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Circulation 1987;75;323-330
Circulation is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 72514
 Print ISSN: 0009-7322. Online ISSN: 1524-4539

Cardiovascular adaptations to intense swim training in sedentary middle-aged men and women

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ABSTRACT Central and peripheral cardiovascular adaptations to 12 weeks of intense swim training were characterized in 12 previously sedentary middle-aged men and women. Peak oxygen uptake ($\dot{V}O_2$) during upright bicycle exercise improved from 29.2 ± 5.6 to 34.7 ± 6.7 ml/kg/min (mean ± SD, $p < .01$) because of similar increases in peak cardiac output (CO) and calculated arteriovenous oxygen difference (both $p = .02$). Peak supine $\dot{V}O_2$ was 10% higher after training ($p < .005$) solely because of enhanced CO ($p = .005$). Peak heart rate decreased in both postures; therefore stroke volume at peak exercise was greater by 10% and 18% in the upright and supine postures, respectively ($p = .05$ and $p = .005$). There was an identical 18% rise ($p = .01$) in peak supine left ventricular end-diastolic volume index by radionuclide ventriculography but no change in left ventricular ejection fraction or end-systolic volume index (ESVI). Peak systolic blood pressure (SBP) was unchanged in the upright posture but was 8% higher ($p = .002$) during recumbency despite a similar total peripheral resistance and SBP/ESVI ratio. Maximal calf conductance (G_{max}), assessed separately by venous occlusion plethysmography after local ischemic exercise to fatigue, was augmented 20% ($p < .02$) by training, resulting in an 18% greater hyperemic blood flow ($p = .05$). Peak $\dot{V}O_2$, CO, and G_{max} were unchanged in five nonexercising control subjects. We conclude that in middle-aged humans, intense swim training for 12 weeks produces adaptations that include a greater capacity for vasodilatation in skeletal muscle and an enhanced cardiac pump capacity.
Circulation 75, No. 3, 323–330, 1987.

ENDURANCE EXERCISE training programs based on running and walking result in an improvement in maximal oxygen uptake ($\dot{V}O_2$), cardiac output (CO), and stroke volume.[1, 2] Swimming is a potentially useful alternate form of conditioning in individuals with neuromuscular or musculoskeletal disabilities of the lower extremities or with bronchospasm induced by nonaquatic exercise. However, little is known about its effects on the cardiovascular system and on the ability to exercise on land. In this investigation, our purposes were (1) to assess whether swimming significantly improves peak bicycle exercise capacity in middle-aged individuals and (2) to characterize the central and peripheral cardiovascular adaptations to swim training. Our hypotheses were that the effects of swim training are transferable to other activities and include both cardiac and peripheral vascular adaptations.

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Supported by NIH Ischemic SCOR grant HL17669 and NIH Training Grant HL07360.

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Received Feb. 24, 1986; revision accepted Oct. 2, 1986.

Methods

Subjects and training. Seventeen subjects were recruited for the study. All were in good health and had normal findings on physical examination and on the exercise electrocardiogram. None exercised regularly. Twelve were chosen at random for the experimental group, which swam 6 days per week and circuit trained with weights three times per week for 12 weeks. There were nine men and three women in the experimental group. The mean age (± SD) was 38 ± 7 years with a range of 30 to 48 years for men and 30 to 42 for women. The control group included three men and two women, with a mean age of 38 ± 8 years (range 31 to 45).

During the first 1 to 2 weeks of training, subjects swam 30 to 45 min per session. For the final 2 weeks of the 12 week program, they averaged 2 miles of swimming per session in a regimen that included both continuous and repetition (interval) training. Approximately 25% of the total distance was com-

prised of kicking drills that primarily activated the lower extremities. Circuit training consisted of a series of 15 individual exercises that involved both the upper and lower extremities and were performed to fatigue in two sets of 10 to 15 repetitions each. The duration of each session was approximately 1 hr.

The study was approved by the Institutional Human Research Review Committee. All subjects gave voluntary written informed consent.

Overall study design. Both before and after the 12 week training or control periods, each subject visited the laboratory four times. The following procedures were performed in random order, each on a separate visit: (1) measurement of $\dot{V}O_2$, CO, heart rate, and blood pressure during seated rest and upright submaximal and peak bicycle exercise; (2) acquisition of the same data in (1) during supine rest and supine submaximal and peak bicycle exercise; (3) multigated radionuclide ventriculographic examinations under conditions described in (2); and (4) determination of resting and maximal local vascular conductance in the calf by venous occlusion plethysmography.

