Surgery for Ischemic Mitral Regurgitation
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Defining the appropriate management of mitral regurgitation secondary to ischemic heart disease — as opposed to primary disease such as rheumatic or myxomatous degeneration — has proved elusive. Coronary artery disease is common, and so is associated mitral regurgitation, at least to some degree. The latter has been recognized as an unfavorable prognostic sign for more than 25 years and thus has been a target of surgical interest, with the goal of negating the adverse effects on late survival without paying too much of an up-front cost in terms of perioperative mortality.

When the regurgitation is severe, debate has centered on the role of mitral-valve repair versus replacement, as recently addressed in the Journal by Acker and colleagues. When the regurgitation is only moderate, however, the question has been whether coronary revascularization alone will suffice or whether intervention on the valve is required. Smith and colleagues now report in the Journal the results of a randomized trial comparing coronary-artery bypass grafting (CABG) alone with CABG plus mitral-valve annuloplasty.

This study is a notable contribution. There are precious few prospective, randomized studies addressing valvular heart disease of any kind and even fewer involving surgical therapies. The Cardiothoracic Surgical Trials Network was established to address this deficiency, and this study is proof that it is capable of doing just that. Admittedly, the negative result is disappointing to the surgeon hoping to “fix the problem” directly and is counter to the popular surgical trend to encourage valvular intervention, but it cannot be said to be entirely unexpected. A number of observational studies over the years have shown much the same. But does this result showing no significant difference between the two surgical approaches put the debate to rest? Unfortunately, I think not.

The trial as described suffers from the same problem of ambiguous language with regard to “ischemic mitral regurgitation” that has plagued this controversy for many years. In the context of coronary artery disease, functional or “secondary” mitral regurgitation, as it is termed in the current American Heart Association–American College of Cardiology guidelines for the management of valvular heart disease, may occur owing to active reversible ischemia or a completed infarction. In either case, impaired ventricular contraction results in wall-motion abnormalities that distort an otherwise structurally normal mitral valve owing to tethering of the leaflets through their chordal attachments to the papillary muscles. It would come as no surprise to discover that ventricular dysfunction due to reversible ischemia may be correctable by revascularization alone, whereas that due to infarction would probably not be. Inclusion of patients with either pathologic feature will muddle the results of any study intended to distinguish the roles of these interventions.

Although not obvious from the moniker, “ischemic mitral regurgitation” is formally defined as that “occurring more than 1 week after myocardial infarction with (1) one or more left ventricular segmental wall motion abnormalities; (2) significant coronary disease in the territory supplying the wall motion abnormality; and (3) structurally normal (mitral-valve) leaflets and chordae tendinae.” Unfortunately, many studies use the term more loosely, including both infarct-related and ischemia-related regurgitation, as it would appear the authors of the current
study have done. Entry into this study required multivessel coronary artery disease and a moderate degree of mitral regurgitation without structural valvular abnormalities; a previous myocardial infarction was not a requirement, and indeed only about 65% of patients had such a history. The inclusion of patients with mitral regurgitation secondary to reversible ischemia may well explain why so many had an improvement in their mitral regurgitation with bypass alone. This may also explain in part why recurrent mitral regurgitation after repair was present in only about 10% of patients, not the 30% reported by others. It is possible that the authors set themselves up to show no significant difference between treatment groups.

Despite these issues, the study is an important one. Although post hoc subgroup analysis is less rigorous scientifically than prespecified subgroup analysis, it will be interesting to follow patients who did and those who did not have improvement in their mitral regurgitation as well as patients who did and those who did not have evidence of a previous myocardial infarction. This is also an important study in helping to establish the feasibility of conducting prospective, randomized surgical trials, so much a part of the culture of cardiology but less so of cardiac surgery. To be sure, it is not easy to admit equipoise when one must act definitively and is expected by one’s patients to express confidence, not uncertainty. But it is important for all of us in the medical community to do exactly this. The authors have helped us along that way.

Disclosure forms provided by the author are available with the full text of this article at NEJM.org.

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