

Another chapter in an enlarging book: Repair degenerative mitral valves

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In this issue of the *Journal*, Gillinov and colleagues¹ provide us with another valuable contribution in the field of mitral valve repair from their large experience at The Cleveland Clinic Foundation available for sophisticated and cutting-edge (ie, Eugene Blackstone) statistical analysis. The superiority of mitral valve repair over replacement in patients with degenerative mitral valve disease is now widely held to be true. It would be surprising to see a single hand raised in a medical audience today if you asked, “Who would prefer to have his or her own valve replaced instead of repaired?” Intuitively it is attractive to keep the parts you were born with. Other potential advantages including better preservation of left ventricular function, avoidance of long-term anticoagulation (mechanical valves) or reoperation (bioprosthetic valves), and *improved survival* all favor mitral valve repair as the gold standard.²⁻⁵ Should the need for a concomitant coronary artery bypass procedure change the paradigm? This is the important question addressed by Gillinov and associates in their analysis of 679 patients with degenerative mitral valve disease and ischemic heart disease undergoing coronary bypass grafting and mitral valve surgery (repair or replacement). It is not surprising that their data provide further confirmation of the superiority of mitral valve repair over replacement in terms of survival, the only end point examined. Impressively, the benefit of mitral valve repair in patients with degenerative mitral regurgitation was noted as early as 2 years after the surgical procedure. This important observation reinforces that the necessity for coronary bypass grafting should not influence our determination to repair rather than replace a degenerative mitral valve.

The authors were challenged by the retrospective nature of their analysis, which included patients undergoing surgery over a 3-decade span: (1) Valve replacements were more prevalent in the early phase of the study, whereas most of the valve repairs occurred after 1990. (2) Likewise, the use of the internal thoracic artery was much more common in later patients in their series, who were also more likely to receive a valve repair. (3) Both replacement and repair procedures underwent significant evolution during the study period. For example, chordal-sparing techniques in the valve replacement group became routine in the late 1980s, but this was not specifically determinable in individual patients. Similarly, valve repair techniques evolved over time, and repairs in their most recent experience were surely improved by the use of annuloplasty prosthetic rings as opposed to annuloplasty using bovine pericardium or non-annuloplasty techniques. (4) Myocardial preservation techniques also evolved and were surely more effective in the later era. (5) The understanding of the pathophysiology of mitral regurgitation and definition of degenerative mitral valve regurgitation also evolved over the study period.

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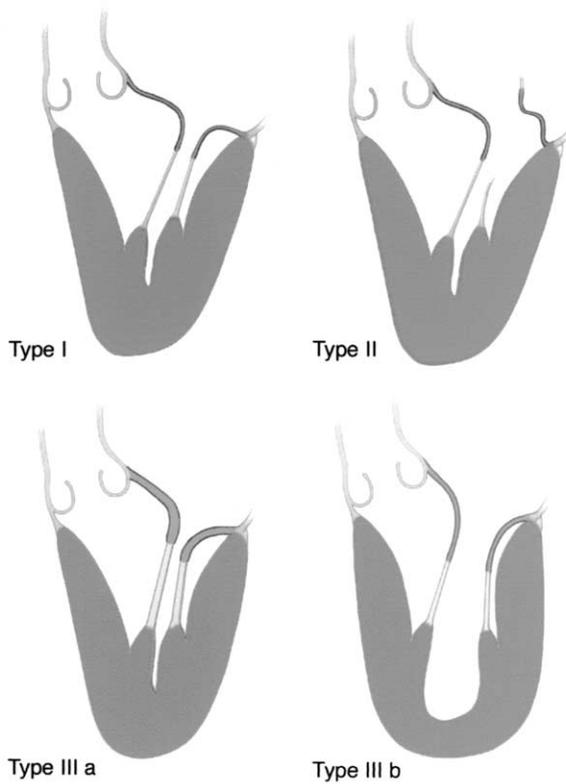


Figure 1. Carpentier's functional classification is based on the opening and closing motions of the mitral leaflets. Type I has normal motion of the leaflets and mitral regurgitation is on the basis of the leaflet perforation or annular dilatation. In type II dysfunction (increased leaflet motion) the free edge of the leaflet travels above the plane of the mitral annulus during systole due to chordal elongation or rupture. Type IIIa dysfunction implies restricted opening leaflet motion during diastole and systole due to rheumatic changes. Type IIIb dysfunction correlates to restricted leaflet motion during systole secondary to papillary muscle displacement.

Craig Miller⁶ has previously provided an excellent editorial comment in the *Journal* regarding the statistical challenges and tools necessary to address the mitral valve repair versus replacement comparison for patients in retrospective nonrandomized trials. Many of the issues Miller discussed are relevant to this article, and we suggest those interested in the nuances of the statistical art refer to it. In the current study by Gillinov and associates, time period was used as a surrogate for chordal-sparing techniques in an attempt to assess the impact of this advance in the replacement group (no advantage was statistically realized.) The potential impact of myocardial protection and the evolution of mitral repair techniques were unfortunately not examined by the authors. For the remainder of issues mentioned previously, there is a lot of “Gene-speak,”⁶ and the authors used sophisticated techniques including multivariable propensity

matching and multivariable, multiphase hazard function analysis to examine factors associated with valve repair or replacement and risk factors for death.^{7,8}