$\dot{V}O_2$. $\dot{V}O_2$ was measured by the Douglas bag technique. After the resting data were obtained, subjects performed two to three 6 min bouts of constant work rate submaximal exercise that progressed in intensity from lightest to heaviest and were separated from each other by approximately 15 min of rest. Peak $\dot{V}O_2$ was studied with a progressive protocol that resulted in exhaustion in 5 to 10 min. Expired air was collected in sequential Douglas bags during the latter 2 to 3 min of submaximal or peak exercise. Respiratory gas concentrations were measured on a Perkin-Elmer 1100 mass spectrometer and ventilatory volumes on a Tissot spirometer. Peak $\dot{V}O_2$ was defined as the highest $\dot{V}O_2$ attained during progressive exhaustive exercise. The respiratory exchange ratio was defined as carbon dioxide production/$\dot{V}O_2$.

CO, heart rate, stroke volume, and blood pressure. CO was determined at rest and during the terminal 15 sec of submaximal or peak exercise by the acetylene (C_2H_2) rebreathing technique.[3] Briefly, the subjects rebreathed a mixture of 0.5% C_2H_2, 10% helium, and 35% oxygen in nitrogen from a closed system anesthesia bag while the respiratory gas C_2H_2 and helium concentrations were continuously monitored by a mass spectrometer interfaced to an LSI-11 computer for storage and processing of the data. CO was calculated from the exponential disappearance rate of C_2H_2 relative to helium in several sequential end expirations. The correlation coefficient between indicator dilution and C_2H_2 rebreathing estimates of CO at rest and during exercise is .94 in our laboratory. The SEM difference is 0.53 liter (4.7%) and is not significantly influenced by the level of CO.[3]

The Frank lead electrocardiogram was monitored continuously by oscilloscope throughout exercise. Indirect arterial blood pressure was measured at rest and during the latter 30 sec of each exercise stage or work bout with a semiautomatic sphygmomanometer equipped with a sound microphone (Narco Biosystems electrosphygmomanometer model PE-300). Concurrent blood pressure, CO, and electrocardiographic data were recorded on a Siemens Mingograf 800 recorder. Systolic and diastolic blood pressure (SBP and DBP) were defined by recorded sound deflections representing Korotkoff phases I and IV, respectively, during simultaneous graphic display of the sphygmomanometer pressure decline. Heart rate (HR) was determined from 10 consecutive RR intervals on the electrocardiogram. Mean blood pressure (MBP) was calculated as (SBP + 2DBP)/3, total peripheral resistance (TPR) as (MBP/CO) × 80 (expressed in dyne-sec-cm^{-5}), stroke volume (SV) as CO/HR, stroke work as (SV × MBP) × 0.0136 (expressed in grammeters), and arteriovenous oxygen difference as $\dot{V}O_2$/CO (expressed in vol%).

Multigated radionuclide ventriculography. A nongeometric equilibrium blood pool scintigraphic technique was used to estimate left ventricular volumes at rest, at two levels of submaximal exercise, and at peak exercise in the supine posture.[4] After red blood cell labeling in vivo with technetium-99m sodium pertechnetate, scintigraphic images were acquired in a modified 35 degree left anterior oblique projection with a standard gamma scintillation camera (Ohio Nuclear Series 100) equipped with an all-purpose parallel-hole collimator. The data were processed by a dedicated on-line computer system (Ohio Nuclear VIP 450) interfaced to the scintillation camera. A detailed description of this technique has been published previously.[4] The correlation coefficient between angiographic and scintigraphic estimates of left ventricular volume is .99 at both end-diastole and end-systole. The standard error of these estimations is 16 and 15 ml, respectively, at end-diastolic volumes ranging from 85 to 768 ml.

Conductance. Conductance, defined as local blood flow/mean blood pressure, was measured in the calf by venous occlusion plethysmography under resting conditions and after ischemic exercise to fatigue. The plethysmographic system, which has been described in detail elsewhere,[5] consisted of an air-filled rubber cuff placed around the calf at its greatest circumference and inflated to 4 cm H_2O. It was connected in series to a Statham pressure transducer and thence to a physiologic recorder. Limb volume changes produced proportional changes in cuff pressure. An occlusion cuff around the ankle was inflated to 300 mm Hg to isolate hemodynamically the foot from the system. Venous occlusion was established by inflation of a proximal occlusion cuff around the thigh to 50 mm Hg at rest and 90 mm Hg after ischemic exercise to fatigue. Maximal calf conductance was elicited by ischemic exercise. The proximal occlusion cuff was inflated to 300 to 400 mm Hg. Alternate heel and toe raise exercise was performed in the upright posture to an end point of severe voluntary fatigue. Hyperemic calf blood flow under these conditions is more than twice that observed after 5 min of ischemia alone.[5] Furthermore, hyperemic flow remains at a plateau for 60 to 90 sec, allowing five to 10 measurements to be made, enhancing reproducibility. In contrast, the situation after occlusive ischemia alone is characterized by a precipitous decline in hyperemic flow such that measurements are critically dependent on the time after reinstitution of perfusion.[5, 6] Maximal hyperemic flow values from two to three trials were averaged in the calculation of results. Conductance was determined from simultaneous calf blood flow and blood pressure data.

