Topol, Eric J., Califf, Robert M., Isner, Jeffrey M., Prystowsky, Eric N., Swain, Judith L., Thomas, James D., Thompson, Paul D., Young, James B.
Textbook of Cardiovascular Medicine, 2nd Edition

ANATOMIC CONSIDERATIONS

Part of "25 - INFECTIVE ENDOCARDITIS"

Vegetations in patients with preexisting valvular lesions are usually located on the atrial surface of incompetent atrioventricular valves or the ventricular surfaces of incompetent semilunar valves. This is likely because of the fact that these surfaces are subjected to injury from regurgitant jets. In patients with ventricular septal defects, vegetations tend to occur on the orifice of the defect, on the right ventricular side of the opening, and secondarily on the tricuspid and pulmonic valves (10). Vegetations may occasionally localize on the chordae tendineae of the anterior leaflet of the mitral valve in patients with aortic insufficiency. Patients with mitral regurgitation may also develop a vegetation (MacCallum's patch) on the wall of the left atrium where the regurgitant jet strikes the atrial wall and results in endocardial thickening (Fig. 25.1).

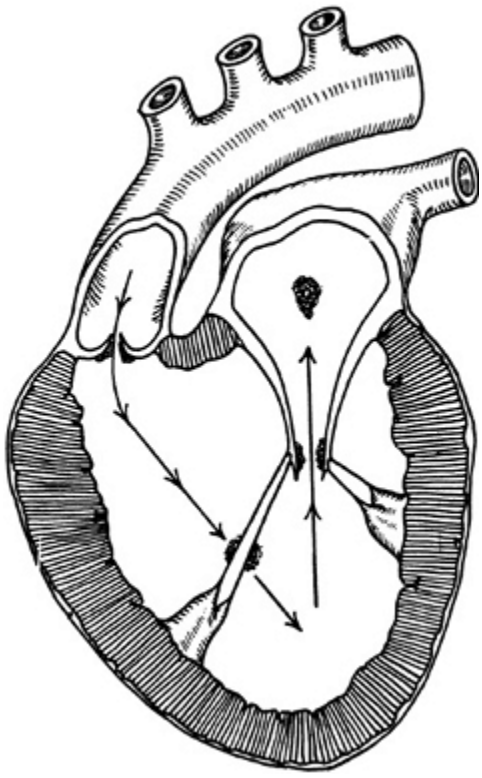

FIGURE 25.1 Sites of high-velocity streams where endocardial injury occurs in mitral and aortic insufficiency. (From Robard S. Blood velocity and endocarditis. *Circulation* 1963;27:24, with

permission.)

The source of infection in some patients with endocarditis may be clinically evident (e.g., an infected vascular catheter, a dental abscess, or an infected skin lesion), but in many patients, there is no history of an antecedent localized

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infection. In most such instances, the source is presumed to be from minor trauma to the oropharyngeal, gastrointestinal, or genitourinary mucosa.

Endocarditis in injection drug users is dominated by one pathogen: *S. aureus. S. aureus* is known to have a predilection for normal as well as abnormal cardiac valves (11). This phenomenon may explain why the majority of injection drug–using patients with endocarditis has no known prior valvular disease (12). Endocarditis in injection drug users is presumed to result as a consequence of the trauma to heart valves from contaminating inorganic debris and bacteria injected along with the illicit drugs, from the paraphernalia used for drug injection, or via bacteria on the skin surface at the site of injection. Tricuspid valve involvement has been noted in 78%, mitral in 24%, and aortic in 8% of drug addicts with endocarditis (12). Simultaneous involvement of more than one valve may occur in approximately 20% of drug-abusing patients (13), and some of these infections are polymicrobial (14).

Approximately three-fourths of all patients with endocarditis have a preexisting structural cardiac abnormality at the time that endocarditis begins (15,16). During the period from 1938 to 1967, rheumatic heart disease was the underlying cardiac lesion in 39% of patients hospitalized with endocarditis at the Presbyterian Hospital in New York (17). In contrast, the authors of a large case series from Tennessee reported that only 6% of patients with endocarditis had underlying rheumatic cardiac lesions (16). Simultaneously, preexisting degenerative valvular lesions such as mitral valve prolapse (usually with coexistent mitral regurgitation) have become relatively more important as a predisposing cause for endocarditis. For example, preexisting mitral valve prolapse was the underlying cardiac lesion in 22% and 29% of cases of endocarditis in two case series (16,18). The estimated risk of endocarditis in patients with mitral valve prolapse and regurgitation has been estimated to be five to eight times higher than the normal population (19,20). However, mitral valve prolapse is a common abnormality and the overall risk of endocarditis in an individual with this lesion is quite small. Aortic valve disease (with stenosis, regurgitation, or both) is a predisposing cause for endocarditis in 12% to 30% of cases (21).

