Blood flow and muscle oxygen uptake at the onset and end of moderate and heavy dynamic forearm exercise

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Van Beekvelt, Mireille C. P., J. Kevin Shoemaker, Michael E. Tschakovsky, Maria T. E. Hopman, and Richard L. Hughson. Blood flow and muscle oxygen uptake at the onset and end of moderate and heavy dynamic forearm exercise. Am J Physiol Regulatory Integrative Comp Physiol 280: R1741–R1747, 2001.—We hypothesized that forearm blood flow (FBF) during moderate intensity dynamic exercise would meet the demands of the exercise and that postexercise FBF would quickly recover. In contrast, during heavy exercise, FBF would be inadequate causing a marked postexercise hyperemia and sustained increase in muscle oxygen uptake (VO2musc). Six subjects did forearm exercise (1-s contraction/relaxation, 1-s pause) for 5 min at 25 and 75% of peak workload. FBF was determined by Doppler ultrasound, and O2 extraction was estimated from venous blood samples. In moderate exercise, FBF and VO2musc increased within 2 min to steady state. Rapid recovery to baseline suggested adequate O2 supply during moderate exercise. In contrast, FBF was not adequate during heavy dynamic exercise. Immediately postexercise, there was an ~50% increase in FBF. Furthermore, we observed for the first time in the recovery period an increase in VO2musc above end-exercise values. During moderate exercise, O2 supply met requirements, but with heavy forearm exercise, inadequate O2 supply during exercise caused accumulation of a large O2 deficit that was repaid during recovery.

At heavier workloads, the tension developed in the muscle will probably totally occlude arterial inflow during contraction (4, 14, 27, 29). Whether the total blood flow during the limited window between contractions is adequate depends on the duration of the recovery pause and the metabolic demand of the exercise (15).

In the recovery period after exercise, the whole body VO2 recovers gradually to resting values. The term “O2 debt” has classically been used to describe this whole process that some investigators have called “excess postexercise O2 consumption” (EPOC) (22). For heavy exercise, the O2 debt exceeds the O2 deficit incurred at the onset of exercise (1, 2). With very short-lasting exhaustive exercise, di Prampero et al. (8) noted that VO2 measured at the mouth remained at the end-exercise level for 12–35 s after stopping. Bangsbo et al. (3) examined both the whole body and the muscle VO2 (VO2musc) at the onset of, and recovery from, exercise that induced exhaustion in ~3.2 min. Recovery values of VO2musc declined rapidly after exercise, but they still exceeded the calculated requirement to repay the O2 deficit (3). In studies of VO2musc in a dog muscle preparation, the magnitude of O2 debt was seen to be equivalent to O2 deficit across a wide range of metabolic demands (7).

There is reason to suspect that the exercise and recovery responses might be different in strenuous forearm exercise compared with leg exercise. Although the knee-extension exercise in the study of Bangsbo et al. (3) was at a high work rate, there was no evidence of an overshoot in the postexercise hyperemia. This contrasts with the response to dynamic (28) and isometric heavy forearm contractions (14, 17, 27, 29) where the blood flow is markedly impaired during contractions (14). None of these studies determined VO2musc. In this study, we examined the rate of change in VO2musc at the onset and end of exercise from continuous measurements of forearm blood flow (FBF) by Doppler ultrasound and repeated blood sampling to determine O2 extraction. We hypothesized that FBF during exercise would be inadequate during heavy but

DURING MODERATE RHYTHMIC EXERCISE of human forearm (12, 15, 26) or leg (28) muscles, the blood flow adapts to a steady value that is appropriate for the metabolic demand. Blood flow is greatly reduced or stopped during muscle contractions and is elevated during the muscle relaxation (15, 28). Over the complete muscle contraction/relaxation schedule, the perfusion pressure gradient and the vascular conductance dictate blood flow. At least for moderate work rates, the flow appears to be adequate as the muscle achieves a steady-state blood flow and rate of oxygen consumption (VO2) (10, 12), and on cessation of exercise, blood flow recovers quickly to the preexercise baseline (28).

