Pathophsiology

Disorder of cardiovascular system

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HYPEREMIA AND CONGESTION

Hyperemia and congestion both refer to an increase in blood volume within a tissue but they have different underlying mechanisms.

HYPEREMIA

- Hyperemia is an active process resulting from arteriolar dilation and increased blood inflow.
- It occurs at sites of inflammation or in exercising skeletal muscle.
- Hyperemic tissues are redder than normal because of engorgement with oxygenated blood.

CONGESTION

- Congestion is a passive process resulting from impaired outflow of venous blood from a tissue.
- It can occur systemically, as in cardiac failure, or locally as a consequence of an isolated venous obstruction.
- Congested tissues have an abnormal blue-red color (cyanosis) that stems from the accumulation of deoxygenated hemoglobin in the affected area.

Edema is extravasation of fluid from vessels into interstitial spaces; the fluid may be protein-poor (transudate) or protein-rich (exudate).

Edema may be caused by:

- increased hydrostatic pressure (e.g., heart failure)
- increased vascular permeability (e.g., inflammation)
- decreased colloid osmotic pressure, due to reduced plasma albumin • decreased synthesis (e.g., liver disease, protein malnutrition) • increased loss (e.g., nephrotic syndrome)
- lymphatic obstruction (e.g., inflammation or neoplasia).
- sodium retention (e.g., renal failure)

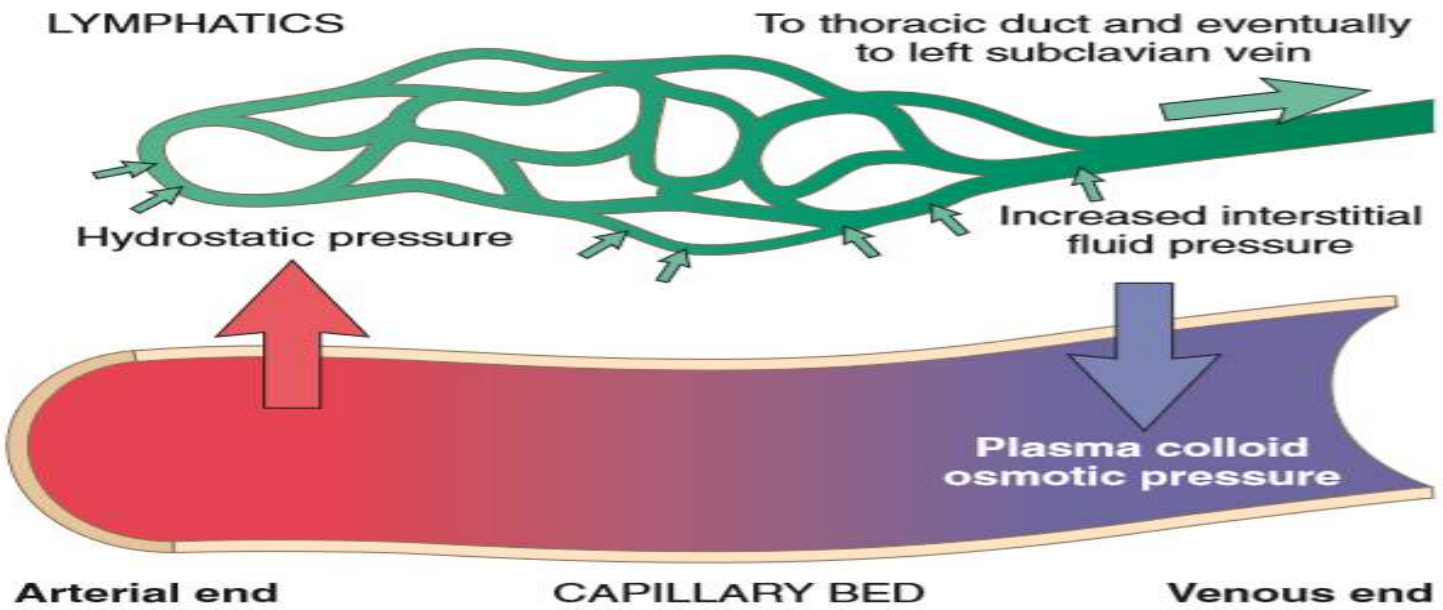


Figure 3–2 Factors influencing fluid movement across capillary walls. Capillary hydrostatic and osmotic forces are normally balanced so there is little net movement of fluid into the interstitium. However, *increased* hydrostatic pressure or *diminished* plasma osmotic pressure leads to extravascular fluid accumulation (edema). Tissue lymphatics drain much of the excess fluid back to the circulation by way of the thoracic duct; however, if the capacity for lymphatic drainage is exceeded, tissue edema results.

Hemostasis and Thrombosis

Normal hemostasis comprises a series of regulated processes that maintain blood in a fluid, clot-free state in normal vessels while rapidly forming a localized hemostatic plug at the site of vascular injury. The pathologic counterpart of hemostasis is thrombosis, the formation of blood clot (thrombus) within intact vessels.

cascade.

Thrombus development usually is related to one or more components of Virchow's triad:

- endothelial injury (e.g., by toxins, hypertension, inflammation, or metabolic products)
- abnormal blood flow, stasis or turbulence (e.g., due to aneurysms, atherosclerotic plaque)
- hypercoagulability: either primary (e.g., factor V Leiden, increased prothrombin synthesis, antithrombin III deficiency) or secondary (e.g., bed rest, tissue damage, malignancy).

• Thrombi may propagate, resolve, become organized, or embolize.

• Thrombosis causes tissue injury by local vascular occlusion or by distal embolization.

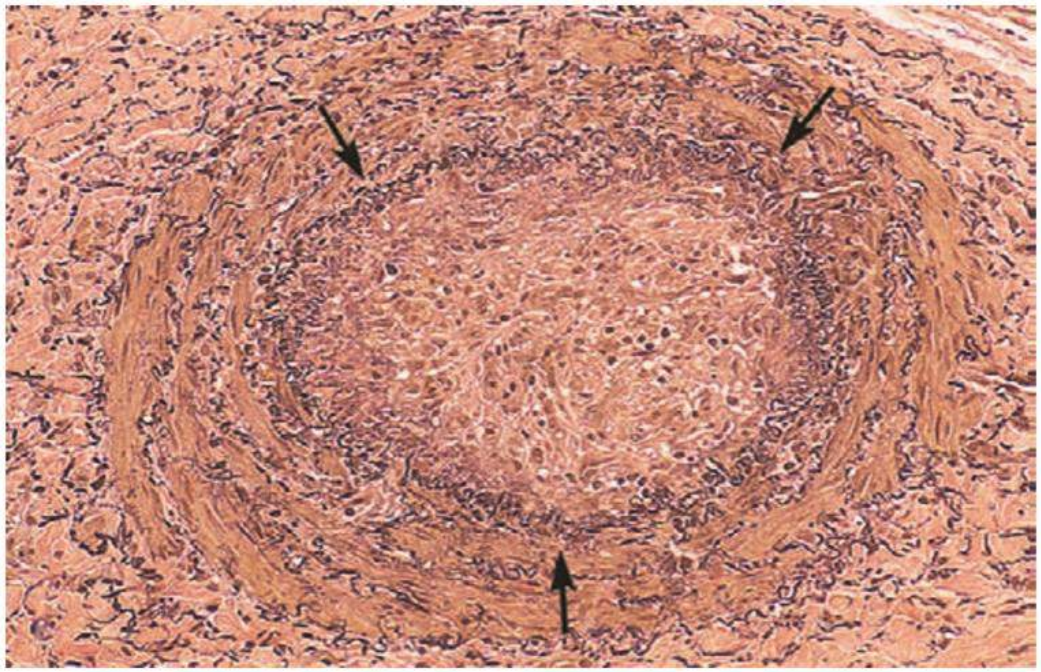


Figure 3–14 Low-power view of a thrombosed artery stained for elastic tissue. The original lumen is delineated by the internal elastic lamina *(arrows)* and is totally filled with organized thrombus.

