Left Ventricular Remodeling in Elite Athletes

To the Editor:

Pelliccia et al should be congratulated for their unique database and their recent paper on ventricular remodeling after cessation of competitive sports in elite athletes. Their conclusion that ventricular cavity size and wall thickness decreased in all athletes after detraining is consistent with reports in previous longitudinal studies. However, their observation that ventricular size does not completely normalize should be further qualified by data that were not clearly reported in the article.

The spread of 1 to 13 years for the interval between cessation of exercise and the reported follow-up measurement may be the greatest confounder of these results. Given that short-term training followed by cessation is associated with normalization of ventricular dimensions, it is reasonable to expect that long-term cumulative changes may regress more slowly. Could it be that the athletes closest to detraining were the ones with persistent cavity enlargement? Furthermore, the duration of performance at elite levels prior to commencement of detraining cannot be gleaned from the report. Athletes with several years of training could be expected to have a bigger left ventricular cavity than those with less training, and therefore require a longer detraining observation period before cavity size normalizes. Was the observed incomplete normalization of volume seen predominantly in those with longer duration of elite performance?

Another confounder is the persistence of light recreational activities in the majority (62.5%) of the former athletes. Hickson et al showed that continued training at a reduced level could prevent regression of the training-induced morphometric changes in former athletes. Although this study reports no difference in the mean left ventricular cavity size between completely deconditioned athletes and those with persistent exercise, a difference cannot be excluded unless the dimensions are indexed to both the dimensions during peak performance and the relative reduction in peak performance represented by detraining activity. This unique database should clarify these important issues.

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Response

Dr Kofo Ogungyankin raises thoughtful questions concerning the incomplete regression of enlarged left ventricular (LV) cavity size in elite athletes after cessation of training and competition, on which we have recently reported.

One of the more intriguing possibilities raised by Dr Ogungyankin is that athletes with persistent LV cavity enlargement after long-term detraining may be those studied closest to the beginning of deconditioning, ie, athletes observed after the shortest period of inactivity following cessation of their competitive career. We have reanalyzed our database searching for statistical evidence of this hypothesis, but we found no significant relationship between the absolute or relative (expressed as percentage of the initial value) reduction in LV cavity dimension and the duration of deconditioning. In addition, we examined another hypothesis raised by Dr Ogungyankin that the period over which athletes trained and competed at a very high (elite) level influenced the extent of reduction in LV cavity size. Here again we found no significant relationship between the period over which the athletes had been competing at national or international level and the extent of LV cavity reduction. Therefore, we found no support for the hypothesis that a higher level of training and longer athletic careers require more prolonged deconditioning periods to reduce LV cavity dimension to within the normal range.

The persistence of light recreational activities in former elite athletes is a possible partial explanation for the incomplete reduction of LV cavity size we observed, as pointed out by Dr Ogungyankin. Our data confirm that residual physical activity (and increased body weight) during the deconditioning period were the only determinants significantly associated with change in LV cavity dimension ($r=0.351$ for residual physical activity and 0.472 for increased body weight). This finding also supports the previous observation of Hickson et al that continued exercise training (although at low intensity) could prevent regression of cardiac enlargement in former highly trained athletes.

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