Effect of altered arterial perfusion pressure on vascular conductance and muscle blood flow dynamic response during exercise in humans

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Submitted 10 September 2012; accepted in final form 4 January 2013

Villar R, Hughson RL. Effect of altered arterial perfusion pressure on vascular conductance and muscle blood flow dynamic response during exercise in humans. J Appl Physiol 114: 620–627, 2013. First published January 10, 2013; doi:10.1152/japplphysiol.01094.2012.—Changes in vascular conductance (VC) are required to counter changes in muscle perfusion pressure (MPP) to maintain muscle blood flow (MBF) during exercise. We investigated the recruitment of VC as a function of peak VC measured in three body positions at two different work rates to test the hypothesis that adaptations in VC compensated changes in MPP at low-power output (LPO), but not at high-power output (HPO). Eleven healthy volunteers exercised at LPO and HPO (repeated plantar flexion contractions at 20–30% maximal voluntary contraction, respectively) in horizontal (HOR), 35° head-down tilt (HDT), and 45° head-up tilt (HUT). Muscle blood flow velocity and popliteal diameter were measured by ultrasound to determine MBF, and VC was estimated by dividing MBF flow by MPP. Peak VC was unaffected by body position. The rates of increase in MBF and VC were significantly faster in HUT and slower in HDT than HOR, and rates were faster in LPO than HPO. During LPO exercise, the increase in, and steady-state values of, MBF were less for HOR and HDT; the increase in VC was less in HUT than HOR and HDT. During HPO exercise, MBF in the HDT was reduced compared with HOR and HUT, even though VC reached 92% VC peak, which was greater than HOR, which was, in turn, greater than HUT. Reduced MBF during HPO exercise had the functional consequence of a significant increase in muscle electromyographic index, revealing the effects of MPP on O₂ delivery during exercise.

Doppler ultrasound; maximal vasodilatory capacity; electromyography

EXERCISING MUSCLES ARE SENSITIVE to changes in limb or body position due to the effects of gravity on muscle perfusion pressure (5). Exercise performed with the limb positioned above and/or below the heart level to alter arterial and venous pressure has been used to challenge muscle blood flow adaptations at the onset of exercise (15, 24, 32), steady state (16), or both (12, 18, 34). Different body positions affect exercise hyperemia by the interaction between changes in muscle perfusion pressure and the mechanical effects of muscle contraction and muscle pump activation, combined with local vasodilatory mechanisms that influence vascular conductance (VC) (15, 23, 24, 28, 30–32, 34).

During low and moderate exercise intensities, muscle blood flow reached the same level, whether comparing supine with head-up tilt (HUT) (18) or arm below vs. above heart level (12). However, with moderate and heavy exercise intensities, peak muscle blood flow (MBFpeak) to the leg might be reduced in upright compared with supine posture (5, 33), and supine cycling work performance was increased by lower body negative pressure and was further enhanced in upright cycling, providing additional evidence that muscle blood flow could be compromised in supine position (6).

The purpose of the present study was to investigate the peak and submaximal muscle blood flow and VC responses in the vascular bed supplied by the popliteal artery during plantar flexion exercise in three different body positions, horizontal (HOR), 35° head-down tilt (HDT), and 45° HUT, that achieved ~100-mmHg difference in muscle perfusion pressure. The aim of the first experiment was to determine peak VC in the lower limb by the sudden release of occlusion following intense, isometric, ischemic muscle contractions. It was hypothesized that peak VC response in the lower limb represents the maximal vasodilatory capacity, and it would not differ between body positions, despite the differences in muscle perfusion pressure. The aim of the second experiment was to compare the impact of altered muscle perfusion pressure on muscle blood flow and VC during transitions from rest to lower and higher power output exercise when limb position was manipulated relative to the heart. It was hypothesized that, following the onset of exercise, the rate of increase in muscle blood flow and VC would be faster during HUT than HOR, and both would be faster than the corresponding response during HDT in both lower and higher power outputs. It was also hypothesized that the adaptations in VC during the lower power output would counteract the changes in perfusion pressure, so that muscle blood flow and the delivery of O₂ to the working muscle would be maintained, regardless of body position, but that limitations to recruitment of VC during the higher power output exercise would compromise muscle blood flow and O₂ delivery when the leg was above the heart in HDT.