Embolism

An embolus is an intravascular solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin.

The vast majority of emboli derive from a dislodged thrombus—hence the term **thromboembolism**.

Less common types of emboli include fat droplets, bubbles of air or nitrogen, atherosclerotic debris **(cholesterol emboli),** tumor fragments, bits of bone marrow, and amniotic fluid.

Inevitably, emboli lodge in vessels too small to permit further passage, resulting in partial or complete vascular occlusion; depending on the site of origin, emboli can lodge anywhere in the vascular tree.

The primary consequence of systemic embolization is ischemic necrosis **(infarction)** of downstream tissues, while embolization in the pulmonary circulation leads to hypoxia, hypotension, and right-sided heart failure.

Figure 3–15 Embolus derived from a lower-extremity deep venous thrombus lodged in a pulmonary artery branch.

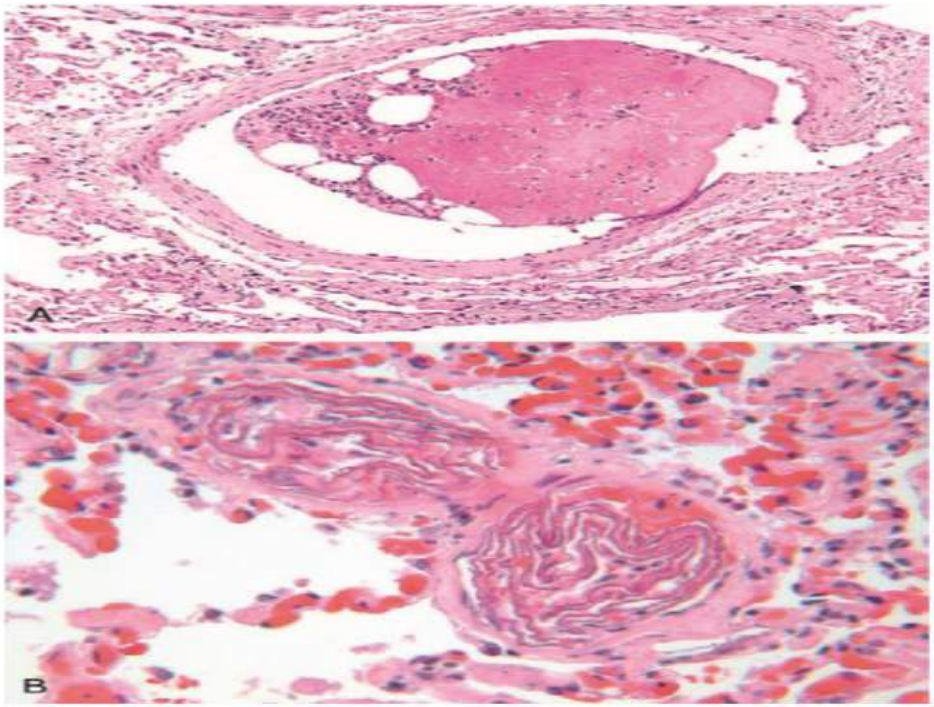


Figure 3–16 Unusual types of emboli. **A,** Bone marrow embolus. The embolus is composed of hematopoietic marrow and marrow fat cells (*clear spaces*) attached to a thrombus. **B,** Amniotic fluid emboli. Two small pulmonary arterioles are packed with laminated swirls of fetal squamous cells. The surrounding lung is edematous and congested.

Infarction

- Infarcts are areas of ischemic necrosis most commonly caused by arterial occlusion (typically due to thrombosis or embolization); venous outflow obstruction is a less frequent cause.
- Infarcts caused by venous occlusion or occurring in spongy tissues typically are hemorrhagic (red); those caused by arterial occlusion in compact tissues typically are pale (white).
- Whether or not vascular occlusion causes tissue infarction is influenced by collateral blood supplies, the rate at which an obstruction develops, intrinsic tissue susceptibility to ischemic injury, and blood oxygenation

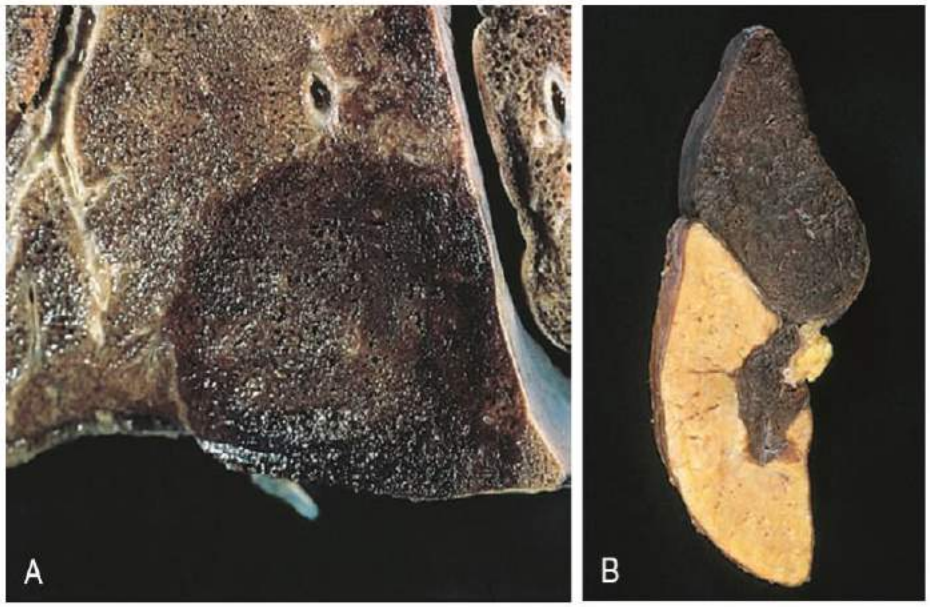


Figure 3–17 Red and white infarcts. **A,** Hemorrhagic, roughly wedge-shaped pulmonary infarct (red infarct). **B,** Sharply demarcated pale infarct in the spleen (white infarct).

Shock

Shock is the final common pathway for several potentially lethal events, including exsanguination, extensive trauma or burns, myocardial infarction, pulmonary embolism, and sepsis.

Shock is **characterized by** systemic hypoperfusion of tissues; it can be caused by diminished cardiac output or by reduced effective circulating blood volume.

The consequences are impaired tissue perfusion and cellular hypoxia. Although shock initially is reversible, prolonged shock eventually leads to irreversible tissue injury that often proves fatal.

Stages of Shock

Unless the insult is massive and rapidly lethal (e.g., exsanguination from a ruptured aortic aneurysm), shock tends to evolve through three general (albeit somewhat artificial) stages. These stages have been documented most clearly in hypovolemic shock but are common to other forms as well:

- An initial nonprogressive stage, during which reflex compensatory mechanisms are activated and vital organ perfusion is maintained
- A progressive stage, characterized by tissue hypoperfusion and onset of worsening circulatory and metabolic derangement, including acidosis
- An irreversible stage, in which cellular and tissue injury is so severe that even if the hemodynamic defects are corrected, survival is not possible .

The most common forms of shock can be grouped into three pathogenic categories :

- **Cardiogenic shock** results from low cardiac output due to myocardial pump failure. It may be caused by myocardial damage (infarction), ventricular arrhythmias, extrinsic compression (cardiac tamponade) , or outflow obstruction (e.g., pulmonary embolism).
- **Hypovolemic shock** results from low cardiac output due to loss of blood or plasma volume (e.g., due to hemorrhage or fluid loss from severe burns).
- **Septic shock** results from arterial vasodilation and venous blood pooling that stems from the systemic immune response to microbial infection.

Pathogenesis of Septic Shock

In septic shock, systemic arterial and venous dilation leads to tissue hypoperfusion, even though cardiac output is preserved or even initially increased. The decreased vascular tone is accompanied by widespread endothelial cell activation, often triggering a hypercoagulable state manifesting as disseminated intravascular coagulation.

