

risks of this technique. This is the classic scientific paradigm: if the current theory does not fit the data, then revise the theory; do not discount reproducible data. The strength and consistency of the observations are compelling enough to give this randomized clinical trial a chance.

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doi:10.1016/j.jacc.2005.05.030

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## Catheter Ablation After Mitral Replacement

Lang et al. (1) report their experience of transcatheter ablation of atrial fibrillation (AF) in patients with mitral valve prostheses (MVP). The investigators claim that patients in both groups were at the extreme end of the spectrum of atrial disease. However, more patients (a total of 14) had paroxysmal AF than did those with chronic AF; this does not necessarily constitute the extreme end of the disease. Did the researchers note any significant differences in the incidences of AF recurrences between those who had paroxysmal and those who had chronic AF?

Lang et al. (1) conclude that the outcomes are similar to those of standard patients undergoing catheter ablation, yet the 73% (75% in controls) sinus conversion rate falls far short of the results achieved by current surgical techniques. The need for subsequent intervention for atrial tachycardia (AT) and recurrent AF was pretty high. Given that AF circuits are unstable, what was the incidence of peri-procedural AF in these patients?

Moreover, the lines of ablation varied within as well as between the groups. Was this variation based on the findings of mapping? It would have been interesting to know what the findings of the mapping were in terms of the sites of the triggers. Given that the lines of ablation were different in these patients, how did the investigators compare the incidences of AF recurrence and AT between the two groups?

Although most studies have concentrated on the conversion to sinus rhythm, AT is emerging as a troublesome complication of most forms of intervention. It is significant that the incidence of AT was 29% in the MVP group, particularly considering that all patients in this group had specific lines of ablation to preclude AT!

Surgical scarring as a cause of AT in these patients is not a tenable explanation as none of them had preablation AT. It is more likely to be a consequence of the inability to create an adequate block at the mitral isthmus owing to the fear of damaging the prosthesis. It is well recognized that the creation of incomplete lines of block will facilitate macro-reentrant arrhythmias. This rate of sinus conversion and prevention of AT is then contingent on our ability to close the mitral isthmus adequately within these

patients, without damaging the prosthesis. We clearly need to refine the ablation technique to address this issue.

None of these patients had a preprocedural diagnosis of AT, suggesting that AT was a consequence of the ablation. Are we then merely replacing one arrhythmia with another? Evidently we need to address this issue.

Notwithstanding these limitations, Lang et al. (1) are to be congratulated for achieving good results in a unique group of patients who are difficult to treat. Ostensibly, the number of such patients will be reduced in the future, as most of these patients will now have AF ablation, concomitant to mitral repair or replacement.

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doi:10.1016/j.jacc.2005.05.027

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## REPLY

We are thankful for the comments made by Dr. Shanmugam as we can further emphasize major points already addressed in the Methods and Discussion sections of our original study (1). Most of all, our goal was to establish for the first time the safety and feasibility of transcatheter ablation of atrial fibrillation (AF) in a very challenging group of patients with mitral valve prosthesis (MVP). We even performed a live satellite broadcast of such a procedure at the last Boston Atrial Fibrillation Symposium on January 14, 2005, in a patient with MVP and chronic AF, where we were able to cardiovert and maintain her into sinus rhythm from the end of the ablation until now.

In our study (1), patients with MVP had both paroxysmal AF (14 patients) and chronic AF (13 patients). Extreme end of the atrial disease was not merely based on the type of AF but on the fact that AF was highly symptomatic and refractory to at least two antiarrhythmic drugs in patients with very enlarged left atrium (55 mm). Also, the 73% (75% in the control group) maintenance rate of sinus rhythm was achieved by percutaneous transcatheter ablation in this selected group of patients. It is not rare to see AF occurrence within the first month following the ablation procedure owing to tissue inflammation, and this does not generally influence the outcomes. Some patients did not have additional lines done in the left atrium as we were in the process of assessing the benefit of these lines, as already mentioned in the original report (1). This was not due to variation in anatomic mapping. Furthermore, 81% of patients in both groups had additional lines performed along the mitral isthmus and in the posterior wall; 12% of the MVP group (vs. 13% of the control group) had only the mitral isthmus line done (again, as we were investigating the benefit of additional lines; data now published [2]), allowing us to compare outcomes in both groups. Postablation left atrial tachycardia occurred in six patients of the MVP group (one in the control group), probably to

incomplete block along the mitral isthmus line because of the risks and inability of getting close to the mitral prosthesis, as we mentioned in the Discussion section of our original study (1). However, surgical scarring in the MVP group, combined with eventual incomplete block, could very well facilitate complex macro-reentrant arrhythmias. Finally, in our Conclusions section, we acknowledge the fact that AF ablation should ideally be performed at the time of MV replacement when feasible.

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doi:10.1016/j.jacc.2005.05.028

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## Myocardial Infarction in the Absence of Obstructive Epicardial Coronary Disease

We read with interest the study by Dokainish et al. (1) reporting characteristics and outcomes of patients with acute coronary syndromes in the absence of obstructive epicardial coronary disease in a post hoc analysis of the TACTICS-TIMI-18 trial. Six percent of patients presenting with an apparent acute coronary syndrome (ACS) and troponin elevation were found to have no significant epicardial disease at angiography. These patients were more likely to be female, and rates of death and reinfarction were lower in this group at six months when compared with those who had angiographically significant coronary disease, regardless of troponin elevation.

The investigators discuss potential mechanisms of myocardial infarction in patients without significant epicardial coronary disease, but they do not consider transient left ventricular apical ballooning syndrome (TLVABS) as a potential explanation in some of these patients (2–6). In a recent systematic review of TLVABS, we and others have pointed out that these patients are typically postmenopausal females who present with acute-onset ischemic cardiac symptoms, electrocardiographic changes, mildly elevated cardiac biomarkers, and characteristic yet transient apical and midventricular wall motion abnormalities in the absence of obstructive epicardial coronary disease (7). The risk of mortality associated with the syndrome appears to be low and recurrence uncommon. It may be that some of the reported patients presenting with an apparent ACS with troponin elevation in the absence of obstructive epicardial coronary disease had TLVABS, which in

turn may explain the observed lower rates of mortality and reinfarction in this patient cohort.

Finally, TLVABS is underrecognized and should be considered in the differential diagnosis of patients presenting with an apparent ACS in the absence of obstructive coronary artery disease. The mechanisms responsible for TLVABS are unknown and deserve further investigation.

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doi:10.1016/j.jacc.2005.05.046

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## Lack of Prognostic Impact of Elevated Troponin Levels in Patients Without Coronary Artery Disease

The study by Dokainish et al. (1) addresses an interesting subgroup of patients with acute coronary syndromes (ACS), namely those with elevated troponin but without significant coronary artery disease (CAD) on angiography. The investigators conclude that a 6.3% incidence of death, reinfarction, and rehospitalization in this subgroup suggests an adverse prognosis as compared to the subgroup without significant CAD and negative troponin.

It is important to note that the figure of 6.3% is based on a total sample size of 32 patients. There were two adverse events in this group including one death and one rehospitalization. The researchers do not report whether these events were cardiovascular or not, information with clear implications for their conclusions. Even if these two events were cardiovascular, the tiny sample size severely limits the conclusions that can be drawn. It is also possible that the bias of knowledge of previous elevated troponin influenced the decision making in the single patient who was rehospitalized.

In addition, data from Table 1 of the Dokainish et al. (1) study