METHODS

Participants

Eleven healthy male volunteers (age 28.0 ± 3.8 yr, height 176.5 ± 5.3 cm, and body mass 79.0 ± 6.9 kg) participated in the two phases of the studies. Their maximal isometric voluntary contraction (MVC) was 60.0 ± 3.5 kg during plantar flexion exercise in the prone HOR position. Participants received complete written and verbal details of the experimental procedures and potential risks involved before signing an information consent form approved by the Office of Research Ethics of the University of Waterloo. They were instructed to refrain from consuming alcohol or caffeinated beverages, engaging in vigorous exercise for 24 h before testing, and from consuming a large meal within 2 h of testing. All tests were conducted with room temperature constant at 19.4 ± 0.9°C to ensure minimal skin blood flow at rest, humidity at 39.9 ± 4.8%, and barometric pressure at 730.8 ± 2.4 mmHg.
**Experimental Design**

Participants reported to the laboratory five times; four times for the transitions from rest to exercise, and once for measurement of peak VC. After arrival in the laboratory, participants assumed a prone position, with their heads supported by a massage table head piece. Shoulder blocks were adjusted accordingly, and their arms were placed with the shoulders and elbows positioned at ~90°. Two belts, one located on the chest and other on the hips, were used to secure the participants during testing. Straps from these belts were attached to the tilt table to prevent sliding. The angles of tilting for HDT (35°) and HUT (45°) accomplished a reduction and increase in muscle perfusion pressure estimated to the middle of the calf muscle of ~44 mmHg and 55 mmHg, respectively. A footplate was connected to the tilt table, with the right foot strapped on this footplate to allow plantar flexion exercise. Instrumentation took place as described below, and then the participants rested before starting the testing for ~30 min.

**Peak VC Protocols**

The peak VC was obtained during the reactive hyperemia that followed release of an arterial occlusion cuff placed around the lower leg just distal to the popliteal fossa. After brief collection of baseline data, the cuff was inflated to 300 mmHg for 2 min. During the inflation period, isometric plantar flexion exercise was performed at 50% MVC for 1 min. The occlusion cuff was rapidly deflated after optimal acquisition of the Doppler ultrasound signal to monitor for peak muscle blood flow velocity (MBVpeak). The peak VC was obtained in the three different body positions administered in random order. Recovery for at least 30 min was allowed between peak VC measurements, and in each case muscle blood flow velocity had returned to baseline. Diameter of the popliteal artery was measured simultaneously, as described below, and used in the calculation of peak muscle blood flow.

**Transitions From Rest to Exercise Protocols**

Preliminary testing revealed that participants could sustain a repeated plantar flexion exercise (3-s duty cycle: 1-s contraction, 1-s relaxation, 1-s rest) with contractions of ~20% MVC (lower, 3.0 ± 0.4 kg, 2.0 ± 0.2 W) and ~30% MVC (higher, 6.0 ± 0.7 kg, 4.0 ± 0.5 W) for 5 min with some challenge, but without fatigue. The angle of movement was ~20°, representing a displacement of 10 cm. The foot plate movement was set in the rotational axis of the ankle to isolate contractions to the calf muscles. Upon changing the angle of the tilt table, the tension in the cable attached to the footplate was altered. To achieve the same tension in all three positions, the load attached was reduced by 0.6 kg in HDT and increased by 0.8 kg in HUT. The equivalence of work rates was confirmed by monitoring muscle electromyography (EMG), as described below.