It is interesting to note the confusing terminology, typical of the literature, used by the authors to define degenerative mitral valve disease. Carpentier⁹ has affectionately referred to the usage of multiple terminologies to identify patients with degenerative disease as the “Babel syndrome,” for example, “myxomatous degeneration,” “floppy leaflets,” “billowing leaflets,” and “leaflet prolapse.” It is much more useful to think about mitral valve disease in terms of his *pathophysiologic triad* that includes the type of valve dysfunction, the lesions, and the etiology.⁹ *Valve dysfunction* correlates to Carpentier's functional classification (see Figure 1). Patients with degenerative mitral valve disease most commonly have type II dysfunction, which corresponds to the overriding of the free edge of the leaflet above the plane of the mitral annulus during systole (leaflet prolapse). *Associated* annular dilatation is a common finding in these patients; however, isolated annular dilatation (type I dysfunction) with normal motion of both leaflets has also been reported. Of note, we have not observed leaflet restriction (type III dysfunction) in patients with degenerative disease due to an “abnormal chordal mesh” of the posterior leaflet as described by the authors. *Lesions* most commonly responsible for valve dysfunction in degenerative mitral valve disease include chordal elongation and chordal rupture. *Etiologies* of degenerative mitral valve disease include fibroelastic deficiency (first described by Carpentier), Barlow disease (characterized by myxoid degeneration), and Marfan disease.¹⁰ Fibroelastic deficiency is most common in elderly patients with a relatively short history of valve dysfunction. Intraoperative analysis typically shows transparent leaflets with no excess tissue except in the prolapsing segment, and elongated, thin and frail, often ruptured, chordae. The annulus is often dilated, and may be calcified. In contrast, Barlow disease appears early in life, and patients typically have a long history of a systolic murmur. The valve leaflets are typically thick with marked excess tissue. The chordae are thickened, elongated, and may be ruptured. Papillary muscles are also occasionally elongated. The annulus is dilated and sometimes calcified. Marfan disease with mitral regurgitation is characterized by excess leaflet tissue, which may be thickened (without myxoid degeneration), and a dilated annulus that is rarely calcified. In some patients with degenerative mitral valve disease, the exact etiology of valvular regurgitation remains undetermined.

The importance of a uniform method to identify patients with mitral valve disease is best exemplified by comparing the current study by Gillinov and colleagues¹ in patients with degenerative mitral valve disease and coexisting coronary artery disease with another report by this group¹¹ describing the durability of mitral valve repair in patients

with *isolated* degenerative mitral disease. In the current series (mean age 67 years, 73% male), annular dilatation is not reported as an isolated mitral valve pathologic finding. In contrast, the authors noted annular dilatation as the primary pathologic lesion in 16% of 1072 patients (mean age 58 years, 65% male) with degenerative mitral valve disease undergoing isolated mitral valve repair between 1985 and 1997. It is unlikely that the mitral degenerative process varies in patients with or without coexisting coronary artery disease or that the mean age difference between the two series explains the observed findings. We suspect a more plausible explanation is a variance in their pathologic methodology over time. We also take issue with the concept presented by the authors “that elderly patients have more advanced degenerative disease with more prolapse than do younger patients with isolated mitral valve degeneration.”¹ In our experience Barlow disease and Marfan disease are usually associated with severe mitral regurgitation due to multiple prolapsing leaflet segments and are more prevalent in younger patients undergoing mitral valve surgery.

It is important to emphasize that the authors were very careful to exclude patients with ischemic mitral regurgitation in their study. Patients with this condition usually have type IIIb dysfunction with leaflet restriction in systole, due most commonly to papillary muscle displacement from ventricular dilatation. Their long-term prognosis is significantly worse than that of patients with degenerative mitral valve disease.¹²⁻¹⁵ In the future, we advocate a more uniform adoption in the literature of classification of patients with mitral valve disease according to valve dysfunctions, lesions, and etiologies of disease. These precise distinctions are relevant because the repair strategy depends on valve dysfunction and lesions, whereas long-term prognosis depends on etiology.

As is the case with many retrospective studies, some of the reported findings are not particularly relevant since they do not reflect current practice patterns. For example, the authors observed replacement was more likely in older patients with degenerative mitral valve disease. The safety and efficacy of mitral valve repair in patients with advanced age is now well documented in the literature.^{16,17} Similarly, it is a safe bet that bileaflet prolapse is no longer associated with a significantly increased incidence of replacement in skilled mitral valve repair centers like The Cleveland Clinic.^{2,3,11} One final caveat: we strongly disagree with the concept of an *algorithm* to choose mitral valve surgical strategy as presented by the authors in Figure 4 and Appendix 2. “*Replacement benefit*” is an oxymoron in patients with degenerative mitral valve disease. The only consideration in degenerative mitral regurgitation for repair or replacement should be the surgeon’s ability to achieve a successful repair. There may be isolated patients with advanced left ventricular dysfunction or other comorbidities in

whom the survival benefit of a mitral valve repair is not obvious; however, it is important to emphasize the authors did not assess the quality of valve repair, quality of life, left ventricular function over time, valve-related complications, valve durability, or reoperation in this study.

The methodology of data analysis was complex, and the authors deserve substantial praise for a statistical tour de force to demonstrate the benefit of mitral valve repair in patients with degenerative disease and concomitant coronary artery disease. They have provided further evidence that our patients deserve our best effort to preserve the native mitral valve whenever possible.

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