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QUANTITY OF MUSCLE MASS TRAINED: THE EFFECT ON CENTRAL CARDIOVASCULAR ADAPTATIONS AND THE TRANSFER OF FITNESS TO UNTRAINED LIMBS

A Thesis

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Degree

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ABSTRACT

Clausen (8) suggests that there exists a direct relationship between the quantity of leg muscle trained and gains in arm and two-leg aerobic power; he argues that this relationship hinges on central cardiovascular adaptations. The question addressed here is whether or not varying the amount of leg muscle mass trained affects arm and two-leg aerobic power. Three groups were tested, a control group and two training groups. Seven subjects trained with both legs simultaneously, 30 minutes a day, 4 days a week for 4 weeks. Nine subjects trained each leg separately, 15 minutes a day, 4 days a week for 4 weeks. Both groups trained at an intensity equal to 75% of two-leg maximal aerobic power. Arm and leg oxygen uptake tests were conducted before and after training. Only the two-leg training induced significant gains in arm aerobic power ($p<.0003$), while both modes of training promoted significant improvement in two-leg aerobic power ($p<.0008$). The conclusions reached: 1. Two-leg training induces greater central cardiovascular adaptations than does one leg training, perhaps due to the fact that more muscle mass is metabolically active; 2. One leg training leads to a similar improvement in two-leg aerobic power as does two-leg training, with the gains appearing to be independent of central cardiovascular adaptations and dependent on leg muscle changes.

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CHAPTER I

INTRODUCTION

Aerobic training induces changes in the skeletal muscle and the cardiovascular system that allow the body to increase maximal aerobic power (6, 7, 15, 16, 18, 19, 33). Scientists have tried to determine which of these systems primarily limits the increase in aerobic power. In general there is more support for the cardiovascular system as the limiting factor than for the skeletal muscular system (30). One unresolved issue is whether it is central cardiovascular function, i.e. maximum cardiac output, or peripheral blood flow that limits maximal aerobic power (8, 37).

Clausen et al. (7) argue that changes in the central cardiovascular system are paramount to increases in maximal oxygen consumption. This argument stems from the results of an investigation that showed that aerobic leg training led to a 10% increase in maximal aerobic power of the arms, despite the untrained condition of the arms. Clausen et al. (7) suggested that the leg training caused an increase in maximal cardiac output that permitted higher blood to the arms with no change in arm vascular resistance.

In a subsequent review, Clausen (8), used the results of Davies and Sargent's one-leg cycle training study (10) to modify his position. Davies and Sargent found that one-leg cycle training caused only a 5% increase in two-leg aerobic power, one third the

increase usually measured after two-leg training. Clausen reasoned that one leg training provided insufficient stimulus to the central cardiovascular system and as a result, little change in the heart's pumping capabilities. Clausen (8) concluded that to induce changes in the central cardiovascular system a critical amount of muscle mass needs to be involved in the training.

The purpose of the present study was to test Clausen's claim that aerobic training of a critical quantity of muscle mass promotes central cardiovascular adaptations, as manifested by an increase in aerobic power in trained as well as in sedentary muscle groups. In this study, three groups of subjects were compared. One group trained both legs simultaneously, a training regimen thought to cause central cardiovascular adaptations that permit an increase in leg and arm maximal aerobic power. Another group trained both legs, but only one at a time. Theoretically, this mode of training should fail to sufficiently stimulate the central cardiovascular system, resulting in smaller gains in leg and arm maximal aerobic power. The two experimental groups were compared to a control, the third group. The following hypotheses were tested: 1. no difference exists in the transfer of fitness to the arms between the two training groups; 2. no difference exists in the gain of leg aerobic power between the two trained groups.

CHAPTER II

REVIEW OF RELATED LITERATURE

The body adapts to aerobic training in many ways. A direct relationship exists between the intensity, duration and frequency of the stimulus and the extent of the adaptations (21, 22, 26). One result of aerobic training involves changes in the exercised skeletal muscle (15,28). Holloszy (15), in a review of skeletal muscle adaptations to aerobic training, discusses these changes and suggests that two important alterations include increases in both mitochondrial density and enzyme concentration. The significance of the latter change lies in the fact that these enzymes catalyze the reactions of the Krebs cycle, the electron transport system, and fatty acid oxidation. Holloszy claims that such adaptations allow aerobically trained muscle to use more oxygen; the muscle has a greater oxidative work capacity.

