Effect of fitness on arm vascular and metabolic responses to upper body exercise

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Volianitis, S., C. C. Yoshiga, P. Nissen, and N. H. Secher. Effect of fitness on arm vascular and metabolic responses to upper body exercise. Am J Physiol Heart Circ Physiol 286: H1736–H1741, 2004.—We investigated arm perfusion and metabolism during upper body exercise. Eight average, fit subjects and seven rowers, mean ± SE maximal oxygen uptake (V\textsubscript{O}\textsubscript{2 max}) 157 ± 7 and 223 ± 14 ml O\textsubscript{2} kg\textsuperscript{-0.7} min\textsuperscript{-1}, respectively, performed incremental arm cranking to exhaustion. Arm blood flow (ABF) was measured with thermodilution and arm muscle mass was estimated by dual-energy X-ray absorptiometry. During maximal arm cranking, pulmonary V\textsubscript{O}\textsubscript{2} was ~45% higher in the rowers compared with the untrained subjects and peak ABF was 6.44 ± 0.40 and 4.55 ± 0.26 l/min, respectively (P < 0.05). The arm muscle mass for the rowers and the untrained subjects was 3.5 ± 0.4 and 3.3 ± 0.1 kg, i.e., arm perfusion was 1.9 ± 0.2 and 1.4 ± 0.1 l blood·kg\textsuperscript{-1}·min\textsuperscript{-1}, respectively (P < 0.05). The arteriovenous O\textsubscript{2} difference was 156 ± 7 and 120 ± 8 ml/l, respectively, and arm V\textsubscript{O}2 was 0.98 ± 0.08 and 0.60 ± 0.04 l/min corresponding with 281 ± 22 and 181 ± 12 ml/kg, while arm O\textsubscript{2} conductive conductance was 49.9 ± 4.3 and 18.6 ± 3.2 ml·min\textsuperscript{-1}·mmHg\textsuperscript{-1}, respectively (P < 0.05). Also, lactate release in the rowers was almost three times higher than in the unretrained subjects (26.4 ± 7.9 vs. 9.5 ± 4.4 mmol/min, P < 0.05). The energy requirement of an ~50% larger arm work capacity after long-term arm endurance training is covered by an ~60% increase in aerobic metabolism and an almost tripling of the anaerobic capacity.

arm exercise; blood pressure; lactate; oxygen diffusion; oxygen uptake.

CHRONIC ENDURANCE EXERCISE induces central and peripheral changes that enhance cardiac output and maximal oxygen uptake (V\textsubscript{O}2 max) (9). The important contribution of peripheral circulatory changes to this adaptive response is demonstrated by the hyperbolic relationship between V\textsubscript{O}2 max and total vascular resistance both in cross-sectional and longitudinal studies (8). Because during maximal exercise ~85% of cardiac output is directed to the working skeletal muscles, it would be expected that the ~50% greater cardiac output after endurance training would result in an increase in muscle blood flow. Yet, although animal data support this assumption (19), it has been difficult to demonstrate a similar increase in muscle blood flow in trained humans (8). One explanation may be that a training-induced increase in muscle mass makes an estimate of blood flow per unit of muscle relatively stable. Another consideration is that leg muscle blood flow has been evaluated after relatively short training periods (18, 23).

In contrast to the legs, only specifically arm-trained subjects use their arms intensively. Thus arm cranking elicits a lower V\textsubscript{O}2 max than leg exercise with a typical ratio between the two values of ~0.7 (26). Conversely, in arm-trained subjects, including rowers, V\textsubscript{O}2 max during arm cranking reaches similar or even higher values than those obtained with their trained legs. During arm cranking at ~80% of V\textsubscript{O}2 max, an arm blood flow (ABF) of ~2.4 l/min is reported in the untrained subjects (1) and ~3.8 l/min in arm-trained subjects (16), suggesting that arm training does increase the blood flow capacity of the arms.

