potential of viridans streptococci is directly related to the thickness of the capsule, which permits adherence to damaged cardiac valves. Viridans streptococci are normal inhabitants of the mouth and gastrointestinal tract. Invasive dental procedures frequently cause transient bacteremias that may result in SBE on damaged but not normal cardiac valves. Transient bacteremias of viridans streptococci may form a vegetation in the sterile platelet and fibrin thrombus covering an area of damaged endothelium. The gastrointestinal or genitourinary tract is the usual source of bacteremia in cases of native valve SBE due to group D enterococci.

Diagnosis

Clinical Features

The cardinal clinical features of IE are fever (90% of cases) and heart murmur (85%). In the antibiotic era, fever may not be present if the patient has been taking antibiotics for another reason. SBE often manifests with sweats, malaise, and anorexia. The course of SBE tends to be more indolent and may be accompanied by back pain, joint pains (>50% of patients), or embolic stroke. As SBE progresses, circulating immune complexes may deposit in the kidney, causing interstitial nephritis, glomerulonephritis, and even renal failure. Osler's nodes (painful, subcutaneous nodules on the distal pads of the fingers or toes), Janeway's lesions (hemorrhagic, nonpainful macules on the palms and soles) and Roth's spots (retinal hemorrhages with small central clearing) are classic findings related to microemboli and SBE immune-mediated vasculitis.

Patients with ABE tend to have a more fulminant course because of the greater virulence of the pathogen. The fever of ABE is usually high (>102° F) and is often accompanied by rigors. If there is mechanical dysfunction of the valve, symptoms of congestive heart failure will predominate. Often, a presenting feature of right-sided ABE is septic pulmonary emboli with pleuritic chest pain. The clinical findings of SBE and ABE are presented in Table 93-1.

Clinically, PVE may be considered as early (<2 months after implantation of the valve) or late (>2 months). Early PVE is caused by virulent pathogens (e.g., *S. aureus*) that infect the prosthetic valve before endothelialization is complete. Endothelialization of a mechanical valve is partially protective against transient bacteremias in late PVE. Over time, bioprosthetic valves have the same IE potential as mechanical ones.

An otherwise unexplained high-grade or continuous bacteremia and murmur should suggest IE. An acute versus subacute presentation correlates with the virulence of the IE pathogen. If blood cultures are negative, a diagnosis of infectious culture-negative endocarditis (CNE) should be considered if a murmur, vegetation, and peripheral manifestations of IE are present. The clinical diagnosis of IE relies on a combination of clinical, laboratory, and echocardiographic findings. Epidemiologic clues to the potential IE pathogens are outlined in Table 93-2. The most important finding in IE is the demonstration of continuous bacteremia, usually by multiple positive blood cultures. Table 93-3 contains the modified Duke criteria that are frequently used to predict the likelihood that a patient has IE.

TABLE 93-1 CLINICAL FINDINGS FOR SUBACUTE BACTERIAL ENDOCARDITIS (SBE) AND ACUTE BACTERIAL ENDOCARDITIS (ABE)

SYMPTOMS AND FINDINGS*	ABE	SBE
Anorexia	–	+
Weight loss	–	±
Myalgias or arthralgias	+	±
Fatigue	–	+
Dyspnea	+	–
Pleuritic chest pain†	+	–
Low back pain	+	+
Headache	+	±
Mental status changes	+	±
Acute confusion	+	–
Cerebrovascular accident	–	+
Sudden unilateral blindness	–	+
Left upper quadrant pain	Splenic abscess	Splenic infarct
Fever	>102° F‡	<102° F
New or changing heart murmur	±	–
Splenomegaly	–	+
Petechiae	+	+
Osler's nodes	–	+
Janeway's lesions	+	–
Splinter hemorrhages	±	+
Roth's spots	–	+
Congestive heart failure (LVF)	+	–

Modified from Cunha BA, Gill MV, Lazar JM: Acute infective endocarditis: diagnostic and therapeutic approach, Infect Dis Clin North Am 10:811–834, 1996.
LVF, Left ventricular fibrillation; +, present; –, absent; ±, present or absent.
*Otherwise unexplained.
†With septic pulmonary emboli from tricuspid valve ABE.
‡Fever may be <102° F in intravenous drug abusers with ABE.

Early PVE pathogens such as *S. aureus and Pseudomonas aeruginosa* are typically highly virulent and invasive. Late PVE more closely resembles SBE, is caused by less virulent pathogens, and has a more indolent course. The most common etiologic agents are coagulase-negative staphylococci, but viridans streptococci also cause late PVE. Nosocomial IE results from invasive intravascular or intracardiac procedures that damage the endothelium or the valves; it can also be caused by direct extension of infection, such as from ABE associated with a pacemaker wire. The organisms causing nosocomial IE originate from the skin (e.g., *S. aureus*, coagulase-negative staphylococci), from gastrointestinal or genitourinary procedures (e.g., group D enterococci), or from central venous catheters, ports, or hemodialysis catheters (e.g., *Candida* spp, aerobic gram-negative bacilli). IE related to total parenteral nutrition (TPN) is most often caused by *Candida* spp; other TPN-associated fungemias cause IE less frequently. In intravenous drug abusers, tricuspid valve ABE is usually caused by *S. aureus* or *P. aeruginosa* (depending on the geography and drug-related materials).

Infectious CNE is caused by organisms that are difficult to culture, such as *Legionella* spp, *Brucella* spp, *Tropheryma whipplei*, and *Coxiella burnetii* (which produces Q fever). Legionnaires' disease may cause NVE or PVE. CNE due to *Brucella* spp can be a difficult diagnosis, but an antecedent history of contact with livestock or consumption of unpasteurized dairy products should suggest the diagnosis, and echocardiography often reveals large