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METHODS

Six healthy young subjects (4 males, 2 females) volunteered to take part in this study after receiving full written and verbal details of the experimental procedures and signing a consent form approved by the Office of Research Ethics of the University. The average (±SE) physical characteristics were age 27.8 ± 1.8 yr, height 180 ± 3 cm, and weight 71.2 ± 6.1 kg.

Experimental design. All testing took place in the supine position with the arm extended to be at heart level. The first test for all subjects was an incremental maximal test to exhaustion. The subjects raised and lowered a bucket that was being continuously filled with water at a rate of 1 kg/min, through a distance of 5 cm by squeezing a handgrip device. This weight was raised during a period of ~0.5 s and lowered during the next ~0.5 s with a 1-s rest pause between contractions. Thus the exercise was dynamic with no static component. The test began with a load of 1 kg and continued until the subjects were not able to lift the weight. The workloads for subsequent constant load testing were picked as 25 (2.4 ± 0.3 kg) and 75% (7.1 ± 0.9 kg) of the peak workload achieved during the incremental exercise (9.4 ± 1.2 kg).

The constant load tests were conducted as two trials on each of 2 separate days. The first trial of each day was at 25%, whereas the second trial was at 75% of the peak workload. At least 30 min separated the two tests on each day. Each trial consisted of a 5-min monitoring period at rest, with data collected over the final minute of this period, a 5-min period of exercise, and a 20-min recovery after the exercise. To eliminate anticipatory responses, the subjects were not aware of the time during any trial.

Data acquisition. FBF was measured beat by beat with combined pulsed Doppler ultrasound (model 500V, Multigon Industries) and echo Doppler imaging (model SSH-140A, Toshiba) (26). The mean blood velocity was digitally recorded at 100 Hz after analysis with a Doppler signal processor (26). The pulsed Doppler probe was a flat 4-MHz crystal with an angle of insonation fixed at 45° relative to the skin. The exact angle to the brachial artery from the skin was confirmed by echo Doppler imaging. Throughout one trial at each workload, the diameter of the brachial artery was obtained by Doppler imaging. Vessel diameter was measured three times at rest, at times centered on 10-s intervals during the first minute of exercise, then at 1-min intervals to the end of exercise. All measurements were obtained at the same point of the cardiac cycle at end diastole. From the measured diameter values, a curve of best fit was obtained in an attempt to minimize random variation and to allow calculation of diameter for each cardiac cycle. Instantaneous FBF was determined from the product of mean blood velocity over a cardiac cycle and the simultaneous cross-sectional area of the vessel.

Arterial blood pressure was monitored continuously with a finger-cuff photoplethysmograph device (Finapres, Ohmeda) placed on the middle finger of the nonexercising hand. The cuff was maintained at heart level. The analog signal was recorded with the other variables at 100 Hz. Mean arterial pressure was determined from 1/3 systolic + 2/3 diastolic blood pressure.

Blood sampling. A catheter (21-gauge, 1-in. Angiocath) was inserted into a vein draining from deep within the forearm. A three-way stopcock was attached to allow for frequent drawing of blood into heparinized 1-ml syringes for measurement of blood gases (ABL-30, Radiometer) and of blood lactate concentration by a fluorimetric method (21).

RESULTS

FBF. At the onset of 25% exercise, FBF increased rapidly (Fig. 1). One-way ANOVA plus post hoc tests detected a statistically significant difference from baseline after 1.5 min of exercise. A plateau was reached, with flow increasing about fourfold over the resting values. Immediately on cessation of exercise at the 25% load, there was a small, nonsignificant, increase in FBF above the end-exercise value only during the first 10-s sample period. FBF during recovery remained above the preexercise baseline for 1.25 min.

