LETTERS TO THE EDITOR

Letters to the Editor will be published when suitable and as space permits. They should not exceed 1,000 words (typed double spaced) in length, and may be subject to editing or abridgment.

Exercise and CAD

To the Editor:

I read with interest the article by Ehsani et al.1 describing the effects of 12 months of primarily aerobic exercise in patients with coronary artery disease. The findings of an elevated double-product threshold for ischemic ST-segment depression, decreased ST-segment depression at the same double product, and reduced or unchanged maximum ST-segment depression despite a “large increase in maximum double product” were not unexpected. I take exception to the author’s explanation that the latter two findings may be due to “exercise-induced improvement in myocardial oxygenation.” The discussion discounted volume and contractility changes that may have decreased myocardial oxygen demands after training. However, factors other than volume, contractility and double product were involved in the investigation that the author did not consider.

Indirect indexes of MVO₂ do not incorporate all of the factors known to influence myocardial oxygen demand. MVO₂ is due principally to left ventricular tension development, the contractile state of the myocardium and heart rate. Ventricular wall tension is dependent upon systolic pressure development, ventricular chamber size and ventricular wall thickness.2,3 Myocardial tension, and thus oxygen demand, would be decreased with smaller ventricular volume or with a thicker ventricle (increased ventricular mass). The data reveal that the ratio of left ventricular end-diastolic dimension (LVEDD) to left ventricular posterior wall thickness (LVPWT) decreased, from 5.96 to 5.63, as did the ratio of estimated left ventricular end-diastolic volume index to left ventricular mass index, from 0.709 to 0.614, after training. This structural change in left ventricular volume and thickness appears likely to have decreased ventricular wall tension. Since geometric change in the left ventricle influences MVO₂,4 the favorable volume/mass relationship after training probably permitted an increased double product after training without much ST-segment alteration. By decreasing wall tension, a higher posttraining heart rate and blood pressure were permitted. The authors seem to have overlooked this likely explanation.

Local changes in the trained muscles are important for reduction in myocardial pressure-work caused by physical conditioning.5 Neither augmented myocardial oxygen delivery6 nor development of collateral circulation7 has been demonstrated after aerobic exercise training. It is possible, but unlikely, that improved oxygenation of the myocardium occurred in this study after training. Myocardial oxygen demand, however, has been shown to decrease after appropriate training.5 In the past, most of the improved endurance capabilities were thought to be secondary to only skeletal muscle adaptations post training.5 8 9 I believe the importance of this investigation is that not only double product will decrease at the same work load after training, but also that a higher double product can be in part attained by a beneficial alteration of the left ventricular volume/mass relationship.

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References


The author replies:

To the Editor:

Dr. Goldberg asserts that the increase in left ventricular wall thickness in our patients should have resulted in a decrease in left ventricular wall tension and, thus, myocardial O₂ consumption (MVO₂) and that this was the mechanism responsible for the improvement in ECG criteria of myocardial ischemia after training. The increase in posterior wall thickness in our patients was proportional to the increase in end-diastolic diameter since the left ventricular wall thickness to radius (h/r) ratio did not change after training (0.36 ± 0.06 vs 0.37 ± 0.05). Obviously, the changes in left ventricular end-diastolic dimension to wall thickness ratio were also not significant (5.8 ± 1.2 vs 5.5 ± 1). These findings are compatible with the pattern of compensated chronic volume overload hypertrophy typically associated with increased wall tension. 1 Dr. Goldberg disregards the changes in the other determinants of left ventricular wall tension which augment wall stress. Quantitative assessment of left ventricular wall tension during maximal exercise in man, with the use of the currently available noninvasive techniques, is extremely difficult since it would require simultaneous measurements of pressure (P), radius (r) and wall thickness (h). However, if one assumes that left ventricular wall thickness and radius at maximal exercise are similar to values observed at rest, as Dr. Goldberg seems to imply, the estimated values for left ventricular wall tension (σ = Pr/2h) would actually be higher after training (302 ± 62 vs 333 ± 35 g/cm²; p < 0.05). In addition, an increase in muscle mass would be expected to raise total left ventricular oxygen consumption. Therefore, it is highly unlikely that regression of ischemic ST-segment changes was mediated primarily by a lower MVO₂ after training, particularly in view of recent observations indicating that left ventricular contractile function, which increases MVO₂, improves after prolonged training.2

The thickness to radius (h/r) ratio at rest may be quite different from that at maximal exercise since acute changes in systolic blood pressure can affect it profoundly.3 Therefore, it is unjustified to attribute the higher maximal exercise double product attained after training to the changes in h/r ratio. Although there is a relationship between h/r ratio and systolic blood pressure at rest in different forms of cardiac hypertrophy,4 no relationship between this ratio at rest and rate-pressure product at maximal exercise has been established.

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References

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