

**Figure 12-24** Comparison of the four major forms of vegetative endocarditis. The rheumatic fever phase of rheumatic heart disease (RHD) is marked by small, warty vegetations along the lines of closure of the valve leaflets. Infective endocarditis (IE) is characterized by large, irregular masses on the valve cusps that can extend onto the chordae (see Fig. 12-25A). Nonbacterial thrombotic endocarditis (NBTE) typically exhibits small, bland vegetations, usually attached at the line of closure. One or many may be present (see Figure 12-26). Libman-Sacks endocarditis (LSE) has small or medium-sized vegetations on either or both sides of the valve leaflets.

bacilli and fungi. In about 10% of all cases of endocarditis, no organism can be isolated from the blood (“culture-negative” endocarditis); reasons include prior antibiotic therapy, difficulties in isolating the offending agent, or because deeply embedded organisms within the enlarging vegetation are not released into the blood.

Foremost among the factors predisposing to endocarditis are those that cause microorganism seeding into the blood stream (bacteremia or fungemia). The source may be an obvious infection elsewhere, a dental or surgical procedure, a contaminated needle shared by intravenous drug users, or seemingly trivial breaks in the epithelial barriers of the gut, oral cavity, or skin. In patients with valve abnormalities, or with known bacteremia, IE risk can be lowered by antibiotic prophylaxis.

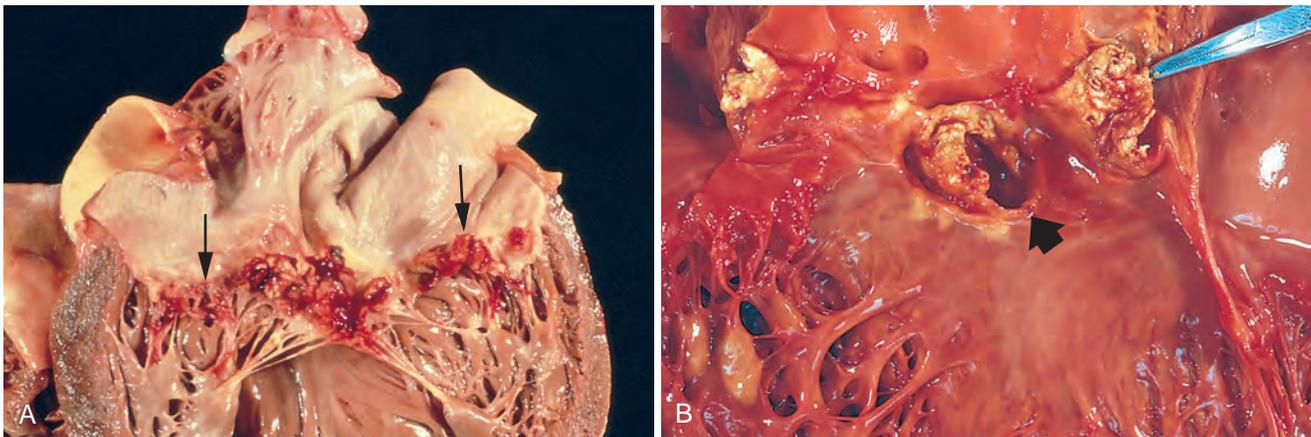
common sites of infection, although the valves of the right heart may also be involved, particularly in intravenous drug abusers. Vegetations can be single or multiple and may involve more than one valve; they can occasionally erode into the underlying myocardium and produce an abscess (ring abscess; Fig. 12-25B). Vegetations are prone to embolization; because the embolic fragments often contain virulent organisms, abscesses frequently develop where they lodge, leading to sequelae such as septic infarcts or mycotic aneurysms.

The vegetations of subacute endocarditis are associated with less valvular destruction than those of acute endocarditis, although the distinction can be subtle. Microscopically, the vegetations of subacute IE typically exhibit granulation tissue at their bases indicative of healing. With time, fibrosis, calcification, and a chronic inflammatory infiltrate can develop.

## MORPHOLOGY

**Vegetations on heart valves** are the classic hallmark of IE; these are friable, bulky, potentially destructive lesions containing fibrin, inflammatory cells, and bacteria or other organisms (Figs. 12-24 and 12-25). The aortic and mitral valves are the most

**Clinical Features.** Acute endocarditis has a stormy onset with rapidly developing fever, chills, weakness, and lassitude. Although fever is the most consistent sign of IE, it can be slight or absent, particularly in older adults, and the only manifestations may be nonspecific fatigue, loss of



**Figure 12-25** Infective (bacterial) endocarditis. **A**, Endocarditis of mitral valve (subacute, caused by *Streptococcus viridans*). The large, friable vegetations are denoted by arrows. **B**, Acute endocarditis of congenitally bicuspid aortic valve (caused by *Staphylococcus aureus*) with extensive cuspal destruction and ring abscess (arrow).