Each participant performed six different protocols; three tests per day on 4 different days, randomized by blocks and counterbalanced among the participants. Between each individual test on each day, participants were allowed to come off the tilt table and rest before the next test bout. The order for test block A was as follows: higher power output exercise in HUT (HPOHUT), lower power output exercise in HDT (LPOHDT), and higher power output exercise in HOR (HPOHOR). The order for test block B was as follows: lower power output exercise in HOR (LPOHOR), higher power output exercise in HDT (HPOHDT), and lower power output exercise in HUT (LPOHUT). The 4 days of testing were performed two times per week, separated by at least 48 h.

**Data Acquisition**

Muscle blood flow velocity was determined from the intensity weighted mean of a 4-MHz pulsed Doppler ultrasound probe (Neurovision Doppler Ultrasound, model 500, Multigon Industries, Mt. Vernon, NY). The flat ultrasound probe was secured by surgical tape to the skin surface over the popliteal artery embedded at a 45° angle of insonation relative to the skin and adjusted according to the measured angle between the skin and the popliteal artery.

Diameter of the popliteal artery was measured with an 8- to 12-MHz echo Doppler ultrasound probe (Diagnostic Ultrasound system, model M5, Shenzen Mindray Bio-medical Electronics, Shenzen, China), recorded and stored for analysis. Arterial diameter measurements were performed in B-mode with the echo Doppler ultrasound probe positioned just proximal to the site of velocity measurements. Popliteal arterial diameter images were taken as the average of three separate measurements during diastole at baseline and at the end of recovery and during the relaxation phase between contractions in the 4th min of exercise. Preliminary testing revealed that muscle blood flow and VC were repeatable, as demonstrated by the lower variability, good agreement, and consistency between 2 days of testing. For muscle blood flow, the coefficient of variation was between 5.0 and 9.7%, with intraclass correlation coefficients between 0.7 and 0.98 in both lower and higher power outputs during HOR, HDT, and HUT positions. For VC, the coefficient of variation was between 6.1 and 9.1%, with intraclass correlation coefficients between 0.76 and 0.98 in HOR, HDT, and HUT for both lower and higher power outputs.

Arterial blood pressure was estimated from a photoplethysmograph cuff placed around the middle finger of the left hand (Finometer, Finapres Medical Systems, Arnhem, the Netherlands). Mean arterial pressure was determined as the average between successive heartbeats. Heart rate was continuously calculated from the R-R interval obtained from an electrocardiogram (Pilot 9200, Colin Medical Instruments, San Antonio, TX). Oxygen saturation was obtained by a pulse oximeter with the probe placed in the index finger of the left hand (Ohmeda 3740 Pulse oximeter, Louisville, CO).

Muscle activity was measured by EMG through six skin surface disposable electrodes, pregelled (Blue Sensor, Medicost, Olstykke, Denmark), and placed longitudinally on the distal half of the medial and lateral gastrocnemius and soleus muscles in the right leg in a bipolar configuration, with the interelectrode distance of 2 cm. Before the data collection, participants were shaved, abraded, and cleaned with isopropyl alcohol to reduce skin impedance. The reference electrode was placed over the head of the fibula. EMG electrode placement was noted on the skin by a pen marker and recorded to allow better comparison between tests. A custom-built amplifier (bandwidth 20–500 Hz, common mode rejection ratio > 90 dB, input impedance 2 MΩ) was used to amplify the raw EMG.

Muscle blood flow velocity, mean arterial pressure, heart rate, EMG, and oxygen saturation were continuously collected at 1,000 Hz using a data acquisition system (Powerlab, ADInstruments, Colorado Springs, CO). EMG data were processed with custom software (Matlab, version 7.0, The Mathworks, Natick, MA).

**Data Analysis**

**Vascular conductance.** The MBVpeak was detected as the average of the three highest consecutive beats after cuff release. Popliteal arterial diameter value used was the closest to the time of the peak velocity (MBVpeak). Then MBFpeak (in ml/min) was determined as the product of MBVpeak and popliteal arterial diameter cross-sectional area as $\text{MBF}_{\text{peak}} = \text{MBV}_{\text{peak}} \times \pi r^2 \times 60$, where $r$ is the vessel radius. To calculate peak VC (ml·min$^{-1}$·mmHg$^{-1}$), $\text{MBF}_{\text{peak}}$ was divided by muscle perfusion pressure measured over the same beats.

