exercise on cognitive function and brain plasticity (9). However, because poor adherence to exercise training is relatively common, effective strategies for optimizing patient adherence are needed (10).

It also should be noted that, though there is an extensive literature documenting the value of exercise in patients with coronary artery disease (CAD), data regarding the significance of stress-management training in improving clinical outcomes are limited. A nonrandomized trial of patients with stable CHD showed that stress-management training was associated with reduced ischemia, fewer cardiac events, and lower medical costs compared to usual care controls (11,12). In the absence of data from large multicenter randomized clinical trials with "hard" clinical end points, we advocate for smaller studies using intermediate biomarkers of cardiovascular risk (13). For example, in a recent study (14) both exercise and stress-management training were found to reduce myocardial ischemia and improve vascular endothelial function, compared to usual care. Furthermore, stress management was actually superior to exercise training in improving measures of heart rate variability and baroreflex sensitivity. These data would strongly support the potential clinical benefits of both exercise and stress management in the routine care of patients with CHD.

*Alan Rozanski, MD
James Blumenthal, PhD
*St. Luke's/Roosevelt Hospital
Department of Medicine
1111 Amsterdam Avenue
New York, New York 10025-1716
E-mail: ar77@columbia.edu

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Hemodynamic Phenomenon or Geometric Discrepancy?

The recent study by Li et al. (1) has several limitations. The absence of information on functional capacities, symptoms, preoperative pressure data, or left ventricular (LV) function indices limits the significance of the data.

Perioperative mortality increases when pulmonary artery (PA) pressures exceed 60 mm Hg, which is the cutoff in risk-stratification systems like the Euroscore. A cutoff of 40 mm Hg overestimates the incidence of severe pulmonary hypertension (PHT). Significantly, 15 of 40 patients with prosthesis-patient mismatch (PPM) had postoperative atrial fibrillation (AF). The difference in mean gradients between the PPM and non-PPM groups is merely statistical, but insignificant. The investigators suggest a "simple" strategy of implanting a prosthesis to obtain an indexed effective orifice area (EOA) [EOAI] >1.2 cm²/m². This arbitrary classification based on EOAI has no surgical significance. There is no mention of valve annular diameters, which is an important consideration in the choice of prosthesis size. The EOAI for the different prosthesis sizes are unavailable.

Most patients have body surface areas (BSAs) between 1.5 and 2 m². This translates to absolute EOAs between 1.8 and 2.4 cm². The minimum absolute EOAI of any size 23 prosthesis is 2.54 cm², which produces an EOAI of 1.2 cm²/m². However, a 23-mm prosthesis would produce an EOAI >1.2 cm²/m². Therefore, a 23-mm mitral prosthesis is clearly hemodynamically restrictive. Most surgeons would implant larger valves than these; it follows that no patient should have PPM if an EOAI of 1.2 cm²/m² is considered the minimum! In consequence, the recommendations by Li et al. (1) are nonspecific and impractical.

Twenty-one of 32 patients with preoperative PHT had PPM. In essence, the smallest valves were implanted in those with large BSAs and preoperative PHT. Naturally, many patients would have residual PHT. Could the investigators have actually undersized the prosthesis in many patients?

Native annular diameter places a major restriction on the maximum implantable prosthesis size. Problems with disproportionately large mitral prostheses include LV outflow obstruction, restriction of prosthetic mobility, circumflex artery and conduction system injury. Complications like atrioventricular groove dehiscence and ventricular rupture with large valves are every surgeon's nightmare. A murine annulus will not take an elephantine prosthesis!
We thank Dr. Shanmugam for his interest in our study (1). Most of the limitations he raises have been discussed in detail in our report. We have never suggested that a cutoff of 40 mmHg was equivalent to severe pulmonary hypertension. Nonetheless, as we have also alluded to in our study, such levels of pulmonary pressures, equivalent to mild/moderate pulmonary hypertension, have been associated with significantly worse outcomes. Moreover, the fact that such levels of pressure would persist in patients with prosthesis–patient mismatch (PPM), whereas they would regress in most patients without PPM, indeed confirms that levels above 40 mmHg are clearly abnormal.

The indexed effective orifice area (EOA) is a physiological parameter that relates to the intrinsic hemodynamic performance of the prosthesis and has nothing to do with valve annular diameters. The threshold value of 1.2 cm²/m² was chosen to identify PPM because it was the most discriminative value to identify patients with persisting pulmonary artery hypertension after mitral valve replacement (MVR), and it is consistent with previous in vitro and in vivo studies on mitral PPM. As we have emphasized, the pressure gradient is a much less appropriate parameter with which to assess the consequences of PPM, especially in the mitral position, because it is highly influenced by chronotropic conditions and because mitral flow tends to decrease when pulmonary resistances are increased.

The statement that “the minimum absolute valve EOA of any size–23 prosthesis is 2.54 cm²” denotes a gross misunderstanding of valve prosthesis physiology and is equivalent to saying that all prostheses of a given labeled size would have similar hemodynamic performance. Indeed, it is well known that labeled sizes have no relevance to valve hemodynamics and that they grossly overestimate the actual EOA, which may vary from one type of prosthesis to another. In this context, it is interesting to note that the normal reference values of EOA for 27-mm mitral prostheses range from 1.6 to 2.2 cm² (2). Hence, it is not surprising that PPM defined as an indexed EOA ≤1.2 cm²/m² can be a frequent occurrence in patients undergoing MVR.

We agree with Dr. Shanmugam that the prevention of PPM in the mitral position is a particularly demanding challenge for the surgeon and that there are not as many options as in the aortic position. Nonetheless, and as we have shown, it is not a rare occurrence and definitely warrants further documentation. Our results also provide impetus for the development of better performing mitral prostheses.

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Drug-Eluting Stent Thrombosis: A Pooled Analysis

With great interest I read the study by Moreno et al. (1) regarding drug-eluting stent thrombosis. The investigators showed a significant relation between the rate of drug-eluting stent thrombosis and the mean stented length in each trial. However, the mean stented length may not represent the stent length of the actual cases, especially in these few occurrences of thrombosis (~0.5%). In their study, only 15 cases suffered from drug-eluting stent thrombosis. Collecting individual patient data will provide the least biased and most reliable means of addressing questions (2).

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We thank Dr. Kaneda for his interest in our study. We agree with the affirmation that the mean stented length for each study may not necessarily represent the stent length of the actual cases of stent thrombosis. Because of that, as we described in the Methods section (Statistical Analysis), we contacted the principal investigators of all studies in which at least one drug-eluting stent thrombosis was documented, requesting the total stent length for