Combined Cardiopulmonary Exercise Testing and 99mTC Sestamibi Myocardial Imaging in Trained vs Untrained Patients with Coronary Artery Disease

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Combined Cardiopulmonary Exercise Testing and $^{99m}$TC Sestamibi Myocardial Imaging in Trained vs Untrained Patients with Coronary Artery Disease

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**Background:** The benefit of exercise training in patients (pts) with coronary artery disease (CAD) is widely conceptual while mentioning several improving systems. Methods: Cardiopulmonary exercise test (CPET) parameters and simultaneous evaluation of myocardial ischemia were performed in 15 trained and 20 untrained pts with CAD and 9 control subjects without coronary disease. The degree of ischemia was evaluated in all 44 subjects, using a $^{99m}$Tc sestamibi myocardial perfusion imaging carried out simultaneously with the CPET.

**Results:** Peak oxygen consumption (peak-V.O2) was significantly higher ($p < 0.001$) and the ischemia score was significantly lower ($p < 0.001$) in the trained pts (1989 ± 422 ml/min and 0.8 ± 0.65, respectively) than in untrained pts (1608 ± 296 ml/min and 1.79 ± 0.95, respectively). The peak oxygen pulse (peak-O2-P) was significantly higher (139 ± 29 % vs 94 ± 11 % of predicted values; $p < 0.001$) and the decrease in peak heart rate (peak-HR) was also more marked in the trained group (70 ± 11 % vs 84 ± 9 % of predicted values; $p < 0.001$).

**Conclusion:** The improvement in ischemia score in the trained pts correlates with increased stroke volume (related to the O2-P) together with the concomitant reduction in peak-HR, while allowing even higher cardiac output in these pts. Such an effect is metabolically energy-efficient for the ischemic myocardium and it is sufficient to explain the improvement in cardiopulmonary function in CAD.

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**Key words:** cardiopulmonary exercise testing, sestamibi myocardial imaging, training effect, coronary artery disease
The study protocol was approved by the local ethics committee, and written informed consent was obtained from each participant in the study.

### Cardiopulmonary Exercise Test

An upright symptom-limited test was performed on an electronically braked cycle ergometer (Ergoline 800). Exercise was initiated after a 3-minute rest and 2 minutes of free pedaling at a rate of 60 rpm. The effort was then progressively increased by 10–20 Watt/min until the predefined end-point was reached, namely, symptoms, volitional fatigue, or attainment of the target HR. Cardiopulmonary data were collected by an online metabolic unit (CPX MedGraphics, Minnesota, USA).

Pts breathed through a low-resistance, two-way valve (Hans Rudolph, Missouri, USA) connected to the expiratory limb. The breath-by-breath signals were integrated by a computer to yield 30-sec and averages of HR, minute ventilation, VO₂, VCO₂, and O₂-P (VO₂/HR). VAT was defined as the point at which the ventilatory equivalent of O₂ (VE/VO₂) increased in the absence of a similar increase in the ventilatory equivalent of CO₂ (VE/VCO₂), as described by Beaver et al [14]. Blood pressure was measured at rest, every 2 minutes, and at peak exercise.

### MIBI-SPECT Protocol

MIBI myocardial perfusion tomography was performed with a same-day “rest-stress” imaging protocol [15]. The first myocardial perfusion imaging was done at rest, 1 hour after injection of MIBI 7–8 mc. The second imaging was done 1–2 hours later, after exercise and additional injection of MIBI 21–22 mc at peak exercise. Tomographic imaging acquisition was performed over a 180-degree arc, from the –45-degree right anterior oblique to the +135-degree left posterior oblique with an S-P-4× digital camera (Elscint Ltd, Haifa, Israel) fitted with an all-purpose collimator. The MIBI images were interpreted by consensus of two experienced observers blinded to the clinical data and the other diagnostic studies. The degree of ischemia was scored on a 4-point scale: 0 – none; 1 – mild; 2 – moderate; 3 – severe but reversible. Stress image defects that showed partial or complete resolution on corresponding rest images were considered “reversible ischemia”.

### Statistical Analysis

Mean ± standard deviation was calculated for each parameter. The paired Student’s t-test was used for intergroup comparison. P values < 0.05 were considered statistically significant.