The relative vasodilation during the rest-to-exercise transitions was estimated from the effective VC during relaxation, calculated from complete cardiac cycles during the relaxation phase of the
duty cycle from the muscle blood flow and the corresponding muscle perfusion pressure, and then expressed as a percentage of the peak VC.

**Transitions from rest to exercise.** Beat-by-beat data were time aligned, linearly interpolated, and averaged over two complete contraction/relaxation cycles (6 s) and over two trials to produce a single data set per person for each of the six conditions. The characteristics of the dynamic response of muscle blood flow and VC were determined by the time to reach 63% of the steady state response \( T_{63\%} \), gain or amplitude of the increase above baseline and exercise response in the last 2 min of exercise. Muscle blood flow was calculated from muscle blood flow velocity and the corresponding popliteal arterial diameter. Muscle perfusion pressure (mmHg) was estimated by mean arterial pressure (mmHg) and corresponding popliteal arterial diameter. Muscle perfusion pressure was higher during 45° HUT, but lower and higher power outputs. During HUT, muscle perfusion pressure increased from baseline (138.7 ± 7.1 mmHg) compared with lower (142.5 ± 7.7 mmHg, \( P < 0.05 \)) and higher power outputs (143 ± 7.4 mmHg, \( P < 0.05 \)), with no differences between lower and higher power outputs. During HDT, muscle perfusion pressure increased from baseline (44.4 ± 6.7 mmHg) to lower (51.1 ± 7.5 mmHg, \( P < 0.05 \)) and higher power outputs (50.6 ± 10 mmHg, \( P < 0.05 \)), but there were no differences between lower and higher power outputs.

**Muscle blood flow and VC increased rapidly after the onset of exercise for all body positions and power outputs.** The post hoc analysis indicated that the rate of increase in muscle blood flow and VC was faster in the HUT and slower in HDT for lower and higher power outputs, as observed in the patterns of the responses and significant effect of body position \( P < 0.05 \), Fig. 1, A and B) and the more rapid \( T_{63\%} \) \( P < 0.05 \), Table 2. Muscle blood flow values during exercise were higher during LPO_{HOR} than LPO_{HDT} and LPO_{HUT} \( P < 0.05 \), Table 2), with no differences between LPO_{HDT} and LPO_{HUT} (Fig. 1A). However, muscle blood flow exercise values were not different between HPO_{HOR} and HPO_{HUT}, but were lower in HPO_{HDT} \( P < 0.05 \), Table 2 and Fig. 1C). VC values during exercise were higher in HDT \( P < 0.05 \), but were lower during HUT.
for lower and higher power outputs (P < 0.05, Table 2 and Fig. 1, B and D). The last minute of recovery after exercise, muscle blood flow and VC responses were greater in HDT and lower in HUT for both lower and higher power outputs (P < 0.05, Table 2). In recovery, muscle blood flow and VC returned to the baseline levels for that specific body position during lower power output (Fig. 1, A and B) and during HPOHOR and HPOHUT, but did not in HPOHUT (Fig. 1, C and D).

Table 2. Dynamic responses of muscle blood flow and vascular conductance during exercise

