Editorial

Cardiac Magnetic Resonance of Left Ventricular Trabeculation
The New Normal

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Historically, left ventricular noncompaction (LVNC) was considered to be a rare form of cardiomyopathy; however, during the past decade, there has been a significant increase in reports of patients with LVNC. Using either echocardiography or cardiac magnetic resonance (CMR) imaging, LVNC is relatively straightforward to diagnose in individuals expressing a typical phenotype. However, the diagnosis may be challenging to make in those individuals who express more subtle phenotypes of LVNC. CMR imaging offers a more detailed examination of myocardial structure and composition compared with other imaging modalities, such as echocardiography. With more common use of CMR, there has been increased appreciation of the degree of hypertrabeculation. Persistence of the left ventricle (LV) trabeculae in the postnatal myocardium may be associated with cardiomyopathy, and LVNC is classified as a cardiomyopathy.2,3

LVNC Diagnosis

There is a lack of consensus surrounding LVNC diagnostic criteria. The imaging criteria for LVNC differ depending on whether echocardiography or CMR is used. Both imaging modalities use the ratio of the thickness of the trabeculated layer to the thickness of the compacted layer (T/C). However, a commonly used diagnostic criterion for echocardiography requires the T/C to be >2.0 at end systole,4 whereas the diagnostic criterion for CMR is T/C >2.3 at end diastole.5 Stollberger and Finsterer6 also proposed echocardiographic criteria requiring the presence of at least 3 deep trabeculae that are contiguous with the ventricular lumen and not related to the papillary muscles. Autopsy examination for hypertrabeculation showed 4% of normal hearts with evidence of hypertrabeculation and even mural thrombi.7 These diagnostic modalities and criteria each have limitations, including autopsy. Evaluation at a single time point and the postmortem state without flow may challenge the identification of trabeculae. Further complicating matters is that it is unknown which imaging plane is best to most accurately measure the thickness of the trabeculated and compacted layers. In this issue of Circulation: Cardiovascular Imaging, Dawson et al8 measured the LV in the short-axis plane; however, previous investigators have used the long-axis imaging plane.3 The current work evaluated CMR findings from 120 normal healthy individuals of varying ages and both sexes to establish the norms for the compacted and trabecular layers of LV myocardium.8 Wall thickness was not found to differ between men and women, and only in the oldest group was there evidence of significant increase in wall thickness. However, with progressive age, the trabecular layer thinned and the compact myocardium thickened. The compact layer was thicker with increasing age at both end diastole and end systole. The compacted layer was significantly different between men and women at both end systole and end diastole. The trabecular layer did not differ between men and women.

A trabecular layer was visible in all hearts, although it was visible on some segments more than others.8 The endocardial layer of the septum was least likely to demonstrate a trabecular layer, whereas the anterior and lateral segments were most likely to display obvious trabeculation. Four individuals in this noncardiomyopathic population were noted to have increased trabeculation, although this was observed at end diastole where thickness is greater than at end systole. However, the finding that 4 of 120 individuals demonstrated increased trabeculation is consistent with the autopsy findings of Boyd et al.7 This number also is consistent with the findings of Kohli et al,9 who noted in their heart failure population that increased trabeculation was more likely to occur in those of remote African descent. However, the ethnicity of the population studied by Dawson et al8 was not described.10 Together, these findings may suggest that in some circumstances, hypertrabeculation may be a normal variant.11

Genetics and LVNC

The findings that certain ethnic groups may have increased LV trabeculation supports that genetic variants may play a
role in the morphology of LV trabeculation. Certain genetic variants are known to predispose to cardiomyopathy and LVNC. Mutations in the genes encoding α-dystrobrevin and tafazzin have been identified in children with LVNC. Mutations in sarcomere genes also have been found in families with LVNC and patients with cardiomyopathy and hypertrabeculation. Acquired LVNC has been described in the setting of myopathy. The Dawson et al study places these findings in context because it can now be appreciated that there is progressive change of the compact layer with age and that this may influence the appearance of LV trabeculae.

An important diagnostic dilemma is the differentiation of LVNC from dilated cardiomyopathy, which occasionally can also have a pronounced trabecular layer. In fact, Kohli et al suggested that up to 24% of patients with dilated cardiomyopathy fulfill the diagnostic criteria for LVNC, but it is unclear whether this is because the diagnostic criteria for LVNC are too sensitive or whether LVNC is underdiagnosed. The ability to distinguish these disorders may require the evaluation of other features, such as LV geometry and sphericity, overall wall thickness, ejection fraction, the presence of an extensive meshwork of trabeculae.

Clinical Implications and Future Directions

The Dawson et al study significantly increases our knowledge of normal ventricular wall morphology. The limitations of the study are that the same patients were not imaged over time, and therefore, progression must be inferred. Because this study was of a normal healthy population, the future outcome of individuals in the younger age groups is not known. The family history and ethnicity was not reported. The criteria for LVNC and its classification as a cardiomyopathy will continue to be debated until longitudinal cohort studies are carried out. For those subjects with hypertrabeculation, further examination of first-degree relatives to determine whether these findings may be inherited.

The current study improves our understanding of the normal variation that exists in the regional extent of LV trabeculation as a function of age and sex. These new data show that the compacted layer is thicker in men than in women. They also suggest that the apparent trabeculated layer becomes thinner with age, whereas the apparent compacted layer becomes thicker. The authors suggest that this finding may be due to hypertrophy of the trabeculae, making it more difficult to differentiate the trabeculated layer from the compacted layer rather than an actual change in the composition of the layers. We also learn that the thickness of the apparent trabeculated layer and the compacted layer changes during the cardiac cycle. Once again, the authors suggest that this is a secondary finding related to an inability to accurately differentiate the layers at end systole. Regardless of the mechanism responsible for the apparent changes in the T/C ratio seen at end systole, these findings raise the possibility that the diagnostic cutoff values of the T/C ratio may need to be adjusted according to age and sex, depending on the cardiac phase selected for making measurements.

There still remain questions with regard to management. If a patient is found to have deep trabeculae, irrespective of imaging modality, how should this condition be managed? In the setting of cardiomyopathy, it has been suggested that anticoagulation be considered to reduce embolic risk. However, in the absence of cardiomyopathy, it remains unclear whether there is increased risk associated with deep trabeculae and the need for serial imaging in an otherwise healthy patient. Further studies are warranted, but the current study provides the necessary background.

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None.

References


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