

McCance: Pathophysiology, 6th Edition

Chapter 30: Alterations of Cardiovascular Function

Key Points – Print

SUMMARY REVIEW

Diseases of the Arteries and Veins

1. Varicosities are areas of veins in which blood has pooled, usually in the saphenous veins. Varicosities may be caused by damaged valves as a result of trauma to the valve or by chronic venous distention involving gravity and venous constriction.
2. Chronic venous insufficiency is inadequate venous return over a long period that causes pathologic ischemic changes in the vasculature, skin, and supporting tissues.
3. Venous stasis ulcers follow the development of chronic venous insufficiency and probably develop as a result of the borderline metabolic state of the cells in the affected extremities.
4. DVT occurs in individuals who have venous stasis (immobility, age, left heart failure), spinal cord injury, vein wall damage (trauma, intravenous medications), or hypercoagulable states (pregnancy, oral contraceptives, malignancy, genetic coagulopathies).
5. DVT is often asymptomatic but may lead to fatal pulmonary emboli; prevention and careful assessment in individuals at risk are crucial.
6. An aneurysm is a localized dilation of a vessel wall to which the aorta is particularly susceptible.
7. A thrombus is a clot that remains attached to a vascular wall.
8. An embolus is a mobile aggregate of a variety of substances that occludes the vasculature. Sources of emboli include thrombi, air, amniotic fluid, bacteria, fat, and foreign matter.
9. The most common sources of arterial thrombotic emboli from the heart are mitral and aortic valvular disease and atrial fibrillation. Tissues affected include the lower extremities, the brain, and the heart.
10. Emboli to the central organs cause tissue death in lungs, kidneys, and mesentery.
11. The generation of air emboli requires a connection between the vascular compartment and a source of air. These emboli cause ischemia and necrosis when a vessel is totally blocked.
12. Amniotic fluid may be forced into the bloodstream and generate an embolus during the labor and delivery of pregnancy.
13. Aggregates of bacteria in the vasculature may be large enough to form an embolus.
14. Fat emboli are caused mainly by trauma to the long bones, either through defective fat metabolism after trauma or through the release of fat globules from bone marrow exposed by fracture.
15. The introduction of foreign matter into the vasculature can occur with trauma and also can occur in a hospital setting in which intravenous and intra-arterial lines are being used.

16. Vasospastic disorders include Raynaud disease, involving arterioles of the extremities; variant angina, involving coronary arteries; and Buerger disease, involving arteries of the hands and feet.
17. Hypertension is a sustained elevation of the system arterial blood pressure resulting from increases in cardiac output or total peripheral resistance or both. Hypertension can be primary (without known cause) or secondary (caused by disease or drugs). Systolic hypertension is the most significant factor in causing target organ damage.
18. The risk factors for hypertension include a positive family history; male gender; advanced age; black race; obesity; high sodium intake; low potassium, calcium, and magnesium intake; diabetes mellitus; labile blood pressure; cigarette smoking; and heavy alcohol consumption.
19. Primary hypertension is the result of extremely complicated interactions of genetics and the environment mediated by a host of neurohumoral effects. These genes interact with diet, smoking, age, and the other risk factors to cause chronic changes in vasomotor tone and blood volume.
20. The most frequently cited theories of the pathogenesis of primary hypertension include overactivity of the SNS; overactivity of the RAAS; alterations in other neurohumoral mediators of blood volume and vasomotor tone such as ANP, BNP, and adrenomedullin; inflammation; a complex interaction involving insulin resistance and endothelial function; and obesity-related hormonal changes.
21. Clinical manifestations of hypertension result from damage of organs and tissues outside the vascular system. These include heart disease, renal disease, CNS problems, and retinal changes.
22. Hypertension is managed pharmacologically, using diuretics, adrenergic blockers, calcium channel blockers, ACE inhibitors, and Ang II receptor blockers. Nonpharmacologic methods include cessation of smoking, dietary modifications, and exercise.
23. Orthostatic hypotension is a drop in blood pressure that occurs on standing. The compensatory vasoconstriction response to standing is altered by a marked vasodilation and blood pooling in the muscle vasculature.
24. Orthostatic hypotension may be acute or chronic. The acute form is caused by a delay in the normal regulatory mechanisms. The chronic forms are secondary to a specific disease or are idiopathic in nature.
25. The clinical manifestations of orthostatic hypotension include fainting and may involve cardiovascular symptoms, as well as impotence and bowel and bladder dysfunction.
26. Atherosclerosis is a form of arteriosclerosis and is the leading cause of coronary artery and cerebrovascular disease.
27. Atherosclerosis is an inflammatory disease that begins with endothelial and progresses through several stages to become a fibrotic plaque.
28. Traditional risk factors include age, family history, gender, smoking, dyslipidemia, hypertension, and diabetes.