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Variables</th>
<th>LPOHOR</th>
<th>LPOHDT</th>
<th>LPOHUT</th>
<th>HPOHOR</th>
<th>HPOHDT</th>
<th>HPOHUT</th>
</tr>
</thead>
<tbody>
<tr>
<td>MBF, ml/min</td>
<td>Baseline</td>
<td>51.5 ± 25.5</td>
<td>41.8 ± 15.3</td>
<td>28.6 ± 14.0</td>
<td>44.2 ± 14.0</td>
<td>46.7 ± 17.7</td>
<td>32.8 ± 10.7</td>
</tr>
<tr>
<td>T_{63%}, s</td>
<td>16.5 ± 10.9</td>
<td>57.6 ± 15.5</td>
<td>9.5 ± 6.3</td>
<td>25.3 ± 8.9</td>
<td>44.5 ± 9.7</td>
<td>13.4 ± 6.7</td>
<td></td>
</tr>
<tr>
<td>G</td>
<td>133.4 ± 65.0</td>
<td>105.1 ± 57.0</td>
<td>92.3 ± 48.1</td>
<td>197.2 ± 83.9</td>
<td>135.9 ± 55.6</td>
<td>187.0 ± 60.6</td>
<td></td>
</tr>
<tr>
<td>EndEx</td>
<td>185.9 ± 70.2</td>
<td>146.9 ± 62.1</td>
<td>120.9 ± 54.8</td>
<td>241.4 ± 93.2</td>
<td>182.6 ± 54.6</td>
<td>219.8 ± 64.2</td>
<td></td>
</tr>
<tr>
<td>Recovery</td>
<td>53.6 ± 32.8</td>
<td>49.0 ± 28.4</td>
<td>26.6 ± 9.9</td>
<td>47.7 ± 19.0</td>
<td>118.1 ± 59.4</td>
<td>38.0 ± 16.2</td>
<td></td>
</tr>
<tr>
<td>VC, ml·min⁻¹·mmHg⁻¹</td>
<td>Baseline</td>
<td>0.7 ± 0.4</td>
<td>1.0 ± 0.4</td>
<td>0.2 ± 0.1</td>
<td>0.6 ± 0.2</td>
<td>1.2 ± 0.8</td>
<td>0.2 ± 0.1</td>
</tr>
<tr>
<td>T_{63%}, s</td>
<td>16.9 ± 9.8</td>
<td>35.6 ± 14.1</td>
<td>9.9 ± 7.6</td>
<td>23.9 ± 10.1</td>
<td>39.0 ± 15.1</td>
<td>13.3 ± 6.7</td>
<td></td>
</tr>
<tr>
<td>G</td>
<td>1.5 ± 0.7</td>
<td>2.0 ± 1.1</td>
<td>0.6 ± 0.4</td>
<td>2.2 ± 1.1</td>
<td>2.5 ± 1.1</td>
<td>1.3 ± 0.5</td>
<td></td>
</tr>
<tr>
<td>EndEx</td>
<td>2.2 ± 0.9</td>
<td>2.9 ± 1.3</td>
<td>0.9 ± 0.4</td>
<td>2.8 ± 1.2</td>
<td>3.7 ± 1.2</td>
<td>1.5 ± 0.5</td>
<td></td>
</tr>
<tr>
<td>Recovery</td>
<td>0.7 ± 0.4</td>
<td>1.1 ± 0.6</td>
<td>0.2 ± 0.1</td>
<td>0.7 ± 0.3</td>
<td>2.9 ± 1.8</td>
<td>0.3 ± 0.1</td>
<td></td>
</tr>
<tr>
<td>Test block order</td>
<td>B1</td>
<td>A2</td>
<td>B3</td>
<td>A3</td>
<td>B2</td>
<td>A1</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SD for 11 participants. LPOHOR, lower power output HOR; LPOHDT, lower power output HDT; LPOHUT, lower power output HUT; HPOHOR, higher power output HOR; HPOHDT, higher power output HDT; HPOHUT, higher power output HUT; T_{63%}, time to reach 63% of the response; G, gain of the response; EndEx, end of exercise values from the last 2 min of exercise. Statistically significant compared with *LPOHOR, †LPOHDT, ‡LPOHUT, §HPOHOR, ¶HPOHDT, and ¶¶baseline and recovery in the same condition: P < 0.05. Test block order indicates, for each of the blocks, the order of that specific condition for comparison of baseline data.

