

EDITORIAL COMMENT

Are Trabeculae and Papillary Muscles an Integral Part of Cardiac Anatomy

Or Annoying Features to Exclude While Tracing Endocardial Boundaries?*

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During the last decade, all cardiac imaging modalities have enjoyed substantial improvements in spatial and temporal resolution, resulting in better visualization of the intricacies of ventricular anatomy. Despite these improvements, visual interpretation of cardiac chamber volumes and function still has well-known shortcomings, irrespective of imaging modality. As a result, referring physicians more frequently expect quantitative evaluations to be conducted. Accurate quantification of chamber volume relies on the delineation of endocardial

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boundaries, which can be achieved by tedious multiplane manual tracing and, more recently, by a variety of time-saving, automated algorithms aimed at bringing this method into routine clinical practice. One of the difficulties with accurate identification of ventricular endocardium is the presence of endocardial trabeculae and papillary muscles (TPM) that give the endocardium its “fuzzy” appearance, which complicates this theoretically simple task.

Because many of the automated techniques frequently fail when image quality is suboptimal, the manual or semimanual analysis remains the method of choice. But because this task is tedious and time-consuming, it is frequently delegated at aca-

demic institutions to trainees and less skilled personnel, under the excuse that “anyone can do this” and that the time of the more qualified readers is too precious to be spent “drawing circles.” However, more often than not, the novice tracers encounter images that either trigger questions or result in gross inaccuracies as judged later by their superiors.

Few would argue that volume quantification would not be simpler in a theoretical, TPM-free ventricle. In fact, in such an ideal world, automated identification of the endocardial boundaries would probably have been achieved long ago. However, in the real world, TPM remains a real issue, irrespective of imaging modality and the level of the reader’s experience. In fact, the variable ability of the cardiac imaging modalities to differentiate the endocardium from the TPM is a major source of intermodality measurement inconsistency. The TPM are particularly easy to identify using cardiac magnetic resonance (CMR) and cardiac computed tomography techniques. In fact, these modalities have demonstrated that TPM are far more extensive than previously appreciated. The improved identification of TPM and lack of knowledge about their normal distribution and extent has had the unexpected consequence of overdiagnosing disorders such as ventricular noncompaction, a condition characterized by increased trabeculation. This condition has recently received a lot of attention in the literature because of its enigmatic nature (1–5).

As many difficult-to-resolve issues are frequently resolved by convention, one has been reached to include TPM in the blood-filled cavity when ventricular volume is measured. Although this convention makes as much sense as the opposite approach would, it has been used in most published studies.

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However, in the last decade, as normal reference values of ventricular volumes and ejection fraction were being established for various populations using different imaging modalities, this issue has been sporadically revisited by investigators who have questioned the validity of the convention and its impact on what is considered to be normal anatomy and function. Nevertheless, few published studies were specifically designed to address this question (6,7).

The interesting study by Chuang et al. (8) in this issue of *JACC* focuses on the relative contribution of the TPM to left ventricular (LV) volume, mass, and ejection fraction as measured from CMR images. The authors studied a large cohort of human subjects over a wide range of ages, using automated software, which allowed them to measure TPM volume. This method is in contrast to techniques previously used to assess the extent of trabeculation by measuring the thickness of the trabecular layer in different views (5,7). The authors indexed TPM volume according to end-diastolic LV volume and by LV mass. Although both these parameters were found to decrease with age, interestingly, only TPM volume normalized by LV mass inversely correlated with body mass index (BMI), probably because of the relationship between LV mass and BMI. These findings are consistent with a recent study that focused on the normal relation between LV trabeculation and demographic characteristics, and showed that with age, the trabecular layer thinned and the compact myocardium thickened (5). Another recent study found that the thickness of the trabecular layer is directly related to LV size (7).

