Ventricular septal defect

Clinical features of VSD vary with the defect's size, the shunting's effect on the pulmonary vasculature, and the infant's age. In a small VSD, shunting is minimal, and pulmonary artery pressure and heart size remain normal. Such defects may eventually close spontaneously without ever causing symptoms. Initially, large VSD shunts cause left atrial and left ventricular hypertrophy. Later, an uncorrected VSD will cause right ventricular hypertrophy due to increasing pulmonary vascular resistance. Eventually, biventricular heart failure and cyanosis (from reversal of shunt direction) occur. Resulting cardiac hypertrophy may make the anterior chest wall prominent. A large VSD increases the risk of pneumonia. Infants with large VSDs are thin and small and gain weight slowly. They may develop heart failure with dusky skin; liver, heart, and spleen enlargement because of systemic venous congestion; diaphoresis; feeding difficulties; rapid, grunting respirations; and increased heart rate. They may also develop severe pulmonary hypertension. Fixed pulmonary hypertension may occur much later in life with right-to-left shunt (Eisenmenger's syndrome), causing cyanosis and clubbing of the nail beds. The typical murmur associated with a VSD is blowing or rumbling and varies in frequency. In the neonate, a moderately loud early systolic murmur may be heard along the lower left sternal border. About the second or third day after birth, the murmur may become louder and longer. In infants, the murmur may be loudest near the heart's base and may suggest pulmonary stenosis. A small VSD may produce a functional murmur or a characteristic loud, harsh systolic murmur. Larger VSDs produce audible murmurs (at least a grade 3 pansystolic), loudest at the fourth intercostal space, usually with a thrill; however, a large VSD with minimal pressure gradient may have no audible murmur. In addition, the pulmonic component of S2 sounds loud and is widely split. Palpation reveals displacement of the point of maximal impulse to the left. When fixed pulmonary hypertension is present, a diastolic murmur may be audible on auscultation, the systolic murmur becomes quieter, and S2 is greatly accentuated.

Atrial septal defect

ASD commonly goes undetected in preschoolers; such children may complain about feeling tired only after extreme exertion and may have frequent respiratory tract infections but otherwise appear normal and healthy. However, they may show growth retardation if they have large shunts. Children with ASD seldom develop heart failure, pulmonary hypertension, infective endocarditis, or other complications. However, as adults, they usually manifest pronounced symptoms, such as fatigability and dyspnea on exertion, frequently to the point of severe limitation of activity (especially after age 40). In children, auscultation reveals an early to midsystolic murmur, superficial in quality, heard at the second or third left intercostal space. In patients with large shunts (resulting from increased tricuspid valve flow), a low-pitched diastolic murmur is heard at the lower left sternal border, which becomes more pronounced on inspiration. Although the murmur's intensity is a rough indicator of the size of the left-to-right shunt, its low pitch sometimes makes it difficult to hear and, if the pressure gradient is relatively low, a murmur may not be detectable. Other signs include a fixed, widely split S2 , caused by delayed closure of the pulmonic valve, and a systolic click or late systolic murmur at the apex, resulting from mitral valve prolapse, which occasionally affects older children with ASD. In older patients with large, uncorrected defects and fixed pulmonary artery hypertension, auscultation reveals an accentuated S2 . A pulmonary ejection click and an audible S4 may also be present. Clubbing and cyanosis become evident; syncope and hemoptysis may occur with severe pulmonary vascular disease.

Coarctation of the aorta

Clinical features vary with age. During the first year of life, when aortic coarctation may cause heart failure, the infant displays tachypnea, dyspnea, pulmonary edema, pallor, tachycardia, failure to thrive, cardiomegaly, and hepatomegaly. In most cases, heart sounds are normal unless a coexisting cardiac defect is present. Femoral pulses are absent or diminished. If coarctation is asymptomatic in infancy, it usually remains so throughout adolescence, as collateral circulation develops to bypass the narrowed segment. During adolescence, this defect may produce dyspnea, claudication, headaches, epistaxis, and hypertension in the upper extremities despite collateral circulation. It commonly causes resting systolic hypertension and wide pulse pressure; high diastolic pressure readings are the same in both the arms and legs. Coarctation may also produce a visible aortic pulsation in the suprasternal notch, a continuous systolic murmur, an accentuated S2 , and an S4 .