In addition, septic shock is associated with perturbations of metabolism that directly suppress cell and tissue function. The net effect of these abnormalities is hypoperfusion and dysfunction of multiple organs.

Hemorrhage extravasation of blood from vessels, occurs in a variety of settings.

The risk of hemorrhage is increased in a wide variety of clinical disorders collectively called hemorrhagic diatheses.

Trauma, atherosclerosis, or inflammatory or neoplastic erosion of a vessel wall also may lead to hemorrhage, which may be extensive if the affected vessel is a large vein or artery.

Hemorrhage may be manifested by different appearances and clinical consequences.

➢ Hemorrhage may be external or accumulate within a tissue as a hematoma, which ranges in significance from trivial (e.g., a bruise) to fatal (e.g., a massive retroperitoneal hematoma resulting from rupture of a dissecting aortic aneurysm) . Large bleeds into body cavities are given various names according to location—hemothorax, hemopericardium, hemoperitoneum, or hemarthrosis (in joints). Extensive hemorrhages can occasionally result in jaundice from the massive breakdown of red cells and hemoglobin.

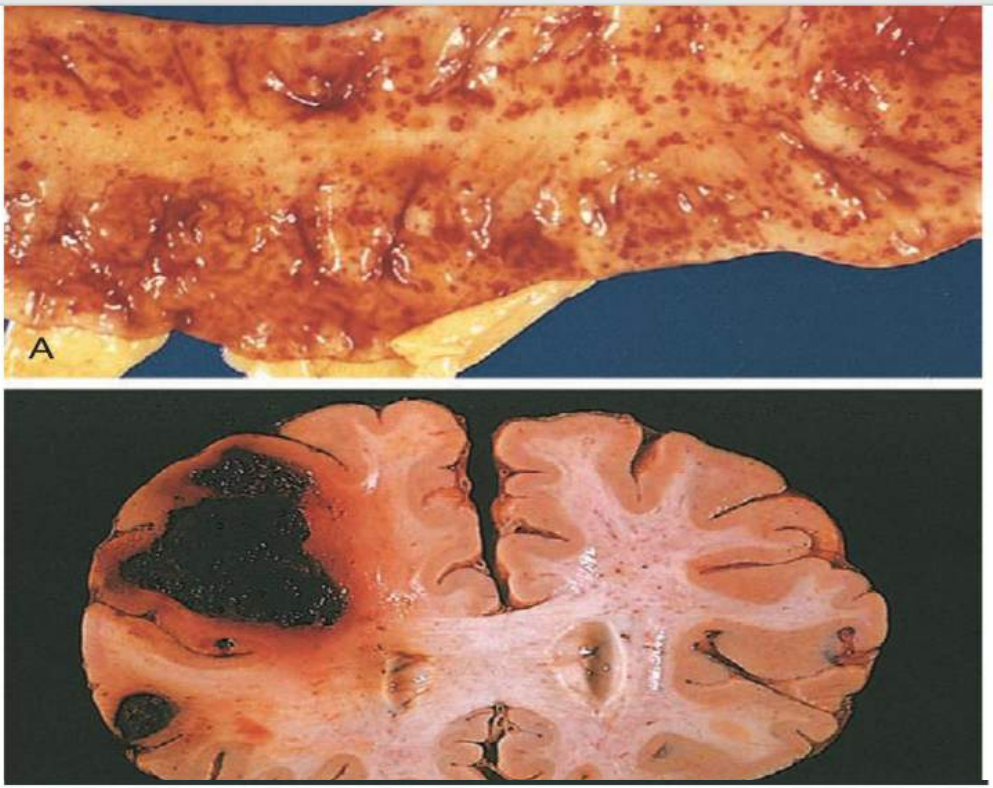


Figure 3–4 A, Punctate petechial hemorrhages of the colonic mucosa, a consequence of thrombocytopenia. **B,** Fatal intracerebral hemorrhage.

➢Petechiae are minute (1 to 2 mm in diameter) hemorrhages into skin, mucous membranes, or serosal surfaces causes include low platelet counts (thrombocy topenia), defective platelet function, and loss of vascular wall support, as in vitamin C deficiency .

➢ Purpura are slightly larger (3 to 5 mm) hemorrhages. Purpura can result from the same disorders that cause petechiae, as well as trauma, vascular inflammation .

Coronary Artery Disease

The term coronary artery disease (CAD) describes heart disease caused by impaired coronary blood flow. Diseases of the coronary arteries can cause a spectrum of disorders ranging from myocardial ischemia and angina to myocardial infarction or heart attack, conduction defects, heart failure, and sudden death.

Ischemic Heart Disease

The manifestations of IHD are a direct consequence of the insufficient blood supply to the heart.

The clinical presentation may include one or more of the following cardiac syndromes:

- **Angina pectoris** (literally, “chest pain”): Ischemia induces pain but is insufficient to cause myocyte death. Angina can be stable (occurring predictably at certain levels of exertion), can be caused by vessel spasm (Prinzmetal angina), or can be unstable (occurring with progressively less exertion or even at rest).
- **Acute myocardial infarction** (MI): The severity or duration of ischemia is sufficient to cause cardiomyocyte death.
- **Chronic IHD with CHF**: Progressive cardiac decompensation after acute MI, or secondary to accumulated small ischemic insults, eventually precipitates mechanical pump failure.

➢**Sudden cardiac death (SCD):** This can occur as a consequence of tissue damage from MI, but most commonly results from a lethal arrhythmia without myocyte necrosis .The term acute coronary syndrome is applied to any of the three catastrophic manifestations of IHD—unstable angina, acute MI, and SCD.

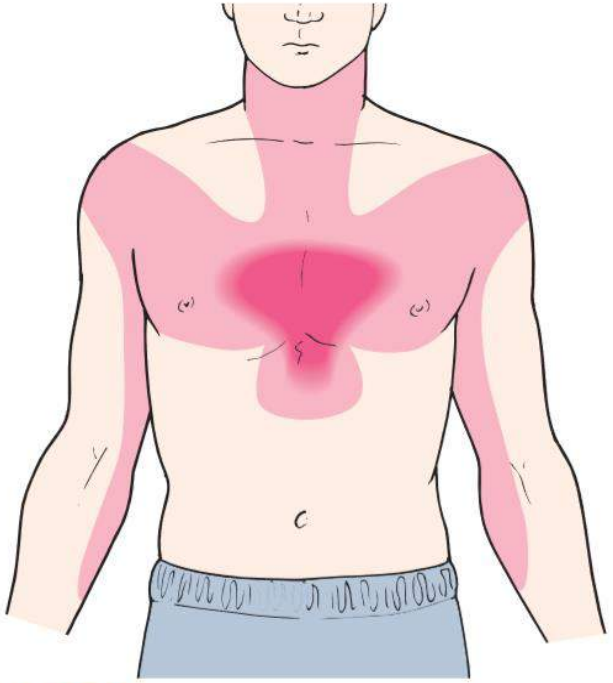


FIGURE 19-11. Areas of pain due to angina.

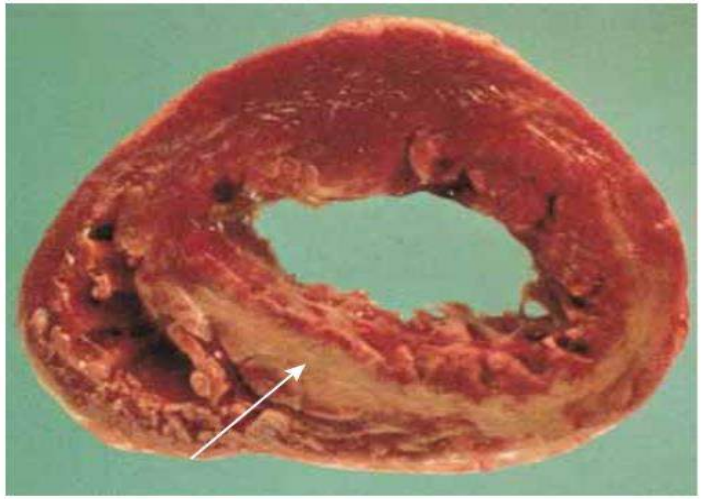


FIGURE 19-6. Acute myocardial infarct. A cross-section of the ventricles of a man who died a few days after the onset of severe chest pain shows a transmural infarct in the posterior and septal regions of the left ventricle. The necrotic myocardium is soft, yellowish, and sharply demarcated. (From Rubin E., Farber J.L. [1999]. *Pathology* [3rd ed., p. 558]. Philadelphia: Lippincott Williams & Wilkins.)

