCI is an excellent alternative revascularization technique where available quickly (door-to-needle insertion time of less than 30 min). This should be strongly considered in patients with contraindications to thrombolytic therapy. If skilled operators and facilities for rapid institution are available, the outcome of patients with primary PTCA appears equivalent to or better than that obtained with thrombolytic therapy.
6. Beta-blockers
   Beta-blockers are useful in preventing tachydysrhythmias and in reducing myocardial oxygen consumption. Early intravenous beta-blockade followed by oral maintenance therapy reduces recurrent ischemia and infarction, even in patients receiving thrombolytic therapy. Patients without contraindications should receive these agents, as they also prevent recurrent ischemia and life-threatening ventricular dysrhythmias. (See Table 3.1.)
7. Angiotensin-Converting Enzyme (ACE) Inhibitors
   ACE inhibitors in the setting of large acute MI may have an impact on LV remodeling and improve survival in patients with LV dysfunction. These drugs should probably not be administered in the first few hours after infarction.
8. Patients should be classified clinically for prognosis as well as to determine therapy (see Tables 3.4 and 3.5).
9. Additional management of patients may be based on the hemodynamic subset in which they fall.
   a. Uncomplicated MI
      (1). In addition to the therapeutic regimens mentioned above, IV nitroglycerin should be used in pain control. Clinical studies have suggested that mortality and infarct size may be reduced by the use of nitrates. Therapy should be started at 10 μg/min and increased until the patient is free of pain, or the systolic blood pressure falls below 100 mmHg, or a maximal dose of approximately 200 μg/min has been achieved.

Table 3.4. Killip Classification of Acute MI

Class I	No heart failure: mortality <10%.
Class II	Mild heart failure: mortality 10–20%.
Class III	Severe heart failure: rales 50% of lung fields. Mortality 35–50%.
Class IV	Cardiogenic shock: mortality 80%.

Adapted from Killip PT: *Am J Cardiol* 1967;20:457.

Table 3.5. Hemodynamic Subsets After Acute MI

Subset	*Cardiac index (L/m^2)*	*Wedge pressure (torr)*
No pulmonary congestion or peripheral hypoperfusion.	2.7 + 0.5	12 ± 7
Isolated pulmonary congestion.	2.3 ± 0.4	23 ± 5
Isolated peripheral hypoperfusion.	1.9 ± 0.4	12 ± 5
Pulmonary congestion and hypoperfusion.	1.6 ± 0.6	27 ± 8

Adapted from Forrester GA: *Am J Cardiol* 1977;39:137.

(2). Morphine sulfate in 2-mg (IV) increments as needed for pain unrelieved by nitroglycerin.
(3). Heparin 5,000 SQ q8–12 h or low molecular weight heparin in deep venous thrombosis (DVT) prophylaxis doses in patients without contraindications and who are not receiving full-dose heparin should be given. *Note:* Patients with anterior wall MI have a lower incidence of LV thrombosis if full heparinization is used.
(4). Statin therapy to lower cholesterol.
(5). Strict bed rest for 24 h followed by gradual increase in activity.
(6). Stool softener, commonly docusate sodium (Colace) 100 mg PO qd.
(7). A low-cholesterol, no-added salt diet should be prescribed.

b. Complicated MI (see Table 3.5)
(1). Left Ventricular Dysfunction Manifested by Pulmonary Congestion
(a). Decrease left ventricular end-diastolic pressure with IV nitroglycerin, consider dobutamine, diuretics, or other vasodilators (see dosages below).
(2). Patients with Hypoperfusion Without Pulmonary Congestion
(a). Careful IV hydration with normal saline. Pulmonary capillary wedge pressure is targeted at approximately 18 mmHg.
(b). Right ventricular MI accompanying inferior infarct may present in this manner. Diagnosis may be made using right-sided precordial chest ECG leads. Significant volume administration may be required for adequate LV preload.
(3). Severe LV Dysfunction
Pulmonary artery cannulation should be performed.
(a). If the systolic blood pressure is >100 mmHg, dobutamine up to 20 μg/kg/min intravenously should be started. Milrinone can also be used. If the patient demonstrates hypotension with systolic blood pressure (BP) <70–100 mmHg, dopamine in alpha-agonist doses or vasopressin 1–4 U/h IV or norepinephrine 0.5–30 μg/min IV should be administered.

(b). Hypertensive patients should be treated with IV nitroglycerin beginning at 10 μg/min. Dihydropyridine calcium-channel blockers (i.e., clevidipine, nicardipine) can also be used.
(c). Mechanical support can be done with
i. IABP
ii. Left ventricular- and biventricular-assist devices
iii. Percutaneous left atrial-to-femoral arterial ventricular-assist device
iv. Extracorporeal membrane oxygenation

c. Other Complications Following MI
(1). Mitral Regurgitation: This is characterized by the sudden appearance of a systolic murmur (radiating to the axilla) and worsening CHF.
(a). Diagnostic Studies: Physical examination will demonstrate a systolic murmur and worsening pulmonary congestion. Cardiac catheterization will demonstrate giant V waves in the pulmonary wedge tracing.
(b). Therapy: Afterload reduction (i.e., IV sodium nitroprusside) to decrease pulmonary capillary wedge pressure. Hypotensive patients may require catecholamines (i.e., dopamine and/or dobutamine).
(c). IABP may improve coronary perfusion and ventricular emptying.
(d). Surgical Repair.
(2). Ventricular Septal Defect (VSD): VSD is an event occurring in <1% of Q-wave MIs and may occur at any point from several hours to several days after the onset of symptoms. It is most commonly seen during the first 7 days.
(a). Diagnosis: Acute VSD results in a loud holosystolic murmur and sudden severe CHF with cardiogenic shock. Right heart catheterization with oxygen saturation measurements will exhibit an oxygen saturation step-up between the right atrium and right ventricle, and "contrast" echocardiography will many times identify the defect.
(b). Treatment: Acute afterload reduction with IV sodium nitroprusside and IABP is required for acute VSD with subsequent surgical repair.

d. Dysrhythmias Following MI: 90% of the patients suffering from acute MI will have dysrhythmias during the first 24 h.
(1). Sinus Bradycardia: The most commonly seen dysrhythmia in acute MI. It should be treated only when signs of diminished cardiac output are present. Atropine 0.5–1 mg IV q3–5 min until a total dose of 0.04 mg/kg has been given. If this proves ineffective, dopamine up to 20 μg/kg/min and epinephrine 10 μg/min should be considered.
(2). Supraventricular Dysrhythmias: Sinus tachycardia should be addressed by treating the underlying cause. Pain relief and sedation many times are all that is required. Patients with atrial fibrillation or flutter in emergent conditions may require acute cardioversion. Stable patients should be treated with calcium-channel blockers, beta-blockers, etc.

(3). Paroxysmal Supraventricular Tachycardia: This should be approached initially with vagal maneuvers and if these are unsuccessful, with adenosine 6 mg rapid IV push; if unsuccessful, adenosine 12 mg rapid IV push; followed by verapamil 2.5–5 mg IV push. Beta-blockers, amiodarone, or procainamide are alternatives.

(4). Ventricular Dysrhythmias: Prophylactic therapy with lidocaine does not result in improvement of overall survival and thus *is not indicated* in patients with acute MI. In the patient showing stable ventricular tachycardia with normal LV function, amiodarone is given (250 mg over 10 min, followed by an intravenous infusion 1 mg/h for 6 h and then 0.5 mg/min). Other choices include procainamide, administered at 20–30 mg/min to a maximum dose of 17 mg/kg/h. Endpoints for therapy include abolition of dysrhythmia, 50% widening of the QRS complex, and/or hypotension. Amiodarone is a preferred agent if depressed LV function is present.

(5). Magnesium sulfate has also been demonstrated to be useful, particularly in polymorphic ventricular tachycardia: 1–2 g over 1–2 min, IV.

e. Conduction Disturbances Accompanying Acute MI

(1). Atrioventricular (A-V) Conduction Disturbances

(a). First-Degree A-V Block: Occurs in 4–14% of acute MIs.

(b). Second-Degree A-V block, Mobitz Type I: This is progressive prolongation of the PR interval with intermittent nonconduction of an atrial beat. It is commonly seen in inferior infarction and rarely progresses to complete heart block.

(c). Second-Degree A-V block, Mobitz Type II: Represents 10% of all second-degree blocks during acute MI. This is commonly seen in anterior infarction and infrequently progresses to complete heart block.

