In Reply We thank Drs Gati, Merghani, and Sharma for their interest in our discussion about the challenges of left ventricular hypertrabeculation in athletes. Like many rare diseases, the diagnostic criteria for left ventricular noncompaction (LVNC) are derived from small cohorts, which inherently lead to poor specificity. Given the low prevalence of this disease, a long-term registry is likely needed before diagnostic criteria with high specificity can be produced. Our understanding of incidental T-wave inversions on ECG is evolving. In the precordial leads, T-wave inversions could be a manifestation of arrhythmogenic right ventricular cardiomyopathy (ARVC). T-wave inversion and reduced systolic function appear useful as part of the diagnostic criteria for LVNC as well. Of note, a recent paper by Brosnan et al describes T-wave inversions in healthy endurance athletes recorded in leads V2-3. They hypothesized that this is likely secondary to displacement of the RV toward the axilla rather than RV dilation or hypertrophy and therefore unlikely to be pathologic.3

Interesting study findings like those from Brosnan et al imply that magnetic resonance imaging (MRI) results should be part of this reformed criteria for LVNC. The majority of studies used to develop diagnostic criteria to date have used echocardiography as the imaging modality of choice, but MRI has been shown useful at distinguishing clinically relevant noncompaction and should be utilized in future criteria.4

Using pregnancy as a model, Gati et al have suggested that left ventricular hypertrabeculation is an epiphenomenon occurring in response to stress or increased preload. If this is the case, as with the pregnancy model, hypertrabeculation in the athlete should regress, if not resolve, when the preload conditions change.5 This would be in opposition to LVNC that, as an inherited disorder, is likely present regardless of circulatory conditions and therefore less responsive to detraining. It remains unclear how hypertrabeculation in the athlete progresses over time along with changes in training intensity or with medical treatment. Using MRI, a group from the Netherlands reported a marked decrease in trabeculations in a 58-year-old male after standard heart failure treatment was started.6 Would it be possible to reverse hypertrabeculation in the healthy athlete through detraining or even medical treatment? In our patient, detraining has been considered. But limiting conditioning and/or practice for several weeks (or more) even in the off-season are not easy tasks. The field of sports cardiology must continue to seek how best to identify those athletes with high-risk features from those with natural adaptation to high-intensity exercise.

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