Many exercise physiologists tend to ignore the possibility that skeletal muscle adaptations might cause gains in maximal aerobic power. Hoppeler et al. (16) suggests that skeletal muscle adaptations warrant further attention. These researchers found that 15 hours of heavy submaximal cycle training caused a 40% increase in the trained muscle's mitochondrial density. Hoppeler et al. claim that such an increase elevates the muscle's oxidative capacity, which allows for an increased mechanical power output from the muscle. The group suggests that enhanced mechanical

power output can account for changes in maximal aerobic power. The researchers do not clarify this point, although it appears they are suggesting that the working muscle extracts more oxygen from the blood, resulting in an increased aerobic power.

Aerobic training also causes changes in the cardiovascular system, which is divided into two parts: the peripheral and the central. Much of the discussion on the increases in, and limitations of, maximal oxygen consumption concerns the changes in the peripheral and central cardiovascular systems. The central cardiovascular system refers to the heart and its' capacity to pump blood. Clausen (8) claims that aerobic training of a large muscle mass alters this capacity, because such training increases left ventricular volume and enhances myocardial performance. Works by Keul et al. and Thompson et al. provide evidence that supports Clausen's claim.

Keul et al. (17) used echocardiagraphic measurements of successful endurance athletes, untrained individuals, and patients with hypertension and cardiomyopathy to conclude that endurance athletes experience increases in left ventricular volume and myocardial performance. The researchers' principal piece of evidence consists of the fact that endurance athletes have a larger stroke volume to heart rate ratio than sedentary persons. Thompson et al. (39) used various techniques to measure cardiac dimensions and performance. They saw no significant change in left ventricular volume but they did find an increase in myocardial performance. The researchers claim that the enhanced

performance resulted from an increase in heart muscle contractility that was due to an adaptation of the muscle and not of the nervous system. They based their assertion on the fact that left ventricular emptying time increases after chemical blockade of the heart's autonomic control centers.

Work conducted by Clausen et al. (7) and Klausen et al. (20) provides additional evidence for an increase in myocardial performance after aerobic training. Clausen et al. (7) trained men on cycle ergometers in order to see how the cycle training effected arm and leg maximal oxygen consumption, indocyanine green clearance, maximum heart rate, mean arterial blood pressure and maximal cardiac output. The cycle training caused a 15% increase in leg maximal oxygen consumption and a 10% increase in arm maximal oxygen consumption, but produced no changes in either indocyanine green clearance or maximum heart rate during performance tests. These data led Clausen et al. to suggest that the gains in arm aerobic power resulted from increased blood flow to the arms. They argued that a greater cardiac output to the arms rather than a decrease in arm vascular resistance permitted the increase in blood flow. Clausen et. al. maintained that arm vascular resistance remained constant because no change in indocyanine green clearance occurred after the cycle training. The lack of change in maximal heart rate prompted them to conclude that gains in either left ventricular stroke volume or myocardial performance allowed for the increase in cardiac output.

Clausen, in a review (8), modified the position he elaborated in his earlier work (7). He used data generated by Davies and Sargent (10) to claim that increases in myocardial performance occur only after aerobic training involving a sufficient quantity of muscle mass. Davies and Sargent found that one-legged cycle training caused a 5% increase in two-leg maximal aerobic power and a 11% increase in one-leg maximal aerobic power. Clausen argued that one-leg training insufficiently stimulated the central cardiovascular system. The result of such training: little or no gain in myocardial performance and small improvements in two-leg aerobic power in comparison to the gains in aerobic power experienced by each leg separately.

The data of Klausen et al. (20) provides additional support to Clausen's claims. Klausen et al. used a one-leg training design similar to the one used by Davies and Sargent (10). The Klausen group looked at how one-leg cycle training altered one and two-leg aerobic power, maximal cardiac output, total peripheral resistance, leg blood flow and leg arterio-venous oxygen difference. The researchers found that the effect of one-leg cycle training on both legs included an 11% increase in aerobic power, no change in leg blood flow, and an increase in leg arterio-venous oxygen difference. The one-leg training effects on one-leg performance included a 19% increase in aerobic power, an increase in leg blood flow and little change in leg arterio-venous oxygen difference.