During exercise, vascular resistance is mediated by a competition between metabolic vasodilatation and sympathetic vasoconstriction (5) that tightly links peripheral vascular conductance, including skeletal muscles (21) and the brain (13), with the available cardiac output so that mean arterial pressure (MAP) is maintained or rises. Thus blood flow to the arms of untrained subjects (30, 31) and to the legs of one-leg-trained subjects is compromised when the active muscle mass is increased (18). However, it is not known whether similar cardiovascular adjustments occur after endurance training when such hemodynamic balance is established on the background of both an enhanced vascular conductance (27) due to increased capillary density (3) and an enhanced cardiac output.

The aim of this study was to determine peak arm perfusion, O\textsubscript{2} conductive conductance (D\textsubscript{O}2), and O\textsubscript{2} uptake during maximal upper body exercise in rowers and average, fit subjects to evaluate the range of local adaptive response to endurance training. Peak arm perfusion was calculated from the ABF and estimates of active muscle, whereas lactate release from the arms indicated the anaerobic contribution to the work performed. A secondary objective was to determine the effect of whole body exercise on the ABF of arm-trained subjects. We hypothesized that a comparison between rowers and untrained subjects would reveal an enhanced arm aerobic metabolic capacity as a result of both a greater arteriovenous O\textsubscript{2} difference (a-v O\textsubscript{2} diff) over the working arm and a larger ABF. A second hypothesis was that chronic endurance training might affect the balance between cardiac output and vascular conductance in a way that ABF is preserved during whole body exercise.

MATERIALS AND METHODS

Subjects. Fifteen men voluntarily participated in the investigation. Incremental cycle ergometry to exhaustion was performed...
from a work rate of 70 + 35-W increments. Eight subjects (157 ± 7 ml·kg⁻¹·min⁻¹) (15) had not been performing regular aerobic exercise (<5 h/wk) and seven competitive rowers (223 ± 14 ml·kg⁻¹·min⁻¹) were members of the Danish national team and had performed regular endurance arm training (>20 h/wk) for at least 4 yr. All subjects were asymptomatic for cardiovascular and respiratory disease, normotensive, and currently not taking prescription or over-the-counter medications. Written consent was provided by all subjects after being informed of the risks and discomforts associated with the experiment. Also, they were requested to abstain from caffeinated beverages, strenuous physical activity, and alcohol up to 24 h before the study. The study was in accordance with the Declaration of Helsinki for the use of human subjects in research and approved by the Copenhagen Ethics Committee (KF 01-314/97).

**Arm cranking.** The subjects were seated on a mechanically braked cycle ergometer (Monark, Stockholm, Sweden) modified for arm cranking. The seat’s height and distance from the ergometer were adjusted so that full extension of the arms was achieved at the horizontal shoulder-level position. The protocol consisted of several 5- to 6-min exercise periods, starting from an unloaded condition and increments of 35 W for every subsequent period interspaced by 5- to 10-min rest intervals. Subjects were asked to equally use both arms and maintain constant crank rate of 70 rpm. The protocol was terminated at the workload that the subjects failed to maintain for 5 min. Pulmonary ventilation and expired gas concentrations were measured by an Oxyscreen metabolic cart (model CPX/D; Medical Graphics, St. Paul, MN), and values were reported as the average of 15-s intervals.

**Combined arm and leg exercise.** After a recovery period of at least 15 min, the rowers performed an exercise trial of combined arm cranking and leg cycling (A + L) using another mechanically braked cycle ergometer (Monark) placed under the arm cranking ergometer. During A + L, the work intensity for arm exercise was 80% of the arm work capacity, whereas L at ~60% of the leg work capacity was added. All subjects reached their limits of exercise tolerance in 5–6 min during the A + L trial that required ~95% of a subject’s \( \dot{V}_{\text{O}_2, \text{max}} \). Subjects were allowed sufficient practice during preliminary testing to become familiar with the exercise modality.

**ABF, heart rate, and blood pressure.** A bolus thermodilution technique was used for ABF. Under local anesthesia, a pulmonary artery catheter (model 132F5; Baxter Healthcare, Irvine, CA) was introduced into vena basilica at the elbow and advanced so that the thermodilution catheter was lying in the axillary vein. Three milliliters of room-temperature saline boluses were infused, and the temperature change was monitored with an Explorer Cardiopulmonary Hemodynamic Monitor (Baxter) to ensure a monophasic thermodilution curve with an exponential decay. This bolus method provides similar ABF with that obtained by constant infusion of cooled saline (30).

