thickening, fusion, and the amount of calcification. Those have helped us in planning our repair. In cases of dense calcification involving both leaflets, we probably would not attempt a repair, although it is feasible to go for if the rewards of repairing the valve in that particular patient were high. We tried to look at leaflet thickening, and, to us, it is difficult to predict that when we compare it with intraoperative findings.

To have a preoperative predictor of success of feasibility of repair, right now we would attempt to repair every regurgitant rheumatic disease short of those that are heavily calcified or totally rigid. Any leaflet that has some amount of pliability, not with a heavy amount of calcification, we would try to repair.

Dr Steven Bolling (Ann Arbor, Mich). That was a great series. I have a technical question. How far out do you take that commissurotomy? Do you take it past the disease point? Do you actually take it into the annulus on both sides?

Dr Dillon. I take it just short of approximately 2 to 3 mm from the annulus. Earlier in my experience, I took it right up to the annulus. I ended up having to put a stitch there, because it frequently leaked there. So what I do is start just short of the annulus and look at the underside of it, look at the fusion of the chords, fusion of papillary muscle, and release the fused leaflet and subvalvular apparatus. Perhaps you could share your experience.

Dr Bolling. So pretty far beyond the natural commissure, almost all the way?

Dr Dillon. Exactly, almost all the way but not quite reaching the annulus.

EDITORIAL COMMENTARY

Mitral valve repair in patients with rheumatic heart disease: What are the limits?

Hartzell V. Schaff, MD

Mitrval valve repair is preferred over prosthetic replacement because, for most patients, successful operation not only relieves the hemodynamic burden of valve regurgitation and resulting symptoms, but also frees them from the hazards of chronic anticoagulation. Moreover, numerous studies have demonstrated that repair of severe mitral valve regurgitation improves late survival, even for those patients who have few or no symptoms.1,2 However, most outcome studies of valve repair have focused on patients with degenerative mitral valve disease, and patients with rheumatic heart disease present special challenges.

The study by Dillon and colleagues3 from the National Heart Institute in Malaysia is important for several reasons. First, the authors have focused on adult patients, for whom the option of valve replacement might be more acceptable than it is for children and young adults, where anticoagulation is especially problematic. Second, they compared their large series of patients with rheumatic valvular disease undergoing mitral valve repair with patients having repair of mitral valve regurgitation that is caused by degenerative valve disease. The good results that they demonstrate, 1% early mortality for patients undergoing isolated mitral valve repair, and 98% freedom from reoperation at 5 and 10 years postoperatively, reflect the skill and experience of their clinical team. In almost every respect, outcomes of patients with rheumatic heart disease having valve repair were similar to outcomes of patients having valvuloplasty for degenerative mitral valve disease.

A few caveats apply. First, even though their study population included only patients aged >40 years (“burnt-out” rheumatics), the average age of these patients was 54 years, and the average age of their patients with degenerative valve disease was only 55 years. These ages are a decade younger than those of the patients we see who are referred for repair of mitral valve regurgitation.1 Whether the same results demonstrated by these authors can be achieved in patients who present later in life is unclear.

Second, Dillon and his surgical colleagues have selected patients carefully, as they should. Among all patients aged >40 years undergoing mitral valve procedures with rheumatic heart disease, mitral valve repair was undertaken in slightly >40% of patients, and this rate of repair was

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even lower, 34.5%, in patients aged >55 years. Most of the patients who had valve replacement presumably had more severe stenosis than regurgitation, but some patients in the present series had mild or moderate stenosis requiring commissurotomy at the time that mitral valve regurgitation was corrected. Thus, a subset of patients with rheumatic heart disease and mitral regurgitation have very damaged, fibrotic valves; appropriate treatment—repair or replacement—depends on the judgment of the operating surgeon. Techniques of leaflet shaving, patch augmentation to extend the leaflet, and chordal replacement, may broaden options for valve repair in rheumatic heart disease, but experience has shown that the more complex the repair, the less durable the late results.

The issue of late outcome of valve repair is especially important in patients who have rheumatic disease leading to valvular regurgitation. In the present study, the risk of reoperation was similar for those who had repair of rheumatic mitral valve regurgitation compared with those who had repair of valve leakage resulting from degenerative causes. These results are encouraging, but readers should recognize that although a few patients were available for assessment 10 years after their operation, the mean follow-up time was slightly >4 years, and valve failure, defined as worsening of regurgitation or reoperation, seemed to be progressive over the first 10 years postoperatively. The lower freedom from valve failure among rheumatic patients was not statistically significant, but it does seem to be an important trend. Further follow-up is necessary to learn whether repaired valves are subject to progressive scarring and fibrosis produced by earlier rheumatic disease.

A few technical points presented by the authors deserve special consideration. They emphasize that when a commissural fusion occurs, splitting fused or fan chordae is important, and when necessary, incision of the papillary muscle, to improve leaflet mobility. These methods are standard for commissurotomy of stenotic valves, but in addition, they are useful in patients with some leaflet fusion and predominate regurgitation. Further, the authors advocate use of artificial chordae, rather than chordal shortening or chordal transfer, for correction of leaflet prolapse, and this lesson has been learned in repairing anterior leaflet prolapse in patients with degenerative valve disease. However, they make the additional point that chordal replacement can be used after division of primary chords to improve leaflet mobility.

Finally, they emphasize the importance of intraoperative echocardiographic guidance, to identify not only residual mitral regurgitation ≥1+, which is a predictor of late repair failure, but also residual eccentric mitral regurgitation, which can cause late hemolysis. Unfortunately, most patients with hemolysis late after mitral valve repair do not have eccentric regurgitation intraoperatively. An important goal for future studies is to document the frequency at which these problems and leaflet coaptation <5 mm are identified and corrected during a second period of cardiopulmonary bypass.

None of the previous comments should detract from the excellent results the authors have achieved in surgical repair of rheumatic mitral valve regurgitation. The group from the National Heart Institute in Kuala Lumpur, as well as other surgeons in Asia, continue to improve surgical techniques and expand the potential for mitral valve repair in patients with rheumatic heart disease.

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