Patent ductus arteriosus

In neonates, especially those who are premature, a large PDA usually produces respiratory distress, with signs of heart failure due to the tremendous volume of blood shunted to the lungs through a patent ductus and the increased workload on the left side of the heart. Other characteristic features may include heightened susceptibility to respiratory tract infections, slow motor development, and failure to thrive. Most children with PDA have no symptoms except cardiac ones. Others may exhibit signs of heart disease, such as physical underdevelopment, fatigability, and frequent respiratory tract infections. Adults with undetected PDA may develop pulmonary vascular disease and, by age 40, may display fatigability and dyspnea on exertion. About 10% of them also develop infective endocarditis. Auscultation reveals the classic machinery murmur (Gibson murmur): a continuous murmur (during systole and diastole) best heard at the heart's base, at the second left intercostal space under the left clavicle in 85% of children with PDA. This murmur may obscure S2 . However, with a right-to-left shunt, such a murmur may be absent. Palpation may reveal a thrill at the left sternal border and a prominent left ventricular impulse. Peripheral arterial pulses are bounding (Corrigan's pulse); pulse pressure is widened because of an elevation in systolic blood pressure and, primarily, a drop in diastolic pressure.

Tetralogy of Fallot

The degree of pulmonary stenosis, interacting with the VSD's size and location, determines the clinical and hemodynamic effects of this complex defect. The VSD usually lies in the outflow tract of the right ventricle and is generally large enough to permit equalization of right and left ventricular pressures. However, the ratio of systemic vascular resistance to pulmonary stenosis affects the direction and magnitude of shunt flow across the VSD. Severe obstruction of right ventricular outflow produces a right-to-left shunt, causing decreased systemic arterial oxygen saturation, cyanosis, reduced pulmonary blood flow, and hypoplasia of the entire pulmonary vasculature. Increased right ventricular pressure causes right ventricular hypertrophy. Milder forms of pulmonary stenosis result in a left-to-right shunt or no shunt at all. Generally, the hallmark of the disorder is cyanosis, which usually becomes evident within several months after birth but may be present at birth if the neonate has severe pulmonary stenosis. Between ages 2 months and 2 years, children with tetralogy of Fallot may experience cyanotic or “blue” spells. Such spells result from increased right-to-left shunting, possibly caused by spasm of the right ventricular outflow tract, increased

systemic venous return, or decreased systemic arterial resistance. Exercise, crying, straining, infection, or fever can precipitate blue spells. Blue spells are characterized by dyspnea; deep, sighing respirations; bradycardia; fainting; seizures; and loss of consciousness. Older children may also develop other signs of poor oxygenation, such as clubbing, diminished exercise tolerance, increasing dyspnea on exertion, growth retardation, and eating difficulties. These children habitually squat when they feel short of breath; this is thought to decrease venous return of unoxygenated blood from the legs and increase systemic arterial resistance. Children with tetralogy of Fallot also risk developing cerebral abscesses, pulmonary thrombosis, venous thrombosis or cerebral embolism, and infective endocarditis. In females with tetralogy of Fallot who live to childbearing age, incidence of spontaneous abortion, premature births, and low birth weight rises.

Transposition of the great arteries

Within the first few hours after birth, neonates with transposition of the great arteries and no other heart defects generally show cyanosis and tachypnea, which worsen with crying. After several days or weeks, such neonates usually develop signs of heart failure (gallop rhythm, tachycardia, dyspnea, hepatomegaly, and cardiomegaly). S2 is louder than normal because the anteriorly transposed aorta is directly behind the sternum; in many cases, however, no murmur can be heard during the first few days of life. Associated defects (ASD, VSD, or PDA) cause their typical murmurs and may minimize cyanosis but may also cause other complications (especially severe heart failure). VSD with PS produces a characteristic murmur and severe cyanosis. As infants with this defect grow older, cyanosis is their most prominent abnormality. However, they also develop diminished exercise tolerance, fatigability, coughing, clubbing, and more pronounced murmurs if ASD, VSD, PDA, or PS is present.

Myocarditis

Myocarditis usually causes nonspecific symptoms—such as fatigue, dyspnea, palpitations, and fever— that reflect the accompanying systemic infection. Occasionally, it may produce mild, continuous pressure or soreness in the chest (unlike the recurring, stress-related pain of angina pectoris). Although myocarditis is usually self-limiting, it may induce myofibril degeneration that results in right- and left-sided heart failure, with cardiomegaly, jugular vein distention, dyspnea, persistent fever with resting or exertional tachycardia disproportionate to the degree of fever, and supraventricular and ventricular arrhythmias. Sometimes myocarditis recurs or produces chronic valvulitis (when it results from rheumatic fever), cardiomyopathy, arrhythmias, and thromboembolism.

