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iVIEW

EDITOR'S PAGE

Is Seeing The Same As Knowing

The Dilemma of Imaging

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Not everything that can be counted counts, and not everything that counts can be counted.
—Attributed to Albert Einstein (1)

Traditionally, many diseases have been defined by their morphology and what better technique is there to demonstrate morphology in the living individual than imaging? Some diseases such as hypertrophic cardiomyopathy (HCM) started with a morphological sine qua non (2), with firm data about other aspects of their natural history coming much later. Many other morphological signatures of cardiac diseases are following a similar trajectory, especially with the widespread availability of sophisticated imaging. One such condition is left ventricular noncompaction (LV-NC)—a diagnosis primarily based on morphology—and unlike HCM, therein may lay a cautionary tale.

Imaging is a powerful technology, and with great power comes great responsibility and the need to use it wisely. The ability to see more and measure more does not necessarily increase one's ability to diagnose better or label pathology that has a good reason for needing a label. One has to only look at the coevolution of the availability of 2-dimensional (2D) echocardiography and mitral valve prolapse to understand how we can go astray; the advent of M mode and then 2D echocardiography created an epidemic of mitral valve prolapse (MVP) in young women, and the prevalence of MVP reached alarming proportions—many were labeled with pathology, and many symptoms, both serious as well as trivial, were immediately attributed to it (3). The frequency of this diagnosis magically abated once we better understood the saddle shape of the mitral valve and the importance of limiting our diagnosis to MVP found only on imaging in the correct plane. History is possibly repeating itself, and we are perhaps entering a similar phase with the diagnosis of LV-NC!

In this issue of *iJACC*, a group of investigators from Brussels (4) evaluated the prognostic import of the 2 traditional cardiac magnetic resonance criteria for LV-NC (noncompacted [NC] segment/compacted [C] length in the long axis and NC/C mass in the short axis, respectively) in 162 patients with classic dilated cardiomyopathy (DCM) and 48 healthy controls. Of the control subjects, 17% had morphology consistent with hypertrabeculation, whereas 37% and 45% of DCM patients met the NC/C length and NC/C mass criteria, respectively, for LV-NC; 27% fulfilled both criteria. Most importantly, however, LV-NC defined this way did not impact cardiac outcomes. This once again illustrates the dilemma among evangelists in the imaging community, that is, the trouble with defining disease based solely on seeing more. Better imaging of morphology may show too many variations from normal that may not need defining. In fact, even the 2 methods, thought to be fairly accepted parameters for diagnosing LV-NC, significantly differed in their ability to classify patients into high or low degree of LV-NC. Lacking a gold standard, normal trabeculation may blend into hypertrabeculation and then bleed into a noncompacted cardiomyopathy without appropriate milestone markers to identify this transition. What needs a label and what remains a curiosity that does not need identification will thus remain a challenge until we have more studies that establish a core set of diagnostic criteria that can then be married to imaging.

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The other reason to publish this study is our declared commitment (5) to highlight the importance of negative findings, something that has been sorely lacking in the literature, possibly to our detriment in seeking the truth. There has been a deluge of papers about LV-NC, and this morphology was soon described across the spectrum in many different subsets of pathology. Indeed, LV hypertrabeculation has been described in the general population and similar to the current study in DCM, without much detriment. Like any new and controversial condition, reports portending the bad overpopulate the literature early on, more often than those reports that are more measured about it, and a similar phenomenon seems to have occurred with LV-NC. This report, pointing toward the possibly less ominous nature of trabeculations in the failing heart, might serve to temper our enthusiasm to prematurely place a pathology label without letting the froth settle down. Of course, this is not the final word. The investigators excluded subjects with neuromuscular disease, a group thought to have a richer association with LV-NC. This study, therefore, does not exclude the classic LV-NC syndrome with more sinister outcomes, nor does it exclude the fact that our definition of LV-NC based solely on morphology might be deficient. Nevertheless, that tried and trusted parameters for prognosis in DCM such as volumes and ejection fraction predicted outcomes in this reasonable-sized study, whereas the more exotic parameters such as NC/C ratio did not, is also reassuring that imaging morphology is useful; it is just that in our excitement to describe more things on imaging, we should not get carried away with new nomenclature until robust data show the usefulness of our descriptors. Imaging is under scrutiny for excess of use and possible misuse. This would be one way to avoid sowing confusion with a plethora of descriptive traits that are not matched by a unique association with proven disease and with clear prognostic significance. Even if we cannot decide on the prognostic aspects of hypertrabeculation, imaging may yet allow us to question and then hopefully find an answer as to what does it all mean. Hypertrabeculation remains fascinating. What is the genesis of hypertrabeculation in DCM, which was more common than in normal subjects in this study? Is it that a pathology such as DCM affects the compacted segment more than the trabeculated segments, and does the increased ratio of NC/C represent thinning of the compacted segment rather than an increased trabeculation per se. In the extreme, is it even possibly some form of reversion to a fetal morphology phenotype, à la reversion of genetic programming to fetal phenotype in the failing heart. It would be interesting to know what the stimuli are if it is growth of trabeculae rather than thinning of the compacted wall, and why does wall stress not affect both similarly. It is interesting that patients in this study with a lower NC/C mass (<31%) had more systolic hypertension, thicker LV walls, and a larger LV mass index.

Imaging is revealing more and more details, but having an "oil immersion" view is not always useful over conventional imaging. The wisdom in imaging is beyond the image itself—it is in understanding what an image means to the patient and his treatment and in separating the signal from the noise. Hopefully, *iJACC* can continue to be your partner in figuring out this important detail. Please write to us about what you think about LV-NC and if it is a real disease or just a passing fad! Would genetics be the only determinant of truthfully imaged morphology?

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