(d). Third-Degree A-V Block: Occurs in 6% of patients. Mortality with inferior MI is 20–25%; mortality with anterior MI is even greater.

(e). Intraventricular Block: Refers to abnormalities within the three divisions of the intraventricular conduction system. These blocks may progress to higher degrees of heart block. One in five patients with bundle branch block in acute MI will develop second-degree or third-degree A-V block. Mortality rates are double of those who do not.

(f). Complete Heart Block: Occurs frequently in MIs with right bundle branch block plus block of the anterior fascicle or posterior fascicle and less frequently, an isolated left or right bundle branch block. Similarly, patients with alternating forms of bundle branch block have a high incidence of complete heart block.

(2). Therapy

(a). Atrioventricular Block

i. First-Degree A-V Block: no specific therapy.

ii. Mobitz Type I Second-Degree A-V Block: Unless unusually slow ventricular rates occur, therapy is not needed.

Atropine is given (as for bradycardia) followed by temporary transvenous pacemaker insertion in those patients who are symptomatic.

iii. Mobitz Type II Second-Degree A-V Block: Particularly when associated with anterior MI, should result in placement of transvenous pacemaker.

iv. Complete Heart Block: Temporary transvenous pacemaker (some would advocate pacemaker therapy in inferior MI for hemodynamically compromised individuals only).

v. Intraventricular Conduction Disturbances: A transvenous pacemaker should be inserted for right bundle branch block plus either anterior fascicular, posterior fascicular, or alternating bundle branch blocks. Patients with first-degree A-V block and new-onset right or left bundle branch block also should receive transvenous pacing.

III. Cardiac Pacemakers

A. Definition. Cardiac pacemakers are complicated devices that may be used to accelerate cardiac rate, bypass, block conduction tissue, and/or disrupt dysrhythmias. Advancing technology has resulted in new modes of operation, with dual-chamber pacing being used more often. A five-position code has been developed to describe clinical pacing mode (see Table 3.6), *The North American Society of Pacing and Electrophysiology/British Pacing Electrophysiology Group Generic Pacemaker Code.*

B. Pacemaker Evaluation. Rhythm strips and 12-lead ECGs can be useful in determining the mode of functioning of cardiac pacemakers placed in ICU patients. Patients should be examined for failure to sense, as indicated by inappropriate pacemaker spikes, and failure to capture, as indicated by pacemaker spikes without subsequent chamber depolarizations. More detailed information can be obtained by querying appropriately equipped pacemakers and examining pulse characteristics with appropriate devices.

IV. Congestive Heart Failure

A. Definition. CHF is the clinical state that occurs when the heart cannot pump sufficient oxygenated blood to meet the metabolic needs of the tissues.

B. Etiology. CHF may result from the failure of either the left ventricle or the right ventricle. In many instances, both pumping chambers of the heart fail. Common causes of left ventricular failure include heart disease (aortic stenosis [AS], aortic regurgitation [AR], mitral regurgitation [MR], hypertension, ischemic heart disease, cardiomyopathy, myocarditis). Common causes of right ventricular failure include pulmonary hypertension (primary and secondary), cardiomyopathy, and right ventricular infarction. Biventricular failure commonly results from left ventricular failure. Additional causes of CHF include dysrhythmias, anemia, thyrotoxicosis, medication, and arteriovenous fistulas.

C. Clinical Manifestations

1. Shortness of breath
2. Orthopnea

   Increased venous return associated with a recumbent position leads to worsening shortness of breath.

Table 3.6. Pacemaker Codes

Chamber paced	*Chamber sensed*	*Response to sensing*	*Programmability*	*Antitachycardic functions*
O = none	O = none	O = none	O = none	O = none
A = atrium	A = atrium	I = inhibited	S = simple programmable	P = antitachycardia
V = ventricle	V = ventricle	T = triggered	M = multiprogrammable C = communicating	S = shock
D = dual	D = dual	D = dual	R = rate modulation	D = dual

3. Paroxysmal nocturnal dyspnea
   This is the result of a number of physiologic factors including the increased venous return in patients who are recumbent.
4. Fatigue and lethargy
   These are due to a low cardiac output.

D. Physical Examination. Signs of left ventricular failure on physical examination include tachycardia and tachypnea. Pulmonary venous congestion results in rales bilaterally. S3 and S4 gallop sounds may be heard. Patients with valvular heart disease may manifest cardiac murmurs. Signs of right heart failure include venous distension in the jugular veins, peripheral edema, ascites, and congestive hepatomegaly with hepatojugular reflux.

E. Classification of CHF. Functional classification is commonly reported per the New York Heart Association criteria (see Table 3.7). Another common way to classify CHF is based on systolic or diastolic dysfunction. Diastolic heart failure is present in patients who have symptoms and signs of CHF, normal or near-normal left ventricular systolic function, and evidence of diastolic dysfunction (e.g., abnormal left ventricular filling and elevated filling pressures).

F. Chest X-Ray. Cardiomegaly with enlargement of involved heart chambers may be seen. Pulmonary vascular congestion progressing to alveolar edema.

G. Therapy
   1. Correct and identify the underlying cause (i.e., treat anemia, infections, hypertension, control ventricular rate in patients with atrial fibrillation).
   2. Decrease cardiac workload with bed rest.
   3. Supplemental oxygen is given as necessary.
      a. If respiratory distress and/or hypoxia persist, we suggest noninvasive positive pressure ventilation as long as the patient does not have a contraindication
   4. Sodium restriction.
   5. Preload reduction.
      a. Nitrates: Venous dilatation associated with nitrates results in prompt improvement of symptoms in many patients with CHF. (See angina section in the beginning of this chapter for dosing recommendations.)
      b. Diuretic Agents
         (1). Loop-Acting Agents
            (a). Furosemide (Lasix) 10–240 mg IV or PO or a continuous IV infusion induces a prompt diuresis and results in venodilatation with rapid improvement in patient symptomatology.
            (b). Bumetanide (Bumex) 0.5–1 mg IV or .5–2 mg PO.
            (c). Thiazides (i.e., hydrochlorothiazide 25–50 mg) are less potent diuretics that may be of value in mild-to-moderate CHF.

Table 3.7. New York Heart Association Classification of CHF

Class I	Symptomatic with extraordinary activity
Class II	Symptomatic with ordinary activity
Class III	Symptomatic with minimum activity
Class IV	Symptomatic at rest

(d). Metolazone (Zaroxolyn) may potentiate the effect of loop-acting diuretics in doses of 2.5–10 mg.

(e). Morphine sulfate has traditionally been used in the management of severe pulmonary edema because of its venodilatory properties and its anxiolytic effects. This agent may depress respirations, and thus, other vasoactive substances may be preferable. If used, increments of 2 mg IV, titrated to effect, are recommended.

6. Arteriodilators (afterload reduction)
   a. ACE Inhibitors: These agents result in dilatation of the arteriolar resistant vessels and also increase venous capacity, having effects on both preload and afterload. They decrease mortality in patients with CHF. Enalaprilat is available for IV administration (1.25 mg IV over several minutes). Oral ACE inhibitors are also available.
   b. Nesiritide: This recombinant human BNP has been used with some success. However, some studies have raised concerns about adverse impact on mortality rate and a potential risk of worsening renal function.
7. Inotropic Agents
   Intravenous inotropes (i.e., milrinone or dobutamine) may be used to relieve symptoms and improve end-organ function, particularly if these patients have marginal systolic blood pressure (<90 mmHg), have symptomatic hypotension despite adequate filling pressure, or are unresponsive to, or intolerant of, intravenous vasodilators.
8. Digitalis Glycosides and Beta-Blockers
   These agents have limited value in the acute setting of pulmonary edema. Beta-blockers reduce mortality in the long-term management of these patients.
9. In severe unresponsive cases or in cases where a different procedure is indicated due to erythrocytosis, phlebotomy may be used.

V. Cardiomyopathies

A. Dilated Congestive Cardiomyopathy

1. Etiologies
   Primary disorders of heart muscle in which dilatation of the ventricles and enlargement of the heart occurs (see Table 3.8).
2. Symptoms of CHF, dysrhythmias, and pulmonary and systemic embolization.
3. Physical Examination
   Signs of CHF are commonly seen. A laterally placed point of maximal impulse [PMI] may be noted along with gallop sounds.

