REPLY: Going Over LEGACY With a Pinch of Salt

We thank Dr. Valentova and colleagues for highlighting some of the key findings of the LEGACY (Long-term Effect of Goal-directed weight management in an Atrial fibrillation Cohort studY). This study demonstrates that in overweight and obese individuals with symptomatic atrial fibrillation (AF), weight loss had a dose-dependent effect on the long-term freedom from AF (1). This was an observational registry in which measurement and sampling bias was minimized through standardized processes, evaluation by blinded operators, and the routine collection of outcome data. Details of patient flow and attrition are provided in the manuscript.

Controversy does exist regarding long-term sustainability of weight loss. Indeed, greater weight loss corresponded with higher participation in the weight management clinic. In addition, the weight loss achieved in patients attending the dedicated clinic was progressive and sustained with less fluctuation. It is incorrect to suggest that the results of this study are an epiphenomenon due to higher compliance. Improved lipid profile, blood pressure, and glycemic control were evident with weight loss, with the use of fewer medications. In this series we did not have consistent data on follow-up polysomnography; however, in a smaller series we have presented that weight loss reduced sleep apnea (2).

The numbers and type of antiarrhythmic drugs used were not different at baseline. At final follow-up the number of antiarrhythmic drugs used was significantly lower with increasing weight loss. No patient remained on amiodarone. The numbers of ablation procedures performed in 3 groups were not different. There were no deaths in this study.

It has been suggested that propensity score matching (PSM) be used to mitigate potential biases to maintain a balanced design. The baseline characteristics of this cohort demonstrated no differences in means, proportions, and distributions between the 3 weight loss groups. Thus, using PSM would not have resulted in a significantly higher balance to that which is already observed. The most important reservation in using PSM in the current analysis is the sample size. PSM is a large sample tool and its use in the current dataset may in fact increase the unbalance among the covariates. This loss was considered unwise given the potential nonsignificant improvement in the balance between the groups.

Finally, we agree on the need for a randomized control multicenter trial—which is underway. However, it is important to realize that the impact of weight fluctuation on AF burden cannot be evaluated in a randomized study design. While we can argue on the mechanisms and vacillate, given the expanding epidemic of obesity and AF, it is prudent that primary and secondary prevention strategies be instigated urgently. Evidently, aggressive risk factor management with weight loss and increased physical activity are crucial elements in the management of AF (2,3).

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Secondary Mitral Regurgitation

In their review of secondary mitral regurgitation (MR), Asgar et al. (1) attribute abnormal leaflet tethering largely to apical and lateral papillary muscle displacement. To our knowledge the first comprehensive quantitative echocardiographic study of this topic was reported in 1983 (2). In that study no difference was found in chordal eccentricity, papillary muscle tethering length, and left ventricular volume between MR and no-MR groups. Instead, the abnormal, apically displaced tethering associated with MR derived almost exclusively from annular dilation, in association with