How to Follow Patients with Mitral and Aortic Valve Disease

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KEYWORDS

• Mitral valve • Aortic valve • Valvular heart disease • Myocardium

KEY POINTS

- The onset of symptoms for any valvular heart disease is ominous.
- Echocardiography is the mainstay of diagnosis.
- Surgery to correct valve disease must occur before irreversible left ventricular damage has occurred and the indicators for such damage must be recognized.

GENERAL PRINCIPLES

All valvular heart diseases (VHDs) place a hemodynamic load on the left ventricle (LV) and/or right ventricle (RV) that, if severe, prolonged, and untreated, damages the myocardium, leading to heart failure and death. Because all VHDs are mechanical problems, definitive therapy almost always (except for secondary mitral regurgitation [MR]) requires a mechanical solution in the form of valve repair or replacement. As with other realms of medicine, these beneficial therapies have inherent risks, including the risk of surgical or percutaneous valve replacement and the risks of valve prostheses, which include structural failure, thromboembolism, and infection. The goals of following patients with VHDs are to assess disease severity and to time mechanical intervention to optimize this risk/benefit ratio. In order to do this, mechanical therapy must occur early enough in the course of disease to avoid hemodynamically mediated myocardial damage and/or sudden death but late enough to avoid unnecessary procedures and unneeded exposure to the risks of prosthetic heart valves. Although it is not perfect, clinical science has evolved so that careful patient surveillance can fairly accurately time this optimum moment for intervention, which is the topic of this article.

AORTIC STENOSIS

Relationship of Pathophysiology to Symptoms

Although rheumatic heart disease is a common cause of aortic stenosis (AS) in developing countries, in developed countries leaflet calcification resulting from an active

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Med Clin N Am 99 (2015) 739–757
http://dx.doi.org/10.1016/j.mcna.2015.02.005 **medical.theclinics.com**
0025-7125/15/$ – see front matter

inflammatory process akin to atherosclerosis is the usual cause of AS.[1] Calcified nodules accrue on the aortic side of the valve causing it to stiffen, reducing its orifice area (**Fig. 1**).[2] Little hemodynamic consequence occurs as valve area decreases from its normal 3.0 cm^2 to one-half that area. However, as orifice area decreases further, left ventricular pressure must increase to drive output past the narrowed valve. At an orifice area of 1.0 cm^2, a mean pressure gradient of 25 mm Hg typically exists between the LV and the aorta. At a valve area of 0.7 cm^2, the gradient increases to 50 mm Hg and at a valve area of 0.5 cm^2 the typical pressure gradient is 100 mm Hg. This pressure overload in turn causes the LV to develop concentric LV hypertrophy (LVH), which is usually viewed initially as a compensatory mechanism.[3] Left ventricular afterload is often described as wall stress (σ), according to the Laplace law, which states that $\sigma = P \times r/2h$, where *P* is LV pressure, *r* is LV radius, and *h* is LV thickness. As the pressure term increases in the Laplace numerator it can be offset by increased thickness in the denominator, maintaining normal afterload and thus normal ejection fraction (EF). However, LVH also results in pathologic consequences and symptoms. The classic symptoms of AS are angina, syncope, and dyspnea. As shown in **Fig. 2**, symptom onset dramatically changes the untreated natural history from a risk of death of less than 1.0% per year in the absence of symptoms to about 25% per year after symptom onset. In turn, symptom onset is linked to hypertrophy.[3,4] Wall thickening may outpace capillary growth, resulting in reduced coronary blood flow reserve potentiating the onset of the symptom of angina.[5,6] Because it takes greater distending pressure to fill a thicker chamber, concentric LVH inherently causes diastolic dysfunction, further exacerbated when collagen content increases as the disease process progresses.[7,8] Systolic function is also compromised in advanced AS, both because wall stress eventually increases and because of an intrinsic loss of contractility.[9] The mechanisms causing contractile dysfunction are under investigation but include abnormal calcium handling and exercise-induced ischemia.[10,11] The presence of diastolic and systolic dysfunction causes the symptoms of dyspnea and other symptoms of heart failure. The pathophysiology of syncope is less certain but often attributed to reduced cardiac output and decreasing peripheral resistance during exercise in the presence of reduced LV cavity size,[12] or to a vasodepressor response caused by exercise-induced obliteration of the hypertrophied LV cavity.[13]

Physical Examination

The diagnosis of AS may first be suspected when the typically harsh systolic ejection murmur is detected during physical examination. This murmur is often described as a crescendo-decrescendo murmur, but, as stenosis worsens, the murmur peaks progressively later in systole until it is primarily a crescendo murmur that radiates to

Fig. 1. Pathology specimens of stenotic aortic valves. From left: a bicuspid valve, a rheumatic valve, and a tricuspid valve. (*From* Sorajja P, Nishimura R. Aortic stenosis. In: Wang A, Bashore TM, editors. Valvular heart disease. New York: Humana Press; 2009; with permission.)