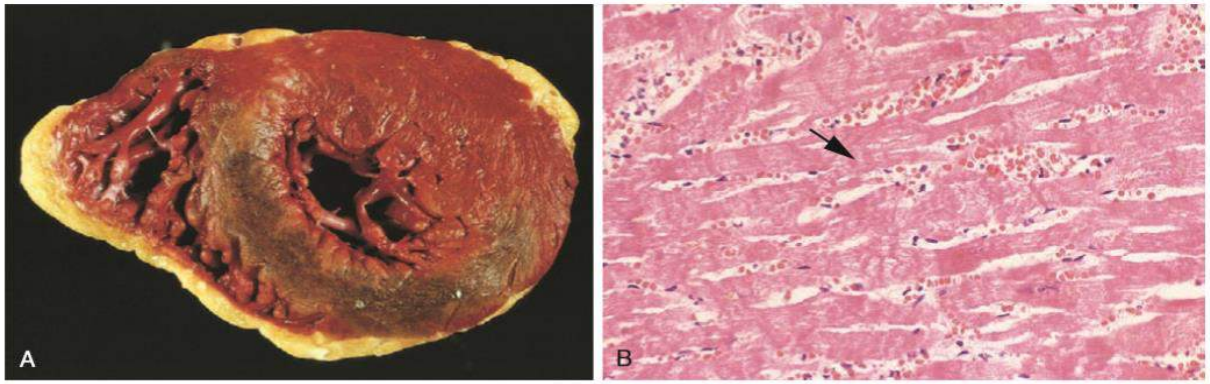


Figure 10–12 Reperfused myocardial infarction. **A,** The transverse heart slice (stained with triphenyl tetrazolium chloride) exhibits a large anterior wall myocardial infarction that is hemorrhagic because of bleeding from damaged vessels. Posterior wall is at *top*. **B,** Hemorrhage and contraction bands, visible as prominent hypereosinophilic cross-striations spanning myofibers (*arrow*), are seen microscopically.

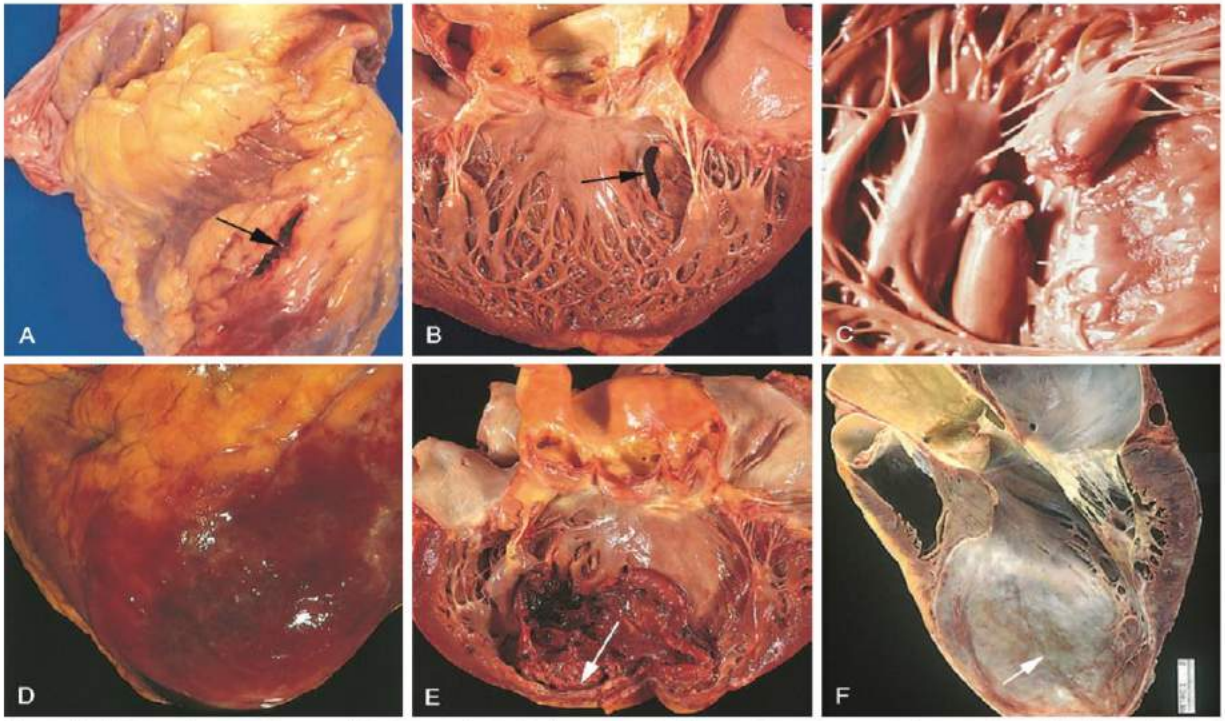


Figure 10–14 Complications of myocardial infarction. **A–C,** Cardiac rupture. **A,** Anterior free wall myocardial rupture (*arrow*). **B,** Ventricular septal rupture (*arrow*). **C,** Papillary muscle rupture. **D,** Fibrinous pericarditis, with a hemorrhagic, roughened epicardial surface overlying an acute infarct. **E,** Recent expansion of an anteroapical infarct with wall stretching and thinning (*arrow*) and mural thrombus. **F,** Large apical left ventricular aneurysm (*arrow*).

(A–E, Reproduced by permission from Schoen FJ: Interventional and Surgical Cardiovascular Pathology: Clinical Correlations and Basic Principles. Philadelphia, WB Saunders, 1989; F, Courtesy of

Rheumatic Valvular Disease

Rheumatic fever is an acute, immunologically mediated, multisystem inflammatory disease that occurs after group A β-hemolytic streptococcal infections (usually pharyngitis, but also rarely with infections at other sites such as skin).

Rheumatic heart disease is the cardiac manifestation of rheumatic fever.

It is associated with inflammation of all parts of the heart, but valvular inflammation and scarring produces the most important clinical features.

The valvular disease principally takes the form of deforming fibrotic mitral stenosis; indeed rheumatic heart disease is essentially the only cause of acquired mitral stenosis.

Pathogenesis

Acute rheumatic fever is a hypersensitivity reaction classically attributed to antibodies directed against group A streptococcal molecules that also are cross-reactive with host antigens.

In particular, antibodies against M proteins of certain streptococcal strains bind to proteins in the myocardium and cardiac valves and cause injury through the activation of complement and Fc receptor–bearing cells (including macrophages). CD4+ T cells that recognize streptococcal peptides also can cross-react with host antigens and elicit cytokine-mediated inflammatory responses.

The characteristic 2- to 3-week delay in symptom onset after infection is explained by the time needed to generate an immune response; streptococci are completely absent from the lesions. Since only a small minority of infected patients develop rheumatic fever (estimated at 3%), a genetic susceptibility is likely to influence the development of the crossreactive immune responses. The chronic fibrotic lesions are the predictable consequence of healing and scarring associated with the resolution of the acute inflammation.