Table 3.8. Etiologies of Dilated Cardiomyopathy

Idiopathic
Collagen vascular disease
Postmyocarditis
Peripartum
Familial
Toxins and nutritional deficiency
Radiation

4. Diagnostic Studies
   a. Chest X-Ray: Cardiac enlargement may be seen, pulmonary congestion with interstitial edema, pleural effusion, etc., also may be seen.
   b. ECG: Dysrhythmias may be seen, as may conduction abnormalities, chamber enlargement/hypertrophy, and nonspecific repolarization abnormalities.
   c. Echocardiography: May demonstrate a low ejection fraction and global hypokinesis and chamber enlargement.
   d. Cardiac catheterization and myocardial biopsy
5. Therapy
   a. Treat the underlying cause.
   b. Management of CHF as noted above.
   c. Prevent thromboembolism.
   d. Consider low-dose beta-blockade.
   e. Consider transplantation with potential mechanical support as bridging maneuver (i.e., left ventricular assist device).

B. Restrictive Cardiomyopathy. This is a myocardial disorder characterized by decreased ventricular compliance.
1. Etiology
   Infiltrative disorders (sarcoidosis, hemochromatosis, amyloidosis, etc.), radiation, endocardial fibroelastosis, endomyocardial fibrosis, scleroderma.
2. Symptoms
   Right-sided CHF signs, fatigue, and weakness.
3. Specific Diagnostic Studies
   Echocardiogram or magnetic resonance imaging (MRI) may help distinguish restrictive cardiomyopathy from constrictive pericarditis (pericardial thickening). Cardiac catheterization and/or biopsy may be used.
4. Therapy
   Control of CHF as previously noted. Special attention of volume status.

C. Hypertrophic Cardiomyopathy. Familial or sporadic disorder with marked hypertrophy of the myocardium. Focal or diffuse forms of hypertrophy may occur.
1. Symptoms: Syncope, dyspnea, chest pain, palpitations, sudden death.
2. Physical Findings
   a. Crescendo-decrescendo murmur at the left sternal border, which increases with Valsalvas maneuver
   b. S4 gallop sound
3. Diagnostic Studies
   Chest x-ray may be normal; ECG may show left ventricular hypertrophy, abnormal Q waves (anterior, lateral, and inferior leads); echocardiography demonstrates ventricular hypertrophy.
4. Treatment
   a. Beta-blockers and/or verapamil. These agents slow the heart rate and prolong diastole, allowing increased ventricular filling.
   b. Surgical and nonsurgical myectomy should be used when optimal medical therapy has failed in appropriately selected patients. Ethanol septal infusion reduces aortic gradient and symptoms in a large proportion of cases and is rapidly becoming the therapy of choice in severely symptomatic cases.

c. Digitalis, nitrates, diuretics, and vasodilators *may worsen the clinical condition* of this subset of patients.

VI. Myocarditis

Myocarditis is an inflammatory condition of the myocardium.

A. Etiology
1. Infection
   a. Viral (echovirus, adenovirus, etc.)
   b. Bacterial
   c. Mycoplasma
   d. Mycotic
   e. Rickettsial
   f. Spirochetal
   g. Parasitic (trichinella, *Trypanosoma cruzi*)
2. Toxins and drugs (i.e., cocaine)
3. Collagen vascular disease (scleroderma, systemic lupus erythematosus, rheumatic fever, sarcoidosis)

B. Symptoms
1. Dyspnea
2. Chest discomfort

C. Physical Examination
1. Tachycardia
2. Pericardial friction rub (in the presence of coexistent pericarditis)
3. Evidence of CHF

D. Therapy
1. Supportive Care
   a. Treatment of CHF
   b. Treatment of dysrhythmias, as necessary
   c. Anticoagulation to prevent thromboembolism
2. Treat the underlying cause (the use of corticosteroids, immunoglobulins, and immunosuppressive therapy in selective populations with inflammatory infiltrates on endomyocardial biopsy may be warranted).

VII. Pericarditis

Inflammation of the pericardium associated with many different etiologic factors.

A. Etiology (see Table 3.9)

B. Symptoms
1. Anterior chest pain, commonly radiating to arms and back, which classically increases with inspiration and is relieved by sitting up or leaning forward. Palpitations and tachycardia may also occur.

C. Physical Examination
1. Pericardial friction rub is best heard with the patient upright and leaning forward.
2. Tachycardia or other dysrhythmias may be auscultated.
3. If pericardial tamponade occurs, low blood pressure, narrow pulse pressure, and accentuated *pulsus paradoxus* may be seen.

D. Diagnostic Studies
1. ECG (see Figure 3.1)

Table 3.9. Etiologies of Pericarditis

1. Idiopathic
2. Infectious (tuberculosis, bacterial, viral, fungal, protozoal)
3. Collagen vascular disease
4. Drug induced
5. Trauma
6. Acute MI and post MI (Dressler's syndrome)
7. Uremia
8. Postradiation
9. Rheumatic fever
10. Neoplasms

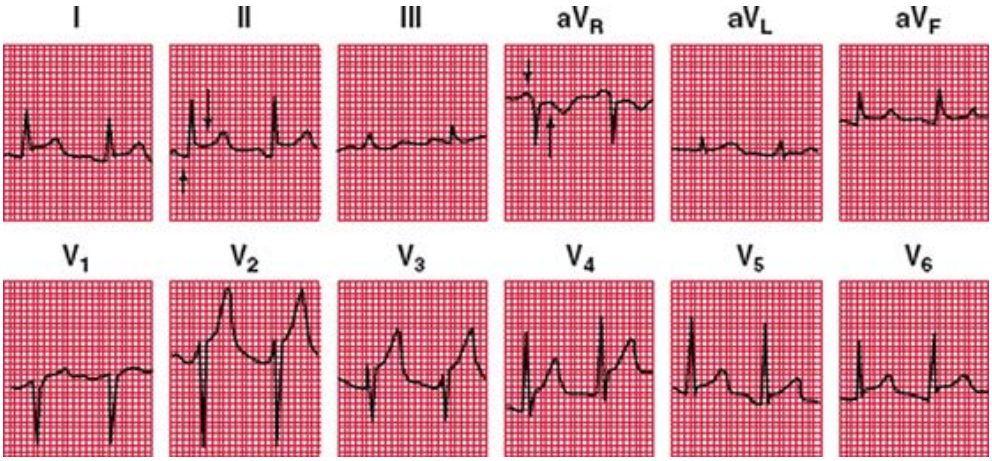

Figure 3.1. Pericarditis. Diffuse ST segment elevation, which is concave upward, is present in all leads except aV_R and V_1. Depression of the PR segment, an electrocardiographic abnormality that is common in patients with acute pericarditis, is not evident because of the short PR interval. (Braunwald, E: *Heart Disease: A Textbook of Cardiovascular Medicine*, 5th ed. Saunders, 1997. Used with permission.)

An acute MI evolutionary pattern of ECG is seen with initial ST-segment elevations with concavity upward, subsequent T-wave inversion, and finally, late resolution of the repolarization abnormalities. PR segment depression may also be seen.

2. Laboratory Evaluation
   MI should be ruled out as noted above. Other potential useful studies might include erythrocyte sedimentation rate (ESR), anti-nuclear antibodies (ANA), rheumatoid factor, viral titers, and tuberculin skin test (PPD).
3. Echocardiogram
   To document pericardial effusion (may not be present).

E. Treatment

1. Anti-inflammatories (i.e., indomethacin 25–50 mg PO q8 h or salicylates 2–5 g/d). In severe cases, corticosteroids (i.e., prednisone 60 mg PO qd).

2. Analgesia for pain unrelieved by anti-inflammatories.
3. Observation for signs of cardiac tamponade.
4. Treatment of underlying causes.

F. Complications

1. Cardiac Tamponade
   Accumulation of pericardial fluid may impair cardiac function, mainly through thinning of diastolic filling.
   a. Symptoms: Dyspnea, orthopnea, and fatigue
   b. Physical Findings
      (1). Neck pain
      (2). Distant heart sounds
      (3). Tachycardia
      (4). Pulsus paradoxus
      (5). Hypotension and a narrow pulse pressure
   c. Diagnostic Studies
      (1). ECG: Decreased QRS amplitude and beat-to-beat changes in the R wave.
      (2). Echocardiography: Demonstrates effusions and early right ventricle diastolic collapse.
      (3). Cardiac Catheterization: Right heart catheterization will reveal equalization of diastolic pressures, which includes pericardial pressure if measured.
   d. Therapy
      (1). Pericardiocentesis (see Chapter 15, Special Techniques). Removal of a relatively small amount of pericardial fluid will improve diastolic filling in the ventricle and greatly improve the patient's symptomatology. A drainage catheter may also be left in place. Fluid obtained should be tested for protein, lactic dehydrogenase (LDH), cell count, Gram's stain, acid-fast bacilli stain (AFB), culture/sensitivity, and cytology.
      (2). Pericardiectomy, Pericardial Window: These surgical procedures may be performed to relieve pericardial tamponade.

