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JUNE 2018:923–32

The 2 approaches can yield significantly different results.

The aim of this report is to provide consensus for segmenting the left ventricle based on CMR images with close alignment to the original report.

For the definition of segments within a cardiac level, the consensus panel suggests the use of both RV insertion points to define 2 major axes. For the basal and mid-cavity level, the septal and the lateral area are then further divided using an equiangular line generating 6 segments (**Figure 1Ai**). Although the resulting segments are not equiangular and thus represent different amounts of myocardium, the other alternatives would result in a misalignment of segments either at the anterior or at the inferior RV insertion point not consistent with clinical practice. Of note, the amount of myocardium for the apical, mid-, and basal slice is also different with the original suggestion due to the different mass of each slice.

Segment 17 is defined as the apex of the heart from the tip of the epicardium to the endocardium. Wall motion can be described as thickening of this segment, but no blood volume or endocardial border is assigned to this segment (**Figure 1Ci**).

For definition of the cardiac levels (base, mid-cavity, apex), the remainder of the LV volume is divided into 3 levels with identical thickness. As such, each level describes one-third of the remaining myocardium. These levels are adapted to the cardiac cycle (i.e., different at end-diastole and end-systole) following approximately the long axis motion of the myocardium (**Figure 1Di**).

Any short axis acquired should be assigned to the respective level. It should be stated at which time-point in the cardiac cycle the assignment was performed. Assignment of short-axis slices may change over the cardiac cycle accounting for longitudinal shortening (**Figure 1D**).

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https://doi.org/10.1016/j.jcmg.2017.09.010

Please note: Dr. Bluemke is a consultant for Siemens. Dr. Ferrari is on the editorial boards for the *Journal of Cardiovascular Magnetic Resonance* and *ACCEL*. Dr. Friedrich is an officer and advisor for Circle Cardiovascular Imaging. Dr. Kramer is a consultant for Abbott and Biotelemetry. Dr. Young has received consulting fees from Siemens. Dr. Kim is the cofounder of HeartIT, LLC; and has received a grant from Siemens Health Care. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose. Zahi A. Fayad, PhD, served as the Guest Editor for this paper.

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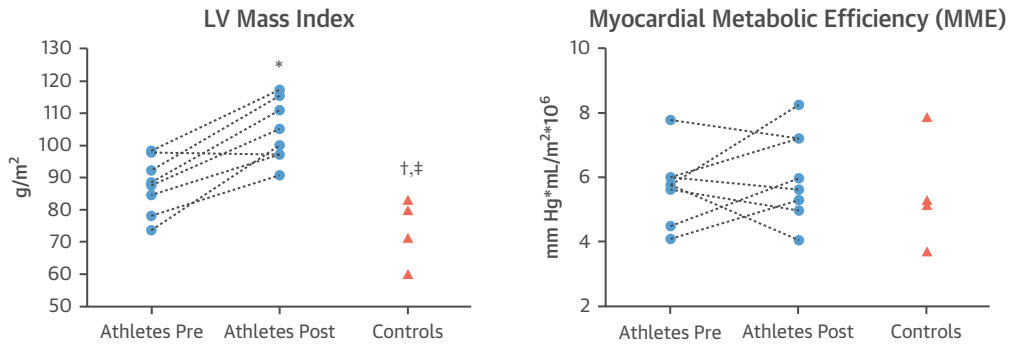
Myocardial Metabolism in Endurance Exercise-Induced Left Ventricular Hypertrophy

Pathologic forms of left ventricular hypertrophy (LVH) are associated with impaired myocardial metabolic efficiency (MME), the ratio of myocardial work to oxygen consumption (MVO_2), which may serve as a key mechanistic link between LVH and the development of heart failure syndromes (1,2). At present, myocardial metabolism in adaptive forms of left ventricular (LV) remodeling, specifically exercise-induced (EI) LVH, remains incompletely understood. We therefore conducted a repeated measures, longitudinal pilot study to determine the impact of EI-LVH on myocardial metabolism and blood flow (MBF).

Male collegiate rowers (n = 8) were studied at baseline (college matriculation) and after a 3-month period of intensive endurance exercise training. Healthy, normally active (<3 h exercise/week) male controls (n = 4) were identically studied. Cardiac positron-emission tomography (PET) (Discovery Rx VCT, GE Healthcare, Milwaukee, Wisconsin) with ^{11}C-acetate tracer (20 mCi) and serial imaging over 30 min were used to generate time activity curves (FlowQuant, TriFoil Imaging, Chatsworth, California)

FIGURE 1 Left Ventricular Mass and Myocardial Metabolic Efficiency in Athletes Before and After Exercise Training and in Controls



*p < 0.05 athletes pre-training versus post-training. †p < 0.05 athletes pre-training versus controls. ‡p < 0.05 athletes post-training versus controls. LV = left ventricular.

to measure MBF, using a 1-compartment kinetic model (3), and LV MVO_2, through derivation of k_{mono} (4). Myocardial oxygen extraction was calculated as: k_{mono}/MBF. The work-metabolic index was used to evaluate MME defined as: (HR × indexed SV × SBP)/k_{mono}, where HR is heart rate, SV is stroke volume, and SBP is systolic blood pressure. Transthoracic echocardiography (Vivid-I, GE Healthcare) was used to measure cardiac structure and function, with LV mass calculated by the area-length method.