To assess whether the change in maximal calf blood flow could be explained by loss of fat tissue that may have a lower capacity for vasodilatation than skeletal muscle, percent lean tissue cross-sectional area in the calf was estimated in five subjects before and after training. Calf circumference (c) was measured with a flexible tape at an identical site on each occasion. The skinfold thickness of the anterior, posterior, medial, and lateral aspects of the calf was determined at the same point with a Lang caliper. Total and lean tissue cross-sectional areas (A) were then calculated from the formula $A = \pi r^2$, where $r = c/2\pi$. The radius for lean tissue area was determined by subtraction of mean skinfold thickness (four-position arithmetic average) from the total calf radius. Percent lean tissue cross-sectional area was the quotient of lean and total tissue areas × 100%. These calculations were based on assumptions that tissue cross-sectional area was circular and that skinfold thickness was proportional to local fat tissue cross-sectional area.

Statistics. Statistically significant differences before and after training were determined by Student's t test for paired observations. Relationships between physiologic variables were evaluated by linear regression analysis. Results were expressed as the mean ± SD.

Results

Oxygen uptake. Peak $\dot{V}O_2$ was slightly higher in the upright than in the supine posture before training (2.36 ± 0.42 vs 2.09 ± 0.29 liters/min; $p < .002$). Swimming resulted in an improvement in bicycle exercise capacity in all 12 subjects. The mean increase in peak upright $\dot{V}O_2$ was 16% (2.74 ± 0.52 vs 2.36 ± 0.42 liters/min; $p < .001$) whereas peak supine $\dot{V}O_2$ improved 10% (2.29 ± 0.30 vs 2.09 ± 0.29 liters/min; $p < .005$). A slight decline in body weight was not statistically significant (79.4 ± 10.8 vs 81.7 ± 11.6 kg). The rise in weight-corrected peak upright $\dot{V}O_2$ was 19% (34.4 ± 6.9 vs 28.9 ± 5.8 ml/kg/min; $p < .001$).

Weight-corrected peak $\dot{V}O_2$ measurements were similar in the male and female experimental subgroups at the control study (28.7 ± 9.7 vs 29.4 ± 4.4 ml/kg/min during upright and 27.4 ± 9.0 vs 25.7 ± 3.3 ml/kg/min during supine peak exercise). The degree of improvement was also similar with training-induced increases in peak $\dot{V}O_2$ of 17% and 19%, respectively, during upright and 9% and 12%, respectively, during supine exercise.

The significant rise in peak $\dot{V}O_2$ was associated with lower peak heart rate after training, providing supportive evidence that changes in peak $\dot{V}O_2$ were related to a training effect rather than to motivational factors (figure 1).

Respiratory exchange ratio (RER). RER data were also consistent with the attainment of peak $\dot{V}O_2$ during both tests in each posture. In the upright position, peak RER was 1.12 ± 0.08 before and 1.16 ± 0.07 after training (NS). Results in the supine position were 1.16 ± 0.08 and 1.15 ± 0.09, respectively (NS).

Calf conductance and blood flow. A trend toward an increase in resting calf conductance after training did not reach statistical significance (table 1). Similar results were observed for resting calf blood flow (table 1). Ischemic exercise to fatigue resulted in a 25-fold rise in calf blood flow that remained at the plateau value for nearly 90 sec both before and after training (figure 2). Maximal calf conductance (ml/100 ml tissue/min/mm Hg) increased 20% from 0.66 ± 0.19 to 0.79 ± 0.19 ($p < .02$) after conditioning, resulting in a higher peak calf blood flow (76.0 ± 16.3 vs 64.4 ± 16.6 ml/100 ml tissue/min; $p = .05$) at a slightly but

