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

strokes was the same in both experimental groups, and the numbers of neurologic events and thromboembolism and thrombosis were higher in the test group (2.07%/patient-year and 1.46%/patient-year). Thus the real benefit of lower anticoagulation levels with warfarin and aspirin is a reduction in bleeding. Thromboembolic episodes still occur.

For the past 13 years, my institution has followed up patients (n = 215) with MAPs who are being treated with the antiplatelet agent clopidogrel bisulfate (INN clopidogrel) or prasugrel hydrochloride (INN prasugrel) plus aspirin as sole anticoagulants. This protocol is based on the principle that embolism after aortic valve replacement is a platelet-mediated event, as described by Becker and associates.² This idea is not new, because the concept was tested with excellent results in 1986.³

In our series of 215 patients (791.1 patient-years) on a regimen of the antiplatelet agent clopidogrel or prasugrel plus aspirin, the incidence of bleeding has been 1.1%/patient-year, that of transient ischemic attack has been 0.3% patient-year, and that of stroke has been 0.88%/patient-year.⁴ When the patients respond to the antiplatelet agent, take their medication, and are compliant, no strokes or transient ischemic attacks have been observed.⁴

With the advent of prasugrel in 2009, we resolved this issue of clopidogrel hyporesponsiveness, and all patients are fully protected from thromboembolism within 24 hours. Patients are loaded with clopidogrel and aspirin. They are tested with the Accumetrics assay (Accumetrics, San Diego, Calif) and thromboelastographic platelet mapping 6 hours after loading. If the patient does not respond to clopidogrel (>50% platelet inhibition) even after a reloading dose of clopidogrel, a loading dose of prasugrel is given. All patients have responded to prasugrel. The maintenance regimen depends on which agent the patient responds to and is

either clopidogrel at 75 mg or prasugrel at 10 mg and aspirin at 81 mg.

In 2009, we reported our initial experience with antiplatelet therapy for patients with MAP.⁵ At that time, clopidogrel resistance was difficult to deal with, and an algorithm was developed. More recent results, incorporating the prasugrel experience, were presented in a poster at the 2013 Congress of the American Heart Association.⁵ The 13-year summary was submitted to the annual meeting of the European Association for Cardio-Thoracic Surgery.

Because of the specificity and success of antiplatelet therapy (treating platelet-mediated events with antiplatelet agents), it is desirable that prospective randomized studies be performed to confirm our results and settle the issue the presumed efficacy of antiplatelet agents for thromboprophylaxis.

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RE: PREDICTORS OF OUTCOME AND DIFFERENT MANAGEMENT OF AORTOBRONCHIAL AND AORTOESOPHAGEAL FISTULAS

To the Editor:

In their recent article in the *Journal*, Mosquera and colleagues¹ reported an interesting single-center historic cohort study on their experience in the treatment of aortobronchial (ABFs) and aorto-esophageal fistulas (AEFs).

AEFs and ABFs are uncommon lesions. If untreated, they carry a mortality approaching 100% as a result of exsanguination. The treatment of choice is still not standardized. Open surgical repair (extra-anatomical or in situ reconstruction) with synthetic vascular prosthesis or cryopreserved homograft, aortic stent-graft placement with thoracic endovascular aortic repair (TEVAR), and conservative medical approaches are all available. Open surgical repair is associated with a high mortality, ranging from 25% to 41%, and TEVAR provides a less-invasive yet still lifesaving and reliable method of treatment for AEFs and ABFs.²⁻⁵

In the study of Mosquera and colleagues,¹ 11 patients did not undergo any surgical treatment; of these, 9 patients were deemed not to be candidates for surgical or endovascular repair because of malignancy, age, or premorbid conditions. In-hospital surgical mortalities were 26.7% in the open surgery group and 37.5% in the TEVAR group, and those patients who had undergone open repair had better actuarial survivals at 1 and 5 years ($P = .04$).

One potential life-threatening complication is prosthetic infection. Although a stent-graft prosthesis may be considered a potential infection carrier, TEVAR has been successfully used to treat AEFs and ABFs.