During the first 3 min of each trial, measurements of ABF were made every 15 s. Blood samples were obtained during the fourth minute and, to ensure steady-state additional flow measurements, were made after sampling until the completion of the trial. Thus for each trial, ABF was expressed as the average of 10–12 separate measurements. Although no statistical difference was found between measurements taken before and after blood sampling, values from the first minute were not used in the calculation to ensure that flow had reached a steady state. Heart rate (HR) was measured with a Vantage NV pulse watch (Polar Electro OY; Kempele, Finland).

MAP was obtained from the arterial catheter connected to a monitoring kit (Baxter) positioned at the level of the heart with continuous infusion of isotonic saline (3 ml/h). Arm vascular conductance (AVC) was the ratio between ABF and MAP, assuming that the influence of muscle contractions on blood flow was the same at a given work load.

**Blood analyses.** Samples (3–10 ml) of arterial and venous blood were drawn anaerobically from the tip of the venous catheter and from a catheter placed in the radial artery of the same arm for blood-gas variables and metabolic analyses. Hemoglobin concentration ([Hb]), oxygen and carbon dioxide tensions (\( \text{PO}_2 \) and \( \text{PCO}_2 \)), oxygen saturation (\( \text{SO}_2 \)), and plasma lactate, glucose, potassium, sodium, and calcium were determined on an ABL blood gas analyzer (model 725; Radiometer, Copenhagen, Denmark). Blood gas measurements were made at 37°C and corrected to the temperature measured during the sampling period by the thermistor probe in the axillary vein. The a-v \( \text{D}_2 \) diff was calculated from the \( \text{O}_2 \) content in arterial (\( \text{CaO}_2 \)) and venous blood (\( \text{CV}_2 \)) derived from the sum of bound (1.39 × [Hb] × \( \text{O}_2 \) saturation) and dissolved \( \text{O}_2 \) (0.003 × \( \text{PO}_2 \)). Arm \( \dot{V}_{\text{O}_2} \) was the product of ABF and the a-v \( \text{D}_2 \) diff, whereas net uptake or release of the metabolites was the product of ABF and the a-v \( \text{D}_2 \) diff. The relative contribution of anaerobic glycolysis and oxidative phosphorylation to the ATP requirements was estimated from lactate release (3 mol ATP/mol lactate) and arm \( \dot{V}_{\text{O}_2} \) (6.5 mol ATP/mol \( \text{O}_2 \)), respectively.

**RESULTS**

**Arm cranking.** Peak work rate and \( \dot{V}_{\text{O}_2, \text{max}} \) for the rowers were ~50 and 45%, respectively, which was higher than the untrained subjects, and a whole body mechanical efficiency of ~17% was maintained (Table 2). The ABF increased linearly with work intensity (Fig. 1) with maximal values varying between 3.7 and 5.7 l/min for the untrained subjects and between 5.2 and 7.3 l/min for the rowers.

Estimated active muscle mass of the rowers was ~6% larger than for the untrained subjects (3.5 ± 0.4 and 3.3 ± 0.1 kg, respectively), i.e., there was an ~30% increase in perfusion.
(1.8 ± 0.2 and 1.4 ± 0.2 l/kg/min, respectively). The HR, MAP, and AVC increased linearly with exercise intensity in both groups (Fig. 2), but with HR and MAP having similar end points, AVC was ~35% higher in the rowers.

The SaO₂ was close to 97% over the entire range of workloads and with some hemococoncentration during exercise, the CaO₂ increased from ~20% at rest to ~22% at the highest workload. During maximal exercise, the axillary SvO₂ of the rowers was almost halved compared with that of the untrained subjects and contributed to an ~65% higher arm Vo₂, i.e., Do₂ was ~170% higher. Both ABF and arm Vo₂ were linearly related with workload (r = 0.99) and whole body Vo₂ (r = 0.97).