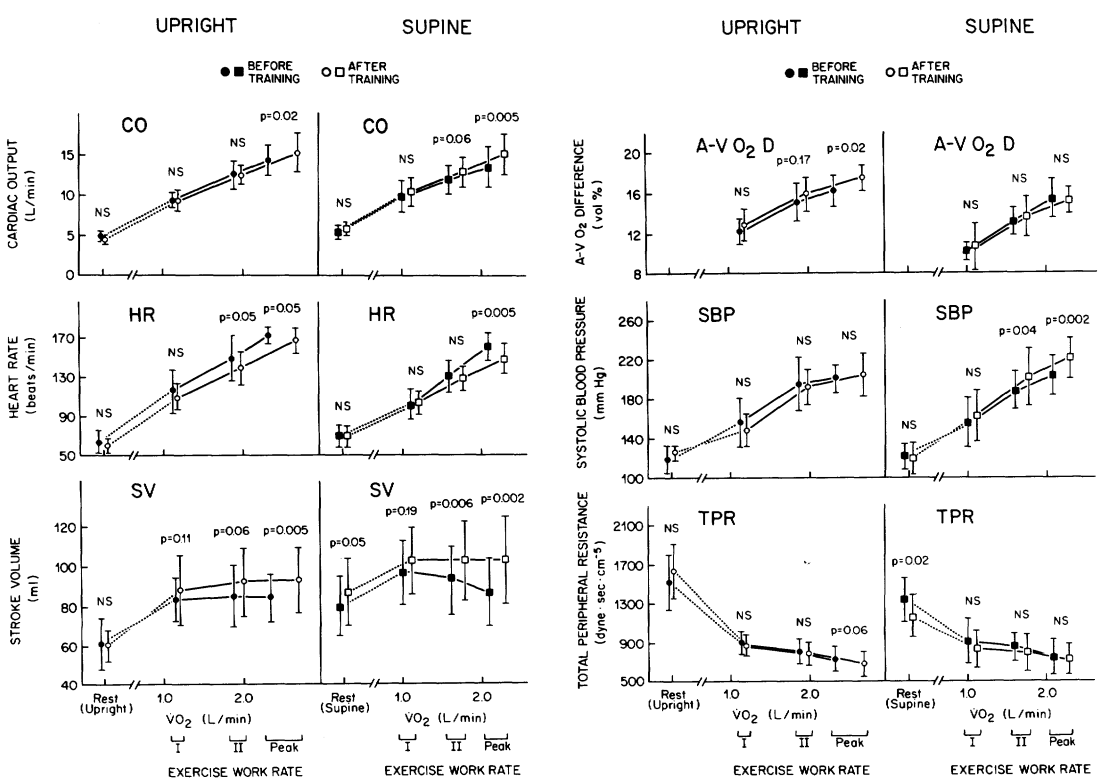

FIGURE 1. Effect of swim training on CO, heart rate (HR), stroke volume (SV), arteriovenous O_2 difference (A-V O_2 D), SBP, and TPR at rest and during submaximal and peak bicycle exercise in the upright and supine postures. Exercise intensity is expressed in terms of $\dot{V}O_2$. Training-induced differences in $\dot{V}O_2$ were significant only at the peak upright ($p < .001$) and peak supine ($p < .005$) levels. p values refer to statistically significant differences between hemodynamic measurements before and after training.

TABLE 1
Resting hemodynamic data for 12 experimental subjects (aged 38 ± 7 years) tested in the upright and supine posture before and after swim training

	Upright		Supine	
	Before training	After training	Before training	After training
Cardiac output (liters/min)	4.7 ± 0.7	4.5 ± 0.6	5.5 ± 0.9	5.9 ± 0.9[B]
Heart rate (beats/min)	76 ± 11	78 ± 10	69 ± 11[B]	69 ± 11[B]
Stroke volume (ml)	63 ± 13	59 ± 9	80 ± 15[B]	87 ± 17[A,B]
Blood pressure (mm Hg)	119 ± 15	126 ± 8	123 ± 13	118 ± 15
	76 ± 12	76 ± 16	74 ± 12	67 ± 10
Calf blood flow (ml/100 ml tissue/min)			2.58 ± 0.99	3.12 ± 1.43
Calf conductance (ml/100 ml tissue/min/mm Hg)			0.027 ± 0.012	0.036 ± 0.016

[A]p = .05 after vs before training; [B]p < .05 supine vs upright.

not significantly lower mean blood pressure (98 ± 12 vs 100 ± 10 mm Hg; NS) (figure 3). The relationship between maximal calf conductance and peak upright $\dot{V}O_2$ was statistically significant ($p < .01$) after but not before training (figure 4).

The heart rate and blood pressure data during ischemic exercise indicate that the improvement in maximal calf conductance was not related to greater subject effort. Mean heart rate during this procedure was 74 ± 11 beats/min before and 71 ± 11 beats/min after training (NS). Blood pressure did not change.

Variability for maximal calf conductance was similar in magnitude to that reported by Siggaard-Anderson[6] for air plethysmography. The coefficient of variation, defined as (SD/mean) × 100% was 9.4% for trials in the same subjects on the same day.

Aquatic training resulted in a slight decline in total calf tissue cross-sectional area (108.4 ± 7.6 vs 118.8 ± 8.7 cm^2; $p < .02$) but no change in lean tissue area. Therefore percent lean tissue cross-sectional area increased slightly from 69.1 ± 12.2 to 72.7 ± 11.7 ($p < .05$). In the same subjects, maximal hyperemic calf blood flow and maximal conductance increased markedly (29% and 24%, respectively; both $p < .05$). There was no significant relationship between percent change in lean calf tissue cross-sectional area and either maximal hyperemic calf blood flow or maximal conductance.

Resting systemic hemodynamics. Resting CO was similar in the seated and supine postures before training (table 1) but supine stroke volume was higher and heart rate slightly lower. Training resulted in a significant increase in supine stroke volume, a trend toward a decline in blood pressure, and a significant fall in TPR in the supine position. There were no significant training-induced changes at rest in the upright position (figure 1 and table 1).

