

HEART FAILURE

The heart is a truly remarkable organ, beating more than 40 million times a year and pumping over 7500 liters of blood a day; in a typical lifespan, the cumulative volume would fill three “supertanker” ships.

OVERVIEW OF HEART DISEASE

Although a host of diseases can affect the cardiovascular system, the pathophysiologic pathways that result in a “broken” heart distill down to six principal mechanisms:

- **Failure of the pump.** In the most common situation, the cardiac muscle contracts weakly and the chambers cannot empty properly—so-called *systolic dysfunction*. In some cases, the muscle cannot relax sufficiently to permit ventricular filling, resulting in *diastolic dysfunction*.
- **Obstruction to flow.** Lesions that prevent valve opening (e.g., calcific aortic valve stenosis) or cause increased ventricular chamber pressures (e.g., systemic hypertension or aortic coarctation) can overwork the myocardium, which has to pump against the obstruction.
- **Regurgitant flow.** Valve lesions that allow backward flow of blood create conditions that add increased volume workload to the affected chambers with each contraction.
- **Shunted flow.** Defects (congenital or acquired) that divert blood inappropriately from one chamber to another, or from one vessel to another, lead to pressure and volume overloads.
- **Disorders of cardiac conduction.** Uncoordinated cardiac impulses or blocked conduction pathways can cause arrhythmias that reduce contraction frequency or diminish effective cardiac output.
- **Rupture of the heart or major vessel.** Loss of circulatory continuity (e.g., gunshot wound through the thoracic aorta) leads to exsanguination, hypotensive shock, and death.

HEART FAILURE

Heart failure generally is referred to as *congestive heart failure* (CHF). CHF is the common end point for many forms of cardiac disease and typically is a progressive.

Clinical Features

Dyspnea (shortness of breath) on exertion is usually the earliest and most significant symptom of left-sided heart failure; cough also is common as a consequence of fluid transudation into air spaces.

Paroxysmal nocturnal dyspnea is a particularly dramatic form of breathlessness, awakening patients from sleep with extreme dyspnea bordering on feelings of suffocation.

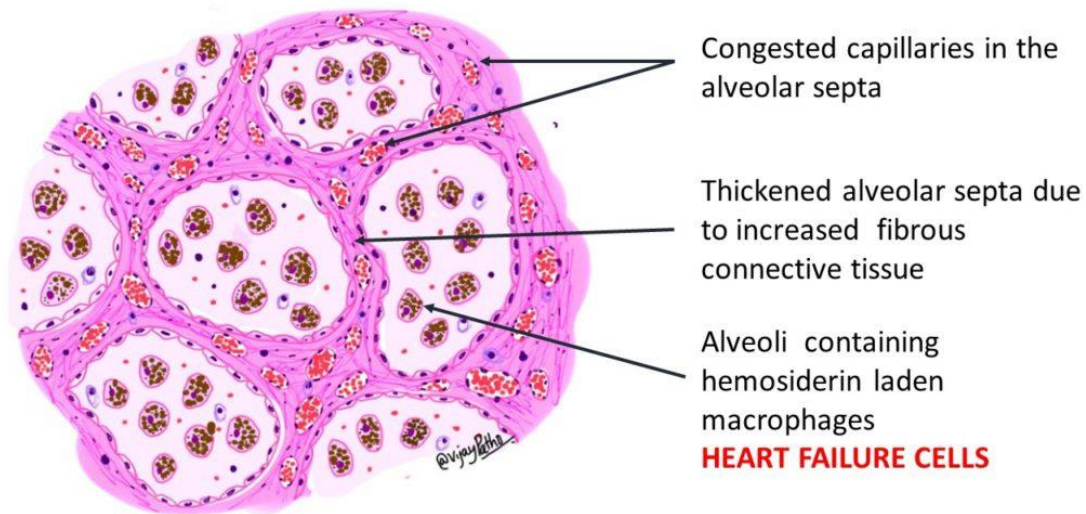
Other manifestations of left ventricular failure include an enlarged heart (cardiomegaly), tachycardia.

MORPHOLOGY

The **microscopic** changes in heart failure are nonspecific, consisting primarily of myocyte hypertrophy with interstitial fibrosis of variable severity.

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.

CHRONIC VENOUS CONGESTION : LUNG**Right-Sided Heart Failure**

Right heart failure usually is the consequence of left-sided heart failure. Isolated right-sided heart failure also can occur in a few diseases.

Clinical Features

Unlike left-sided heart failure, pure right-sided heart failure typically is associated with very few respiratory symptoms. Instead, the clinical manifestations are related to systemic and portal venous congestion, including hepatic and splenic enlargement, peripheral edema, pleural effusion, and ascites.

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.

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.

Clinical Features

Acute rheumatic fever occurs most often in children; the principal clinical manifestation is carditis. Nevertheless, about 20% of first attacks occur in adults, with arthritis being the predominant feature.

Symptoms in all age groups typically begin 2 to 3 weeks after streptococcal infection, and are heralded by fever and migratory polyarthritis— one large joint after another becomes painful and swollen for a period of days, followed by spontaneous resolution with no residual disability.