Endocarditis

Early clinical features of endocarditis are usually nonspecific and include malaise, weakness, fatigue, weight loss, anorexia, arthralgia, night sweats, chills, valvular insufficiency and, in 90% of patients, an intermittent fever that may recur for weeks. A more acute onset is associated with organisms of high pathogenicity such as S. aureus. Endocarditis commonly causes a loud, regurgitant murmur typical of the underlying heart lesion. A suddenly changing murmur or the discovery of a new murmur in the presence of fever is a classic physical sign of endocarditis. In about 30% of patients, embolization from vegetating lesions or diseased valvular tissue may produce typical features of splenic, renal, cerebral, or pulmonary infarction or of peripheral vascular occlusion: splenic infarction—pain in the

left upper quadrant, radiating to the left shoulder; and abdominal rigidity renal infarction—hematuria, pyuria, flank pain, and decreased urine output cerebral infarction—hemiparesis, aphasia, or other neurologic deficits pulmonary infarction (most common in right-sided endocarditis, which commonly occurs among I.V. drug abusers and after cardiac surgery)—cough, pleuritic pain, pleural friction rub, dyspnea, and hemoptysis peripheral vascular occlusion—numbness and tingling in an arm, leg, finger, or toe, or signs of impending peripheral gangrene. Other signs may include splenomegaly; petechiae of the skin (especially common on the upper anterior trunk) and the buccal, pharyngeal, or conjunctival mucosa; and splinter hemorrhages under the nails. Rarely, endocarditis produces Osler's nodes (tender, raised, subcutaneous lesions on the fingers or toes), Roth's spots (hemorrhagic areas with white centers on the retina), and Janeway lesions (purplish macules on the palms or soles).

Pericarditis

Acute pericarditis typically produces a sharp and often sudden pain that usually starts over the sternum and radiates to the neck, shoulders, back, and arms. However, unlike the pain of MI, pericardial pain is often pleuritic, increasing with deep inspiration and decreasing when the patient sits up and leans forward, pulling the heart away from the diaphragmatic pleurae of the lungs. Pericardial effusion, the major complication of acute pericarditis, may produce effects of heart failure (such as dyspnea, orthopnea, and tachycardia), ill-defined substernal chest pain, and a feeling of fullness in the chest. (See Patterns of cardiac pain.)

Chronic constrictive pericarditis causes a gradual increase in systemic venous pressure and produces symptoms similar to those of chronic right-sided heart failure (fluid retention, ascites, and hepatomegaly).

Rheumatic fever and rheumatic heart disease

In 95% of patients, rheumatic fever characteristically follows a streptococcal infection that appeared a few days to 6 weeks earlier. A temperature of at least 100.4° F (38° C) occurs, and most patients complain of migratory joint pain or polyarthritis. Swelling, redness, and signs of effusion usually accompany such pain, which most commonly affects the knees, ankles, elbows, or hips. In 5% of patients (generally those with carditis), rheumatic fever causes skin lesions such as erythema marginatum, a nonpruritic, macular, transient rash that gives rise to red lesions with blanched centers. Rheumatic fever may also produce firm, movable, nontender, subcutaneous nodules about 3 mm to 2 cm in diameter, usually near tendons or bony prominences of joints (especially the elbows, knuckles, wrists, and knees) and less often on the scalp and backs of the hands. These nodules persist for a few days to several weeks and, like erythema marginatum, often accompany carditis. Later, rheumatic fever may cause transient chorea, which develops up to 6 months after the original streptococcal infection. Mild chorea may produce hyperirritability, a deterioration in handwriting, or an inability to concentrate. Severe chorea (Sydenham's chorea) causes purposeless, nonrepetitive, involuntary muscle spasms; poor muscle coordination; and weakness. Chorea always resolves without residual neurologic damage. The most destructive effect of rheumatic fever is carditis, which develops in up to 50% of patients and may affect the endocardium, myocardium, pericardium, or the heart valves. Pericarditis causes a pericardial friction rub and, occasionally, pain and effusion. Myocarditis produces characteristic lesions called Aschoff bodies (in the acute stages)