Exercise systemic hemodynamics. Exercise CO results are shown in figure 1. There were no postural differences in peak CO before training. The CO vs $\dot{V}O_2$ relationship was not affected by conditioning; however, peak CO increased 10% and 7% in the supine and

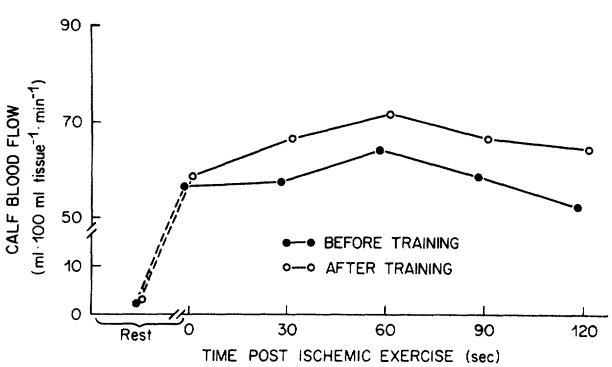

FIGURE 2. Calf blood flow at rest and at various times after ischemic exercise to fatigue.

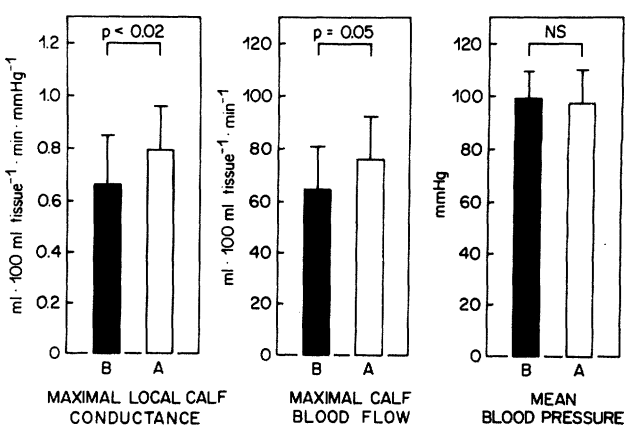

FIGURE 3. Effect of swim training on maximal local calf conductance, blood flow, and simultaneously determined mean blood pressure during hyperemia after ischemic lower leg exercise to fatigue. Statistically significant differences between measurements before (B) and after (A) training are indicated by p values.

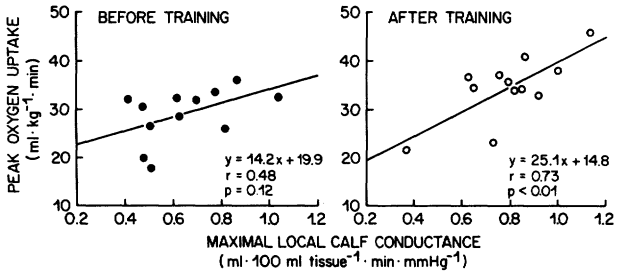

FIGURE 4. Correlation between maximal local calf conductance and upright peak $\dot{V}O_2$ in the same subjects studied before and after swim training.

upright postures, respectively ($p = .005$ and $p = .02$).

Before training, peak heart rate was 172 ± 9 beats/min during upright exercise vs 160 ± 13 beats/min during recumbency ($p < .05$). Aquatic training resulted in a reduction in peak heart rate that was particularly pronounced in the supine position (148 ± 13 vs 160 ± 13 beats/min; $p = .005$) but that was also observed with upright exercise (167 ± 12 vs 172 ± 9 beats/min; $p = .05$).

Stroke volume was initially somewhat higher in the supine than in the upright posture, particularly during submaximal work (figure 1). The rise in peak CO with swimming was a result of augmented stroke volume. Differences in stroke volume after vs before training were related to exercise intensity. At peak exercise, stroke volume was 18% higher in the supine posture ($p = .002$) and 10% higher in the upright posture ($p = .005$) after conditioning.

The enhanced stroke volume and CO during peak supine bicycle exercise could not be explained by reduced afterload, measured either as SBP or TPR. Swim training resulted in an 8% greater peak supine SBP (221 ± 21 vs 204 ± 20 mm Hg; $p = .002$) (figure 1) whereas TPR under these conditions was similar before and after training. As a result, peak supine stroke work was 35% greater after training (183.4 ± 32.8 vs 135.9 ± 27.7 g-m; $p < .002$). There were no significant changes in peak upright SBP (206 ± 22 vs 203 ± 28 mm Hg; NS), stroke work (157.6 ± 33.3 vs 144.5 ± 20.9 g-m; NS), or TPR (figure 1).

The improvement in peak $\dot{V}O_2$ in the supine position was related exclusively to augmented CO, whereas rises in peak CO and arteriovenous oxygen difference contributed equally (each approximately 8%) to the enhanced peak $\dot{V}O_2$ in the upright posture (figure 1).