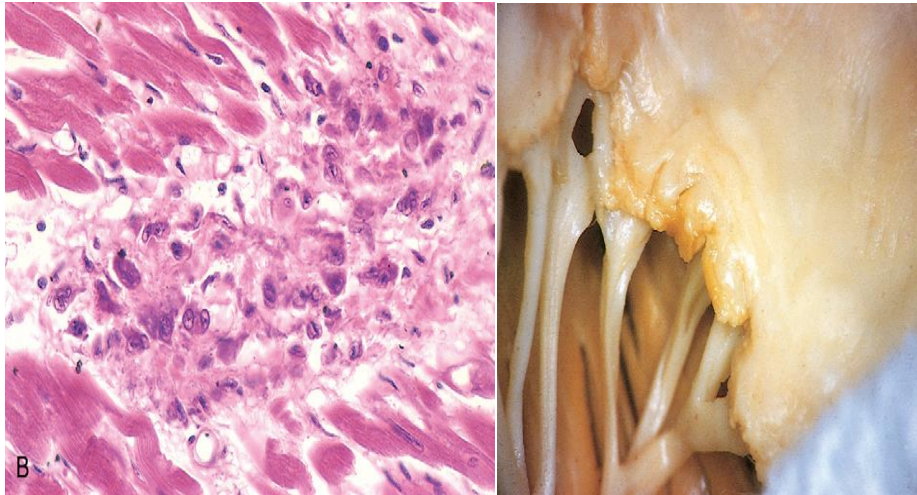
Diagnosis

The diagnosis of acute rheumatic fever is made based on serologic evidence of previous streptococcal infection in conjunction with two or more of the so-called ***Jones criteria***:

(1) carditis; (2) migratory polyarthritis of large joints; (3) subcutaneous nodules; (4) erythema marginatum skin rashes; and (5) Sydenham chorea, a neurologic disorder characterized by involuntary purposeless, rapid movements (also called *St. Vitus dance*).

Minor criteria such as fever, arthralgia's, ECG changes, or elevated acute phase reactants also can help support the diagnosis.

After an initial attack and the generation of immunologic memory, patients are increasingly vulnerable to disease reactivation with subsequent streptococcal infections.



Infective Endocarditis

Infective endocarditis is a serious infection mandating prompt diagnosis and intervention.

Microbial invasion of heart valves or mural endocardium—often with destruction of the underlying cardiac tissues—characteristically results in bulky, friable *vegetation's* composed of necrotic debris, thrombus, and organisms.

The aorta, aneurysmal sacs, other blood vessels and prosthetic devices also can become infected.

The vast majority of cases are caused by extracellular bacteria.

Infective endocarditis can be classified into **acute and sub-acute forms**,

- **Acute endocarditis** refers to tumultuous, destructive infections, frequently involving a highly virulent organism attacking a previously normal valve, and capable of causing substantial morbidity and mortality even with appropriate antibiotic therapy and/or surgery.

- **Sub acute endocarditis** refers to infections by organisms of low virulence involving a previously abnormal heart, especially scarred or deformed valves. The disease typically appears insidiously and—even untreated—follows a protracted course of weeks to months; most patients recover after appropriate antibiotic therapy.

PATHOGENESIS

Infective endocarditis can develop on previously normal valves, but cardiac abnormalities predispose to such infections; rheumatic heart disease, mitral valve prolapse, bicuspid aortic valves, and calcific valvular stenosis are all common substrates.

Prosthetic heart valves now account for 10% to 20% of all cases of infective endocarditis.

Host factors such as neutropenia, immunodeficiency, malignancy, diabetes mellitus, and alcohol or intravenous drug abuse also increase the risk of infective endocarditis, as well as adversely affecting outcomes.

The causative organisms differ depending on the underlying risk factors. Fifty percent to 60% of cases of endocarditis occurring on damaged or deformed valves are caused by *Streptococcus viridans*, a relatively banal group of normal oral flora.

By contrast, the more virulent *S. aureus* (common to skin) can attack deformed **as well as healthy** valves and is responsible for 10% to 20% of cases overall; it also is the major offender in infections occurring in intravenous drug abusers.

Foremost among the factors predisposing to endocarditis is seeding of the blood with microbes.

The mechanism or portal of entry of the agent into the bloodstream may be an obvious infection elsewhere, a dental or surgical procedure that causes a transient bacteremia, injection of contaminated material directly into the bloodstream by intravenous drug users, or an occult source from the gut, or oral cavity.

Recognition of predisposing anatomic substrates and clinical conditions causing bacteremia allows appropriate antibiotic prophylaxis.

Clinical Features

Fever is the most consistent sign of infective endocarditis. However, in sub-acute disease (particularly in the elderly), fever may be absent, and the only manifestations may be nonspecific fatigue, weight loss, and a flulike syndrome; splenomegaly also is common in sub-acute cases.

By contrast, acute endocarditis often manifests with a stormy onset including rapidly developing fever, chills, weakness, and lassitude.

Murmurs are present in 90% of patients with left-sided lesions; microemboli can give rise to petechia, nail bed (*splinter*) hemorrhages, retinal hemorrhages (*Roth spots*).

Diagnosis

Is confirmed by positive blood cultures and echocardiographic findings. Left untreated, infective endocarditis generally is fatal.

However, with appropriate long-term (6 weeks or more) antibiotic therapy and/or valve replacement, mortality is reduced.