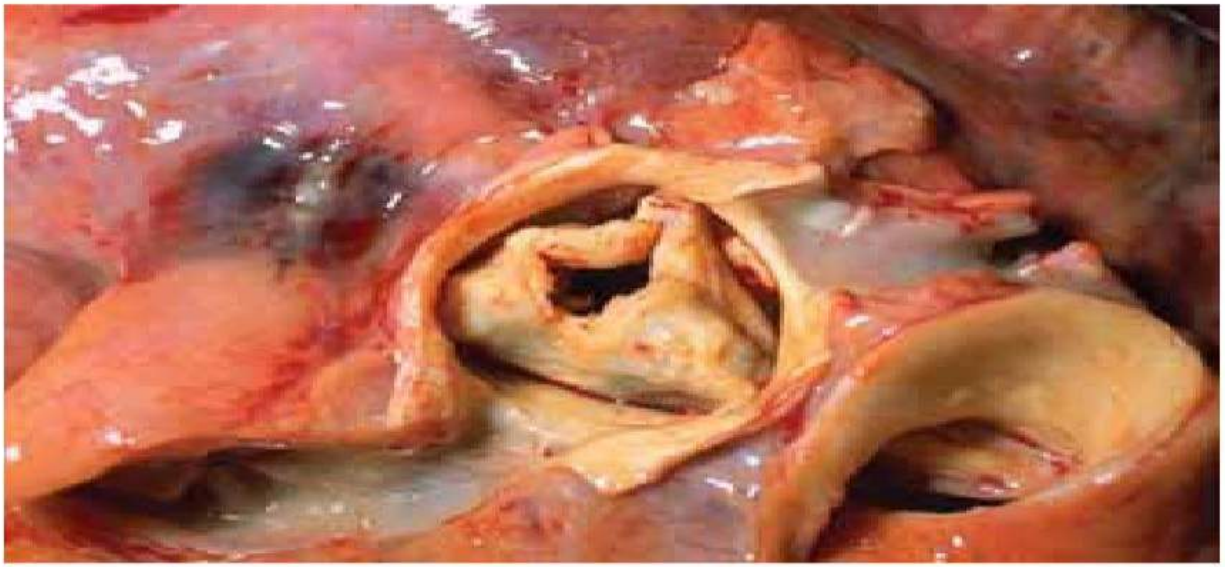


FIGURE 19-16. Gross pathology of rheumatic heart disease: aortic stenosis. Fused aortic valve leaflets and opened coronary arteries from above. (From Centers for Disease Control and Prevention Public Images Library. [Online].)

Atherosclerosis

- Atherosclerosis is characterized by the presence of intimal lesions called atheromas (or atheromatous or atherosclerotic plaques). Atheromatous plaques are raised lesions composed of soft grumous lipid cores (mainly cholesterol and cholesterol esters, with necrotic debris) covered by fibrous caps .
- Atherogenesis is driven by an interplay of vessel wall injury and inflammation. The multiple risk factors for atherosclerosis all cause endothelial cell dysfunction and influence smooth muscle cell recruitment and stimulation.
- Atherosclerotic plaques develop and grow slowly over decades. Stable plaques can produce symptoms related to chronic ischemia by narrowing vessels, whereas unstable plaques can cause dramatic and potentially fatal ischemic complications related to acute plaque rupture, thrombosis, or embolization.
- Stable plaques tend to have a dense fibrous cap, minimal lipid accumulation, and little inflammation, whereas “vulnerable” unstable plaques have thin caps, large lipid cores, and relatively dense inflammatory infiltrates.

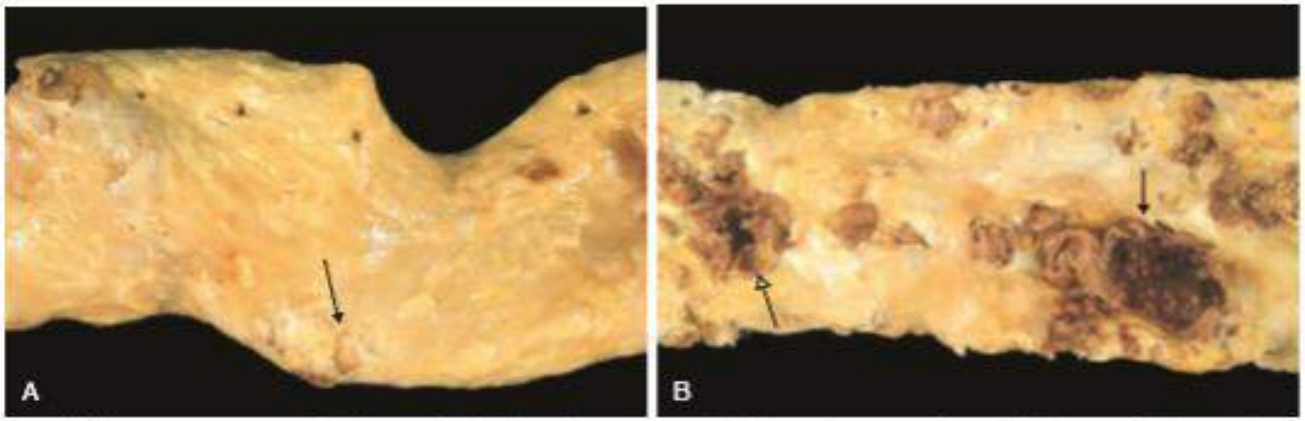


Figure 9–12 Atherosclerotic lesions. **A,** Aorta with mild atherosclerosis composed of fibrous plaques, one denoted by the *arrow*. **B,** Aorta with severe diffuse complicated lesions, including an ulcerated plaque (*open arrow*), and a lesion with overlying thrombus (*closed arrow*).

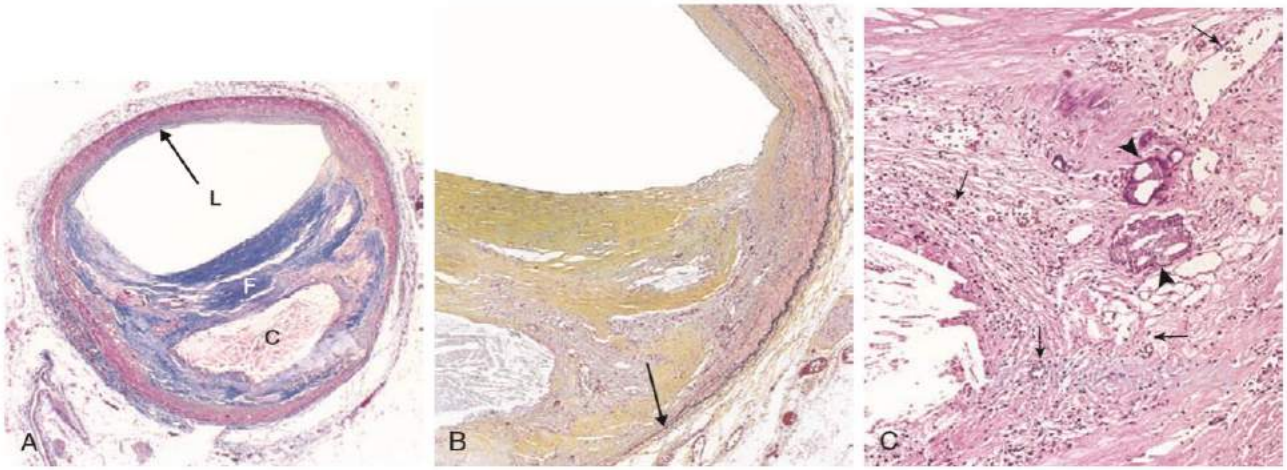


Figure 9–13 Atherosclerotic plaque in the coronary artery. **A,** Overall architecture demonstrating fibrous cap (F) and a central necrotic (largely lipid) core (C); collagen (*blue*) is stained with Masson trichrome. The lumen (L) is moderately narrowed by this eccentric lesion, which leaves part of the vessel wall unaffected (*arrow*). **B,** Moderate-power view of the plaque shown in **A,** stained for elastin (*black*); the internal and external elastic membranes are attenuated and the media of the artery is thinned under the most advanced plaque (*arrow*). **C,** High-power view of the junction of the fibrous cap and core, showing scattered inflammatory cells, calcification (*arrowheads*), and neovascularization (*small arrows*).

