diagnosed LVHT is more often visible on CMR in patients with an enlarged left ventricle and systolic dysfunction than in those with normal-size, well-contracting left ventricles (4). Did any of the patients undergo follow-up CMR studies and did the ratios of noncompacted to compacted layers and the trabecular mass change? 2) From pathoanatomic investigation we know that endocardial fibrosis is frequently found in LVHT patients (5). Was that phenomenon also visible on CMR? 3) Because of unknown reasons, LVHT was diagnosed more frequently in male than female patients. How can the relatively high proportion of female patients in the present study be explained?

In conclusion, only close cooperation between cardiologists, radiologists, neurologists, and cardiac pathologists will solve the enigmas of LVHT.

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REPLY: Diagnostic Problem of LV Hypertrabeculation/Noncompaction?

We thank Drs. Stollberger and Finsterer for their interest in our recent publication (1). They raised several points that will help clarify future discussions regarding left ventricular noncompaction (LVNC).

Although the criteria for LVNC cardiomyopathy are evolving, the phenotypic characterization has been previously reported. We used standard criteria based on the end-systolic (2), end-diastolic (3), trabecular mass (4) ratio and ensured the presence of apical trabeculations with blood noted in trabecular recesses. Furthermore, we observed 88% concordance between systolic criteria with cardiac magnetic resonance and interpretable echocardiography in our patient cohort. In our experience, the most common cause of overdiagnosis is the singular dependence on the long-axis views. An obliquely imaged papillary muscle in the long axis may give the appearance of trabeculation instead of papillary muscle because the long-axis cardiac images are optimized for wall motion analysis and not papillary muscle anatomy. By using the short- and long-axis views together, an interpreter has the opportunity to distinguish between papillary muscle and trabeculation. As to whether some trabeculation may represent normal variants, 43% of the MESA (Multi-Ethnic Study of Atherosclerosis) population had 1 segment with an end-diastolic noncompacted to compacted ratio >2.3 (5). The clinical relevance of a single-segment pattern is uncertain. Our patients had multiple segments involved and occasional right ventricular involvement. Of note, no LVNC pattern criteria were associated with late gadolinium enhancement, making infiltrative or idiopathic disease such as endomyocardial fibrosis unlikely. Among the 8 patients who had serial cardiac magnetic resonance evaluation, no substantive changes in the pattern of LVNC were appreciated.

We agree that a pathoanatomic gold standard is lacking at this time; however, increased recognition and longitudinal clinical care may yield a pathway to obtaining tissue and address these concerns. Patients in our study did not have diagnoses of neuromuscular disorders or congenital heart disease and were not biologically related. Although a longer duration of clinical follow-up would be ideal, a mean follow-up of 500 days in our cohort does provide clinically useful short- and intermediate-term information. Concerns regarding genotypic myopathies as a clinical subset are not answered by our data, and future proposed collaboration may help to investigate these associations.

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