29. Novel risk factors include elevated CRP, increased serum fibrinogen, oxidative stress, infection, and periodontal disease.
30. Once a plaque has formed, it can rupture resulting in thrombosis and vasoconstriction leading to obstruction of the lumen and inadequate perfusion of distal tissues.
31. PAD is atherosclerosis of arteries that perfuse the limbs, especially the lower extremities.
32. PAD is often asymptomatic but can present with intermittent claudication (pain in leg on walking). Treatment includes risk factor reduction and antiplatelet therapy.
33. CAD is spasm or occlusion of the coronary arteries and is most often the result of atherosclerotic lesions that limit the flow of blood to the heart.
34. Many risk factors contribute to the onset and escalation of CAD, including advanced age, male gender (younger than age 60), hypertension, dyslipidemia (including elevated Lp[a]), diabetes mellitus, smoking, obesity, sedentary lifestyle, psychosocial factors, elevated CRP, and possibly infectious agents.
35. CAD results in an imbalance between coronary supply of blood and myocardial demand for oxygen and nutrients such that reversible myocardial ischemia or irreversible infarction may result.
36. Reversible myocardial ischemia presents clinically in several ways. Chronic coronary obstruction results in recurrent predictable chest pain called *stable angina*. Abnormal vasospasm of coronary vessels results in unpredictable chest pain called *Prinzmetal angina*. Myocardial ischemia that does not cause detectable symptoms is called *silent ischemia*.
37. Stable angina is evaluated by noninvasive techniques of assessing coronary flow with or without exercise (stress ECG, thallium, or SPECT). Management may include lifestyle changes, vasodilators, antithrombotics, PCI, or CABG surgery.
38. When there is sudden coronary obstruction because of thrombosis formation over a ruptured atherosclerotic plaque, the acute coronary syndromes result. Unstable angina causes reversible myocardial ischemia and is a harbinger of impending infarction. Myocardial infarction results when prolonged ischemia causes irreversible damage to the heart muscle. Sudden cardiac death can occur in any of the acute coronary syndromes.
39. Unstable angina occurs because of transient episodes of thrombotic vessel occlusion and vasoconstriction at the site of plaque damage, with return of perfusion before significant myocardial necrosis occurs. This must be managed aggressively with antithrombotic agents to prevent myocardial infarction.
40. When coronary blood flow is interrupted for an extended period, myocyte necrosis occurs; this is called MI. Pathologically, there are two major types of myocardial infarction: subendocardial infarction and transmural infarction. In addition to myocyte necrosis, other changes in the heart with MI include hibernating, stunning, and remodeling of the myocardium.
41. Acute coronary syndromes are assessed by measuring serum enzymes, such as creatinine kinase and troponins, as well as looking for characteristic changes in the ECG. Those individuals at highest risk for complications present with ST segment elevations on the ECG.

(STEMI) and require immediate intervention. Smaller subendocardial infarctions are not associated with ST segment elevations (non-STEMI) but suggest that additional myocardium is still at risk for recurrent ischemia and infarction. Management may include thrombolytic drugs, antithrombotic drugs, vasodilators, PCI, or immediate surgery.

42. Dysrhythmias, congestive heart failure, and sudden death are the most common complications of the acute coronary syndromes.

