Effect of Training on Myocardial Oxygen Supply/Demand Balance

R. James Barnard, Ph.D., Rex MacAlpin, M.D.,
Albert A. Kattus, M.D., and Gerald D. Buckberg, M.D.

SUMMARY In five well-trained and five sedentary control subjects potential subendocardial blood supply was estimated from the diastolic pressure time index (DPTI) and myocardial oxygen demands from the tension time index (TTI) during a progressive near-maximal treadmill test. DPTI/TTI was used to assess the effects of training on myocardial oxygen supply/demand balance. DPTI/TTI was significantly higher in trained subjects at rest and comparable workloads. At 6.4 km/hr, 18% grade (maximum for the controls), TTI was significantly lower (4300 ± 76 vs 4495 ± 99 mm Hg · sec/min) and DPTI significantly higher (2534 ± 86 vs 2295 ± 91 mm Hg · sec/min) in the trained subjects; DPTI/TTI was significantly higher (0.59 ± 0.02 vs 0.50 ± 0.03). At near-maximal heart rates both groups achieved the same supply/demand balance (0.50); however, the trained subjects were working at higher workloads. We conclude that endurance conditioning increases work capacity, reduces myocardial oxygen demands, increases potential oxygen supply and improves the supply/demand balance at any given submaximal workload which reduces the risk of ischemia.

EXERCISE TRAINING is known to reduce myocardial oxygen demands at any given submaximal workload. Attention also has been focused on the possible development of coronary collaterals to enhance oxygen delivery to the myocardium. Several studies have shown that collaterals do develop in patients with coronary artery disease but that exercise training does not enhance their development. These studies concluded that the increased work capacity resulting from training in some patients with coronary artery disease is due solely to a reduction in myocardial oxygen demands. Previous work by Buckberg et al. has shown that potential subendocardial blood supply can be estimated from the diastolic pressure time index (DPTI). If training increases DPTI at any given workload then potential subendocardial blood supply would be increased. In this report we describe the effects of training on potential subendocardial blood supply (DPTI) and myocardial oxygen demands estimated from the tension time index (TTI). We have previously shown that the ratio of these indices of supply and demand (DPTI/TTI) predicted ischemia when sudden, strenuous exercise was performed without prior warm-up. The present study tests the effects of training on these supply/demand indices during a progressive, near-maximal treadmill test.

Methods

We studied ten men; the five trained volunteers had been involved in an active jogging program (30–60 min/day 3–5 days/week) for at least six months and the five similar age (35 ± 4 vs 37 ± 6 yrs) control volunteers did no organized exercise. Using 1% lidocaine anesthesia, a polyethylene catheter (0.045 inch i.d.) was introduced percutaneously into the brachial artery and advanced six inches into the axillary artery. Arterial blood pressure was measured (using the 4th rib-ternal junction as the 0 reference level) by connecting the catheter to a Statham P23dB transducer and recording on a Honeywell 906 Visicorder. A transthoracic electrocardiogram (V1-RV1) was recorded simultaneously using Sanborn exercise electrodes.

Control arterial blood pressure and electrocardiographic recordings were made after a 15 minute rest period and while the subjects were standing beside the treadmill. They then performed a progressive multi-stage treadmill test exercising for three minutes at each of the following workloads: 4.8 km/hr, 10% grade; 6.4 km/hr, 10% grade, 6.4 km/hr, 14% grade; 6.4 km/hr, 18% grade; 6.4 km/hr, 22% grade; 8.1 km/hr, 24% grade. We ended the test when the subjects reached near-maximal heart rates for their age. Arterial blood pressure recordings from three consecutive heart beats were analyzed by planimetry to obtain an average value for TTI and DPTI (fig. 1). Left atrial pressure was assumed to be 5 mm Hg for the DPTI measurements. The t-test was used for statistical analysis and P ≤ 0.05 was used for statistical significance.

Results

The subjects in our study who were involved in an active jogging program demonstrated the classic signs of training. Their resting heart rates were significantly lower (76 ± 2 vs 84 ± 5); however, there was no significant difference in near-maximal heart rates (182 ± 3 vs 178 ± 3). They were capable of working to higher workloads before achieving near-maximal heart rate and had lower heart rates at any given submaximal workload (fig. 2).

Myocardial oxygen requirements at rest, estimated from the tension time index, were 18% lower in trained than untrained subjects. This reduction in tension time index was due to a lower systolic pressure (120 ± 4 vs 130 ± 4) as well as a lower resting heart rate. During exercise, at comparable workloads, the tension time index remained lower in trained than untrained subjects (fig. 3). The tension time index rose to comparable levels in all subjects when they exercised to reach their near-maximal heart rates. Trained subjects, however, were able to walk faster and up steeper grades before reaching this level of oxygen requirement.

Diastolic pressure time index during rest was higher in trained than untrained subjects (3611 ± 93 vs 3425 ± 104 mm Hg · sec/min) but the difference was not statistically significant. Exercise caused a significant reduction in DPTI in both groups as tachycardia shortened the diastolic filling
period of the coronary arteries. Trained subjects, however, maintained a significantly higher potential subendocardial blood supply (DPTI) than untrained subjects at comparable workloads. As a result, they were able to work at higher levels before reducing their diastolic pressure time indices to levels seen in untrained subjects at near-maximal exercise (fig. 4).