Although arterial and axillary venous blood pH decreased similarly, the H⁺ release was doubled in the rowers compared with the untrained subjects during maximal exercise (Table 3). The arterial lactate concentration in the rowers was ~35% lower but the lactate release was almost thrice compared with the untrained subjects during maximal exercise. The venous plasma glucose concentration was higher than the arterial concentration, a finding that, given the little difference between the plasma and intracellular values,
indicates a net glucose release from the arm in both groups during maximal exercise and suggests that the metabolic substrate was provided by muscle glycogen breakdown. During maximal exercise, the glycolytic contribution to the total energy requirements doubled from the resting value that was higher in the rowers (Table 2). Ionized calcium release was 2.5-fold higher, whereas the potassium release was more than three times higher in the rowers compared with the untrained subjects during maximal exercise (Table 3). In contrast, plasma sodium release did not change, whereas the potassium release was reduced when the legs were added to arm exercise.

**DISCUSSION**

During maximal arm cranking, in arm endurance-trained subjects, ABF was ~40% higher, whereas arm oxygen uptake was elevated by ~60% and lactate release by ~180% compared with the values established in untrained subjects, explaining why the rowers could perform ~50% more work. In agreement with findings by Ahlborg and Jensen-Urstad (1, 16), there was no significant difference in ABF or oxygen uptake between the arm-trained and untrained subjects during submaximal exercise at the same absolute workload. Also, during arm and leg exercise, there appears to be a limitation in ABF (and oxygen uptake) in arm-trained subjects as established for untrained subjects (30, 31).

A greater peak blood flow to the working muscles is anticipated as a consequence of the greater cardiac output after endurance training (8). Exceptionally high muscle blood flows should be evident in highly trained endurance athletes who have very high aerobic capacities (9). However, determination of muscle blood flow during maximal exercise has involved technical difficulties and/or methodological limitations. For example, muscle blood flow determined with xenon washout leads to an underestimate, especially at high flow rates (7). However, indirect evidence of increased vasodilatory capacity obtained by venous occlusion plethysmography (27) and increased leg blood flow obtained by dye indicator dilution after one-leg training (18) implies that endurance training does lead to a greater muscle blood flow capacity. Roca et al. (23) demonstrated an ~20% increase in leg blood flow after endurance training, but muscle mass was not evaluated, and the reported limb blood flow increase expressed as muscle perfusion was likely to be smaller.

The higher VO₂ arm in the rowers was the result of both a higher aerobic and anaerobic capacity. However, determination of muscle blood flow during maximal exercise has involved technical difficulties and/or methodological limitations. For example, muscle blood flow determined with xenon washout leads to an underestimate, especially at high flow rates (7). However, indirect evidence of increased vasodilatory capacity obtained by venous occlusion plethysmography (27) and increased leg blood flow obtained by dye indicator dilution after one-leg training (18) implies that endurance training does lead to a greater muscle blood flow capacity. Roca et al. (23) demonstrated an ~20% increase in leg blood flow after endurance training, but muscle mass was not evaluated, and the reported limb blood flow increase expressed as muscle perfusion was likely to be smaller.

The higher VO₂ arm in the rowers was the result of both a higher ABF and a larger O₂ extraction as indicated by the lower SvO₂. A lower pH could have contributed via the Bohr effect to a greater Hb unloading but during maximal exercise, it was similar in both groups. The low SvO₂ in the rowers is attributed to the lower PVo₂ and, therefore, a higher muscle DO₂ that is determined primarily by the capillary surface area (4).