Klausen et al.(20) concluded that one-leg training fails to promote central cardiovascular adaptations because one-leg aerobic power increased more than two-leg aerobic power. They suggested that if there were sufficient cardiovascular adaptations then the gains in two-leg aerobic power should equal those experienced by each leg individually. The group contends that the failure of the body to augment leg blood flow during the post-training two-leg performance tests supports their claim that no central cardiovascular adaptations occurred. The scientists reasoned that one-leg training insufficiently taxed the heart, not inducing large enough increases in the heart's capability to pump blood to permit a large increase in two-leg blood flow and two-leg aerobic power.

Peripheral circulatory adaptations, on the other hand, deal with the interplay between local metabolic vasodilation and centrally controlled sympathetic vasoconstriction (6). Many of the exercise physiologists who argue that peripheral circulatory adjustments play an important role in determining maximal aerobic power base their claims on data collected from one type of experimental design. This design entails adding arm work to maximal leg work. Secher et al. (36) and Gleser et al. (12) undertook such studies.

Secher et al. (36) found that the addition of heavy arm work to maximal leg work reduced leg blood flow and leg aerobic power, but caused two leg arterio-venous oxygen difference to increase. Secher et al. reasoned that increases in leg vascular resistance

led to a drop in leg blood flow, and that the body attempted to compensate by extracting more oxygen from the arterial blood. The group concluded that the peripheral circulatory system limits maximal aerobic power because the addition of arm work to leg work results in a decrease in leg blood flow before the legs achieve maximal aerobic power. The scientists also argued that if central cardiovascular adjustments limit maximal aerobic power, then a switch from leg work to combined arm and leg work should induce a reduction in mean arterial blood pressure. They reasoned that if all the limbs are metabolically active then the heart might not be able to pump enough blood to maintain mean arterial blood pressure. The researchers failed to find such a decrease.

Gleser et al. (12) concurred with Secher et al. (36). Gleser et al. added arm work to maximal leg work and found that maximal aerobic power increased by 10%. The scientists attributed this increase to an elevated cardiac output. Both research groups claimed that leg work by itself fails to elicit maximal cardiac output because adding arm work to the leg work increases maximal aerobic power.

The data they generated also supports an alternate hypothesis. When the arms begin to work, the amount of metabolically active muscle tissue increases, and, as a result, more blood flows to the arms. Increased physical activity induces locally mediated vasodilation in the working arm muscles. If cardiac output can not meet this increased demand for blood flow by the arms without a fall in mean arterial blood pressure, then blood flow must be

fall in mean arterial blood pressure, then blood flow must be redistributed. Increased leg vascular resistance reduces blood flow to the legs, which an elevated arterial oxygen extraction nearly offsets, and more blood flows to the arms. The result: gains in total body aerobic power independent of increases in cardiac output.

Lewis et al. (21) suggested that the data generated from the combined arm and leg work experimental design be viewed skeptically. Lewis et al. (21) noted that a subject's fitness and familiarity with either leg work or arm work or both affects results. Secher et al. (35), in a study that preceded his work discussed previously (36), found that if highly trained cyclists attempted arm exercise while performing maximal leg work, then aerobic power falls. This finding suggests that highly trained cyclists reach maximal cardiac output during maximal aerobic power tests on the cycle ergometer.

In summary, aerobic exercise induces many physiological adaptations. These changes include alterations in skeletal muscle and the circulatory system that permit increases in maximal aerobic power. It appears that many exercise physiologists, such as Rowell (30), Clausen (8), and Blomqvist and Saltin (6), believe that changes in the cardiovascular system are more responsible for increases in aerobic power than changes in the skeletal muscle. However, the debate over which part of the cardiovascular system is more important in limiting gains in aerobic power is not settled. Some physiologists now argue that it may be a

mistake to differentiate between the central and peripheral circulatory systems (6, 30). Blomqvist and Saltin (6) claim that increases in cardiac output occur only if total peripheral resistance decreases, because as vascular resistance falls more blood returns to the heart after each beat, and, hence, the heart can pump more blood. They conclude that central and peripheral circulatory factors work synergistically to permit increases in maximal aerobic power.