### Results

The basic data and summary of results for all 3 groups are shown in Table 1, and the extent of disease in the trained and untrained pts is shown in Table 2. There was an almost equal distribution of 1-, 2- and 3-vessel diseases within groups II and III with no significant difference in extent of disease between the groups (Table 2). Significantly less ischemia was observed in the trained pts (group III) in comparison to the untrained pts is shown in Table 2. There was an almost equal distribution of 1-, 2- and 3-vessel diseases within groups II and III with no significant difference in extent of disease between the groups (Table 2). Significantly less ischemia was observed in the trained pts (group III) in comparison to the untrained pts (group II) (0.8 ± 0.65 vs 1.79 ± 0.95; p < 0.001), as demonstrated also in Figure 1, along with a significantly greater peak-VO₂ (1989 ± 422 vs 1608 ± 296 ml/min; p < 0.001). Since peak-VO₂ equals peak-O₂-P x HR, as mentioned above, a highly significant increase in peak-O₂-P was observed in the trained vs the untrained group (139 ± 29 % vs 94 ± 11 % of predicted values; p < 0.001) concomitantly with a significantly lower peak-HR (70 ± 11 % vs 84 ± 9 % of predicted values; p < 0.001).

Peak-VO₂ and O₂-P were not significantly different in the trained group (group III) vs the control group (group I). The comparison of the relationship among the peak-VO₂, peak- O₂-P and peak-HR within the 3 groups is demonstrated in Figure 2.

### Discussion

Exercise is a highly valuable non-pharmacological treatment for pts with chronic stable angina. Exercise training reduces myocardial ischemia, and, on the clinical level, reduces the frequency of angina attacks while improving functional capacity and long-term outcomes [16]. As regular exercise has been shown to improve myocardial perfusion and to retard disease progression in pts with stable CAD and even with ischemic cardiomyopathy [17], Hambrecht et al [18] conducted a randomized study to compare the effects of exercise

![Figure 1. Comparison of number of occluded coronary vessels and scoring of the ischemic degree (as evaluated by Tc-99m-Sestamibi test) between groups II (untrained) and III (trained).](image-url)
cardiopulmonary data suggests that the beneficial effect of the trained group at the same degree of exercise than in the [19]. Simultaneously, peak-HR is also significantly lower in trained group than in the untrained one, indicating an improvement of myocardium that accrues from exercise training. Completely, to the improved metabolic-physiologic state of the subjects without CAD (group I = control). However, the contribution of this study is in showing that the reduced ischemic burden seen on perfusion studies is compatible with either of these possibilities.)

The present study does not deny the former possibility (increased myocardial perfusion) since it does not address this issue at all. However, the contribution of this study is in showing that the reduced ischemic burden seen on perfusion studies can be attributed to a substantial degree, if not completely, to the improved metabolic-physiologic state of the myocardium that accrues from exercise training. We indeed show in the present study that SV (as indicated by O2-P) is significantly higher in CAD patients after a 3–6-month exercise program prescribed according to HR at the VAT (group III) than in group-II patients, who were similar to them in severity of coronary artery disease but were not subjected to exercise training, and it is also similar to the SV of the subjects without CAD (group I = control).

Furthermore, peak-VO2 is also significantly higher in the trained group than in the untrained one, indicating an improvement in functional capacity as described by Arena et al [19]. Simultaneously, peak-HR is also significantly lower in the trained group at the same degree of exercise than in the untrained group (as demonstrated in Figure 2). This peak-HR decrease indicates that exercise training decreases myocardial oxygen demand, and the combination of this with the cardiopulmonary data suggests that the beneficial effect of exercise training is mediated, at least in a substantial way, by a significant decrease in peak-HR along with the significant increase in peak-O2-P (related to SV) and peak-VO2 (functional capacity). This metabolic-physiologic advantage to the myocardium is expressed by the significant improvement of ischemia that was demonstrated by MIBI-SPECT imaging at the peak-HR that was simultaneously reached during the same exercise testing while performing the CPET. Thus, it is not at all necessary to invoke an improvement in myocardial blood flow to explain the beneficial effect of exercise training in chronic CAD.

The results of the present study will have to be further validated by means of a larger study. It will also be desirable to compare, in future studies, maximal heart rate, peak VO2 and peak O2 pulse (as an index of stroke volume) at the same degree of exercise intensity before and after a training program in the same patients.

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