Heart Failure

- CHF occurs when the heart is unable to provide adequate perfusion to meet the metabolic requirements of peripheral tissues; inadequate cardiac output usually is accompanied by increased congestion of the venous circulation.
- Left-sided heart failure is most commonly secondary to ischemic heart disease, systemic hypertension, mitral or aortic valve disease, or primary diseases of the myocardium; symptoms are mainly a consequence of pulmonary congestion and edema, although systemic hypoperfusion can cause renal and cerebral dysfunction.
- Right-sided heart failure is due most often to left heart failure and, less commonly, to primary pulmonary disorders; signs and symptoms are related chiefly to peripheral edema and visceral congestion.

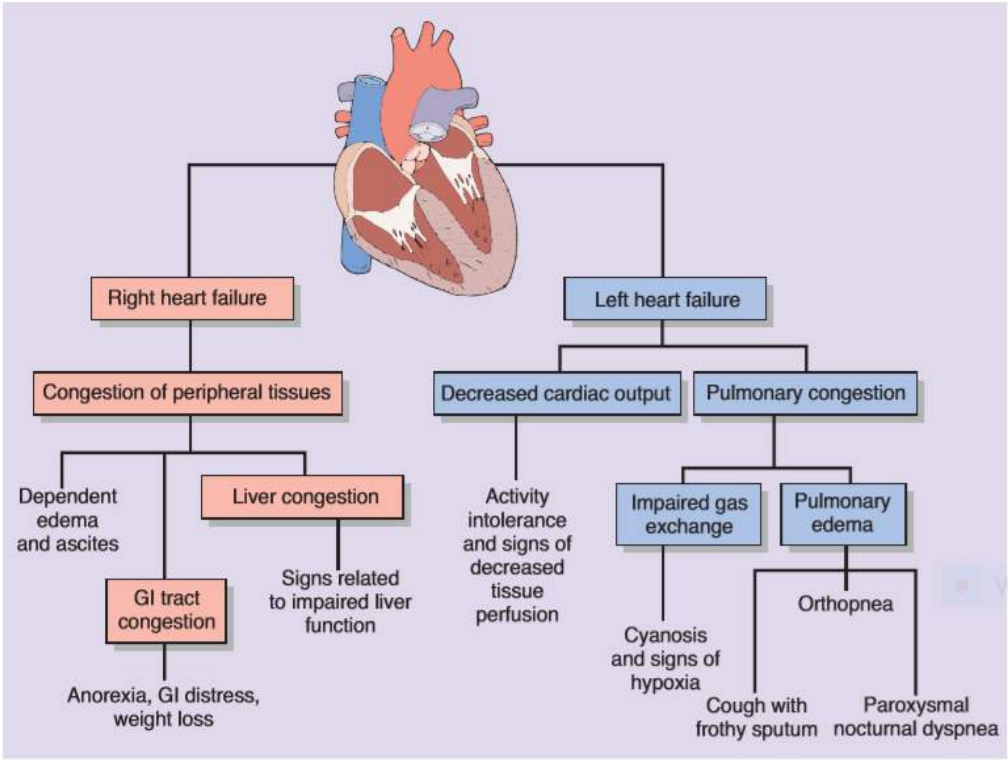


FIGURE 20-2. Manifestations of right- and left-sided heart failure. GI, gastrointestinal.

Acute Pulmonary Edema

Acute pulmonary edema is the most dramatic symptom of CHF. It is a life-threatening condition in which capillary fluid moves into the alveoli.

The accumulated fluid in the alveoli and airways causes lung stiffness, makes lung expansion more difficult, and impairs the gas exchange function of the lung. With the decreased ability of the lungs to oxygenate the blood, the hemoglobin leaves the pulmonary circulation without being fully oxygenated, resulting in shortness of breath and cyanosis.

and coarser.

Pathogenesis of CPE

Left sided heart failure

↓

Decrease pumping ability to the systemic circulation

↓

Congestion & accumulation of blood in the pulmonary area

↓

Fluid leaks out of the intravascular space to the interstitium

↓

Accumulation of fluid

↓

Pulmonary edema

Morphology

Lungs. Rising pressure in the pulmonary veins is ultimately transmitted back to the capillaries and arteries of the lungs, resulting in congestion and edema as well as pleural effusion due to an increase in hydrostatic pressure in the venules of the visceral pleura. The lungs are heavy and boggy, and **microscopically** show perivascular and interstitial transudates, alveolar septal edema, and accumulation of edema fluid in the alveolar spaces. In addition, variable numbers of red cells extravasate from the leaky capillaries into alveolar spaces, where they are phagocytosed by macrophages The subsequent breakdown of red cells and hemoglobin leads to the appearance of hemosiderin-laden alveolar macrophages— **so-called heart failure cells**—that reflect previous episodes of pulmonary edema.

Hypertensive Vascular Disease

Hypertension is a major health problem in the developed world. Although it occasionally manifests in an acute aggressive form, high blood pressure is much more often asymptomatic for many years.

This insidious condition is sometimes referred to as benign hypertension, but it is in fact far from harmless. Besides increasing the risk of stroke and atherosclerotic coronary heart disease, hypertension can lead to cardiac hypertrophy and heart failure (hypertensive heart disease), aortic dissection, multi-infarct dementia, and renal failure.

Table 9–2 Types and Causes of Hypertension (Systolic and Diastolic)

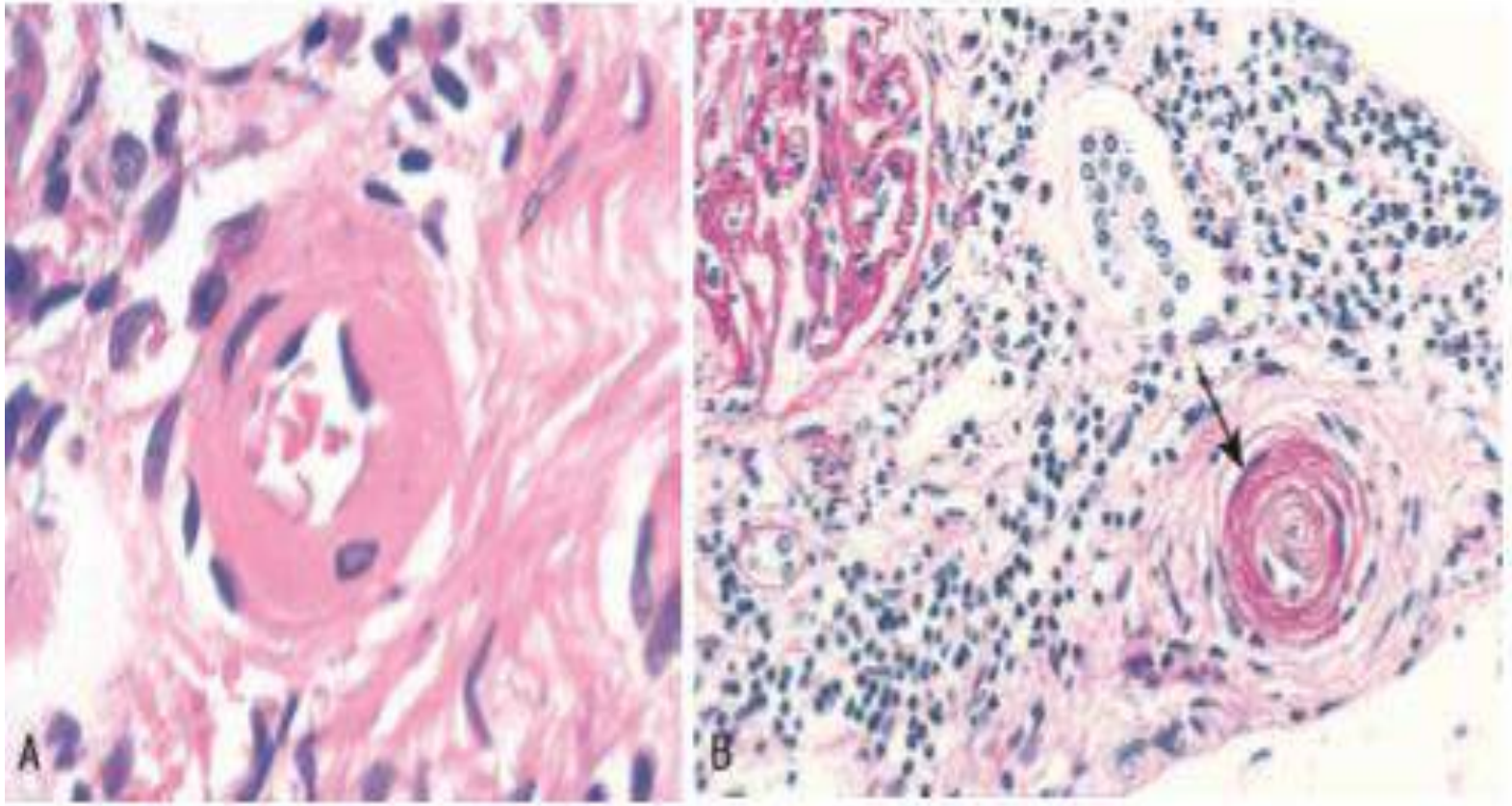
Essential Hypertension
Accounts for 90% to 95% of all cases
Secondary Hypertension
Renal
Acute glomerulonephritis Chronic renal disease Polycystic disease Renal artery stenosis Renal vasculitis Renin-producing tumors
Endocrine
Adrenocortical hyperfunction (Cushing syndrome, primary aldosteronism, congenital adrenal hyperplasia, licorice ingestion) Exogenous hormones (glucocorticoids, estrogen [including pregnancy-induced and oral contraceptives], sympathomimetics and tyramine-containing foods, monoamine oxidase inhibitors) Pheochromocytoma Acromegaly Hypothyroidism (myxedema) Hyperthyroidism (thyrotoxicosis) Pregnancy-induced (pre-eclampsia)
Cardiovascular
Coarctation of aorta Polyarteritis nodosa Increased intravascular volume Increased cardiac output Rigidity of the aorta
Neurologic
Psychogenic Increased intracranial pressure Sleep apnea Acute stress, including surgery