Multigated radionuclide ventriculography. The greater supine resting stroke volume after swim training resulted entirely from an increased left ventricular end-diastolic volume index (LVEDVI) ($p = .01$; figure 5). There was a minimal rise in resting left ventricular end-systolic volume index (LVESVI), a slight fall in SBP/LVESVI (figure 6), and no change in left ventricular ejection fraction (figure 5). Identical 18% improvements were observed in stroke volume measured at peak supine exercise by C_2H_2 rebreathing and LVEDVI measured during submaximal and peak supine exercise by multigated radionuclide ventriculography (figures 1 and 5). Both before and after training, there was a slight rise in LVEDVI as exercise intensity and heart rate increased. During peak supine exercise, the LVESVI, ejection fraction, and SBP/LVESVI were all similar before and after training despite the 8% greater peak SBP and 18% larger stroke volume and LVEDVI after training.

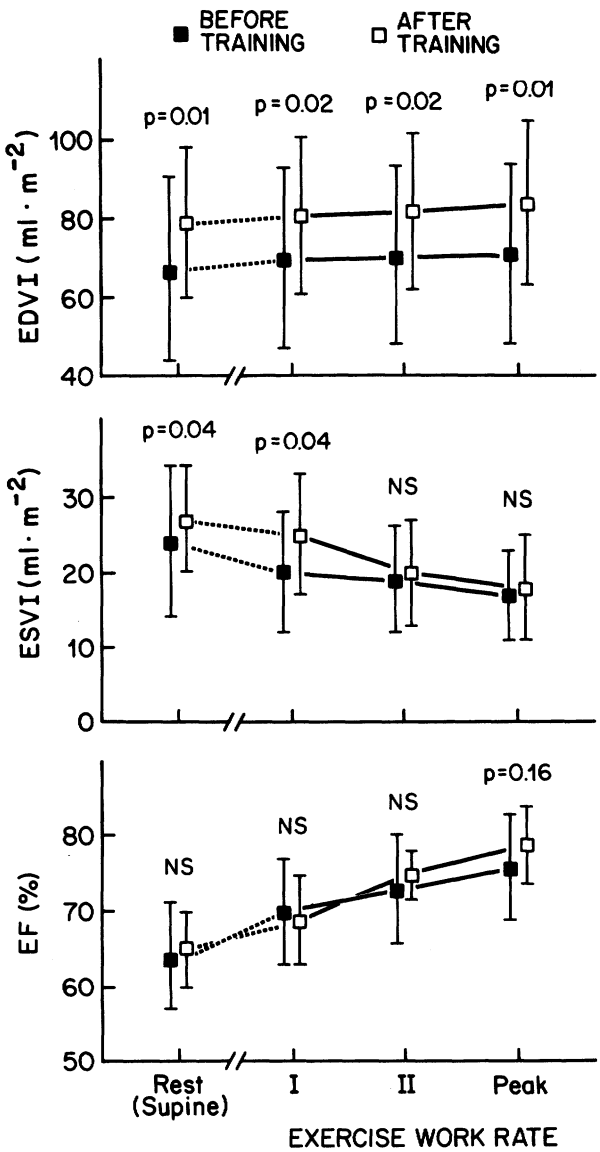

FIGURE 5. Effect of swim training on the left ventricular end-diastolic volume index (EDVI), end-systolic volume index (ESVI), and ejection fraction (EF) at rest and during submaximal and peak bicycle exercise in the supine posture. p values refer to statistically significant differences between measurements before and after training.

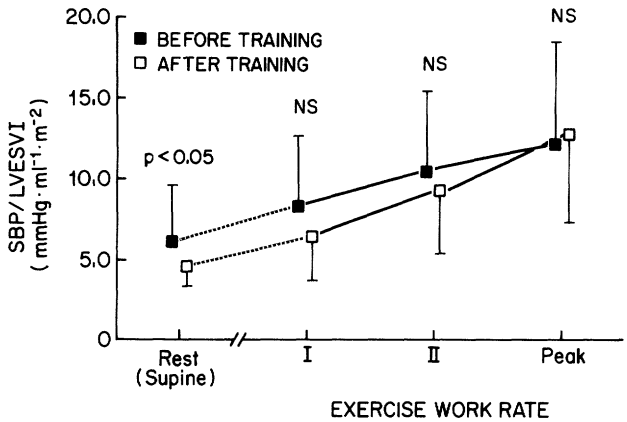

FIGURE 6. Effect of swim training on the systolic blood pressure to end-systolic volume index ratio (SBP/LVESVI) at rest and during submaximal and peak bicycle exercise in the supine posture. p values indicate statistically significant differences between pre- and posttraining measurements.

