Response to Letter Regarding Article, “Familial Clustering of Mitral Valve Prolapse in the Community”

We thank Dr. Barison and colleagues for their interest in our article.1 In our manuscript, we demonstrate that both parental mitral valve prolapse (MVP) and parental mild, nondiagnostic MVP are associated with increased prevalence of MVP in the offspring in the Framingham Heart Study community. In their letter, Barison et al. underline the importance of age when assessing MVP prevalence, stating that MVP is more common among older participants in the offspring or generation 2 (Gen 2) than in the younger generation 3 (Gen 3). Based on published Framingham Heart Study literature,2 the prevalence of MVP in Gen 2 is 2.4% at their fifth examination cycle. This percentage is slightly higher in the same cohort at their sixth examination cycle (98/3380 or 2.9%) and at their eighth examination cycle (94/2725 or 3.4%; ie, 11 to 17 years after the fifth examination). In addition, in the younger Gen 3 (average age of 40 years at their first examination cycle) the prevalence of MVP is 1.4% (56/4061) and lower than in Gen 2 (average 55–67 years of age at their sixth and eight examination cycles, respectively). Of note, the proportion 49 of 3679 or 1.3% reported in our article is not the prevalence of MVP among all Gen 3 participants, but only that among those with available parental information. Overall, these findings are consistent with an age-related increase in the prevalence of MVP. In reevaluation of MVP status at follow-up examination cycles of Gen 3 may help confirm these initial observations.

We agree with Barison et al. that only longitudinal data can answer the questions about the echocardiographic progression and long-term prognostic significance of MVP in the community. To date, MVP progression has been shown to be rapid, although previously published data relied on tertiary care samples3 or nonrandomly selected cohorts.4 Barison and colleagues also note the similarities in our article between Gen 3 participants with and without parental MVP with regard to left ventricular size and systolic function. A more direct comparison between MVP/nondiagnostic MVP morphologies and no MVP (rather than between parental and nonparental MVP) in the Framingham Heart Study Gen 2 also showed similar left-sided chamber size and function in the 2 groups.5 These results simply suggest that MVP and nondiagnostic MVP morphologies are related to true valvular disease rather than the consequence of a small, hyperdynamic ventricle. The question whether MVP is primarily a genetically determined disorder with mechanical/electric myocardial involvement is more complex and cannot be answered by our observations. Indeed, additional larger studies are warranted to address the interesting points raised by Barison et al.

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Disclosures

None.

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