and cellular swelling and fragmentation of interstitial collagen, leading to formation of a progressively fibrotic nodule and interstitial scars. Endocarditis causes valve leaflet swelling, erosion along the lines of leaflet closure, and blood, platelet, and fibrin deposits, which form beadlike vegetations. Endocarditis affects the mitral valve most often in females; the aortic, most often in males. In both females and males, endocarditis affects the tricuspid valves occasionally and the pulmonic only rarely. Severe rheumatic carditis may cause heart failure with dyspnea; right upper quadrant pain; tachycardia; tachypnea; a hacking, nonproductive cough; edema; and significant mitral and aortic murmurs. The most common of such murmurs include: a systolic murmur of mitral insufficiency (high-pitched, blowing, holosystolic, loudest at apex, possibly radiating to the anterior axillary line) a midsystolic murmur due to stiffening and swelling of the mitral leaflet occasionally, a diastolic murmur of aortic insufficiency (low-pitched, rumbling, almost inaudible). Valvular disease may eventually result in chronic valvular stenosis and insufficiency, including mitral stenosis and insufficiency, and aortic insufficiency. In children, mitral insufficiency remains the major sequela of rheumatic heart disease.

Hypertension

Hypertension usually doesn't produce clinical effects until vascular changes in the heart, brain, or kidneys occur. Severely elevated blood pressure damages the intima of small vessels, resulting in fibrin accumulation in the vessels, development of local edema and, possibly, intravascular clotting. Symptoms produced by this process depend on the location of the damaged vessels:

brain — stroke

retina — blindness

heart — myocardial infarction

kidneys — proteinuria, edema and, eventually, renal failure.

Hypertension increases the heart's workload, causing left ventricular hypertrophy and, later, left- and right-sided heart failure and pulmonary edema.

Coronary artery disease

The classic symptom of CAD is angina, the direct result of inadequate oxygen flow to the myocardium. Anginal pain is usually described as a burning, squeezing, or tight feeling in the substernal or precordial chest that may radiate to the left arm, neck, jaw, or shoulder blade. Typically, the patient clenches his fist over his chest or rubs his left arm when describing the pain, which may be accompanied by nausea, vomiting, fainting, sweating, and cool extremities. Anginal episodes most often follow physical exertion but may also follow emotional excitement, exposure to cold, or a large meal. Some patients, particularly those with diabetes, may not experience typical anginal pain but may have dyspnea, fatigue, diaphoreses, or more vague symptoms. Angina has four major forms: stable (pain is predictable in frequency and duration and can be relieved with nitrates and rest), unstable (pain increases in frequency and duration and is more easily induced), Prinzmetal's or variant (from unpredictable coronary artery spasm), and microvascular (in which impairment of vasodilator reserve causes angina-like chest pain in a patient with normal P coronary

arteries). Severe and prolonged anginal pain generally suggests MI, with potentially fatal arrhythmias and mechanical failure.

Myocardial infarction

The cardinal symptom of MI is persistent, crushing substernal pain that may radiate to the left arm, jaw, neck, or shoulder blades. Such pain is usually described as heavy, squeezing, or crushing, and may persist for 12 hours or more. However, in some MI patients — particularly elderly people or those with diabetes — pain may not occur at all; in others, it may be mild and confused with indigestion. In patients with coronary artery disease, angina of increasing frequency, severity, or duration (especially if not provoked by exertion, a heavy meal, or cold and wind) may signal impending infarction.

Other clinical effects include a feeling of impending doom, fatigue, nausea, vomiting, and shortness of breath. Some patients may have no symptoms. The patient may experience catecholamine responses, such as coolness in extremities, perspiration, anxiety, and restlessness. Fever is unusual at the onset of an MI, but a low-grade temperature elevation may develop during the next few days. Blood pressure varies; hypotension or hypertension may be present. The most common post-MI complications include recurrent or persistent chest pain, arrhythmias, leftsided heart failure (resulting in heart failure or acute pulmonary edema), and cardiogenic shock. Unusual but potentially lethal complications that may develop soon after infarction include thromboembolism; papillary muscle dysfunction or rupture, causing mitral insufficiency; rupture of the ventricular septum, causing ventricular septal defect; rupture of the myocardium; and ventricular aneurysm. Up to several months after infarction, Dressler's syndrome may develop (pericarditis, pericardial friction rub, chest pain, fever, leukocytosis and, possibly, pleurisy or pneumonitis).