VIII. Valvular Heart Disease

A. Aortic Stenosis

1. Etiology
   a. Rheumatic inflammation of the aortic valve
   b. Progressive stenosis secondary to congenital bicuspid valve
   c. Congenital aortic stenosis
   d. Idiopathic calcification stenosis of the aortic valve
2. Pathophysiology
   Stenosis of the aortic valve results in increased resistance to ventricular ejection and increased left ventricular pressure. Hypertrophy of the ventricle will occur. Normal aortic valve area is approximately 3 cm^2. Aortic valves of <1 cm^2 generally produce symptoms, and those with <0.5 cm^2 with pressure gradients of ≥ 50 mmHg are considered severe.
3. Symptoms
   a. Syncope: Commonly with exertion and frequently associated with vasodilatation in muscle beds, leading to cerebral ischemia.
   b. Transient Dysrhythmias
   c. Angina
   d. CHF

4. Physical Findings
   a. Slow-rising, delayed carotid upstroke with decreased amplitude
   b. Narrowing of pulse pressure
   c. Loud systolic ejection murmur heard at the base of the heart and radiating to the neck, often with a palpable thrill
5. Diagnostic studies
   a. ECG
      (1). Left ventricular hypertrophy
      (2). Nonspecific repolarization abnormalities
   b. Chest x-ray
      (1). Pulmonary congestion in patients with CHF
      (2). Aortic dilatation
      (3). Calcification of the aortic valve
   c. Echocardiography
      (1). Hypertrophy of the left ventricular wall
      (2). Visualization of the abnormal aortic valve
   d. Cardiac catheterization documents severity of disease and calculation of valve area
6. Therapy
   a. Judicious management for CHF and angina as they occur (see appropriate sections as above. These patients may be very preload sensitive).
   b. Valve replacement should be reserved as palliative therapy for patients who are poor surgical risks.

B. Aortic Insufficiency
1. Etiology
   a. Infective endocarditis
   b. Trauma with valvular rupture
   c. Congenital bicuspid aortic valve
   d. Rheumatic fibrosis
   e. Myxomatous degeneration
   f. Accompanying aortic dissection
2. Pathophysiology
   Left ventricular pressure increases secondary to regurgitation of blood from the aorta, resulting in diastolic volume overload and subsequent decompensation.
3. Symptoms
   a. Many patients remain asymptomatic for many years.
   b. Symptoms during decompensation include dyspnea on exertion, syncope, chest pain, and CHF.
4. Physical Findings
   a. Widening pulse pressure with bounding pulses. Rapid rise and sudden fall in arterial pressure may result in head bobbing, capillary pulsations in the nail beds (Quincke's pulse), and "water-hammer" pulse. In addition, a murmur can be heard over the femoral arteries.
   b. PMI may be displaced laterally, and S_3 gallop may be heard. A diastolic blowing decrescendo murmur occurs along the left sternal border.
   c. Austin-Flint murmur (apical diastolic rumble of low pitch secondary to aortic regurgitation, which affects the anterior mitral leaflet).
   d. Systolic apical ejection murmur may also be heard.

5. Diagnostic Studies
   a. Chest X-Ray: May show left ventricular and/or aortic dilation.
   b. ECG: Left ventricular hypertrophy is usually present.
   c. Echocardiogram: Increased left ventricular dimensions and Doppler documentation of regurgitant aortic flow. Fluttering of the anterior mitral leaflet may also be seen.
   d. Cardiac Catheterization: Contrast study of the aortic root will demonstrate aortic regurgitation.
6. Therapy
   a. Medical management of CHF as noted above.
   b. Surgical therapy for patients unresponsive to medical management or with acute aortic regurgitation and left ventricular failure or with a declining ejection fraction.

C. Mitral Stenosis
1. Etiology
   a. Rheumatic fever
   b. Congenital defects
2. Pathophysiology
   The normal mitral orifice is 4–6 cm^2 in area. An obstruction of the orifice results in impedance of flow into the left ventricle. When the orifice area approaches 1 cm^2, symptoms appear.
3. Symptoms
   a. Dyspnea, orthopnea, and paroxysmal nocturnal dyspnea (pulmonary edema may develop following exertion).
   b. Systemic embolization, secondary to thrombi forming in a dilated left atrium.
   c. Dysrhythmias, particularly atrial fibrillation.
   d. Hemoptysis, secondary to persistent pulmonary hypertension.
4. Physical Findings
   a. Auscultation reveals an opening snap in early diastole.
   b. Apical presystolic or mid-diastolic rumble.
   c. Accentuated S_1, Graham-Steel murmur.
   d. Pulmonary regurgitation.
5. Diagnostic Studies
   a. ECG: Right ventricle hypertrophy, right axis deviation, left atrial enlargement, atrial fibrillation
   b. Chest X-Ray
      (1). Left atrial enlargement is seen on the lateral chest and a double density on the chest x-ray.
      (2). Elevation of the left main stem bronchus and widening of the angle between the right and left main stem bronchi.
      (3). Pulmonary arterial prominence.
   c. Echocardiography: Abnormalities of the valve itself may be seen with calcification and reduction of the E-F slope of the anterior mitral leaflet during diastole.
6. Treatment
   a. Control of ventricular rate in patients with atrial fibrillation and anticoagulation to prevent thromboembolism.
   b. Management of CHF as noted above.

c. Surgical therapy if the valve orifice is less than approximately 0.8 cm^2 or if symptoms persist despite optimal therapy.
d. Balloon valvuloplasty may be of value in poor surgical candidates.

D. Mitral Regurgitation
1. Etiology
a. Papillary muscle dysfunction or rupture of the chordae tendineae (i.e., MI)
b. Infective endocarditis
c. Left ventricle dilatation of any cause
d. Mitral valvular calcification
e. Rheumatic heart disease
f. Mitral valve prolapse
g. Idiopathic myxomatous degeneration of the mitral valve
h. Atrial myxoma
2. Symptoms
a. Dyspnea, orthopnea, and CHF of varying severity
b. Hemoptysis
c. Atrial fibrillation
d. Systemic embolization
3. Physical Findings
a. Holosystolic murmur at the apex with radiation to the base or to the left axilla
b. Rarely, early to mid-diastolic rumble secondary to increased mitral blood flow
c. Signs of CHF
d. Left ventricular lift and apical thrill
4. Diagnostic Studies
a. ECG: Left atrial enlargement, left ventricular hypertrophy, atrial fibrillation
b. Chest X-Ray: Left atrial enlargement, left ventricular enlargement, pulmonary congestion
c. Echocardiography
(1). Hyperdynamic left ventricle with enlarged left atrium
(2). Doppler studies demonstrating regurgitant flow
(3). Flail leaflet in patients with ruptured chordae
5. Therapy
a. Medical management of CHF as noted above, with particular attention to afterload reduction and control of ventricular rate.
b. Severity of acute disease may be temporized with IABP or surgical intervention.

IX. Aortic Dissection
A. Definition. Although commonly called aneurysms, this disorder is more appropriately termed *aortic dissection.* This condition results when there is a tear of the aortic intima, dissection of blood into the media, and stripping away of the vessel wall from the adventitia.