Importantly, by isolating a large group of normal controls from the study cohort allowed Chuang et al. (8) to establish age-, sex-, and body mass-related normal values of the variables they used to quantify TPM. These data are of particular importance in the context of LV noncompaction cardiomyopathy. The diagnosis of this condition largely relies on the extent of ventricular trabeculation and has been difficult because of: 1) unavailability of user-friendly measurement tools; 2) lack of normal values; and 3) use of 1-dimensional assessment of the degree of trabeculation (7). This study is important because it simultaneously tackled all 3 of these problems, as the authors developed and tested an automated technique for the quantification of TPM volume and reported age-, sex-, and body mass-related normal values. Another potential clinical use of this tool is to help identify patients with cardiomyopathy who are at highest risk of stroke, as, theoreti-

cally, thrombi could form within the trabecular layer and lead to stroke. Conceivably, future studies using this new tool should be aimed at determining TPM volume thresholds for optimized stroke prevention therapy by balancing its benefits against its adverse effects, as reflected by patient outcomes.

It is worthwhile mentioning, without diminishing the importance of this study (8), that values reported here are specific to CMR. In other words, the same parameters, if measured using other imaging modalities, such as multidetector computed tomography and echocardiography (either 2- or 3-dimensional), would likely yield slightly different values. It is true that CMR imaging is currently considered as the standard reference technique for the evaluation of LV volume and mass, and is used as such in the majority of recent validation studies. This is mostly due to the increasing availability of this modality, the high spatial resolution, and the tissue contrast of the steady-state free-precession images, allowing in most patients clear differentiation between myocardial tissue, blood, and TPM.

Moreover, normal values reported by Chuang et al. (8) are specific to the analysis software used in this study, as different algorithms may well result in slightly different values, depending on where the endocardial boundary is placed in each segment of each slice. Furthermore, even the same software may define the endocardial boundary differently in various segments, in view of the large intersegmental variability in the degree of trabeculation (e.g., increased trabeculation near the apex) (5,7). Nevertheless, because there is no gold standard reference for absolute truth for endocardial boundary position, having normal values according to 1 automated technique is infinitely better than having none.

The study by Chuang et al. (8) reports that TPM accounted for as much as 23% of end-diastolic LV volume and 28% of the LV mass. These are certainly not negligible fractions. Clearly, errors in the delineation of these anatomic structures have the potential to significantly bias the measurements of LV volume and mass, depending on how well defined the interface is between TPM and blood as well as how easily TPM can be differentiated from the myocardial tissue. A study by our group reported that the trabeculae alone accounted for approximately 13% of the LV end-diastolic volume (9), which, assuming that the papillary muscles account for the other 10%, would indicate agreement between the 2 studies.

Furthermore, for any imaging modality, irrespective of its spatial resolution, chamber quantification at end-systole, when endocardial trabeculae are compressed together by the surrounding contracted myocardium, is considerably more challenging compared with end-diastole, when the intertrabecular spaces are filled with blood and thus allow easier differentiation between the trabeculae and the myocardium. Thus, the timing within the cardiac cycle at which TPM volume is measured is important as well. One might suggest that for the latter reason, TPM volume should be measured at end-diastole as described by Chuang et al. (8) However, at this phase of the cardiac cycle, blood in the intertrabecular spaces is likely to account for a significant proportion of the measured TPM volume. Accordingly, there is no ideal phase for this measurement, as each phase is associated with its own sources of error.

Although conventions may change depending on findings of future investigations targeting specific issues, the question of whether TPM are an important part of cardiac anatomy or simply annoying

features to exclude while tracing endocardial boundaries will probably remain with us as long as we believe that measurements of ventricular volume provide clinically useful information. This is unless a solution is found for an error-proof, perfectly accurate, fully automated technique for endocardial border detection. The likelihood of such a miracle solution will increase as image quality improves with future technological developments. However, in our imperfect world, it is more likely that we will continue making judgment calls in the foreseeable future regarding the position of the endocardial boundary, which promise to keep intermeasurement variability alive and well. In this regard, additional knowledge gleaned from studies such as that of Chuang et al. (8) is certainly a step in the right direction.

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