At baseline, LV mass and left ventricular end-diastolic volume (LVEDV) were smaller in controls than athletes (LV mass 74 ± 11 g/m² vs. 88 ± 9 g/m², p = 0.04; LVEDV 72 ± 8 ml/m² vs. 89 ± 7 ml/m²; p =0.002), and in controls, who maintained baseline exercise levels during the study (2.5 ± 0.5 h/week), remained stable. In contrast, athletes' increased exercise training (pre-study = 6.1 ± 3.0 h/week vs. intra-study = 13.0 ± 0.9 h/week; p < 0.0001) and experienced balanced increases in LV mass (87 ± 9 g/m² vs. 104 ± 10 g/m²; p = 0.001 (**Figure 1**) and LVEDV (89 ± 6 ml/m² vs. 101 ± 9 ml/m²; p = 0.009) were consistent with eccentric EI-LVH. Although athletes' resting stroke volume increased (48 ± 4 ml/m² to 54 ± 6 ml/m²; p = 0.01), cardiac work was unchanged due to concomitant nonsignificant reductions in HR and SBP. k_{mono} and MBF were lower at baseline in athletes than controls (k_{mono} 0.0486 ± 0.004 min^{-1} vs. 0.0583 ± 0.007 min^{-1}, p = 0.02; MBF 0.53 ± 0.05 ml/min/g vs. 0.80 ± 0.05 ml/min/g, p = 0.0001), and did not change with exercise training. Despite the development of EI-LVH, MME was preserved and remained similar to controls (**Figure 1**). In addition, both at baseline and follow-up, athletes showed higher myocardial oxygen extraction than controls (0.093 ± 0.009 ml/g vs. 0.073 ± 0.006 ml/g; p = 0.003), an index that was significantly related to lower HR (r^2 = 0.75; p = 0.0001).

The primary finding from this study is that the development of EI-LVH is associated with preserved MME. This attribute differentiates this common form of adaptive cardiac remodeling caused by the intermittent volume and pressure loading of exercise training from pathologic forms of LVH due to genetic causes (2) or chronic pressure/volume overload (1) in which MME is impaired and likely causal in the development of heart failure. This finding advances our understanding of adaptive hypertrophy and suggests a novel clinical role for cardiac PET. Specifically, and pending larger confirmatory studies, cardiac PET-derived MME appears capable of distinguishing between physiologic and pathologic LVH. This technique may therefore prove valuable in the care of athletes who have EI-LVH that overlaps phenotypically with pathologic forms of LVH that carry a risk of sudden death during exercise. In addition, we found that athletes had lower MBF but higher oxygen extraction compared to controls at rest, a pairing that likely represents an acquired increase in substrate delivery and utilization reserve capacities. This difference was present at baseline, before the development of EI-LVH, thereby suggesting that enhancements in myocardial oxygen extraction may occur following lower levels of exercise training than those required for the development of EI-LVH and may be in part driven by acquired bradycardia and resultant longer myocardial capillary transit time. In sum, these findings advance our scientific understanding of adaptive LVH and suggest that

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JUNE 2018:923–32

cardiac PET-derived indices of myocardial metabolism may prove useful for distinguishing physiologic from pathologic LVH in "gray-zone" cases that are commonly encountered in the clinical care of athletes.

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https://doi.org/10.1016/j.jcmg.2017.09.012

Please note: Dr. Wasfy was supported by the American College of Cardiology Foundation/Merck Award. Dr. Di Carli has received consulting fees from General Electric; and has received a grant from Spectrum Dynamics. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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Radiation Exposure of Downstream Testing

The role of testing for suspected coronary disease remains a cornerstone of clinical decision making. As clinicians, we are fortunate to have a variety of tests that provide accurate diagnoses and prognoses. Although there are well-established methods, such as functional treadmill testing, imaging modalities can augment standard test results or supplant them altogether. Costs of imaging are not only monetary but also include exposure to radiation. In the December 2017 issue of *iJACC*, the report by Lubbers et al. (1) evaluated the effectiveness and efficiency of a tiered approach to the patient with suspected coronary disease. Their study compares algorithms based on conventional functional testing with those which incorporate coronary calcium score, coronary computed tomography angiogram (CTA), and computed tomography–myocardial perfusion imaging (CT-MPI). Based on their findings, the authors concluded that a tiered cardiac CT protocol offers a fast and efficient alternative to functional testing. We commend Lubbers et al. (1) for their direct and well-constructed study to highlight the often difficult question regarding testing modalities in patients with chest pain. However, considering downstream testing, questions are raised regarding radiation exposure in the functional testing group. The median radiation exposure was significantly higher for the tiered CT group than for the functional group (respectively, 3.1 mSv [interquartile range: 1.6 to 7.8 mSv] vs. 0 mSv [interquartile range: 0.0 to 7.1 mSv]; $p < 0.001$). As noted in the report, 51 patients in the functional testing group underwent a total of 62 imaging procedures that utilized ionizing radiation. These procedures consisted of 11 CTA, 31 single-photon emission computed tomography (SPECT), and 20 invasive angiograms. Based on standard radiation estimates (1.3 mSv per calcium scan, 3.5 mSv per CTA, 10.6 mSv per CT-MPI) (1); 8.7 mSv per SPECT study (2); and 5.6 mSv per invasive angiogram study (3), the mean downstream radiation exposure for those 51 patients was more than 8.6 mSv. Therefore, 37% of the functional testing group received a significant amount of radiation during their evaluations. Information regarding total radiation exposure from presentation to final diagnosis are valuable data, and although it is statistically accurate to present the median and interquartile range, it does not fully reflect the extent of radiation exposure incurred through testing. This information is especially relevant due to recent technological advances in cardiac CT (4), as well as the emergence of cardiac positron-emission tomography-MPI (5), which have resulted in significant reductions in radiation exposure while maintaining a high diagnostic yield. Thus, the exposure to all patients, and most notably those who undergo downstream testing, can be substantially reduced by applying modern low-radiation imaging modalities. In conclusion, even a functional testing algorithm will inevitably incur downstream testing due to rates of false positives and equivocal studies.