B. Etiology
1. Hypertension (present in 90% of patients)
2. Connective tissue disorders (i.e., Marfan's syndrome, Ehlers-Danlos syndrome)
3. Bicuspid aortic valve

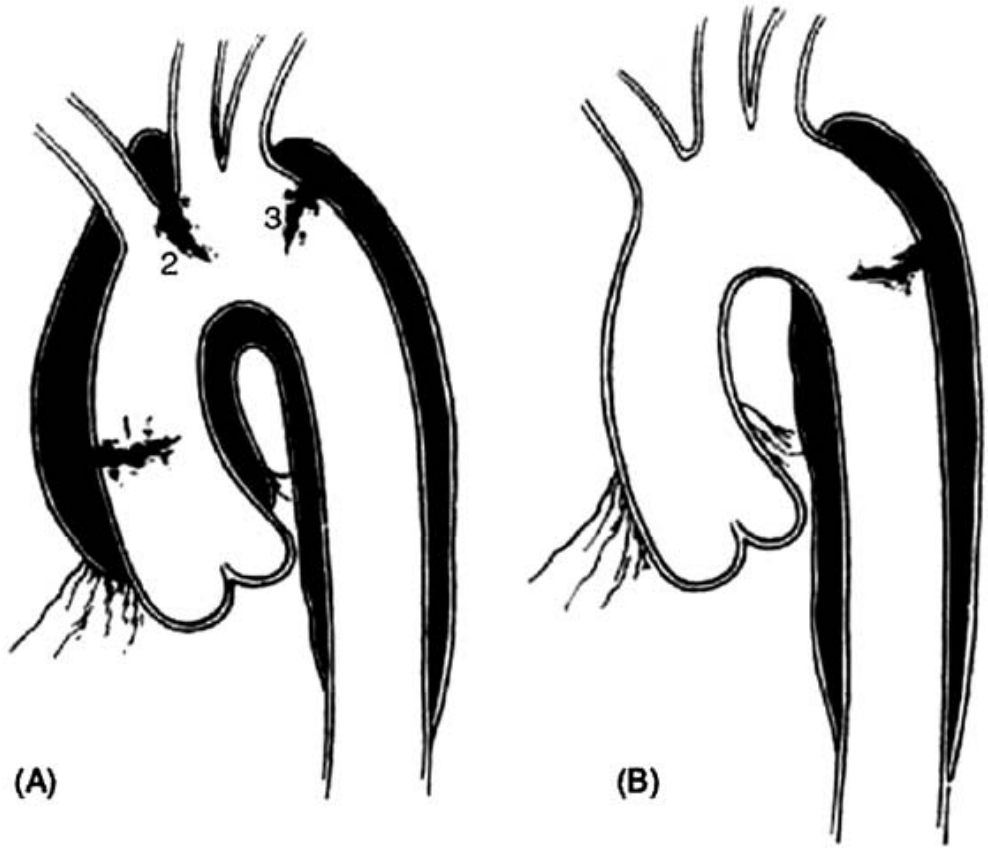

Figure 3.2. Classification of Aortic Dissection (Stanford). In type A, the ascending aorta is dissected ***(A)***. The intimal tear has always been at point 1, but can occur at position 2 or 3. In type B dissection, the dissection is limited to the descending aorta ***(B)***. (Thubrikar, M: *Vascular Mechanics and Pathology*, New York, Springer, 2007. Used with permission.)

4. Granulomatous arteritis and syphilitic aortitis
5. Pregnancy
6. Aortic injury

C. Classification. These lesions are commonly classified by their location (See Figure 3.2). Type A dissections involve the proximal aorta, from the aortic valve to the aortic arch, and Type B dissections arise beyond the takeoff of the left subclavian artery.

D. Symptoms
1. Chest pain (almost always present and usually abrupt, severe, and tearing or burning)
2. Syncope
3. CHF
4. Cerebrovascular accidents

E. Physical Findings
1. Hypotension or hypertension
2. Pulse deficits

3. Aortic regurgitation murmur
4. Pericardial friction rub
5. Neurologic signs
6. Horner's Syndrome and/or hoarseness

F. Diagnostic Studies
1. Chest X-Ray
   a. Abnormal in 90% of aortic dissections
   b. Widened aortic shadow
   c. Pleural effusions
   d. Aortic calcification
2. ECG: Frequently abnormal (90%); however, nondiagnostic.
3. Computed tomography of the chest with or without contrast will reveal the lesion.
4. Echocardiogram: Transthoracic and transesophageal studies may reveal the dissection.
5. Aortogram: The old "gold standard" for diagnosis.
6. Magnetic resonance imaging

G. Therapy
1. Surgical
   a. Proximal dissections (Type A)
   b. Distal dissection, particularly if vital organs are compromised or persistent pain occurs despite medical management
2. Medical
   a. Aggressive control of blood pressure. The typical regimen is administration of IV beta-blocker (traditionally, propranolol 1 mg IV q5 min until evidence of beta-blockade) followed by sodium nitroprusside to maintain systolic blood pressure of approximately 100–120 mmHg. Alternative regimens using clevidipine or nicardipine combined with a beta-blocker have also been used.
   b. Transcatheter stenting techniques are becoming more common and used in some centers.

X. Shock States

Shock can be defined as a state of inadequate tissue perfusion, which, unless reversed, results in progressive organ dysfunction, damage, and death. Mortality rates for shock of many causes still exceed 50%. In early stages of shock, the patient may be relatively asymptomatic. Sympathetic discharge and other compensatory mechanisms may cause tachycardia and mild peripheral vasoconstriction in attempts to maintain blood pressure. When the state of shock worsens, organ hypoperfusion continues and blood pressure declines; signs of organ dysfunction including restlessness and agitation, decreased urine output, and cool and clammy skin become evident.

A. Classification and Etiology. A number of classification schemes for shock states have been devised. These include cardiogenic shock; myopathic (reduced systolic function, i.e., acute MI), mechanical (mitral regurgitation, ventricular septal defect), extracardiac obstructive shock (i.e., pericardial tamponade, massive pulmonary embolus, or severe pulmonary hypertension); oligemic shock (i.e., hemorrhage or fluid depletion); and distributive shock (i.e., septic shock, anaphylaxis, neurogenic shock, etc.).

B. Diagnostic Evaluation
   1. Physical Examination
      Tachycardia, hypotension, and evidence of hypoperfusion (i.e., altered mental status, decreased urine output, cool and clammy skin) are generally present. Other manifestations may be seen on physical examination, depending upon the etiology of the shock state.
   2. Laboratory Evaluation
      a. ECG: Useful for identifying dysrhythmias and acute MI.
      b. Chest X-Ray: Pneumothorax, abnormal cardiac silhouette, and pulmonary edema.
      c. Hematology and Chemistry: CBC, BUN, creatinine, electrolytes, glucose, liver function tests (LFTs), and arterial blood gases should be obtained in the evaluation of any patient with shock.
   3. Monitoring
      a. Foley Catheter: Patients in shock without contraindication should receive catheter insertion for the monitoring of urine output.
      b. Arterial Line: For direct intra-arterial pressure determination and to allow easy vascular access for laboratory and arterial blood gas monitoring.
      c. Central Venous Pressure (CVP) Monitoring Catheter, Oximetric CVP Catheter, or (rarely) PA Catheter: These are commonly employed. Most patients can be managed without pulmonary artery cannulation, and the PA catheter is being used less frequently.

C. Therapy. Primary treatment goals include restoring oxygen transport and organ perfusion (i.e., urine output $^{1}/_{2}$–1 mL/kg/h and the absence of lactic acidosis).
   1. Airway, breathing, and circulation (ABCs), as in all critically ill patients.
   2. Supportive Measures
      a. Two large-bore IV catheters for those patients requiring volume resuscitation. For patients in whom fluid status is normal or elevated, a central IV line for administration of medication will usually be required. For those patients not volume overloaded, the initial management of hypotension and shock is volume administration. Volume challenges of 250 cc to 1 L at a time should be rapidly administered with reassessment of the patient's clinical condition.
      b. Supplemental oxygen appropriate for the patient's clinical status.
      c. Those patients not responding to volume administration should receive beta-receptor stimulants. Dopamine, although commonly used in hypotensive patients, is not ideal as it produces significant shunts and can decrease cerebral perfusion pressure. In the authors' experience, vasopressin 1–6 U/h is the agent of choice in these conditions. Other agents, such as norepinephrine, can also be used.
      d. Mechanical support of the circulation may be necessary in patients with refractory cardiogenic shock and amenable lesions.
      e. Additional interventions and therapeutic goals for management of shock must be based on specific etiologies.