Pathogenesis

most cases (95%) are idiopathic (essential hypertension). This form is compatible with long life unless a myocardial infarction, stroke, or another complication supervenes. Most of the remaining cases **(secondary hypertension)** are due to primary renal disease, renal artery narrowing **(renovascular hypertension),** or adrenal disorders.

Morphology

Hypertension not only accelerates atherogenesis but also causes degenerative changes in the walls of large and mediumsized arteries that can lead to aortic dissection and cerebrovascular hemorrhage. Two forms of small blood vessel disease are hypertension-related: **hyaline arteriolosclerosis and hyperplastic arteriolosclerosis .**



9–5 Hypertensive vascular disease. **A,** Hyaline arteriolosclerosis. The arteriolar wall is thickened with the deposition of amorphous protein-
material (hyalinized), and the lumen is markedly narrowed. **B,** Hyperplastic arteriolosclerosis ("onion-skinning") (arrow) causing luminal obliter-
riodic acid–Schiff stain).

of Helmut Rennke, MD, Brigham and Women's Hospital, Boston, Massachusetts.)

Hypertension represents an elevation in systolic and/or diastolic blood pressure.

■ **Primary or essential hypertension** is characterized by a chronic elevation in blood pressure that occurs without evidence of other disease, and **secondary hypertension** by an elevation of blood pressure that results from some other disorder, such as kidney disease.

The pathogenesis of essential hypertension is thought to include constitutional and environmental factors involving the kidney and its role in regulating extracellular fluid volume through salt and water elimination, sympathetic nervous system hyperreactivity, renin-angiotensin system activity, or intracellular sodium and calcium levels. The medications that are used in the treatment of hypertension exert their effect through one or more of these mechanisms.

■ **Uncontrolled hypertension** produces increased demands on the heart, resulting in left ventricular hypertrophy and heart failure, and on the vessels of the arterial system, leading to atherosclerosis, kidney disease, retinopathy, and stroke.

Aneurysms and Dissection

Aneurysms are congenital or acquired dilations of blood vessels or the heart . **“True” aneurysms** involve all three layers of the artery (intima, media, and adventitia) or the attenuated wall of the heart; these include atherosclerotic and congenital vascular aneurysms, as well as ventricular aneurysms resulting from transmural myocardial infarctions. By comparison, **a false aneurysm** (pseudoaneurysm) results when a wall defect leads to the formation of an extravascular hematoma that communicates with the intravascular space (“pulsating hematoma”). Examples are ventricular ruptures contained by pericardial adhesions and leaks at the junction of a vascular graft with a natural artery. In arterial dissections, pressurized blood gains entry to the arterial wall through a surface defect and then pushes apart the underlying layers. Aneurysms and dissections are important causes of stasis and subsequent thrombosis; they also have a propensity to rupture—often with catastrophic results.

Pathogenesis

Aneurysms occur when the structure or function of the connective tissue is compromised by any of the following factors:

- Inadequate or abnormal connective tissue synthesis. Several rare inherited diseases provide insight into the types of molecular abnormalities that can lead to aneurysm formation.
- Excessive connective tissue degradation. Increased MMP expression, such as by macrophages in atherosclerotic plaque, can contribute to aneurysm development by degrading arterial ECM in the arterial wall; similarly, decreased TIMP expression can also tip the balance toward net ECM degradation. A genetic predisposition to aneurysm formation in the setting of inflammation may be related to MMP and/or TIMP polymorphisms, or to the nature of the local inflammatory response that drives MMP or TIMP production.

Pathogenesis

- Loss of smooth muscle cells or change in the smooth muscle cell synthetic phenotype. Atherosclerotic thickening of the intima can cause ischemia of the inner media by increasing the diffusion distance from the lumen. Conversely, systemic hypertension can cause luminal narrowing of the aortic vasa vasorum, leading to ischemia of the outer media. Such ischemia results in smooth muscle cell loss as well as aortic “degenerative changes,” which include fibrosis (replacing distensible elastic tissue), inadequate ECM synthesis, and accumulation of increasing amounts of amorphous proteoglycans. metabolic syndrome such as scurvy. The two most important causes of aortic aneurysms are atherosclerosis and hypertension.

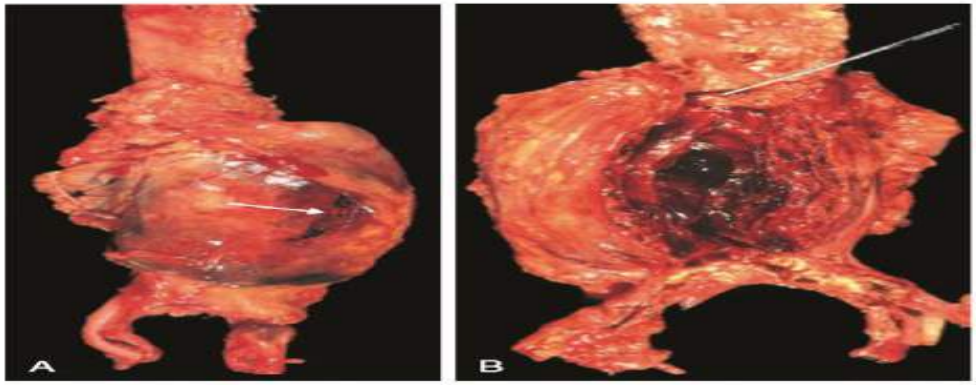


Figure 9–19 Abdominal aortic aneurysm. **A,** External view of a large aortic aneurysm that ruptured at the site is indicated by the *arrow*. **B,** Opened view, with the location of the rupture tract indicated by a *probe*. The wall of the aneurysm is attenuated, and the lumen is filled by a large, layered thrombus.