At the 75% exercise load, FBF increased significantly above baseline by 25 s and continued to increase throughout the exercise, reaching a peak value of ~400 ml/min compared with ~50 ml/min at rest. Immediately on cessation of exercise, FBF increased to more than 600 ml/min. The marked postexercise hyperemia was maintained for over 90 s at values greater than the end-exercise FBF (Fig. 1). The FBF remained much higher than FBF after 25% exercise through the first 10 min of recovery and was still significantly greater than the 25% tests as well as the preexercise baseline at the completion of recovery monitoring.
Throughout the 25% exercise test, there was a small increase in brachial artery diameter (Fig. 2), but the diameter was only significantly greater than baseline in the first minute after exercise. During the 75% test, brachial artery diameter increased significantly above baseline by 25 s, and it remained greater until the end of measurements at the completion of recovery monitoring. Also, immediately after the 75% exercise test, diameter was significantly elevated above the end-exercise value for the first 2 min of recovery.

\( a\text{-}vD\text{O}_2 \). During the first 2 min of exercise at the 75% load, the \( a\text{-}vD\text{O}_2 \) was greater than for the 25% load (Fig. 1). Over the final 3 min of exercise, there were no differences between the \( a\text{-}vD\text{O}_2 \) for the two exercise loads. The \( a\text{-}vD\text{O}_2 \) declined rapidly on cessation of exercise, with a return to preexercise baseline by 1.5 min in the 25% exercise load tests. In the 75% load tests, the \( a\text{-}vD\text{O}_2 \) declined below baseline, not returning to preexercise values until 15 min after the end of exercise.

\( \dot{V}_\text{O}_2\text{musc} \). In both the 25 and 75% exercise load tests, the \( \dot{V}_\text{O}_2\text{musc} \) was significantly elevated above baseline by 35 s of exercise. Furthermore, \( \dot{V}_\text{O}_2\text{musc} \) continued to increase over the first 2–3 min of exercise to an apparent plateau (Fig. 1). In the case of the 25% test, the \( \dot{V}_\text{O}_2\text{musc} \) was not different between the end-exercise value and that observed in the first samples in recovery. The \( \dot{V}_\text{O}_2\text{musc} \) was no longer significantly elevated above preexercise baseline after only 25 s of recovery in the 25% tests.

After the 75% exercise tests, the \( \dot{V}_\text{O}_2\text{musc} \) increased significantly above the end-exercise values at 10 and 20 s of recovery. \( \dot{V}_\text{O}_2\text{musc} \) remained elevated above baseline for the first minute of recovery.

Lactate concentration. The lactate concentration in venous blood increased from just under 1 mmol/l at rest to \( \sim 1.8 \text{ mmol/l} \) during the 25% exercise load test and to \( 3.2 \text{ mmol/l} \) during the 75% test. Return to preexercise levels was delayed until 15 min after exercise in both tests (Fig. 3).

Mean arterial blood pressure. The small increases in mean arterial blood pressure during the 25% exercise tests were not significantly different from the baseline

**Brachial artery diameter.** Throughout the 25% exercise test, there was a small increase in brachial artery diameter (Fig. 2), but the diameter was only significantly greater than baseline in the first minute after exercise. During the 75% test, brachial artery diameter increased significantly above baseline by 25 s, and it remained greater until the end of measurements at the completion of recovery monitoring. Also, immediately after the 75% exercise test, diameter was significantly elevated above the end-exercise value for the first 2 min of recovery.

\( a\text{-}vD\text{O}_2 \). During the first 2 min of exercise at the 75% load, the \( a\text{-}vD\text{O}_2 \) was greater than for the 25% load (Fig. 1). Over the final 3 min of exercise, there were no differences between the \( a\text{-}vD\text{O}_2 \) for the two exercise loads. The \( a\text{-}vD\text{O}_2 \) declined rapidly on cessation of exercise, with a return to preexercise baseline by 1.5 min in the 25% exercise load tests. In the 75% load tests, the \( a\text{-}vD\text{O}_2 \) declined below baseline, not returning to preexercise values until 15 min after the end of exercise.

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After the 75% exercise tests, the \( \dot{V}_\text{O}_2\text{musc} \) increased significantly above the end-exercise values at 10 and 20 s of recovery. \( \dot{V}_\text{O}_2\text{musc} \) remained elevated above baseline for the first minute of recovery.