Control subjects. Data obtained from the control subjects, who were tested on two occasions 12 weeks apart, are shown in table 2. Peak $\dot{V}O_2$ and CO in the upright posture were not significantly different in the two studies. Likewise, there were no changes in cardiac volume or peak supine SBP. Resting and maximal calf conductance and blood flow also remained similar in the absence of training.

Discussion

A major objective of the study was to determine the extent to which physiologic effects of swim training are transferable to other forms of exercise. Magel et al.[7] were unable to show an improvement in treadmill maximal $\dot{V}O_2$ despite an 11% increase in swimming peak $\dot{V}O_2$ in college-age men. In contrast, our posttraining data showed a significant increase in peak $\dot{V}O_2$ measured during bicycle exercise. The change was equal in magnitude (about 20%) to that reported by other investigators for middle-aged men after walking or running programs of similar or longer duration.[8–11]

The specific reasons for the differences between our results and those reported by Magel et al.[7] are uncertain. However, the two studies differed in many respects. The subjects studied by Magel et al.[7] were younger (mean age 21 ± 3 years) and fitter (treadmill peak $\dot{V}O_2$ 55 ± 6 ml/kg/min) and were skilled swimmers before training.

Our program included a significant weight training component but an improvement in peak $\dot{V}O_2$ of this magnitude would not be expected from a regimen limited to weight training.[12, 13] Hurley et al.[13] examined the effects of a 16 week period of high-intensity weight training in a group of men with a mean age of 44 years. Strength training of an intensity sufficient to raise heart rate to an average of 155 beats/min and to increase resting plasma norepinephrine and lactate concentrations by factors of 7 and 16, respectively, produced an increase of lower body strength by 33% but maximal $\dot{V}O_2$ (treadmill) did not change.

Thus swimming appears to enhance maximal aerobic power, at least in middle-aged, previously sedentary individuals. The safety of training based on swimming was also verified. None of the participants suffered from musculoskeletal problems during training, whereas conventional programs of similar intensity and duration may cause injuries in almost 50% of the subjects.[14]

Physical training produces a broad range of central and peripheral adaptations.[15–20] The present study focused on the properties of the peripheral vasculature and on cardiac pump performance. Previous studies have provided strong indirect evidence that training affects the peripheral vasculature at the arteriolar level.[16, 18] Clausen[16] has demonstrated that there is a curvilinear and inverse relationship between maximal $\dot{V}O_2$

TABLE 2
Selected rest and exercise data in five nonexercising control subjects (age 38 ± 8 yr) tested 12 weeks apart in the upright or supine posture

Study conditions	Initial	Final
Rest		
Cardiac output (liters/min)	4.5 ± 0.8	4.2 ± 1.0
Stroke volume (ml)	53 ± 14	53 ± 15
Calf blood flow (ml/100 ml tissue/min)[A]	2.38 ± 0.98	3.00 ± 1.30
Calf conductance (ml/100 ml tissue/min/mm Hg)[A]	0.027 ± 0.013	0.035 ± 0.019
Peak exercise		
$\dot{V}O_2$ (ml/kg/min)	26.9 ± 8.2	25.8 ± 6.7
RER	1.19 ± 0.07	1.15 ± 0.07
Cardiac output (liters/min)	15.8 ± 5.0	14.9 ± 4.1
Stroke volume (ml)	86 ± 26	86 ± 20
Arteriovenous O_2 difference (ml/100 ml)	15.2 ± 3.3	15.8 ± 3.1
End-diastolic volume index (ml/m²)[A]	57 ± 12	54 ± 14
Left ventricular ejection fraction (%)[A]	78 ± 5	77 ± 4
Systolic blood pressure (mm Hg)[A]	180 ± 17	183 ± 39
Ischemic leg exercise		
Maximal hyperemic calf blood flow (ml/100 ml tissue min)[A]	62.5 ± 18.0	62.7 ± 15.7
Maximal calf conductance (ml/100 ml tissue/min/mm Hg)[A]	0.61 ± 0.21	0.69 ± 0.20

[A]Obtained in the supine posture.

and systemic resistance during maximal treadmill or bicycle work.

