lift up the central portion of both leaflets and results in less tethering of leaflets (Figure 1, E). With 3-dimensional analysis by means of multislice computed tomography, we have demonstrated that the IPMD, which reflects the degree of papillary separation, is a strong determinant of severity of FMR. Moreover, we found that when the papillary muscle tethering distance was resolved into apical and nonapical (posteromedial or posteromedial) components, the severity of FMR was strongly correlated with nonapical components. These findings strongly support our concept of papillary separation.

Another important concept is that valvular tenting of mitral leaflets is determined by the positions of the anterior and posterior papillary muscles. Leaflet tethering is geometrically restricted by the length of the main and aligning chords. Excessive mediolateral displacement of unilateral papillary muscle therefore lifts not only the ipsilateral portion of mitral leaflets but also the contralateral portion in cooperation with contralateral papillary muscle.⁶ This could be a possible explanation why the IPMD is strongly correlated with the severity of FMR.

In summary, apical displacement of papillary muscles has less effect on leaflet tethering and severity of FMR than does posteromedial or posterolateral displacement. Papillary separation results from nonapical outward displacement of papillary muscle, lifting up central portions of both mitral leaflets, which leads to insufficient mitral closure. Leaflet tethering of both mitral leaflets is determined by coordination of the anterior and posterior papillary muscles. The IPMD, reflecting the degree of papillary separation and bilateral papillary muscle displacement, could therefore be a plausible dominant determinant of FMR in patients with left ventricular dysfunction.

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MANAGEMENT OF MILD AORTIC VALVE STENOSIS IN RHEUMATIC MITRAL SURGERY

To the Editor:

We read with great interest the article by Hwang and colleagues¹ on the management of mild aortic valve lesions during surgery for rheumatic mitral valve. We are grateful to them for their excellent and meaningful

work, for we are facing the same dilemma occasionally.

In this study, Hwang and colleagues¹ demonstrated that mild rheumatic aortic stenosis frequently progresses in patients undergoing surgery for rheumatic mitral valve, regardless of initial treatment strategy. We agree with their conclusion. In our practice, the aortic valve should be always inspected in this scenario, especially in young patients. If the valve is found to be grossly deformed or with advanced commissural fusion, aortic valve replacement is performed at the same time. Very little information is available about progression of rheumatic aortic stenosis. Rates of progression of mild stenosis are neither uniform nor predictable.² Usually the progression of aortic stenosis is slow, but in certain patients it may be remarkably rapid. Progression of stenosis from the mild to the severe form has been seen in as little as 2 to 3 years.³ Otto and colleagues⁴ found that the presence of mild aortic stenosis signifies a greater degree of aortic valve disease than mild aortic regurgitation and that turbulence caused by a stenotic valve can contribute to leaflet damage and rapid progression of the lesion. We try to avoid a potential reoperation in the near future, which is frequently denied by patients' families because of the costs. We therefore would like to know whether Hwang and colleagues¹ inspected the stenotic aortic valve during the primary mitral operation.

Hwang and colleagues¹ also found that in the 113 patients in the no treatment group who underwent postoperative echocardiography, the grade of the aortic valve lesion improved to less than mild degree and became aggravated to moderate degree in only 1 patient. We would like to know the diagnosis of the mitral valve disorder in the patient with aggravation. In the presence of severe mitral stenosis, the hemodynamic effects of aortic stenosis remain masked. We

think that the pressure gradient across the aortic valve may be increased after operation as a result of increased preload of the left ventricle after mitral replacement or repair.

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ROUTINE AORTIC VALVE REPLACEMENT FOR MILD STENOSIS IS NOT NECESSARY DURING RHEUMATIC MITRAL SURGERY

Reply to the Editor:

We greatly appreciate the interest in our recently published study by Zhang and colleagues in their letter to the Editor.¹ We designed our study with the same question that they raised: should we intervene in the grossly deformed but functionally mild rheumatic aortic valve (AV) to prevent future AV-related events? The answer obtained from our study was “no.” Although rheumatic aortic stenosis (AS) progresses easily as seen in previous studies,² we demonstrated that prophylactic methods such as AV replacement or repair failed to

prevent adverse events after the primary operation. The aortic annulus is often too small to insert a prosthetic AV of sufficient size in patients with mild rheumatic AV lesions and aortic valve repair also does not guarantee favorable outcomes, as shown in previous studies and ours.^{1,3}

Regarding the authors’ first question about inspecting the AV during the primary operation, we have often inspected the AV at the discretion of the operating surgeon during the study period. Unfortunately, however, we could not clearly demonstrate how many patients in the no-treatment (NT) group underwent AV inspection during mitral valve surgery because of the retrospective nature of our study. Their second question came from concerns that the preoperative AS grade might be underestimated in patients with mitral stenosis and the pressure gradient across the AV might increase after mitral valve surgery. Although we did not provide detailed data on this issue in our published article, only 1 of 5 patients who had AS and pure mitral stenosis in the NT group showed moderate AS early after surgery. Among 16 patients (14.2% of the NT group) who showed improved severity of AV lesions, 13 patients had regurgitant AV and only the other 3 had AS. In these 13 patients with regurgitant AV, 9 had mitral stenosis, 3 had mitral regurgitation, and 1 had mixed lesions. In 3 patients with AS, the mitral valve lesion was mixed steno-insufficiency.

As we discussed as a limitation of our study, we believe that the issues on attitude after a mild rheumatic AV lesion should be addressed in future studies with a large number of patients. Once again, we appreciate the comments and questions of Zhang and colleagues.

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ANTIPLATELET THERAPY AS SOLE ANTICOAGULANT IN PATIENTS WITH AORTIC MECHANICAL PROSTHESES

To the Editor:

I read the very interesting article by Puskas and colleagues,¹ “Reduced Anticoagulation After Mechanical Aortic Valve Replacement: Interim Results From the Prospective Randomized On-X Valve Anticoagulation Clinical Trial Randomized Food and Drug Administration Investigational Device Exemption Trial.” This article represents part of the Prospective Randomized On-X Valve Anticoagulation Clinical Trial (PROACT), which is intended to answer existing questions about anticoagulation in patients with mechanical aortic prostheses (MAPs).

Puskas and colleagues¹ concluded that the international normalized ratio can be safely maintained at 1.5 to 2.0 in high-risk patients after implantation of the On-X aortic prosthesis (On-X Life Technologies Inc, Austin, Tex) in patients who are monitoring with a home international normalized ratio monitor. Some of the results reported are still worrisome, however, because the issue of thromboembolism in patients with MAPs who are undergoing warfarin anticoagulation has not been resolved. The number of