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During the 75% exercise test, mean arterial pressure was significantly elevated above baseline by 25 s and did not return to baseline until 2 min after the end of exercise.

DISCUSSION

To the best of our knowledge, this is the first time that VO\textsubscript{2musc} has been observed to increase during the recovery period to values above those measured at the end of dynamic exercise. Consistent with our hypothesis, FBF during the heavy forearm exercise was inadequate for the demands of metabolism and waste removal. This was indicated by a marked and prolonged postexercise hyperemia. Our results differ from those presented by Bangsbo et al. (3) for knee-extension exercise that caused exhaustion in 3.2 min. They did not see an overshoot in blood flow, nor did they see an elevation in VO\textsubscript{2musc} after exercise. There were differences in experimental design that might account for the findings as considered below. The current results for the heavy exercise, but not for the moderate exercise, also differ from those reported for one set of experiments with dog muscle where the time course of VO\textsubscript{2musc} at the onset of exercise mirrored that in recovery (7). Our findings, similar to those of Bangsbo et al. (3), showed a marked asymmetry with a long recovery time for VO\textsubscript{2musc} after heavy exercise. Furthermore, our results are consistent with another set of animal experiments where the peak VO\textsubscript{2musc} was influenced by the type of muscle contraction such that insufficient time for perfusion restricted O\textsubscript{2} supply (4).

Critique of methodology. Combined pulsed and ech Doppler techniques allow for continuous beat-by-beat quantitative determination of FBF (26, 28). In this study, the immediate increase in FBF on cessation of heavy exercise was a function of removal of the high resistance to flow during the contractions. Indeed, we observed (data not shown) that the flow on each complete cardiac cycle that occurred between contractions was identical to that measured in the first 10–20 s of recovery. This confirms previous reports of the beat-by-beat variation in flow to an exercising muscle (15, 26, 28). The thermodilution method as used by Bangsbo et al. (3) is an intermittent approach to the study of muscle blood flow. These authors did not show data until ~30 s after the completion of their exercise challenge and may have missed important information about the magnitude of postexercise hyperemia. In our study, FBF was still markedly elevated above the mean end-exercise value at 30 s of recovery. These data suggest that the metabolic challenge of exercise relative to the muscle perfusion was probably greater in the current experiments than in those of Bangsbo et al. (3).

One factor that might contribute to the relatively greater flow deficit in the current experiments compared with the experiments of Bangsbo et al. (3) is the type of muscle contraction. In their experiments, the subjects performed knee-extension exercise at ~60 per minute. This required ~0.5 s for extension and a similar time for passive recovery. Our subjects performed a handgrip that required ~0.5 s to lift the weight and 0.5 s to lower the weight followed by a 1-s relaxation. That is, even though both exercises were dynamic with no static component and the duty cycle was similar in the two studies, the duration of a contraction was longer in our experiments. This might have been an important factor in restricting blood flow relative to metabolic demand.

In this study, as in our previous report (12), we did not believe we could justify insertion of an arterial catheter simply to confirm that saturation did not vary from 97%. Given the minimal change in central cardiovascular response, there is no reason to believe that arterial saturation might change as it does in some individuals during very heavy exercise (6). In the estimation of a-\text{vDO}_2, venous O\textsubscript{2} content is the greatest...
uncertainty in this study, because it is impossible to isolate the blood from only the exercising muscle and totally exclude the possibility of skin or nonworking muscle contributions with human subjects (5).

An additional concern with the calculation of \( V_{\text{O2musc}} \) by the product of blood flow (measured continuously) and a-vDO\(_2\) (measured intermittently) is the matching of these two variables. Blood was drawn over the final 5 s of each time period. Given the short distance from the exercising muscle to the sample site, this might be expected to be representative of the \( O_2 \) extraction at the midpoint of the 10-s windows that were analyzed in the first minute of exercise and recovery. However, it is appropriate to examine the outcome if the arrival of blood was delayed by one full sample period. It can be appreciated from Fig. 1 that for the 75% exercise challenge, the \( V_{\text{O2musc}} \) would still be elevated above the end-exercise value even if \( V_{\text{O2musc}} \) was calculated as the product of the FBF during the first 10-s period matched with the a-vDO\(_2\) of the second 10-s sample period. Such a large mismatch is unlikely, but even if it did occur, the outcome and interpretation of our data would not be changed.