Heart failure

Left-sided heart failure primarily produces pulmonary signs and symptoms; right-sided heart failure, primarily systemic signs and symptoms. However, heart failure often affects both sides of the heart. Clinical signs of left-sided heart failure include dyspnea, orthopnea, crackles, possibly wheezing, hypoxia, respiratory acidosis, cough, cyanosis or pallor, palpitations, arrhythmias, elevated blood pressure, and pulsus alternans. Clinical signs of right-sided heart failure include dependent peripheral edema, hepatomegaly, splenomegaly, jugular vein distention, ascites, slow weight gain, arrhythmias, positive hepatojugular reflex, abdominal distention, nausea, vomiting, anorexia, weakness, fatigue, dizziness, and syncope.

Dilated cardiomyopathy

In dilated cardiomyopathy, the heart ejects blood less efficiently than normal. Consequently, a large volume of blood remains in the left ventricle after systole, causing signs of heart failure—both leftsided (shortness of breath, orthopnea, dyspnea on exertion, paroxysmal nocturnal dyspnea, fatigue, and an irritating dry cough at night) and right-sided (edema, liver engorgement, and jugular vein distention). Dilated cardiomyopathy also produces peripheral cyanosis and sinus tachycardia or atrial fibrillation at rest in some patients secondary to low cardiac output. Auscultation reveals diffuse apical impulses, pansystolic murmur (mitral and tricuspid insufficiency secondary to cardiomegaly and weak papillary muscles), and S3 and S4 gallop rhythms.

Hypertrophic cardiomyopathy

Clinical features of the disorder may not appear until it's well advanced, when atrial dilation and, possibly, atrial fibrillation abruptly reduce blood flow to the left ventricle. Reduced inflow and subsequent low output may produce angina pectoris, arrhythmias, dyspnea, orthopnea, syncope, heart failure, and death. Auscultation reveals a medium-pitched systolic ejection murmur along the left sternal border and at the apex; palpation reveals a peripheral pulse with a characteristic double impulse (pulsus biferiens) and, with atrial fibrillation, an irregular pulse.

Hypovolemic shock

Hypovolemic shock produces a syndrome of hypotension, with narrowing pulse pressure; decreased sensorium; tachycardia; rapid, shallow respirations; reduced urine output (less than 25 ml/hour); and cold, pale, clammy skin. Metabolic acidosis with an accumulation of lactic acid develops as a result of tissue anoxia, as cellular metabolism shifts from aerobic to anaerobic pathways. Disseminated intravascular coagulation (DIC) is a possible complication of hypovolemic shock.

Cardiogenic shock

Cardiogenic shock produces signs of poor tissue perfusion: cold, pale, clammy skin; a decrease in systolic blood pressure to 30 mm Hg below baseline, or a sustained reading below 80 mm Hg not attributable to medication; tachycardia; rapid, shallow respirations; oliguria (less than 20 ml/ hour); restlessness; mental confusion and obtundation; narrowing pulse pressure; and cyanosis. Although many of these clinical features also occur in heart failure and other shock syndromes, they're usually more profound in cardiogenic shock.

Ventricular aneurysm

Ventricular aneurysm may cause arrhythmias —such as premature ventricular contractions or ventricular tachycardia— palpitations, signs of cardiac dysfunction (weakness on exertion, fatigue, and angina) and, occasionally, a visible or palpable systolic precordial bulge. This condition may also lead to left ventricular dysfunction, with chronic heart failure (dyspnea, fatigue, edema, crackles, gallop rhythm, and jugular vein distention); pulmonary edema; systemic embolization; and, with leftsided heart failure, pulsus alternans. Ventricular aneurysms enlarge but seldom rupture.

Cardiac tamponade

Cardiac tamponade classically produces increased venous pressure with jugular vein distention, reduced arterial blood pressure, muffled heart sounds on auscultation, and pulsus paradoxus (an abnormal inspiratory drop in systemic blood pressure greater than 15 mm Hg). The absence of a preexisting pericardial friction rub may suggest an increase in fluid in the pericardial space. These classic symptoms represent failure of physiologic compensatory mechanisms to override the effects of rapidly rising pericardial pressure, which limits diastolic filling of the ventricles and reduces stroke volume to a critically low level. Generally, ventricular end-systolic volume may drop because of inadequate preload. The increasing pericardial pressure is transmitted equally across the heart cavities, producing a matching rise in intracardiac pressure, especially atrial and end-diastolic ventricular pressures. Cardiac tamponade may also cause dyspnea, diaphoresis, pallor or cyanosis,

anxiety, tachycardia, narrow pulse pressure, restlessness, and hepatomegaly, but the lung fields will be clear. The patient typically sits upright and leans forward.