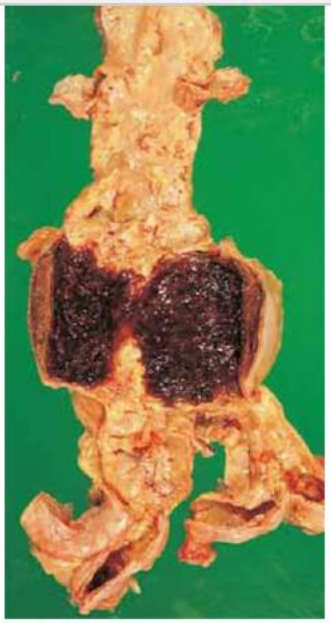


FIGURE 18-11. Atherosclerotic aneurysm of the abdominal aorta. The aneurysm has been opened longitudinally to reveal a large thrombus in the lumen. The aorta and common iliac arteries display complicated lesions of atherosclerosis. (From Gotlieb A.I. [2008]. Blood vessels. In Rubin R., Strayer D.S. [Eds.], *Rubin's pathology: Clinicopathologic foundations of medicine* [5th ed., p. 420]. Philadelphia: Lippincott Williams & Wilkins.)

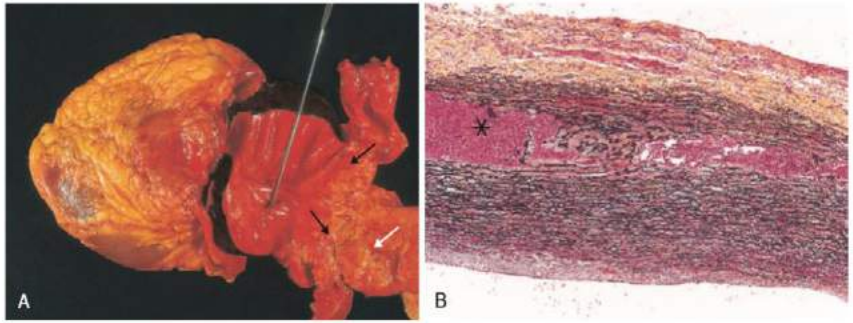


Figure 9–20 Aortic dissection. **A,** An opened aorta with a proximal dissection originating from a small, oblique intimal tear (identified by the *probe*) associated with an intramural hematoma. Note that the intimal tear occurred in a region largely free of atherosclerotic plaque. The distal edge of the intramural hematoma (*black arrows*) lies at the edge of a large area of atherosclerosis (*white arrow*), which arrested the propagation of the dissection. **B,** Histologic preparation showing the dissection and intramural hematoma (*asterisk*). Aortic elastic layers are black and blood is red in this section, stained with the Movat stain.

Varicose Veins of the Extremities

Varicose veins are abnormally dilated tortuous veins produced by chronically increased intraluminal pressures and weakened vessel wall support.

The superficial veins of the upper and lower leg typically are involved. Up to 20% of men and a third of women develop lower extremity varicose veins.

Obesity increases the risk, and the higher incidence in women probably reflects the prolonged elevation in venous pressure caused by compression of the inferior vena cava by the gravid uterus during pregnancy. There is also a familial tendency toward premature varicosities.

Clinical Features of Varicose Veins

Varicose dilation renders the venous valves incompetent and leads to lower extremity stasis, congestion, edema, pain, and thrombosis.

The most disabling sequelae include persistent edema in the extremity and secondary ischemic skin changes, including stasis dermatitis and ulcerations. The latter can become chronic varicose ulcers as a consequence of poor wound healing and superimposed infections.

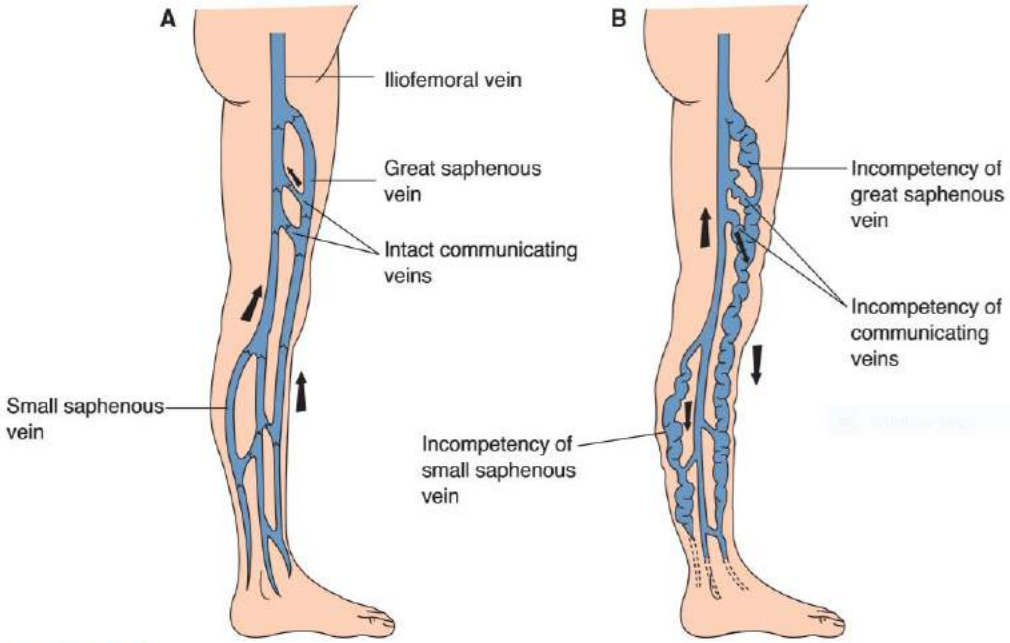


FIGURE 18-17. Superficial and deep venous channels of the leg. **(A)** Normal venous structures and flow patterns. **(B)** Varicosities in the superficial venous system are the result of incompetent valves in the communicating veins. The *arrows* in both views indicate the direction of blood flow. (Modified from Abramson D.I. [1974]. *Vascular disorders of the extremities* [2nd ed.]. New York: Harper & Row.)

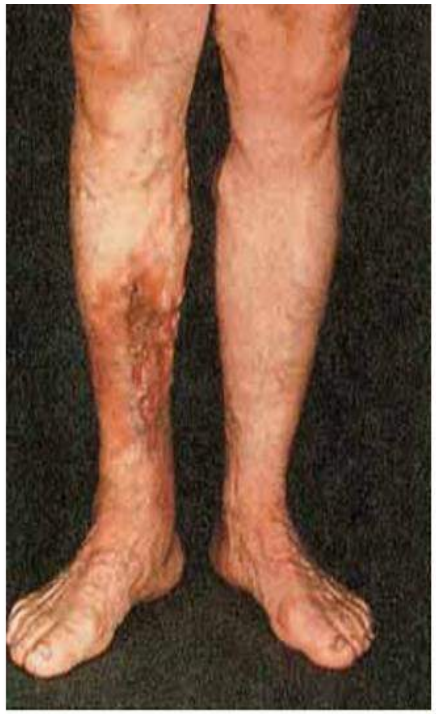


FIGURE 18-19. Varicose veins of the legs. Severe varicosities of the superficial leg veins have led to stasis dermatitis and secondary ulcerations. [From Gotlieb A.I. [2008] Blood vessels. In Rubin R., Strayer D.S. [Eds.], *Rubin's pathology: Clinico-pathologic foundations of medicine* [5th ed., p. 423]. Philadelphia: Lippincott Williams & Wilkins.)

Varicosities of Other Sites

Venous dilations in two other sites merit special attention:

- Esophageal varices. Liver cirrhosis (less frequently, portal vein obstruction or hepatic vein thrombosis) causes portal vein hypertension . This in turn leads to the opening of porto-systemic shunts and increased blood flow into veins at the gastro-esophageal junction (forming esophageal varices), rectum (forming hemorrhoids), and periumbilical veins of the abdominal wall (forming a caput medusae). Esophageal varices are most important since they are prone to ruptures that can lead to massive (even fatal) upper gastrointestinal hemorrhage.

- Hemorrhoids are varicose dilations of the venous plexus at the anorectal junction that result from prolonged pelvic vascular congestion associated with pregnancy or straining to defecate. Hemorrhoids are a source of bleeding and prone to thrombosis and painful ulceration.