XI. Infective Endocarditis

A. Definition. Infection of the endocardial structures of the heart.

B. Etiology
   1. *Streptococcus viridans:* Streptococci is the most common organism isolated, excluding prosthetic valve or right-sided endocarditis.
   2. *Staphylococcus aureus:* The most frequent organism isolated in right-sided endocarditis.
   3. *Staphylococcus epidermidis*
   4. Others
      a. Gonococci
      b. Other bacteria
      c. Fungi

C. Risk Factors. A number of disorders and behaviors are risk factors for the development of endocarditis. These include the following:
   1. Valvular abnormalities
      a. Rheumatic valvulitis
      b. Bicuspid aortic valve
      c. Aortic stenosis or insufficiency
      d. Mitral stenosis, prolapse, or insufficiency
      e. Mechanical heart valves
      f. Previous endocarditis
   2. IV drug abuse
   3. Marfan's syndrome
   4. Instrumentation

D. Diagnosis
   1. History and Physical Examination
      Careful history for underlying risk factors should be elicited.
   2. Physical Examination
      a. Fever: Generally present but may not be noted in elderly or immunocompromised patients.
      b. Cardiac Murmurs: Are usually present but may not be detected, particularly in right-sided endocarditis.
      c. Peripheral Manifestations: These include painless erythematous papules and macules of the soles and palms (Janeway lesions) and painful erythematous subcutaneous papules (Osler's nodes), as well as petechia and splinter hemorrhages of the nail beds.
   3. Laboratory Evaluation
      a. Blood Cultures: Before antibiotic therapy, positive cultures are quite common (85–95%). Reasons for negative cultures include prior antibiotic therapy, slow-growing or very fastidious organisms, or improper collection.
      b. Nonspecific Laboratory Findings
         (1). Includes decreased hemoglobin/hematocrit.
         (2). Elevated, decreased, or normal white blood cell count with a left shift, hematuria on urinalysis, and an elevated sedimentation rate. Rheumatoid factor may be positive in half of the cases by 6 weeks, and assays of teichoic acid antibodies have been advocated for *Staphylococcus aureus* endocarditis.
      c. Echocardiography: Transthoracic and transesophageal echocardiography may reveal valvular damage, impairments of left ventricular function, and valvular vegetations. Transesophageal echocardiography

enhances sensitivity. Some patients will not demonstrate abnormal echocardiographic studies.

E. Major Complications
1. CHF secondary to valvular destruction, dysrhythmias, or myocarditis
2. Embolization
3. Cardiac dysrhythmias
4. Myocarditis and pericarditis

F. Therapy
1. Antibiotics appropriate for the clinical setting. For a valvular endocarditis, penicillin G IV (12–24 million U/d) and gentamicin (dosed by body weight and renal function) are commonly advocated. For IV drug addicts, penicillinase-resistant penicillin or vancomycin plus gentamicin are advocated.
2. Surgical therapy for endocarditis should occur if severe heart failure or valvular obstruction is present or if uncontrolled infection exists. Relative indications for cardiac surgery include two or more embolic events, unusually large vegetations, extension of the infection to other intracardiac structures, or in the case of prosthetic valve endocarditis, periprosthetic leak.

XII. Dysrhythmias (See Also Chapter 2, "The Basics of Critical Care")

A. Supraventricular Dysrhythmias. A group of dysrhythmias whose site of origin and pathway is not confined to the ventricles.
1. Paroxysmal Supraventricular Tachycardia (PSVT)
PSVT commonly originates through a reentrant mechanism in the AV node, characterized by abrupt onset and termination. PSVT may occur in young patients without other evidence of cardiac disease, as well as in patients with acute MI, Wolf-Parkinson-White Syndrome, or other structural heart diseases.
a. ECG Characteristics: Regular tachycardia of 150–220 beats per minute. Atrial activity (P waves) may or may not be seen, depending upon the rate and relationship between atrial and ventricular depolarization. QRS complex is frequently narrow. However, a wide QRS complex may be seen.
(1). Symptoms
(a). Palpitations
(b). May produce hypotension during acute MI or may precipitate CHF.
(2). Therapy
(a). ABCs.
(b). For patients demonstrating clinical instability (i.e., cardiogenic shock, ischemic chest pain, or CHF), synchronized DC countershock should be used.
(c). Treatment of the stable patient should begin with a vagal maneuver. Valsalva or carotid sinus massage following exclusion of carotid disease may abort the dysrhythmia.
(d). Adenosine should be administered to those patients who do not respond to vagal maneuvers, 6 mg rapid IV bolus. A second bolus of 12 mg rapid IV may be given. Methylxanthines (i.e., theophylline, aminophylline, caffeine) are competitive

antagonists, and dipyridamole enhances the pharmacologiceffect of adenosine.

(e). Verapamil 5–10 mg over 5 min (may repeat dose in 20–30 min if ineffective) may be used if adenosine is ineffective in patients with narrow complex PSVT. Pretreatment with a slow injection of 10 mL of 10% calcium chloride may decrease the common hypotensive effects of this drug. Patients with wide-complex tachycardia that cannot be confidently diagnosed as supraventricular should not receive verapamil, nor should patients with depressed ejection fraction. Digoxin or amiodarone as noted above for stable PSVT should be considered.

(f). Additional considerations include digoxin, beta-blockers, propafenone, diltiazem, pace termination, and synchronized cardioversion for patients with preserved ejection fraction.

B. Atrial Fibrillation. Atrial fibrillation is characterized by chaotic atrial activity without an organized atrial rhythm. This dysrhythmia may accompany coronary artery disease, mitral and aortic valvular disease, thyrotoxicosis, peri- and myocarditis, alcoholic heart disease, and MI without evidence of other organic cardiac disease.

1. Electrocardiogram
   Irregular, chaotic atrial activity without an organized rhythmic pattern. Conductive QRS complexes will have an irregularly irregular pattern. However, atrial ventricular block with emergence of a lower pacemaker site may result in irregular ventricular response.
2. Other Diagnostic Studies
   a. Thyroid function tests
   b. Echocardiography
3. Therapy
   a. In unstable patients, as in PSVT, DC cardioversion is indicated.
   b. Digoxin Loading: Digoxin has been the traditional therapy for new-onset atrial fibrillation. Administer 0.5-mg IV loading dose followed by 0.25 mg q3–4 h until the ventricular rate is controlled. For patients with impaired ejection fraction, this remains a good choice.
   c. Alternatives for rapid rate control include
      (1). Diltiazem 20–25 mg IV over 2 min with continuous infusion of 5–15 mg/h.
      (2). Beta-blockers (i.e., propranolol 0.5 mg IV slowly followed by boluses of 1 mg q5 min to a total of 0.1 mg/kg). Atenolol 5 mg IV slowly × 2. Metoprolol 5 mg slow IV q5 min × 3.
      (3). Amiodarone in a dose of 150 mg over 10 min followed by 360 mg over 6 h and 0.5 mg/min is a good choice, particularly with depressed ejection fraction.
   d. Anticoagulation is advisable pre- and postcardioversion, particularly in patients with mitral valve disease or a history of embolic phenomenon. Patients with a duration of atrial fibrillation >48 h should not be converted acutely, if avoidable, because of embolization risk. Three to four weeks of anticoagulation precardioversion is recommended, with at least 4 weeks of anticoagulation postcardioversion suggested.

C. Atrial Flutter. Atrial flutter is characterized by a rapid regular atrial rate of 280–340 beats per minute, generally associated with varying degrees of AV block. This dysrhythmia may occur with coronary artery disease, including MI, thyrotoxicosis, pulmonary embolism, and mitral valve disease.
   1. ECG: Atrial depolarization classically has a "saw-tooth" pattern with varying AV conduction block. Vagal maneuvers may slow the ventricular response rate, making atrial flutter waves more readily apparent.
   2. Therapy
      a. As for atrial fibrillation. In patients who are compromised, cardioversion with DC countershock is indicated.
      b. Commonly responds to the pharmacologic interventions previously denoted for atrial fibrillation. In addition, atrial pacing may also terminate atrial flutter.

D. Multifocal Atrial Tachycardia. In multifocal atrial tachycardia, a chaotic irregular atrial activity is seen, with rates between 100 and 180 and varying p-wave morphology (three consecutive different p-wave morphologies). This disorder commonly accompanies chronic obstructive pulmonary disease, theophylline toxicity, hypoxemia, and/or other metabolic disturbances.
   1. ECG (See Figure 3.3)
      Varying P-to-P intervals and beat-to-beat variability in P-wave morphology.
   2. Therapy
      a. Treat the underlying cause. Rate may be controlled, if necessary, with diltiazem or amiodarone. In the absence of depressed ejection fraction, beta-blockers may be used. In addition, IV magnesium has been advocated by some authorities.

