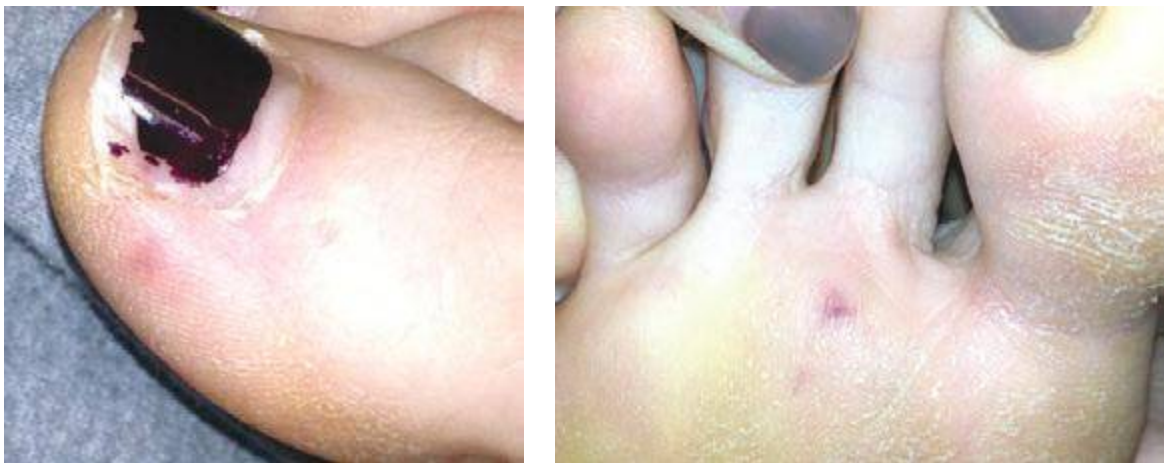
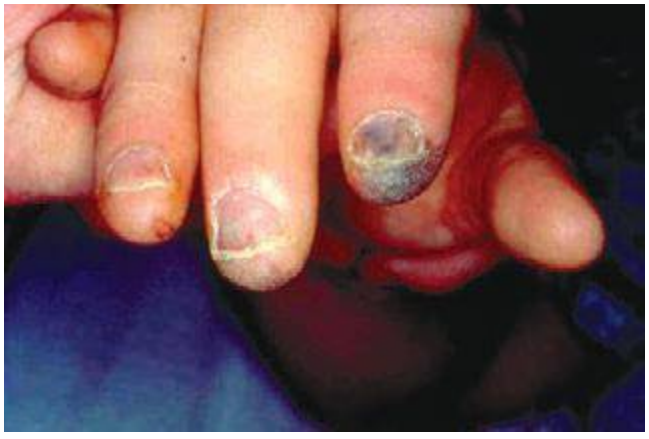
In patients with subacute presentations, fever is typically low-grade and rarely exceeds 39.4°C (103°F); in contrast, temperatures of 39.4°–40°C (103°–104°F) are often noted in acute endocarditis. Fever may be blunted in patients who are elderly, are severely debilitated, or have renal failure.

Cardiac Manifestations Although heart murmurs are usually indicative of the predisposing cardiac pathology rather than of endocarditis, valvular damage and ruptured chordae may result in new regurgitant murmurs. In acute endocarditis involving a normal valve, murmurs may be absent initially but ultimately are detected in 85% of cases. Congestive heart failure (CHF) develops in 30–40% of patients as a consequence of valvular dysfunction. Occasionally, CHF is due to endocarditis-associated myocarditis or an intracardiac fistula. Heart failure due to aortic valve dysfunction progresses more rapidly than does that due to mitral valve dysfunction. Extension of infection beyond valve leaflets into adjacent annular or myocardial tissue results in perivalvular abscesses, which in turn may cause intracardiac fistulae with new murmurs. Abscesses may burrow from the aortic valve annulus through the epicardium, causing pericarditis, or into the upper ventricular septum, where they may interrupt the conduction system, leading to varying degrees of heart block. Mitral perivalvular abscesses, which are usually more distant from the conduction system, only rarely cause conduction abnormalities; if such abnormalities occur in this setting, the conduction pathway is most likely disrupted near the atrioventricular node or in the proximal bundle of His. Emboli to a coronary artery occur in 2% of patients and may result in myocardial infarction.

Noncardiac Manifestations The classic nonsuppurative peripheral manifestations of subacute endocarditis (e.g., Janeway lesions; Fig. 155-2*A*) are related to prolonged infection; with early diagnosis and treatment, these have become infrequent. In contrast, septic embolization mimicking some of these lesions (subungual hemorrhage, Osler's nodes) is common in patients with acute *S. aureus* endocarditis (Fig. 155-2*B*). Musculoskeletal pain usually remits promptly with treatment but must be distinguished from focal metastatic infections (e.g., spondylodiscitis), which may complicate 10–15% of cases. Hematogenously seeded focal infection occurs most often in the skin, spleen, kidneys, skeletal system, and meninges. Arterial emboli, one-half of which precede the diagnosis, are clinically apparent in up to 50% of patients. Endocarditis caused by *S. aureus,* vegetations >10 mm in diameter (as measured by echocardiography), and infection involving the mitral valve, especially the anterior leaflet, are independently associated with an increased risk of embolization. Symptoms, pain, or ischemia-induced dysfunction relate to the organ or area suffering embolic arterial occlusion (e.g., kidney, spleen, bowel, extremity). Cerebrovascular emboli presenting as strokes or occasionally as encephalopathy complicate 15–35% of cases of endocarditis. Again, one-half of these events precede the diagnosis of endocarditis. The frequency of stroke is 8 per 1000 patient-days during the week prior to diagnosis; the figure falls to 4.8 and 1.7 per 1000 patient-days during the first and second weeks of effective antimicrobial therapy, respectively. This decline exceeds that which can be attributed to change in vegetation size. Only 3% of strokes occur after 1 week of effective therapy. Emboli occurring late during or after effective therapy do not in themselves constitute evidence of failed antimicrobial treatment.



A



B

FIGURE 155-2 ***A.*** Janeway lesions on toe (*left*) and plantar surface (*right*) of the foot in subacute *Neisseria mucosa* endocarditis. *(Image courtesy of Rachel Baden, MD.)* ***B.*** Septic emboli with hemorrhage and infarction due to acute *Staphylococcus aureus* endocarditis.