ratio, 2.54; $P = .006$). Stenosis-type SVD developed early (3–4 years) in patients with PPM, whereas incompetent-type SVD developed late (up to 9 years) in patients without PPM. These authors hypothesized a causative relationship of SVD to PPM.

This report by Flameng and colleagues⁴ is the first to suggest a relationship of SVD to PPM (EOAi $< 0.85 \text{ cm}^2/\text{m}^2$). The other independent risk factor for early stenotic-type SVD was valve size of 21 mm or less. Valve size had no relationship to incompetent-type late SVD. Of the 564 patients, 152 had Medtronic Mosaic (Medtronic Inc, Minneapolis, MN) implants, and currently Flameng and colleagues⁴ have 7 patients being followed for early stenosis-type SVD. Flameng and colleagues⁴ conclude by stating “by avoiding PPM, the incidence of SVD should be reduced by $\geq 50\%$.”

We propose an alternative relationship that SVD developing in smaller size valve with reduced EOA may be identified earlier because of PPM advancing over time. The same hypothesis could be related to late incompetent-type SVD with degenerated changes not developing stenosis in larger valves, and consequently the development of leaflet tears and incompetence from late SVD causing problems of stress-related disease. In our reported study on AVR PPM, survival was not influenced by level of PPM, but bioprostheses was a predictive factor.⁵

The challenge to us that Lawton and colleagues³ experience is SVD caused by moderate PPM is not acceptable, and further investigation is required. These valves had no documented bioprosthetic tissue degenerative changes and no mineralization of leaflet tissue on explant radiographs. In our experience, thrombosis developed in a small percentage of Medtronic Mosaic prostheses, possibly because of the abnormal coagulation profile.

We do not agree with Meuris and Flameng¹ that Lawton and colleagues³ made the correct conclusion. We think our observations are correct,

and these valves do not have SVD developing early from moderate PPM (in fact, we consider moderate PPM an EOAI of $> 0.65\text{--}0.75 \text{ cm}^2/\text{m}^2$; 1 of the cases should be classified as mild PPM, 2 cases should be classified just mild at $0.83 \text{ cm}^2/\text{m}^2$, and 1 case should be classified with no PPM at the time of implantation). We agree with Meuris and Flameng¹ that this phenomenon requires further extensive investigation.

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doi:10.1016/j.jtcvs.2010.07.043

ANTICOAGULATION AFTER BIOPROSTHETIC AORTIC VALVE REPLACEMENT

To the Editor:

We read with interest the article by ElBardissi and colleagues.¹ We concur with their conclusions that early anticoagulation after isolated bioprosthetic aortic valve replacement in patients in sinus rhythm does not seem to reduce the risks of thromboembolism, except in high-risk groups, and that current recommendations should be revisited.

TABLE 1. Survey of United Kingdom consultant practice

Regimen	Percent of respondents
Warfarin 3 mo	15%
Warfarin > 3 mo	0%
Aspirin 3 mo	10%
Aspirin > 3 mo	48%
No medication	10%
Other regimens	17%

In a review of this subject, we² learned that data are not uniform owing to the retrospective analysis of the majority of the articles. Only 2 are prospective randomized trials comparing an antiplatelet agent with vitamin K antagonist^{3,4} and unclear anticoagulation and antithrombotic regimens. Furthermore, in many of the studies, the results of aortic and mitral valve replacements are combined. Despite this lack of evidence, guidelines for management of patients with valvular heart disease published by American College of Cardiology, American Heart Association, American College of Chest Physicians, and European Society of Cardiology all recommend the use of an anticoagulation regimen for the first 3 months after bioprosthetic aortic valve replacement. ElBardissi and colleagues¹ have pointed out that practice differs widely from these recommendations. We carried out a survey of 194 United Kingdom consultant cardiac surgeons on antithrombotic therapy after bioprosthetic aortic valve replacement and received 171 (88%) responses (Table 1). Because of the diverse results of this survey and insufficient evidence in the literature, like ElBardissi and colleagues, we believe that a randomized trial would be necessary.

We embarked on a feasibility phase of this trial. If a definitive randomized trial comparing aspirin with warfarin sodium monotherapy is considered, assuming a stroke rate of 1% to 5% per person-year, 1800 patients are required in each group. If a 3-armed trial comparing aspirin with warfarin or with no therapy is considered, approximately 5400 patients are required in

total. In the United Kingdom, approximately 3500 isolated bioprosthetic aortic valve replacements are performed per annum. Assuming 50% of these patients are eligible for the study and all units participate, the study will take more than 4 years to complete.

One of the main difficulties we have encountered in the feasibility phase of this study is recruitment of patients with pure aortic valve disease who do not have any forms of arrhythmia or coronary artery disease, thus already receiving aspirin or warfarin. Ideally, a randomized study is necessary. Unless this is a multicenter international trial, sufficient numbers of patients cannot be recruited. We may then have to rely on antithrombotic therapy in the high-risk groups.

We would like to thank the British Heart Foundation for supporting the feasibility phase of this study.

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doi:10.1016/j.jtcvs.2010.06.058

Reply to the Editor:

We thank the authors for their comments regarding our recent article discussing the necessity of immediate postoperative anticoagulation in patients with bioprosthetic aortic valves.¹ Although this exact topic has been studied repeatedly over the past decade, there continues to be a lack of consensus, primarily owing to the absence of level 1 evidence. As the authors point out, despite strong evidence to advocate for/against antithrombotic therapy after bioprosthetic aortic valve replacement, guidelines from major international cardiology and cardiac surgery societies continue to recommend antithrombotic therapy for 90 days after bioprosthetic aortic valve replacement.

Although there have been attempts at performing a prospective randomized controlled trial to definitively answer this question and establish a standard of care,^{2,3} these studies have been grossly underpowered, containing no more than 150 patients in each group. Depending on the parameters used to perform a power analysis, a conservative estimate for conducting a randomized controlled trial with an effective power ($\beta > 80\%$) would consist of at least 1500 patients in each group. As the authors describe, conducting such a study would require enrollment from multiple high-volume centers.

The controversy and need for level 1 evidence regarding this topic are illustrated by the variability in antithrombotic therapy regimens, which has been documented in the literature (primarily in the United Kingdom).⁴ Anecdotal, in the United States, we know of institutions and surgeons that routinely anticoagulate with intravenous heparin followed by warfarin sodium and those that provide no anticoagulation whatsoever. These varying practices and unclear data should provide incentive to funding agencies to further study this question. On one hand, we may be placing patients at a high risk of major bleeding events and on the other hand, we may not

be adequately protecting patients from the risk of embolism. In the meantime, however, we believe it is important that we use the best available evidence, which is to avoid routine anticoagulation except in high-risk patients.¹

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doi:10.1016/j.jtcvs.2010.07.044

SURGICAL VERSUS ENDOVASCULAR TREATMENT FOR TYPE B AORTIC DISSECTIONS

Supplemental material is available online.

To the Editor:

We read with great interest the manuscript by Xu and associates,¹ in which they presented their early and midterm results of endovascular repair for the treatment of type B aortic dissections.

Although the Stanford classification is frequently used in the current era, the DeBakey classification seems more