E. Bradycardias and AV Conduction Blocks. These are characterized by a low intrinsic rate from the sinus node or blockade of sinus impulses between the AV node, which result in slow ventricular rates.
   1. Etiology
      a. Vagotonia
      b. Ischemic heart disease
      c. Cardiomyopathies
      d. Drugs
      e. Degenerative diseases of the AV conduction system
   2. Treatment
      Patients who are symptomatic due to low ventricular response rate may be treated with the following:

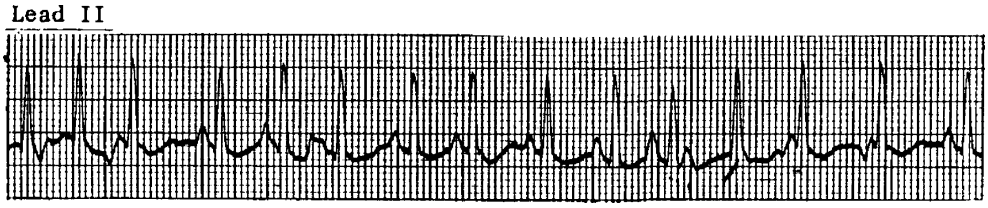

Figure 3.3. Multifocal atrial tachycardia. (Criner, G: *Critical Care Study Guide*, Springer, 2002. Used with permission.)

a. Initially with atropine 0.5–1.0 mg IV, repeated every 3–5 min with a total dose of 0.04 mg/kg.
b. Transcutaneous pacing when available may be employed.
c. Pharmacologic therapy may include dopamine 5–20 μg/kg/min IV infusion or epinephrine 2–10 μg/min IV infusion titrated to heart rate. Patients requiring transcutaneous pacers for high degrees of AV block should receive consideration for urgent transvenous pacemaker placement.

F. Ventricular Tachycardia. Ventricular tachycardia is defined as three or more consecutive beats of ventricular origin. Common rates are between 100 and 200 beats per minute. The differentiation of ventricular and supraventricular dysrhythmia with conduction may be difficult. A good rule of thumb is that wide-complex QRS tachycardia should be considered ventricular tachycardia until proven otherwise.
1. Monomorphic ventricular tachycardia (a single QRS morphology) should be treated as noted in Chapter 3, "The Basics of Critical Care".
2. Polymorphic ventricular tachycardia or "torsade de pointes" may be caused by agents frequently used in the treatment of monomorphic ventricular tachycardia. Electrolyte disturbances including hypokalemia, hypomagnesemia, and the presence of cardiac and psychotropic medications should be sought. Unstable patients should receive electrical therapy as previously outlined. Stable patients may respond to overdrive pacing, IV magnesium, and correction of underlying causes.

XIII. Hypertensive Crises. Hypertensive crises are potentially life-threatening situations that are the result of elevated blood pressure. One percent of all patients with hypertension (HTN) may present with hypertensive crises. Manifestations include hypertension with end-organ dysfunction (see Table 3.10). Rarely occur with blood pressures <130 mmHg diastolic. Hypertensive crises can be further classified into: Hypertensive urgencies (when the BP is elevated but no active organ dysfunction is found) and hypertensive emergencies (when there is evidence of organ dysfunction). The management of hypertensive urgencies differs from that of hypertensive emergencies.

A. Treatment
1. Blood pressure should be promptly reduced in patients with hypertensive emergencies. Most authorities recommend reductions of approximately 15% (10–20%) in the first hour with gradual reduction to diastolic BP of 100–110 mmHg or a reduction of 25% of initial reading over the first 4–24 h.

Table 3.10. End-Organ Dysfunction in Hypertensive Emergencies

Hypertensive encephalopathy
Acute aortic dissection
Acute myocardial infarction
Acute cerebral vascular accident
Acute hypertensive renal injury
Acute congestive heart failure

Table 3.11. Intravenous Antihypertensive Medications

Clevidipine	Start at 2 mg/h and double to the dose every 3 min (max 32 mg/h)
Labetalol	20 mg bolus, 2 mg/min (max 300 mg/day)
Nicardipine	5–20 mg/h
Fenoldopam	0.1–2 μg/kg/min
Nitroglycerin	5 μg/min (increase by 5–10 μg q3–5 min as needed)
Diazoxide	25–150 mg IV over 5 min or infusion of 30 mg/min to effect

2. Reductions in BP may result in ischemia, and thus, these patients must be carefully followed. Parenteral therapy with short-acting agents is initially recommended (see Table 3.11).
3. Patients receiving parenteral therapy commonly need continuous arterial pressure monitoring.
   a. Cyanide poisoning may occur with IV administration of sodium nitroprusside to renal failure patients. Consider this if such patients develop CNS depression, seizures, lactic acidosis, or cardiovascular instability.
      (1). May occur with infusion rate 2 μg/kg/min.
      (2). Infusion rates of 10 μg/kg/min should not be continued for prolonged periods of time because of this hazard.
      (3). If cyanide intoxication suspected, discontinue infusion and treat as described in Chapter 16, "Toxicology."
4. Oral therapy with clonidine (Catapres) (0.1 mg PO q20 min) or a host of other agents may be used in less severe cases of hypertension in the ICU patient.

XIV. Useful Facts and Formulas

A. **Pressure = Flow × Resistance:** This is true in the airways as well as in the cardiovascular system. For example:

Mean arterial pressure = cardiac output × systemic vascular resistance.
Mean pulmonary arterial pressure = cardiac output × pulmonary vascular resistance.

The unmeasured resistance term is usually calculated by solving the equations:

$$\text{systemic vascular resistance} = \frac{\text{mean arterial pressure}}{\text{cardiac output}}$$

B. The Primary Determinants of Cardiovascular Performance
   1. Heart rate
   2. Preload
   3. Afterload
   4. Contractility

C. Other Principles and Conversion Factors

Fluid flow:

$$\text{Fluid flow} = \frac{(\text{pressure difference})(\text{radius})^4}{(\text{vessel length})(\text{fluid viscosity})^8}$$

Conversion to mmHg

$$\text{Pressure in mmHg} = \text{Pressure in cm } H_2O/1.36$$

LaPlace's law:

$$\text{Wall tension} = \text{distending pressure} \times \frac{\text{vessel radius}}{\text{wall thickness}}$$

Ohm's law:

$$\text{Current (I)} = \frac{\text{electromotive force (E)}}{\text{resistance (R)}}$$

Poiseuille's law:

$$Q = v\pi r^2$$

where Q = rate of blood flow (mm/sec); πr^2 = cross-sectional area (cm^2); v = velocity of blood flow.

Vascular capacitance:

$$\text{Vascular compliance (capacitance)} = \frac{\text{increase in volume}}{\text{increase in pressure}}$$

Vascular distensibility:

$$\text{Vascular distensibility} = \frac{\text{increase in volume}}{\text{increase in pressure} \times \text{original volume}}$$

D. Direct measurements of the heart rate are relatively easy. Preload, afterload, and contractility are more difficult to assess clinically. In assessment of cardiovascular performance, the following hemodynamic measurements are commonly measured or calculated:

1. *Arteriovenous Oxygen Content Difference ($avDO_2$)*
   This is the difference between the arterial oxygen content (CaO_2) and the venous oxygen content (CvO_2).

2. *Body Surface Area (BSA)*
   Calculated from height and weight, BSA is generally used to index measured and derived values according to the size of the patient.
3. *Cardiac Index (CI)*
   Calculated as cardiac output/BSA, CI is the prime determinant of hemodynamic function.
4. *Left Ventricular Stroke Work Index (LVSWI)*
   LVSWI is the product of the stroke index (SI) and (mean arterial pressure [MAP]—pulmonary artery occlusion pressure [PAOP]), and a unit correction factor of 0.0136. The LVSWI measures the work of the left ventricle (LV) as it ejects into the aorta.
5. *Mean Arterial Pressure (MAP)*
   The MAP is estimated as one-third of pulse pressure plus the diastolic blood pressure.
6. *Oxygen Consumption ($\dot{V}O_2$)*
   Calculated as $C(a–v)O_2$ H CO H 10, it is the amount of oxygen extracted in mL/min by the tissue from the arterial blood.
7. *Oxygen Delivery ($\dot{D}O_2$)*
   Calculated as (CaO_2) H CO H 10, it is the total oxygen delivered by the cardiorespiratory systems.
8. *Pulmonary Vascular Resistance Index (PVRI)*
   Calculated as (MAP—PAOP)/CI, it measures the resistance in the pulmonary vasculature.
9. *Right Ventricular Stroke Work Index (RVSWI)*
   RVSWI is the product of the SI and (mean pulmonary artery pressure [MPAP]—central venous pressure [CVP]), and a unit correction factor of 0.0136. It measures the work of the right ventricle as it ejects into the pulmonary artery.
10. *Stroke Index (SI)*
    Calculated as CI/heart rate, SI is the average volume of blood ejected by the ventricle with each beat.
11. *Systemic Vascular Resistance Index (SVRI)*
    Calculated as (MAP—CVP)/CI, SVRI is the customary measure of the resistance in the systemic circuit.
12. *Venous Admixture (Qva/Q_T)*
    Calculated as ($C\bar{c}O_2$—CaO_2)/($C\bar{c}O_2$—$C\bar{v}O_2$), it represents the fraction of cardiac output not oxygenated in an idealized lung.