**FBF and \( V_{\text{O2musc}} \).** Our observations of achievement of steady state for FBF and \( V_{\text{O2musc}} \) during the 25% exercise load are consistent with recent reports at the onset of moderate intensity arm (12) and leg exercise (10). In agreement with experiments that investigated the response of isolated dog muscle (7), our data showed that recovery \( V_{\text{O2musc}} \) had a similar time course to that at the onset of light-intensity exercise. Calculated \( O_2 \) debt (16.1 ± 1.6 ml \( O_2 \)) did not differ from the \( O_2 \) deficit incurred at the onset of exercise (15.3 ± 1.4 ml \( O_2 \)). There was a greater total FBF above baseline during recovery (206 ± 20 ml) than there was FBF deficit at the onset of exercise (112 ± 21 ml, \( P < 0.05 \)), suggesting that supply of \( O_2 \) was not the crucial regulatory factor.

Exercise at the 75% workload can be compared with the previous work of Bangsbo et al. (3), who investigated \( V_{\text{O2musc}} \) at the onset of and recovery from heavy knee-extension exercise. There are some important differences in the results. First, we observed a marked and sustained postexercise hyperemia, whereas Bangsbo et al. (3) did not. As considered above, this might have been a consequence of their intermittent method of measurement or differences between leg and arm exercise. Even though we employed dynamic muscle contractions with a 1-s pause between contractions, the pattern of FBF after 75% exercise resembled that after heavy isometric exercise (14, 17, 27, 29) where there is essentially no flow through the contracting muscle during contraction (14). A second major difference between our high-intensity exercise and that studied by Bangsbo et al. (3) was that \( V_{\text{O2musc}} \) increased in our study from ~50 ml/min during exercise to ~70 ml/min immediately after exercise. \( V_{\text{O2musc}} \) then declined slowly to baseline over the next 10–15 min. It was not possible to compute deficit and debt for FBF and \( V_{\text{O2musc}} \) because of uncertainty in the “steady-state” value during exercise. Exploration of muscle metabolism by magnetic resonance spectroscopy to determine intramuscular phosphocreatine and pH during the high-intensity exercise could provide important insight into metabolic control. Previous investigations of human forearm or leg muscles have shown progressive depletion of phosphocreatine as work rate increased, but it is not known how blood flow was affected in these studies (18, 30).

It is appropriate to examine whether our values of FBF and \( V_{\text{O2musc}} \) are realistic in terms of metabolic potential. In single leg knee-extension exercise, values of at least 250 ml·100 g\(^{-1}\)·min\(^{-1}\) have been observed for peak blood flow and 363 ml·kg\(^{-1}\)·min\(^{-1}\) for peak \( V_\text{O2} \) (23). We do not have magnetic resonance imaging data to provide an accurate assessment of active muscle mass in our subjects. If the active forearm muscle mass was less than 500 g (from an average total forearm volume of ~1,100 ml), then peak FBF would be ~150 ml·100 g\(^{-1}\)·min\(^{-1}\) and the \( V_\text{O2} \) peak would be ~140 ml·kg\(^{-1}\)·min\(^{-1}\). The values for peak blood flow for forearm exercise would be expected to be less than those for single leg ergometry for several reasons. The most important reason is that the peak blood flow occurred after stopping the forearm exercise, because even though mean arterial pressure was higher during exercise, each contraction caused vascular occlusion. Furthermore, with the arm at heart level rather than below, as with the leg exercise, perfusion pressure was lower in the arm exercise. It remains to be determined if forearm and leg blood flow responses could be equivalent if tested under similar conditions.

