of patients have persistent symptoms due to left ventricular outflow tract (LVOT) obstruction following ASA, which is likely due to suboptimal procedural result (3–5). A recent study demonstrated that patients undergoing ASA usually end up with a basal inferoseptal (as opposed to the preferred anteroseptal) infarction, as compared with patients who undergo myectomy (6). That study also demonstrated that myectomy resulted in a "more complete" relief of LVOT obstruction than did ASA (6). Another issue that needs attention, and perhaps should be studied in the future, is that of myocardial scarring that is induced by ASA and the potential for ventricular tachyarrhythmias. Also, there are many other potential reasons for suboptimal results following ASA. A recent study (7) highlighted other important mitral subvalvular aspects leading to a failed ASA. It demonstrated the multifactorial etiology of dynamic LVOT obstruction, which includes mitral apparatus abnormalities, such as anterior displacement of the papillary muscles leaflet elongation and anteriorly displaced coaptation of the mitral valve leaflets. We have also observed abnormal chordae attachment to the base of the anterior mitral leaflet resulting in systolic "buckling" of and dynamic LVOT obstruction, even in the setting of a normal-sized upper septum. In fact, 1 large surgical series identified abnormalities of the mitral valve in 19% of patients undergoing surgical myectomy, which required further modification of surgical technique to relieve obstruction (8). A priori identification of such abnormalities might be crucial for an optimal result following ASA.

As aptly described in a recent editorial by Sigwart (9), "ASA was never devised to replace surgery for symptomatic patients with HOCM. It was intended to provide those patients, young and old, who have favorable (and accessible) anatomy, with an alternative to open heart surgery through the induction of a meaningful septal necrosis." In conclusion, we stand by our assertion that at present, open heart surgery through the induction of a meaningful septal necrosis. In conclusion, we stand by our assertion that at present, open heart surgery through the induction of a meaningful septal necrosis. In conclusion, we stand by our assertion that at present, open heart surgery through the induction of a meaningful septal necrosis. In conclusion, we stand by our assertion that at present, open heart surgery through the induction of a meaningful septal necrosis. In conclusion, we stand by our assertion that at present, open heart surgery through the induction of a meaningful septal necrosis. In conclusion, we stand by our assertion that at present, open heart surgery through the induction of a meaningful septal necros...
the VBT group. This group does not reflect the latest practice of VBT: the dose was lower, the patients in the study were treated with stents that were found to be associated with worse results when combined with VBT, and the duration of clopidogrel in that cohort was limited. These issues raise flaws in the methodology because the concurrent enrolled group and the historic group should not be pooled for the analysis.

Second, although the study was not powered to detect differences in hard clinical end points, at 3 years of follow-up, the overall cardiac mortality, Q-wave and non-Q-wave MI were higher in the SES group than in the VBT group. As detailed in Table 2 (1), the overall mortality and incidence of MI increased with SES therapy over 38% and 48%, respectively.

Third, although the TLR was lower in the SES group, major adverse cardiovascular events were similar, suggesting that the rise in major adverse cardiovascular events with SES was related to an increase of death and MI. In addition, the incidence of cumulative stent thrombosis was higher in the SES-treated group (4.2%).

Fourth, Holmes et al. (1) suggested that there is clinical evidence that coronary restenosis after BMS implantation is associated with high incidence of MI and death (2). Currently, most BMS restenoses are treated with drug-eluting stents (DES), which are associated with high rate of late stent thrombosis and perhaps lead to increase in death and MI (3).

In contrast, in the era before DES, BMS restenosis was not associated with major incidence of death or MI. In the head-to-head comparisons of BMS to coronary artery bypass graft, although BMS was associated with increase of TLR when compared with coronary artery bypass graft, this was not associated with increase of mortality or MI. In fact, in a recent meta-analysis from 4 large randomized studies (4), survival was almost identical in both BMS or coronary artery bypass graft groups in spite of repeat revascularization, which was 4 times higher in BMS patients. In addition, in the ERACI III (Argentine Randomized Trial of Coronary Stents versus Bypass Surgery) study registry (5), at 3 years of follow-up, regardless to the reduction of TLR, both DES and BMS groups had similar incidence of death and MI, including diabetic patients. In ERACI III, all BMS data was collected in the era before DES; therefore, any BMS restenoses was treated with DES implantation.

In conclusion, the 3-year report of the SISR (Sirolimus-Eluting Stent Versus Vascular Brachytherapy for In-Stent Restenosis) study alarms us regarding the high rates of stent thrombosis and mortality and raises the question of what is the ideal therapy for BMS restenosis. Clearly when these patients are treated with SES, they should be monitored closely, perhaps with indefinite dual antiplatelet therapy, to prevent late events until we get more data from large randomized clinical trials and registries.

*Alfredo E. Rodriguez, MD, PhD, FACC, FSCAI
Ron Waksman, MD, FACC
*Interventional Cardiology Unit
Otamendi Hospital/Buenos Aires School of Medicine
Azcuenaga 870 (AAC1115) Buenos Aires
Argentina
E-mail: rodrigueza@sanatorio-otamendi.com.ar

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**REFERENCES**


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**Reply**

We thank Drs. Rodriguez and Waksman for the interest in our study (1). Certainly, caution is always mandated as we evaluate the longer-term outcome of strategies of care. Longer-term follow-up was very important for the field of vascular brachytherapy because it documented the late catch-up phenomenon that decreased the long-term effect of vascular brachytherapy and was one of the reasons vascular brachytherapy is rarely used today.

Drs. Rodriguez and Waksman reiterate our published comment that this “trial was neither powered nor designed for long-term follow-up” (1). It was powered for the primary 9-month end point. Although by protocol, patients continue to be followed up for 5 years.

Given the sample size of the population, the usual statistical analysis did not document a significant difference in cardiac mortality. Obviously, longer-term follow-up to 5 years will be important to see if trends emerge.

Drs. Rodriguez and Waksman are correct in that target lesion revascularization was indeed lower in the sirolimus-eluting stent group. There was a difference in absolute percentage of stent thrombosis. We reported any Academic Research Consortium (ARC) thrombosis as well as the other definitions of thrombosis. Drs. Rodriguez and Waksman choose to focus on any ARC thrombosis. However, most investigators prefer to use definite or probable ARC thrombosis rates, which occurred in 2.4% of vascular brachytherapy and 3.5% of sirolimus-eluting stents (p = 0.758).

Drs. Rodriguez and Waksman also support our belief that restenosis after bare-metal stents is not benign. This is not a new finding and we provided references (36) to (38) as further support (see Holmes et al. [1]).

Finally, we share the concerns of Drs. Rodriguez and Waksman. In an era of bare-metal stent in-stent restenosis treated with drug-eluting stents, our patients continue to need vigilant follow-up. More data is certainly needed.

We do appreciate the concerns raised and believe they will only be adequately addressed by larger numbers of patients with very careful follow-up.