Cardiac arrhythmias

Signs and symptoms of cardiac arrhythmias include palpitations, fainting, lightheadedness, dizziness, chest pain, shortness of breath, changes in pulse patterns, paleness, and the temporary absence of breathing. However, the patient with a cardiac arrhythmia may be asymptomatic until the development of sudden cardiac arrest.

Thoracic aortic aneurysm

The most common symptom of thoracic aortic aneurysm is pain. With ascending aneurysm, the pain is described as severe, boring, and ripping and extends to the neck, shoulders, lower back, or abdomen but seldom radiates to the jaw and arms. Pain is more severe on the right side. Other signs of ascending aneurysm may include bradycardia, aortic insufficiency, pericardial friction rub caused by a hemopericardium, unequal intensities of the right carotid and left radial pulses, and a difference in blood pressure between the right and left arms. These signs are absent in descending aneurysm. If dissection involves the carotids, an abrupt onset of neurologic deficits may occur. With descending aneurysm, pain usually starts suddenly between the shoulder blades and may radiate to the chest; it's described as sharp and tearing. Transverse aneurysm causes a sudden, sharp, tearing pain radiating to the shoulders. It may also cause hoarseness, dyspnea, dysphagia, and a dry cough because of compression of surrounding structures in this area.

Abdominal aneurysm

Although abdominal aneurysms usually don't produce symptoms, most are evident (unless the patient is obese) as a pulsating mass in the periumbilical area, accompanied by a systolic bruit over the aorta. Some tenderness may be present on deep palpation. A large aneurysm may produce symptoms that mimic renal calculi, lumbar disk disease, and duodenal compression. Abdominal aneurysms rarely cause diminished peripheral pulses or claudication, unless embolization occurs. Lumbar pain that radiates to the flank and groin from pressure on lumbar nerves may signify enlargement and imminent rupture. A rare but recognized symptom is unrelenting testicular pain with no other cause. If the aneurysm ruptures into the peritoneal cavity, it causes severe, persistent abdominal and back pain, mimicking renal or ureteral colic. Signs of hemorrhage—such as weakness, sweating, tachycardia, and hypotension—may be subtle because rupture into the retroperitoneal space produces a tamponade effect that prevents continued hemorrhage. Patients with such rupture may remain stable for hours before shock and death occur, although 20% die immediately.

Femoral and popliteal aneurysms

Popliteal aneurysms may cause pain in the popliteal space when they're large enough to compress the medial popliteal nerve and edema and venous distention if the vein is compressed. Femoral and popliteal aneurysms can produce symptoms of severe ischemia in the leg or foot due to acute thrombosis within the aneurysmal sac, embolization of mural thrombus fragments and, rarely, rupture. Symptoms of acute aneurysmal thrombosis include severe pain, loss of pulse and color,

coldness in the affected leg or foot, and gangrene. Distal petechial hemorrhages may develop from aneurysmal emboli.

Thrombophlebitis

In both types of thrombophlebitis, clinical features vary with the site and length of the affected vein. Although DVT may occur asymptomatically, it may also produce severe pain, fever, chills, malaise and, possibly, swelling and cyanosis of the affected arm or leg. Superficial thrombophlebitis produces visible and palpable signs, such as heat, pain, swelling, rubor, tenderness, and induration along the length of the affected vein. Varicose veins may also be present. (See Varicose veins.) Extensive vein involvement may cause lymphadenitis.

Raynaud's disease

After exposure to cold or stress, the skin on the fingers typically blanches and then becomes cyanotic before changing to red and before changing from cold to normal temperature. Numbness and tingling may also occur. These symptoms are relieved by warmth. In long-standing disease, trophic changes, such as sclerodactyly, ulcerations, or chronic paronychia, may result. Although it's extremely uncommon, minimal cutaneous gangrene necessitates amputation of one or more phalanges.

Buerger's disease

Buerger's disease typically produces intermittent claudication of the instep, which is aggravated by exercise and relieved by rest. During exposure to low temperature, the feet initially become cold, cyanotic, and numb; later, they redden, become hot, and tingle. Occasionally, Buerger's disease also affects the hands, possibly resulting in painful fingertip ulcerations. Associated signs and symptoms may include impaired peripheral pulses, migratory superficial thrombophlebitis and, in later stages, ulceration, muscle atrophy, and gangrene.