Congenital heart disease is now the underlying lesion in 10% to 20% of cases of endocarditis (10). The most common congenital heart lesions predisposing to endocarditis include bicuspid aortic valves, patent ductus arteriosus, ventricular septal defects, coarctation of the aorta, and tetralogy of Fallot. Unlike most other congenital defects, secundum atrial septal defects are not associated with an increased risk (21).

A succession of new surgical techniques for correction of congenital and acquired valvular lesions has affected the distribution of cardiac abnormalities now seen in patients with

infective endocarditis (IE). The risk of endocarditis in patients with mechanical or bioprosthetic valves is similar. In a multicenter follow-up study of over 1,000 patients who were randomized to receive mechanical or bioprosthetic cardiac valves, the overall rate of prosthetic valve endocarditis was similar in both groups (0.8 cases per year of follow-up) (22). Duration of follow-up averaged 7.7 years; the cumulative percentage of patients who developed prosthetic valve endocarditis was 5.8% (22). However, other authors have suggested that mechanical prosthetic valves are more susceptible to endocarditis initially, whereas after 1 year, bioprosthetic valves are more likely to develop endocarditis (23,23a). In another study, endocarditis was more common after 11 years in patients with mechanical valves than in patients with bioprosthetic valves (23b).

[e in triangle] A limited amount of data exists on the risk of endocarditis in patients with defined cardiac lesions. In a recent study in which 2,401 patients with congenital heart lesions were followed prospectively for a total of 40,855 days, the overall incidence of endocarditis was 135 cases per 100,000 person-years. The highest rates of endocarditis occurred in patients with aortic stenosis (4.8% of 462 cases, or 271 cases, per 100,000 person-years) and with ventricular septal defects (2.4% of 1,347 cases, or 145 cases, per 100,000 person-years). The lowest rate of endocarditis was observed in patients with pulmonic stenosis; only 1 of 592 patients developed endocarditis during the follow-up period (24). Horstkotte estimated that the risk of infective endocarditis in patients with aortic regurgitation was approximately twice as high (73 per 100,000 person-years) (25). The peak gradient across the aortic valve has been linked with the risk of infective endocarditis: The higher the gradient, the higher the risk of developing endocarditis (24).

A history of endocarditis is an additional important predisposing cause for endocarditis. Recurrent endocarditis occurred in 4.5% of patients in a follow-up study of a large cohort of patients who survived their initial episode of endocarditis (26). Other studies have reported rates of recurrence of endocarditis ranging from 2.5% to 9.0% (27). Also, not surprisingly, prosthetic valve endocarditis occurs more frequently when the original indication for the valve replacement was active endocarditis (28).

Other uncommon but notable predisposing causes for endocarditis include pregnancy (10), arteriovenous fistulas used for hemodialysis (29), the use of central venous

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and pulmonary artery catheters (30), the presence of peritoneovenous (LeVeen) shunts for the control of intractable ascites (31), and the use of ventriculoatrial shunts for the management of hydrocephalus (32). In addition, patients with ulcerative lesions of the colon caused by carcinoma or inflammatory bowel disease have a poorly understood predilection to develop endocarditis caused by *S. bovis* (33,34). Infective endocarditis has also been reported in patients undergoing liver, heart, and heart–lung transplantation (35).

A number of cases of endocarditis in patients with human immunodeficiency virus (HIV) have also been reported (36). Some patients with HIV and endocarditis have had infection with unusual organisms such as *Salmonella* and *Listeria* (37,38). Although one study suggested that HIV infection was an independent risk factor for IE in injection drug abusers (39), the results of another study of a large cohort of injection drug users found no evidence that supported this concept (37). Cocaine use may be an additional risk factor for IE in injection drug users. In a study of a large number of injection drug users with fever,

those who used cocaine were significantly more likely to have endocarditis (40).