Myocardial O₂ supply/demand balance (DPTI/TTI) at rest was significantly higher in trained than untrained subjects (1.78 ± 0.07 vs 1.45 ± 0.2). Trained subjects were able to maintain an approximate 17% higher supply/demand index at all comparable levels of work (fig. 5). Untrained subjects reached maximum workloads when the supply/demand index fell to 0.5. Although trained subjects reached maximum work capacity at a comparable supply/demand index, they did not lower their DPTI/TTI to this value until they exercised to a higher capacity. Transthoracic electrocardiograms showed no signs of ischemia in either the trained or control group.

Discussion

Our results show that endurance training a) increases work capacity, b) reduces myocardial oxygen demands, c) increases potential oxygen supply and d) improves the myocardial supply/demand balance so that the risk of myocardial ischemia at any given submaximal workload is lessened. Our observation that training increases work capacity and reduces myocardial oxygen demands at any given submaximal workload is consistent with previous reports of the effects of exercise. These salutary effects occur in both healthy subjects and patients with coronary artery disease. We used the tension time index to estimate myocardial oxygen demands as previous experimental and clinical studies have shown that it correlates well with myocardial oxygen consumption. The lower TTI observed in our trained subjects at any given workload is consistent with the report of Frick et al. who studied their subjects before and after endurance training. The lower tension time indexes in trained subjects occurred as a result of lower heart rates and systolic blood pressures. The myocardial adaptation of a slower heart rate resulted in less tension development per minute. The lower systolic blood pressure suggests that a peripheral muscular adaptation which reduced afterload might have occurred in trained subjects.

The avoidance of ischemia during exercise requires a
matching of oxygen demands with oxygen supply. The subendocardium is most vulnerable to ischemia and can receive its oxygen supply only during diastole; the diastolic pressure time index describes the coronary driving pressure during myocardial relaxation and has been shown to be a reasonable predictor of potential subendocardial blood flow.\(^6\),\(^9\) DPTI fell progressively in both trained and untrained subjects during exercise as a consequence of reduced diastolic filling time resulting from tachycardia. The rate of decline of DPTI was, however, much slower in trained than untrained subjects and the trained subjects consequently had a higher DPTI at any given submaximal workload. The higher DPTI levels occurred as a consequence of slower heart rates at comparable workloads. These observations indicate that trained subjects have higher potential subendocardial blood flow than untrained subjects at comparable levels of physical exertion. This higher potential blood supply may be extremely valuable during exercise since coronary blood flow is likely the sole source of oxygen delivery; the heart extracts almost 70\% of its oxygen at rest and raises its oxygen extraction to 85\% with heavy exertion.\(^9\)

We used the ratio DPTI/TTI to estimate the myocardial oxygen supply/demand balance. During rest, trained subjects had a more favorable balance in that this ratio was 23\% higher than in the controls. The ratio remained higher in trained subjects when comparisons were made between them and untrained subjects at comparable levels of work. DPTI fell less rapidly and tension time indices rose less precipitously. A fall in this ratio occurred with exercise in both groups and indicates an expenditure of vasodilator reserve capacity as myocardial oxygen requirements rise and potential subendocardial blood supply falls. Ischemia was not evident electrocardiographically in either group as DPTI/TTI never fell below 0.4. Our previous study\(^7\) showed that ischemia occurred clinically only at ratios below 0.4. Untrained subjects reached maximum work capacity (near-max heart rate) when their supply/demand index fell to 0.50 and they achieved their near-maximum heart rate. Trained subjects also reduced their DPTI/TTI ratio to this same level at comparable heart rates but were able to walk faster and up steeper inclines before achieving this level.

Our previous experimental\(^8\) and clinical\(^7\) studies demonstrate that any further reduction in this ratio is associated with subendocardial ischemia. These observations suggest that in subjects with patent coronaries, the coronaries are close to being maximally dilated when maximum heart rate and the observed DPTI/TTI levels occur. Ischemia would, of course, occur at higher ratios in patients with coronary disease as DPTI is always reduced beyond the coronary stenosis and their coronaries become maximally dilated at higher DPTI/TTI ratios recorded in the aorta.

Our study shows that training may improve coronary blood flow without affecting the anatomic configuration of the coronary vessels. The diastolic pressure time index was significantly higher in trained than untrained subjects at comparable levels of exercise. When the coronaries become maximally dilated, subendocardial flow is dependent upon the driving pressure (DPTI). Since trained subjects maintained a higher driving pressure during exercise it is reasonable to assume that blood supply through both a major coronary lesion and collateral pathways would be greater in trained than untrained coronary patients doing comparable levels of exercise. Our data show, therefore, that exercise training simultaneously reduces myocardial oxygen requirements and increases potential subendocardial oxygen supply and therefore creates a more favorable supply/demand balance. Our observations help explain a reduced risk of ischemia in some coronary patients who undergo exercise programs. Whether or not patients with other forms of cardiac disease (e.g., valvular heart disease, congenital heart disease) can adapt to exercise training and receive similar benefits remains to be determined.

References

**Figure 5.** Effect of training on myocardial oxygen supply/demand balance (DPTI/TTI) at different workloads.
Effect on training on myocardial oxygen supply/demand balance.
R J Barnard, R MacAlpin, A A Kattus and G D Buckberg

Circulation. 1977;56:289-291
doi: 10.1161/01.CIR.56.2.289

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1977 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://circ.ahajournals.org/content/56/2/289