CHAPTER III

METHODS

I. Selection of Subjects

Twenty three college-age (mean age and weight: 22.8 years and 73 kg) males served as subjects. The subjects read and signed an informed consent form prior to participating in the study.

II. Maximal Oxygen Uptake Tests

One week before the training period began, the subjects' maximal leg and arm oxygen uptakes were measured. The leg tests preceded the arm tests by at least three days.

A mechanically braked Monarch cycle was used for the leg tests. The subjects pedalled at 60 revolutions per minute at an initial work rate of 118 watts, except for one subject. This subject weighed 45 kg and because of his low weight his initial work rate was set at 59 watts. The work rate was increased 30 watts every two minutes. Expired gas was collected every other minute until heart rate reached 150 beats per minute, at which time continuous collections were made.

The arm tests were conducted on a modified Monarch cycle, set up in a similar fashion to the one employed by Sawka et al. (32); this design permits optimal positioning of the crank arms. The subjects cranked the pedals at 70 rpm at an initial work rate of 69 watts, except for the lightweight subject who started at 35

watts. Work rate increased 17 watts every two minutes. Expired gas was collected every other minute until the heart rate reached 150 bpm, at which time continuous collections were made.

In both the leg and arm tests, the pedal frequency was monitored by a modified speedometer attached to the ergometer's flywheel. A metronome was also employed to help the subjects maintain the proper cadence. Test duration did not exceed 10 minutes.

Three days after the last training session the subjects were retested. The legs were tested first and then the arms, with at least three days, but no more than five, between the leg and arm tests. All follow up tests were completed within seven days of the last training session.

III. Group Formation and Training Regime

After the initial testing, the subjects were assigned to one of three groups. Assignments were arranged so that each group had a similar mean value for two-leg aerobic power. The seven subjects assigned to the two-leg training group trained 30 minutes a day, 4 days a week, for 4 weeks at a heart rate that corresponded to 75% of their two-leg $V0_2$ max. The nine subjects assigned to the one-leg group trained each leg 15 minutes a day, 4 days a week for 4 weeks at a heart rate equal to that of the two-leg trained group. To insure proper training intensity in both groups, the subjects' heart rates were checked every 5 minutes. All training took place on mechanically braked Monarch ergometers. The subjects pedaled

at 60 rpm and their cadence was monitored with modified speedometers attached to the ergometers' flywheels.

The remaining seven subjects served as the control group. These subjects were instructed to refrain from engaging in any new activities, although they were permitted to maintain daily routines. All subjects were asked to avoid extra aerobic or weight-lifting activities while involved in the study.

IV. Measurement of Selected Variables

Determination of Oxygen Uptake

Oxygen uptake was measured by the open circuit method using meteorological balloons and a Daniels valve. An Applied Electrochemistry model S-3A-O_2 analyzer and a Beckman model LB2 $C0_2$ analyzer were used to measure the expired gas fractions. The analyzers were calibrated with standardized gases. The calibration gases were checked by the Scholander and Lloyd-Gallenkamp methods. A Parkinson-Cowan CD4 gas meter and Narco Biosystems DMP-4A recorder were used to measure and record inspired ventilation. Oxygen uptake was calculated by the Haldane transformation.

The criteria for $V0_2$ max were an RER greater than 1.10, a heart rate close to the predicted value (220-age), a leveling in the rate of increase in $V0_2$, and the inability of the subject to maintain the proper cadence. In an attempt to help the subjects work for as long as possible, each received vigorous verbal encouragement.

Measurement of Heart Rate

Heart rate was recorded with a Narco Biosystems DMP-4A recorder and cardiac coupler. A standard CM-5 bipolar lead system was used to record the ECG. R waves were counted for the last 12 seconds of each minute to calculate the HR in beats per minute.

V. Statistical Analysis

The nature of this experimental design permitted the use of co-variance analysis. I compared the changes in the experimental groups to the changes in the control group, with the first test score serving as the co-variate. A P-value of $< .05$ was considered significant. In addition to the co-variance test, the arm data were analyzed further using a Bonferroni multiple comparison test. This test permits a more thorough examination of the data, as small differences in the means were expected. The alpha level for this test was set at .05.

