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Short Communication

Exercise-induced left ventricular trabeculation: what's the evidence?

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Cross-sectional studies [1] and small case series [2] have suggested that increased left ventricular (LV) trabeculation may be a manifestation of benign athletic remodeling in predisposed individuals. Interestingly, there is some support that increased LV trabeculation may improve cardiac performance through increases in stroke volume, stroke work and cardiac index (preprint) [3]. The practical implications are that athletes are at risk of over diagnosis of left ventricular non-compaction cardiomyopathy (LVNC). Whilst it has been considered in international consensus criteria for sports participation, that isolated and incidentally discovered LV should not be diagnosed as LVNC in the absence of symptoms, family history, ECG abnormalities or LV impairment [4], there is no gold standard criteria for the diagnosis of LVNC resulting in profound difficulty in case definition and subsequent variation in practice.

The diagnosis of LVNC is often suggested by cardiac imaging for which a cacophony of different findings, measurements and ratios have been proposed to distinguish between normality and LVNC. None of these methods have demonstrated convincing success or superiority in relation to each other [5]. Selections and combinations of these "LVNC imaging criteria" have been used in research to highlight that some groups, including those with the potential for cardiac remodeling based on hemodynamic differences, possess a greater amount of increased LV trabeculation compared to healthy control populations. These groups include those with heart failure [6], sickle cell anemia [7], pregnant women [8] and athletes [1]. The scale of the problem in athletes varies between 1.4% [9] to 18.3% [1] depending on how conservatively or liberally increased LV trabeculation is defined, with general agreement that the proportion of athletes in whom there is real concern of LVNC, based on additional clinical features, is in the order of 0.1% [9] to 0.9% [1].

The findings of cross-sectional studies require cautious interpretation considering the potential influences of confounding factors, selection bias and inability to discern that increased LV trabeculation follows the exposure of high exercise volume when looking at a single time-point. For this reason, we conducted a study in novice marathon runners investigating whether a self-directed increase in exercise, through running training, could result in an increase in LV trabeculation and have reported the findings previously [10].

We recruited 120 first-time marathon runners aged 18-35 years, based on our understanding that older runners would have less myocardial plasticity for remodeling [11]. The final cohort comprised only 68 subjects due to attrition from injury and missed follow up appointments. Multiple measures of LV trabeculation using echocardiography and cardiac magnetic resonance (CMR) were employed, finding no clinically important change in LV trabeculation after 17 weeks of training and completing a marathon. In addition, measures of LV trabeculation had poor agreement with each other, with echocardiographic Chin criteria [12] demonstrating a small increase in LV trabeculation and CMR-based Petersen criteria [13] demonstrating a small drop. Though both of these small changes were statistically significant, they resulted from sub-millimeter differences in layer thicknesses, which are impossible to measure on an individual level and therefore biologically unimportant. Other measures of LV trabeculation did not change, though the prevalence of apical trabeculation, particularly as determined by CMR fractal dimension measurement [14]