The ~50% higher work capacity of the rowers is attributed to both a higher aerobic and anaerobic capacity. How-
Table 3. Blood variables for untrained and arm-trained subjects (rowers) at rest and during maximal arm cranking and combined arm and leg exercise

<table>
<thead>
<tr>
<th></th>
<th>Untrained</th>
<th></th>
<th>Rowers</th>
<th></th>
<th>A 80%</th>
<th>A 80% + L 60%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>A max</td>
<td>Rest</td>
<td>A max</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH Arterial</td>
<td>7.42±0.01</td>
<td>7.33±0.02</td>
<td>7.43±0.02</td>
<td>7.35±0.02</td>
<td>7.36±0.02</td>
<td>7.31±0.02†</td>
</tr>
<tr>
<td>Venous Arterial</td>
<td>7.35±0.02</td>
<td>7.20±0.01</td>
<td>7.39±0.02</td>
<td>7.19±0.01</td>
<td>7.27±0.02</td>
<td>7.16±0.02†</td>
</tr>
<tr>
<td>H+ release, nmol/min</td>
<td>2.6±0.2</td>
<td>74.2±3.4</td>
<td>1.0±0.1</td>
<td>128.2±5.7†</td>
<td>52.0±3.2</td>
<td>80.8±4.3†</td>
</tr>
<tr>
<td>Lac Arterial</td>
<td>1.2±0.1</td>
<td>11.8±1.1</td>
<td>0.5±0.01</td>
<td>7.8±1.1*</td>
<td>6.7±1.2</td>
<td>10.5±1.8†</td>
</tr>
<tr>
<td>Venous Lac</td>
<td>1.4±0.1</td>
<td>13.9±1.4</td>
<td>0.9±0.1</td>
<td>11.9±1.3*</td>
<td>8.5±1.6</td>
<td>14.7±2.1†</td>
</tr>
<tr>
<td>Release, mmol/min</td>
<td>0.0±0.0</td>
<td>9.5±0.4</td>
<td>0.1±0.0</td>
<td>26.4±1.1*</td>
<td>9.4±1.6</td>
<td>16.8±2.1†</td>
</tr>
<tr>
<td>Glu Arterial</td>
<td>5.2±0.3</td>
<td>4.6±0.1</td>
<td>5.1±0.1</td>
<td>4.4±0.1</td>
<td>4.5±0.2</td>
<td>4.1±0.2</td>
</tr>
<tr>
<td>Venous Glu</td>
<td>4.9±0.1</td>
<td>4.8±0.1</td>
<td>4.9±0.1</td>
<td>5.0±0.1</td>
<td>4.7±0.2</td>
<td>4.5±0.2</td>
</tr>
<tr>
<td>Release, mmol/min</td>
<td>−0.1±0.0</td>
<td>0.9±0.1</td>
<td>−0.1±0.0</td>
<td>3.9±0.3*</td>
<td>1.0±0.3</td>
<td>1.6±0.4</td>
</tr>
<tr>
<td>Lac energy contribution, % Ca2+</td>
<td>5±0</td>
<td>12±1</td>
<td>10±1</td>
<td>20±2*</td>
<td>11±1</td>
<td>22±1†</td>
</tr>
<tr>
<td>Arterial Lac</td>
<td>1.14±0.02</td>
<td>1.21±0.02</td>
<td>0.89±0.02</td>
<td>1.13±0.02*</td>
<td>1.00±0.02</td>
<td>1.01±0.02†</td>
</tr>
<tr>
<td>Venous Lac</td>
<td>1.29±0.01</td>
<td>1.31±0.02</td>
<td>1.21±0.02</td>
<td>1.30±0.02</td>
<td>1.25±0.02</td>
<td>1.32±0.02†</td>
</tr>
<tr>
<td>Release, mmol/min</td>
<td>0.02±0.01</td>
<td>0.46±0.06</td>
<td>0.09±0.00</td>
<td>1.09±0.12*</td>
<td>1.3±0.08</td>
<td>1.24±0.07</td>
</tr>
<tr>
<td>K+ Arterial</td>
<td>3.7±0.2</td>
<td>5.2±0.3</td>
<td>3.0±0.02</td>
<td>5.3±0.3</td>
<td>4.7±0.3</td>
<td>5.4±0.3†</td>
</tr>
<tr>
<td>Venous K+</td>
<td>4.5±0.1</td>
<td>5.8±0.3</td>
<td>4.9±0.02</td>
<td>6.6±0.3*</td>
<td>6.0±0.1</td>
<td>6.8±0.4</td>
</tr>
<tr>
<td>Release, mmol/min</td>
<td>0.3±0.0</td>
<td>2.7±1.2</td>
<td>0.5±0.04</td>
<td>8.4±1.0*</td>
<td>6.8±0.1</td>
<td>5.6±0.3†</td>
</tr>
<tr>
<td>Na+ Arterial</td>
<td>140±0</td>
<td>143±1</td>
<td>141±1</td>
<td>145±1</td>
<td>143±1</td>
<td>145±1</td>
</tr>
<tr>
<td>Venous Na+</td>
<td>140±1</td>
<td>144±1</td>
<td>138±1</td>
<td>146±1</td>
<td>144±1</td>
<td>147±1</td>
</tr>
<tr>
<td>Release, mmol/min</td>
<td>0±1</td>
<td>5±1</td>
<td>0±1</td>
<td>6±0</td>
<td>5±1</td>
<td>8±1†</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 8 untrained subjects and 7 rowers. Lac, lactate; Glu, glucose; Lac energy contribution, calculated ATP from lactate related to total ATP from lactate and oxidative phosphorylation. *Different from untrained and †different from A, P < 0.05.