CHAPTER IV

RESULTS

The results are listed in Table 1 and Table 2. The mean value for two-leg maximal aerobic power for the two-leg trained group increased 13.5%, while the one-leg trained men experienced a 15.6% gain. The aerobic power of the controls changed 1.2%. The analysis of co-variance indicated that the increase in the means for the one-leg and two-leg trained men were significantly different from the control group. The P-values are <.0002 and <.0008 respectively.

The mean max arm $V0_2$ for the control group decreased 4.1%. Of the original seven controls, two were dropped from the study because they began weight training. One of the remaining five controls took two post-training arm test. In his first post-training test, VO_2 max decreased by 30%, maximum heart rate decreased 30 bpm, max ventilation decreased 40 liters a minute, and RER dropped from 1.23 to .90. The subject was retested one week later and performed better, although still worse than during his initial test. After training, the mean arm $V0_2$ max for the two-leg trained men increased 18.1%. Two men in this group had large increases(>26%); they claimed that their only activities were the training and some intramural football games. The mean arm $V0_2$ max for the one-leg trained men increased 3.5%. The analysis of co-variance indicated that only the increase in the mean for the two-leg trained was significant, with a P-value < .0003. The P-value for the one-leg

Table 1. Maximum aerobic power, P-values and RER data for the legs for the three different groups before and after training.

	Maximum aerobic power				RER	
Group	Pre	Post	% Change	P-Value	Pre	Post
Control	2.94±.20	2.97±.19	+1.2%		1.16±.04	1.13±.08
Two-legs	2.98±.20	3.37±.20	+13.5%	.0008	1.18±.07	1.14±.03
One-leg	2.89±.11	3.34±.11	+15.6%	.0002	1.22±.05	1.19±.06

NOTE: Values are liters of oxygen per minute expressed as Mean ± SEM
Values for RER are expressed as Mean±SEM

Table 2. Maximum aerobic power, P-values and RER data for the arms for the three different groups before and after the training.

	Maximum aerobic power				RER	
Group	Pre	Post	% Change	P-Value	Pre	Post
Control	2.02±.20	1.92±.15	-4.1%		1.16±.02	1.18±.05
Two-legs	2.04±.13	2.42±.16	+18.1%	.0003	1.13±.07	1.16±.10
One-leg	2.01±.11	2.12±.10	+3.5%	.06	1.16±.06	1.17±.06

NOTE: Values are liters of oxygen per minute expressed as Mean ± SEM
Values for RER are expressed as Mean±SEM

trained men was < .06. A Bonferroni comparison of means indicated that the means for the two trained groups were significantly different at the .05 level and that no significant difference existed between the one-leg trained men and the control group.

CHAPTER V

DISCUSSION

The purpose of this research was to test Clausen's (8) hypothesis that a critical quantity of muscle mass needs to be aerobically trained to cause central cardiovascular adaptations. I found that the training of two-legs together induced a greater gain in arm aerobic power than the training of two-legs, but each one separately. This result supports Clausen's (8) claim that the aerobic training of large muscle groups promotes greater central cardiovascular adaptations than the aerobic training of smaller muscle groups.

A problem exists though, as both the trained groups had a similar increase in two-leg $VO_{2\ max}$; such a result is inconsistent with Clausen's (8) hypothesis. One-leg training should not promote enough central cardiovascular adaptations to induce large gains in two-leg aerobic power, because an insufficient quantity of muscle is metabolically active. This finding is also inconsistent with the work of Klausen et al. (20) and Davies and Sargent (10). These researchers found that one-leg training failed to promote significant improvement in two-leg aerobic power. My results may differ from theirs for several reasons, including subject number, training intensity and research design. Klausen et al. (20) used only six subjects and Davies and Sargent (10) used only seven subjects in their respective studies. While the 4.7% increase in two-leg

aerobic power found by Davies and Sargent (10) was not a significant difference, what is puzzling is that the 11% increase found by Klausen et al. (20) was also not significant. An increase in subject number in the latter study would have increased statistical power and might have allowed Klausen et al. (20) to show that the 11% improvement in two-leg aerobic power was significant. The 4.7% increase found by Davies and Sargent (10) is less than a half to a third of that reported by Klausen et al. (20) and the present study. The small increase found by Davies and Sargent may have been due to the lower training intensity they used in their study.