**\( O_2 \) extraction.** Calculated \( O_2 \) extraction (a-vDO\(_2\)) reflected the change in venous \( O_2 \) content. In the early phase of the 75% exercise load, there was greater \( O_2 \) extraction compared with the 25% load, suggesting a greater metabolic demand. Throughout the final 3 min of exercise, \( O_2 \) extraction was the same at the lower and higher intensities of exercise. This finding is consistent with the observations of others (3, 13, 16, 23) who have shown that venous \( O_2 \) content rarely drops below 50 ml/l during strenuous exercise of a small muscle mass. With intermittent handgrip exercise, blood flow occurs almost exclusively during the pause between contractions. Thus flow rate was quite high during the pause, and this might have contributed to reduced \( O_2 \) extraction.

On cessation of the 25% exercise load, the \( O_2 \) extraction returned to baseline values within 90 s and remained at this level throughout the remainder of the recovery period. In contrast, \( O_2 \) extraction during the recovery after the 75% exercise load declined to values below baseline within 30 s. Until the 15-min point of recovery, the \( O_2 \) extraction was less than baseline, indicating an excess of blood flow relative to \( O_2 \) requirement. The mechanism responsible for this hyperemia was at least in part related to the local metabolic state as evidenced by the sustained increase in venous blood lactate concentration. On the other hand, for the 25% exercise load, venous blood lactate remained elevated slightly above baseline values throughout the
first 5 min of recovery, whereas FBF returned to baseline.

Venous blood lactate. As expected, the increase in venous blood lactate from the forearm during the 25% exercise load was less than during the 75% exercise load. This observation is consistent with other suggestions of inadequate O₂ delivery during the heavier exercise. Venous lactate remained elevated at approximately the end-exercise value for the first 3–4 min of recovery after both the 25 and 75% exercise tasks. Given the very high FBF in the recovery after the 75% exercise task, this suggests a very high venous lactate outflow from muscle and interstitial sources.

Brachial artery diameter, blood pressure, and blood flow. During the 25% exercise load tests, there was only a small, nonsignificant, increase in brachial artery diameter so that the major contributor to the threefold increase in FBF was an increase in blood velocity. Immediately after the 25% exercise load, there was a small significant increase in diameter. The brachial artery diameter response to the 75% exercise load was an exaggeration of the response to the lower-intensity exercise.

It seems to us that the most likely explanation for the dilation of the brachial artery, a main conduit vessel supplying the exercising forearm, is an upstream transmission of dilatory stimuli released by the exercising muscles (24). In line with our previous observations (25), greater dilation occurred with the heavier work rate. Additional factors to consider include release of nitric oxide and/or prostacyclin in response to increases in blood flow velocity stimulating the endothelial cells via increased shear stress (11). Removal of sympathetic restraint that existed during the exercise (20) or a myogenic response to the decrease in arterial pressure after exercise (19) should also be considered as potential explanations for the conduit artery response.

The marked increase in arterial blood pressure during the 75% exercise load indicates that this type of exercise did induce a severe local strain. As with sustained isometric contractions, the 75% load caused at least relative muscle ischemia and the accumulation of metabolites that stimulated the muscle chemoreflex to bring about a sympathetic nervous system-mediated increase in vascular resistance. As indicated above, this sympathetic response might have influenced the conduit vessels as well as the resistance vessels (i.e., arterioles) that are further downstream.

Perspectives

Consistent with our hypotheses, we found that the blood flow response to moderate exercise was adequate to meet the metabolic demands with little need for a postexercise hyperemia. On the other hand, the metabolic demand of repeated higher-intensity contractions could not be met by oxidative mechanisms because of the major impairments to total blood flow during the muscular contraction. This has implications for many tasks in an industry that requires repeated application of high levels of muscular force (9). It is apparent from our data that major deficits can occur in blood flow and VO₂musc that will cause muscle fatigue with the potential to cause injury. Appropriate work-to-rest schedules could be developed based on the duration and intensity of the work task.

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