ever, Pendergast et al. (22), on the basis of similar maximal exercise blood lactate concentration between arm-trained and untrained subjects, concluded that endurance training does not influence the anaerobic capacity of the arms. Yet, calculation on the basis of lactate release from the arms, demonstrates a three times greater glycolytic capacity in the rowers compared with untrained subjects. Such release was possible, because the rowers had a low arterial lactate concentration, suggesting an enhanced lactate clearance from other organs, e.g., the kidneys and the liver (20), less active muscles (25), and the brain (14).

Glucose release from the arm was probably due to rapid glycogenolysis at the onset of intense exercise, which with the glucose transported into the muscle may have exceeded the hexokinase reaction and resulted in a transient glucose efflux (17, 30, 31). Increase in ionized calcium, parallel to the drop in pH in both subject groups, reflects increased calcium release from albumin and depression of calcium reuptake by sarcoplasmic reticulum (6). In addition, a similar HR-systolic blood pressure product suggests that the load to the heart was comparable during maximal exercise.

Peak arm perfusion during maximal arm cranking, even in the rowers, is lower than the values reported for peak leg perfusion during maximal leg exercise (3). One likely factor responsible for this discrepancy is the lower perfusion pressure in the arms although the higher MAP observed during arm exercise compared with leg exercise would partially counterbalance this effect. On the venous side, we assume that the effect of the muscle pump is similarly supporting flow during arm and leg exercise.

The implication of the lower peak arm perfusion values is that during maximal whole body exercise, the cardiac output required to perfuse all active musculature is smaller than suggested by Andersen and Saltin (3). During whole body exercise in which ~30 kg of skeletal muscle are engaged, reduction in muscle blood flow is ~20% (25, 30, 31). Data from the A + L trial indicate that a blood flow reduction of similar magnitude also develops in endurance-trained subjects. This finding is in line with the observation of Klausen et al. (18) in which an increase in the active muscle mass limits the limb vascular conductance of one-leg-trained subjects to pretraining levels. It seems that after endurance training, an enhanced cardiac output allows hemodynamic balance at a higher blood flow level.

The effect of sympathetic outflow on the active muscle vasculature is not without controversy. Strange et al. (28) used neck suction to stimulate the carotid sinus baroreceptors and found little evidence for the effect of sympathoexcitation on leg blood flow during heavy exercise and more recent work (29) presents evidence of sympatholysis in which forearm blood flow is preserved even during heavy handgrip exercise. Equally, a restraint in cardiac output does not compromise brain blood flow during forearm exercise, but it does effect both middle cerebral artery velocity and leg blood flow during whole body exercise (12, 13, 21). Thus there is a balance established between the level of sympathetic activation and requirement for muscle blood flow (24).

In conclusion, endurance training results in peripheral adaptations that increase both the aerobic and anaerobic metabolic capacities reflected in the enhanced work capacity during arm
cranking. However, during maximal whole body exercise, these same adaptations render the elite athlete liable to similar perfusion limitations as observed in untrained subjects.

REFERENCES