Perhaps the one-leg trained group in the present study did not experience central cardiovascular adaptations, as the change in this group's mean value for arm aerobic power failed to differ significantly from the control group's arm aerobic power. The one-leg trained group's significant gain in two-leg $V0_2$ max appears to be due to changes in the leg muscles, and not central cardiovascular adaptations. Some of the changes that training induced in muscle that may have accountted for the increase in aerobic power include increases in mitochondrial density, oxidative enzyme concentrations and muscle capillary density (13, 15, 16, 19, 20). One of these papers, Hoppeler et al. (16), strongly advocates the view that muscle adaptations can account for gains in aerobic power. They reasoned that alterations in muscle infrastructure permit an increase in aerobic capacity at the local level (maintained power) that can account for the increase in aerobic power at the general level (aerobic power).

The possibility that the men in the one-leg trained group experienced greater peripheral muscle adaptations than the men in the two-leg trained group exists. Both groups trained at a heart rate equivalent to 75% of two-leg aerobic power. The work rate that corresponded to this heart rate during the last week of training reached 98.5 watts per leg for the two-leg trained group versus 147 watts per leg for the one-leg trained group. The one-leg trained group worked at a rate that was 50% higher than that rate achieved by the two-leg trained group. Perhaps this difference in training intensity induced greater peripheral muscle adaptations in the one-leg trained group. Additionally, the power output of the one-leg trained group was equivalent to a VO_2 of 2.1 liters per minute, or roughly equal to 66% of their two-leg maximum. This training intensity exceeds the threshold of the exercise intensity needed for a training effect as outlined by the American College of Sports Medicine[1]. One would expect, therefore, that the one-leg trained should group should have experienced some central cardiovascular adaptations, but the arm aerobic power data fails to support this expectation.

The differences in work rate achieved by the two groups raises an interesting question that deals with total work output. I attempted to address this issue by training both groups at the same absolute intensity: 30 minutes of aerobic work at a heart rate equivalent to 75% of two-leg oxygen consumption. Although the

[1]American College of Sports Medicine. (1986) Guidelines for Graded Exercise Testing and Perscription. Third Edition, Philadelphia: Lea and Febiger.

one-leg trained group ended up working at a higher rate per leg than the two-leg trained group, the one-leg trained group failed to perform more total work. On average during the last training session, each leg in the one-leg trained group performed 2206 watts of work (15 minutes at 147 watts). Each leg in the two-leg trained group, on the other hand, performed 2941 watts of work (197 watts divided by 2 and multiplied by 30 minutes). The sum of the work performed by both legs in each group also favored the two-leg trained group; the two-leg trained group completed 25% more work than the one-leg trained. Perhaps this 25% discrepancy in total work between the groups permitted the two-leg trained group to experience greater central cardiovascular adaptations than the one-leg trained group.

In summary, the arm data suggests that two-leg training induces more central cardiovascular adaptations than one-leg training. These additional cardiovascular adaptations may result from the fact that with two-leg training, the heart must supply blood to more muscle mass than it does with one-leg training. The fact that the two-leg trained group performed 25% more work than the one-leg trained group may skew this interpretation. The data also indicate that intense one-leg training induces changes in two-leg aerobic power that equal those experienced by more conventional two-leg training. The mechanism for the large gain in aerobic power in the one-leg trained group appears to be independent of central cardiovascular adaptations and dependent on changes in leg muscle activity.

CHAPTER VI

CONCLUSIONS AND RECOMMENDATIONS

This study supports the following statements: 1) Greater gains in arm VO_2 max occur as the quantity of leg muscle mass involved in a training program increases; 2) Intense, non-simultaneous training of two-legs can permit increases in two-leg VO_2 max that rival gains experienced by men who train both legs simultaneously; 3) Gains in two-leg $V0_2$ max experienced by the one-leg trained group appear to be independent of central cardiovascular adaptations and dependent on peripheral muscle adaptations.

Additional research may provide evidence that could strengthen these conclusions. Several recommended projects include: muscle biopsies, to assess if the one-leg trained group did indeed have greater muscle changes than the two-leg trained group; and measurements of mean arterial blood pressure, arm blood flow, and maximal cardiac output before and after training during the arm aerobic power tests to see how leg training alters arm hemodynamics. I also recommend a study that examines the relationship between gains in aerobic power, total work and training intensity. Finally, I recommend a study that examines the effects of two-leg and one-leg training on one-leg aerobic power.

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