Total peripheral resistance has multiple determinants. At least three different mechanisms may contribute to the lower resistance in fit subjects: anatomic vascular adaptations, attenuation of systemic adrenergic vasoconstrictor drive, and enhancement of metabolic vasodilator drive in active skeletal muscle. Significant adrenergic vasoconstrictor activity is present in skeletal muscle during maximal work if the activity involves a large muscle mass and produces a systemic oxygen uptake approaching maximal oxygen uptake,[16, 18, 20, 21] but the techniques we used to measure maximal calf conductance are likely to produce maximal arteriolar dilatation in skeletal muscle.[5, 6, 22] Maximal conductance is then primarily a function of the size of the arteriolar bed. We have previously demonstrated in cross-sectional studies that a significant linear relationship exists between systemic peak oxygen uptake and maximal calf conductance and that this relationship is enhanced in subjects with a high peak $\dot{V}O_2$. The data from the present study confirm and extend these observations. Training produced a large increase in maximal conductance that cannot be explained simply by a loss of subcutaneous fat and an increase in the lean tissue fraction of the leg. The correlation between systemic peak $\dot{V}O_2$ and maximal calf conductance was also markedly improved by conditioning. However, training-induced changes in total peripheral resistance during peak upright maximal work only approached statistical significance ($p = .06$) and were absent during supine exercise. These data indicate that anatomic vascular adaptations may develop quite rapidly but that their impact on systemic hemodynamics may be limited after a short-term training program at least during activities involving a large muscle mass. Changes in the regulatory mechanisms that control systemic hemodynamics may evolve more slowly than the anatomic adaptations in the peripheral vasculature. The relative contributions of training-induced changes in the anatomic and regulatory mechanisms remain to be defined. Early studies based on the ^{133}Xe clearance technique demonstrated a paradoxical decrease or no effect of training on muscle blood flow even during maximal exercise.[23, 24] It is now apparent that the ^{133}Xe technique often underestimates muscle blood flow.[20, 25–27] Leg blood flow measured by indicator dilution techniques is proportional to leg muscle $\dot{V}O_2$.[20, 27]

The principal changes in our systemic hemodynamic data were increases in stroke volume at supine rest and during submaximal and peak exercise in both positions. Peak CO during supine and upright exercise was also increased. Supine resting stroke volume increased despite evidence for a lower inotropic state, manifest as a decrease in left ventricular systolic pressure/volume ratio and presumably reflecting a reduced level of β-adrenergic cardiac stimulation. Stroke volume data obtained during exercise in the supine position showed close agreement between two independent methods, i.e., radionuclide angiography and the acetylene rebreathing technique. In general, the increase in maximal $\dot{V}O_2$ after training may be attributed to an increase in maximal stroke volume and CO, to an increased systemic arteriovenous oxygen difference, or to a combination of both. An increased arteriovenous oxygen difference has been a consistent finding in longitudinal studies of sedentary young men and patients with ischemic heart disease but not in women or older men.[18] Our findings during peak upright exercise indicated that increases in CO and systemic arteriovenous oxygen difference contributed equally to the improvement in $\dot{V}O_2$.

Few training studies have been conducted in swimmers. Rerych et al.[28] reported a large improvement in peak CO, stroke volume, and left ventricular end-diastolic volume as measured by first-pass radionuclide ventriculography during upright bicycle exercise in a group of varsity college swimmers. Cross-sectional studies in young competitive swimmers have also shown them to have a high maximal $\dot{V}O_2$, stroke volume, and CO.[29, 30] To our knowledge, there are no studies characterizing the cardiovascular adaptations to aquatic training in middle-aged subjects.

An increased cardiac pump capacity after training, manifest as an increased peak CO, may be the result of central or peripheral adaptations or a combination of both.[18] Peripheral adaptations may enhance cardiac pump performance by increasing preload or decreasing afterload. The heart itself may adapt by an increase in myocardial mass and dimensions and/or by an improvement in its intrinsic contractile performance.[18, 31]

Cross-sectional studies have generally shown significantly larger cardiac dimensions in trained than in untrained subjects.[18, 31] Longitudinal studies in human subjects have provided more variable results.[31–34] Experimental studies in various animal species have also generated conflicting results regarding the effect of physical training on heart size and on intrinsic myocardial function.[18, 31] The most convincing evidence for a training-induced effect on the intrinsic properties of the myocardium has been derived from a long series of studies by Scheuer and his colleagues, recently reviewed by Schaible and Scheuer.[31] Alterations in myo-

cardial cell contractile proteins and/or the calcium regulatory system may account for findings of enhanced contractility.

The most striking changes in our subjects were an 18% increase in peak left ventricular end-diastolic volume and an 8% rise in peak systolic blood pressure during supine exercise without any alterations in ejection fraction, end-systolic volume, or end-systolic pressure/volume ratio. The combined ventriculographic and hemodynamic data clearly indicate an enhanced cardiac pump capacity after training, which is at least partially attributable to the greater left ventricular end-diastolic volume. Maintenance of an unchanged ejection fraction and end-systolic volume in the presence of an increased systolic pressure may be interpreted as evidence for improved contractile performance. However, the systolic pressure/volume ratio did not change significantly and the observed changes in systolic function may reflect an increase in myocardial mass rather than a change in the intrinsic myocardial contractile state.

The training-induced decrease in peripheral resistance during peak upright exercise only approached significance, suggesting that cardiac adaptations primarily accounted for the increase in peak CO. However, the increase in leg conductance (which translates into potential for a higher skeletal muscle blood flow at any given arterial pressure) and the increased systemic arteriovenous oxygen difference during peak upright exercise are strong indications that the training program also produced significant peripheral adaptations.

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