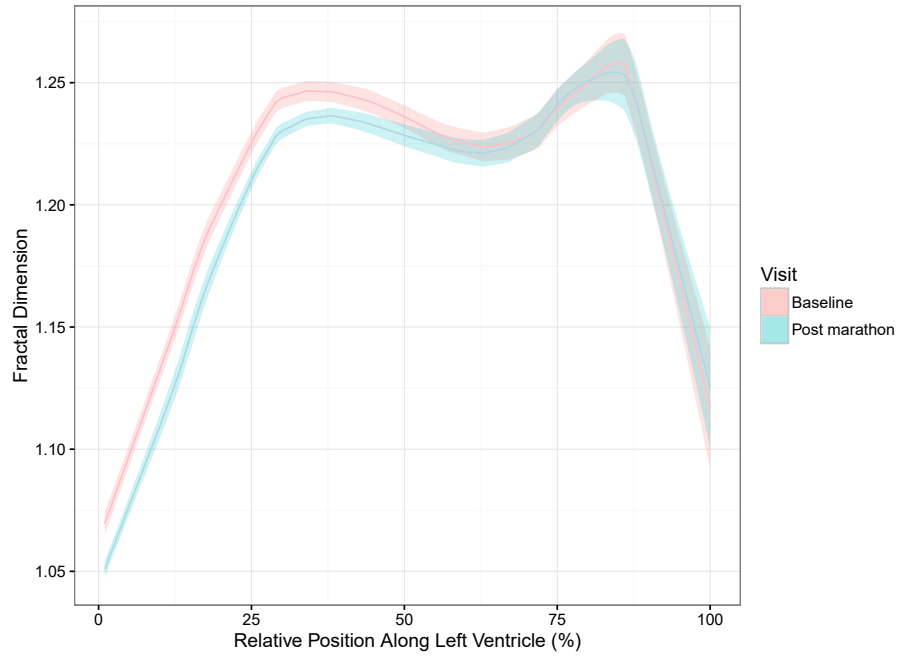

Figure 1: Mean fractal dimension across the entire left ventricle from base (0%) to apex (100%). Baseline (pink) and post marathon (blue) results are displayed together with the respective shaded regions representing 95% confidence intervals of mean values.

was unexpectedly high in this study cohort at baseline. The mean fractal dimension also indicated that endocardial border complexity at the basal LV reduced after the marathon, compared to baseline measurements but beyond the mid-ventricular level, moving towards the apex, fractal dimensions did not change (Figure 1). Speculatively, this may be the result of other structural remodeling changes such as a 3% increase in LV end-diastolic volume and a 4% increase in compact myocardial LV mass, previously reported in this cohort [15].

Though we were unable to demonstrate a meaningful change in LV trabeculation in novice marathon runners, the situation in competitive athletes with substantially higher exercise volume and longer duration of participation at higher intensity, could be drastically different. As we have demonstrated previously, running a first marathon achieving a near average finishing time is not associated with an increase in peak oxygen consumption or cardiac remodeling changes akin to those seen in athletes. Unsurprisingly, a 17-week, unsupervised beginner's training plan is a relatively weak stimulus for cardiac remodeling when considered on the spectrum of exercise-induced remodeling. Consequently, to better address the question of whether athletes develop exercise-induced LV trabeculation, one could study whether adolescent athletes joining academies develop excessive LV trabeculation over years of professional sport participation. The advantages of studying such a group would be that weekly training times would be expected to increase to elite athlete levels and professional athletes may be reassessed on an annual/biennial basis if participating in a regular cardiovascular screening program. Pre-participation screening and surveillance of adolescent athletes for ongoing eligibility has reached its 20th year in the English Football Association cardiac screening program [16] and echocardiograms could be examined retrospectively for LV trabeculation. Examining, with accuracy, whether LV trabeculation increases over time in an athlete's career and whether LV trabeculation regresses with detraining from injury or retirement, could yield important discoveries in exercise-induced LV trabeculation and confirm or refute its existence. By including an age-matched control group, natural changes in LV trabeculation over decades can be compared, as studies demonstrate that LV trabeculation decreases with normal ageing [17,18].

Our study also highlighted a greater reproducibility of trabeculation quantification through measurements conducted by CMR as compared to echocardiography. A key obstacle to accurately quantifying LV trabeculation and detecting small changes over multiple assessment time points is the subjectivity of manual measurements and oversimplification of a 3-dimensional global LV property into a regionally representative ratio of linear measurements. Hence, reliable automated techniques quantifying global and regional trabeculation across the whole LV would represent an important and much needed breakthrough in this area. Such technology could be applied to a large Biobank of healthy individuals to better understand the limits of normal LV trabeculation and address the problem that current proposed thresholds are oversensitive, capturing far more than simply the LVNC population of interest.

In conclusion, it remains unclear whether LV trabeculation can be exercise-induced and is potentially a manifestation of the normal athletic heart. We were unable to demonstrate an increase in LV trabeculation in a cohort of young, unsupervised, novice marathon runners but such a modest change in physical activity for a relatively short period of time may have been an insufficient exercise stimulus. The current evidence from cross-sectional studies and small case series is not conclusive and future studies should employ

highly reproducible, automated techniques of LV trabeculation quantification in longitudinal studies spanning the adolescent to adult competitive athlete career to better address this question. Until such time, caution should be applied before diagnosing LVNC in an athlete on the basis of trabeculation appearances alone.

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