Disorders of the Heart Wall

1. Inflammation of the pericardium (pericarditis) may result from innumerable sources (infection, drug therapy, tumors). Pericarditis presents with symptoms that are physically troublesome, but in and of themselves they are not life threatening.
2. Fluid may collect within the pericardial sac (pericardial effusion). Cardiac function may be severely impaired if a large volume of fluid accumulates rapidly.
3. Cardiomyopathies are a diverse group of primary myocardial disorders that are poorly understood. The cardiomyopathies are categorized as dilated (congestive), restrictive (rigid and noncompliant), and hypertrophic (asymmetric). Size of the cardiac muscle walls and chambers may increase or decrease, depending on the type of cardiomyopathy, thereby altering contractile activity.
4. Hemodynamic integrity of the cardiovascular system depends to a great extent on properly functioning cardiac valves. Congenital or acquired disorders that result in stenosis or incompetence or both can structurally alter the valves.
5. Characteristic heart sounds, cardiac murmurs, and systemic complaints assist in determination of which valve is abnormal. If severely compromised function exists, a prosthetic heart valve may be surgically implanted to replace the faulty one.
6. Mitral valve prolapse is a common finding, especially in young women. Although not grossly abnormal, the mitral valve leaflets do not position themselves properly during systole. MVP may be a completely asymptomatic condition, or it may result in severe subjective symptoms. Afflicted valves may be at greater risk for developing infective endocarditis.
7. Rheumatic fever is an inflammatory disease that results from a delayed autoimmune response to a streptococcal infection. The disorder usually resolves without sequelae if treated early.
8. Severe or untreated cases of rheumatic fever may progress to rheumatic heart disease, a potentially disabling cardiovascular disorder.
9. Infective endocarditis is a general term for infection and inflammation of the endocardium, especially the cardiac valves. A wide range of conditions predisposes one to the development of this disorder. In the mildest cases, valvular function may be slightly impaired by vegetations that collect on the valve leaflets. If infective endocarditis is left unchecked, severe valve abnormalities, chronic bacteremia, and systemic emboli may occur as vegetations break off the valve surface and travel through the bloodstream. Antibiotic therapy can limit the extent of this disease.

10. HIV is associated with cardiac abnormalities, including myocarditis, endocarditis, pericarditis, and cardiomyopathy. Left heart failure is the most common clinical manifestation.

Manifestations of Heart Disease

1. Heart failure is an inability of the heart to supply the metabolism with adequate circulatory volume and pressure.
2. Left heart failure can be categorized as systolic heart failure or diastolic heart failure.
3. Systolic heart failure is defined as an inability of the heart to generate an adequate cardiac output to perfuse vital tissue.
4. Cardiac output depends on the heart rate and stroke volume. Stroke volume is influenced by contractility, preload, and afterload. Myocardial infarction is the most common cause of decreased contractility. Myocardial ischemia results in ventricular remodeling that causes progressive myocyte contractile dysfunction over time.
5. Preload LVEDV is increased when there is decreased contractility or excess plasma volume.
6. Increased afterload is most commonly the result of increased peripheral vascular resistance. This increase in resistance decreases ventricular emptying and makes more workload for the left ventricle, resulting in hypertrophy and ventricular remodeling. The vicious cycle of decreasing contractility, increasing preload, and increasing afterload causes progressive worsening.
7. Neurohumoral mechanisms of CHF include abnormalities in the SNS, RAAS, arginine vasopressin, natriuretic peptides, inflammatory cytokines, and in myocyte metabolism.
8. The clinical manifestations of left heart failure are the result of pulmonary vascular congestion and inadequate systemic perfusion.
9. Management of left heart failure relies on increasing contractility and reducing preload and afterload.
10. Diastolic heart failure can occur singly or with systolic heart failure. The major causes of diastolic dysfunction include hypertension-induced myocardial hypertrophy and ischemia with resultant ventricular remodeling.
11. Right heart failure can result from left heart failure and/or diffuse hypoxic pulmonary disease, such as COPD, cystic fibrosis, and ARDS. These mechanisms are discussed in Chapter 33[AU1].
12. High output failure is the inability of the heart to adequately supply the body with blood-borne nutrients despite adequate volume and normal or elevated myocardial contractility. Common causes are anemia, septicemia, hyperthyroidism, and beriberi.

13. A dysrhythmia (arrhythmia) is a disturbance of heart rhythm. Dysrhythmias range in severity from occasional missed beats or rapid beats to disturbances that impair myocardial contractility and are life threatening.
14. Dysrhythmias can occur because of an abnormal rate of impulse generation or the abnormal conduction of impulses.