E. Cardiac Output Formulas

$$\text{Output of left ventricle} = \frac{O_2 \text{ consumption (mL/min)}}{[AO_2] - [\dot{V}O_2]}$$

It may also be measured by thermodilution techniques:

$$Q = V(Tb - Ti)K/\int Tb(t)dt$$

where Q = cardiac output; V = volume of injectate; Tb = blood temperature; Ti = injectate temperature; K = a constant including the density factor and

catheter characteristics; $\int Tb(t)dt$ = area under the blood-temperature-time curve.

The same principle is applicable for the pulmonary blood flow:

$$\dot{Q} = B/(C\bar{v} - Ca)$$

where Q = pulmonary blood flow; B = rate of loss of the indicator of alveolar gas; $C\bar{v}$ = concentration of the indicator in the venous blood; Ca = concentration of the indicator in the arterial blood.

$$\dot{Q} = \dot{V}/(CaO_2 - cO_2)$$

where $\dot{Q}$ = total pulmonary blood flow; $\dot{V}O_2$ = oxygen uptake; CaO_2 = arterial oxygen concentration; $C\dot{V}O_2$ = mixed venous oxygen concentration.

F. Other Cardiovascular Performance Formulas/Tables

Alveolar-arterial O_2 difference or A-a gradient
= Alveolar pO_2—arterial pO_2
Normal <10 torr (10 mmHg)

Alveolar PO_2 at sea level (PAO_2)
= $(FiO_2 \times 713) - 1.2 \times PaCO_2$

Arterial blood O_2 content (CaO_2)
= $(PaO_2 \times 0.003) + (1.34 \times$ Hb in g $\times$ arterial blood Hb O_2 sat %)
Normal = 18–20 mL/dL

Arteriovenous oxygen difference ($avDO_2$)
= $(CaO_2) - (C\bar{v}O2)$
Normal = 4–5 mL/dL

Cardiac index (CI) = cardiac output/body surface area
Normal = 3.0–3.4 L/min-m^2

Ejection Fraction (EF)

$$= \frac{[\text{end diastolic volume}] - [\text{end systolic volume}]}{\text{end} - \text{diastolic volume}}$$

$= \%$

Mean arterial (or pulmonary) pressure
= DBP + 1/3 (SBP–DBP)

Mean pulmonary arterial pressure
= DPAP + 1/3 (SPAP–DPAP)

O_2 delivery index (DO_2I) = CaO_2 x cardiac index $\times$ 10
Normal = 500–600 mL/min-m2

O_2 consumption index (O_2I)
= Arteriovenous O_2 difference $\times$ cardiac index $\times$ 10
Normal = 120–160 mL/min^2

O_2 extraction (O_2 Ext)
= (arteriovenous O_2 difference/arterial blood O_2 content) $\times$ 100
Normal = 20–30%

Pulmonary vascular resistance index (PVRI)
= 79.92 (Mean PAP – $\overline{\text{PAOP}}$)/CI
Normal = 255–285 dyne-sec/cm/5·m^2

Shunt % = (Q_S/Q_T)

$$Q_S Q_T(\ ) = \frac{CcO_2 - CaO_2}{CcO_2 - C\bar{v}O_2}$$

CcO_2 = Hb in g × 1.34 + (Alveolar pO_2 × 0.003)
Normal <10%
Considerable disease = 20–29%
Life-threatening 30%

Stroke volume (SV)
= (end-diastolic volume) – (end-systolic volume)

Systemic vascular resistance index (SVRI)
= 79.92 (MAP – CVP/CI)
Normal = 1970-230 dyne-sec/cm^5m^2

Venous blood O_2 content ($C\bar{v}O_2$)
= (PvO_2 × 0.003) + (1.34 × Hb in g × venous blood Hb O_2 sat %)
Normal = 13–16 mL/dL
Normal hemodynamic parameters are depicted in Tables 3.12, 3.13, and 3.14 below:

Table 3.12. Normal Hemodynamic Parameters, Adult

Parameter	*Equation*	*Normal range*
Arterial blood pressure (BP)	Systolic (SBP)	90–140 mmHg
	Diastolic (DBP)	60–90 mmHg
Mean arterial pressure (MAP)	[SBP + (2 × DBP)]/3	70–105 mmHg
Right atrial pressure (RAP)		2–6 mmHg
Right ventricular pressure (RVP)	Systolic (RVSP)	15–25 mmHg
	Diastolic (RVDP)	0–8 mmHg
Pulmonary artery pressure (PAP)	Systolic (PASP)	15–25 mmHg
	Diastolic (PADP)	8–15 mmHg
Mean pulmonary artery pressure (MPAP)	[PASP + (2 × PADP)]/3	10–20 mmHg
Pulmonary artery wedge pressure (PAWP)		6–12 mmHg
Left atrial pressure (LAP)		6–12 mmHg
Cardiac output (CO)	HR × SV/1,000	4.0–8.0 L/min
Cardiac index (CI)	CO/BSA	2.5–4.0 L/min/m^2
Stroke volume (SV)	CO/HR × 1,000	60–100 mL/beat
Stroke volume index (SVI)	CI/HR × 1,000	33–47 mL/m^2/beat
Systemic vascular resistance (SVR)	80 × (MAP – RAP)/CO	800–1,200 dyness/cm^5
Systemic vascular resistance index (SVRI)	80 × (MAP – RAP)/CI	1970–2390 dyness/cm^5/m^2
Pulmonary vascular resistance (PVR)	80 × (MPAP – PAWP)/CO	<250 dyness/cm^5
Pulmonary vascular resistance index (PVRI)	80 × (MPAP – PAWP)/CI	255–285 dyness/cm^5/m^2

Table 3.13. Hemodynamic Parameters, Adult

Parameter	*Equation*	*Normal Range*
Left ventricular stroke work (LVSW)	SV × (MAP – PAWP) × 0.0136	58–104 g-m/beat
Left ventricular stroke work index (LVSWI)	SVI × (MAP – PAWP) × 0.0136	50–62 g-m/m^2/beat
Right ventricular stroke work (RVSW)	SV × (MPAP – RAP) × 0.0136	8–16 g-m/beat
Right ventricular stroke work index (RVSWI)	SV × (MPAP – RAP) × 0.0136	5–10 g-m/m^2/beat
Coronary artery perfusion pressure (CPP)	Diastolic BP-PAWP	60–80 mmHg
Right ventricular end-diastolic volume (RVEDV)	SV/EF	100–160 mL
Right ventricular end-systolic volume (RVESV)	EDV-SV	50–100 mL
Right ventricular ejection fraction (RVEF)	SV/EDV	40–60%

Table 3.14. Oxygenation Parameters, Adult

Partial pressure of arterial oxygen (PaO_2)		80–100 mmHg
Partial pressure of arterial CO_2 ($PaCO_2$)		35–45 mmHg
Bicarbonate (HCO_3)		22–28 mEq/L
PH		7.38–7.42
Arterial oxygen saturation (SaO_2)		95–100%
Mixed venous saturation ($S\bar{v}O_2$)		60–80%
Arterial oxygen content (CaO_2)	$(0.0138 \times Hb \times SaO_2) + 0.0031 \times PaO_2$	17–20 mL/dL
Venous oxygen content ($C\bar{v}O_2$)	$(0.0138 \times Hb \times S\bar{v}O_2) + 0.0031 \times P\bar{v}O_2$	12–15 mL/dL
A-V oxygen content (CaO_2)	$CaO_2 - C\bar{v}O_2$	4–6 mL/dL
Oxygen delivery ($\dot{D}O_2$)	$CaO_2 \times CO \times 10$	950–1150 mL/dL
Oxygen delivery index ($\dot{D}O_2I$)	$CaO_2 \times CI \times 10$	500–600 mL/min/m^2
Oxygen consumption ($\dot{V}O_2$)	$(C(a-v)O_2) \times CO \times 10$	200–250 mL/min
Oxygen consumption index ($\dot{V}$ O_2I)	$(C(a-v)O_2) \times CI \times 10$	120–160 mL/min/m^2
Oxygen extraction ratio (O_2ER)	$[(CaO_2 - CvO_2)/CaO_2] \times 100$	22–30%
Oxygen extraction index (O_2EI)	$(SaO_2 - SvO_2)/SaO_2 \times 100$	25–25%