Fig. 1. Time course of changes in muscle blood flow (MBF; A and C) and vascular conductance (VC; B and D) during dynamic plantar flexion exercise performed in lower (LPO; A and B) and higher power outputs (HPO; C and D). Lines indicate group response, symbols indicate the points at which statistical analysis were performed and dashed vertical lines indicate the start and cessation of exercise. Data are the mean analyzed over 6-s time bins, including contraction and relaxation phases of the duty cycles. SD was omitted to improve data visualization, but are presented with statistical comparison in Table 2 along with definition of abbreviations. If necessary, symbols were offset for clarity. HDT, head-down tilt; HOR, horizontal; HUT, head-up tilt.
VC Expressed as Percentage of Peak VC

There were main effects of body position and time and an interaction between body position and time for effective VC during relaxation and effective VC during relaxation expressed as percentage of peak VC ($P < 0.05$). Effective VC during relaxation and effective VC during relaxation expressed as percentage of peak VC were significantly higher during HDT and lower during HUT from baseline to recovery for lower and higher power outputs ($P < 0.05$, Fig. 2).

Muscle Activity Responses During Exercise

EMG was not different during lower power output exercise for all body positions (Fig. 3A) and for HPOHOR and HPOHUT, except for gastrocnemius medial head. However, EMG was higher during HPOHDT for all muscle groups ($P < 0.05$, Fig. 3B). The EMGindex values were not different during lower power output for all body positions and for HPOHOR and HPOHUT, but they were higher during HPOHDT ($P < 0.05$, Fig. 3C).

DISCUSSION

In this study of plantar flexion exercise performed in different body positions, we have demonstrated the encroachment of vascular responses on peak VC as muscle perfusion pressure was reduced and work rate increased. Consistent with our first hypothesis, peak VC was not different between body positions. Following the onset of exercise, recruitment of VC was necessary to achieve appropriate muscle blood flow and, as predicted in the second hypothesis, was faster during HUT and slower during HDT than in the HOR position for both the lower and higher output exercise. The results confirmed that recruitment of VC was able to fully compensate in LPOHDT for the reduced muscle perfusion pressure relative to the HUT position to maintain muscle blood flow. However, as predicted, the recruitment of VC during HPOHDT restricted the increase in muscle blood flow limiting O2 delivery with functional consequences as reflected in the increased EMG only in this condition (1, 9, 35).

Peak VC

Previous investigations of peak VC (17, 25) reported similar increases to those observed in the present study, even though different methods were employed. To the best of our knowledge, this is the first time that peak VC has been assessed across a wide range of perfusion pressures. Finding similar values of peak VC, regardless of body position, was anticipated as the accumulation of metabolites during ischemia plus exercise was expected to achieve maximal vasodilation and to be independent of body position.
Baseline Muscle Blood Flow and VC

A consistent observation from the baseline period for the HUT was a marked reduction in VC, resulting in a lower muscle blood flow, despite the elevated muscle perfusion pressure (up to ~100 mmHg in HDT). VC increased in HDT so that muscle blood flow was not different than the HOR position. These findings, which are consistent with previous research (4, 7, 10, 12, 13, 20, 29, 34), can be explained by the myogenic reflex response (13, 27), activation of the venoarteriolar reflex, inducing local arteriolar vasoconstriction of the corresponding arteriole (8, 29), and activation of the arterial baroreflex, increasing sympathetic vasoconstriction (3, 27). The elevated VC in HDT was a consequence of vascular smooth muscle relaxation, removal of the venoarteriolar reflex (19, 21) and reduced baroreflex activity (26). While endothelial factors probably play a role in modifying VC (22), their role with postural changes are unknown.

Adaptive Responses of VC and Muscle Blood Flow to Exercise

The present study is the first to compare the vascular adaptations to calf muscle exercise in the HOR, HDT, and HUT positions. The rates at which muscle blood flow approached the exercise value were dependent on muscle perfusion pressure, with faster adaptations in HUT and slower in HDT than HOR, as well as faster responses at the lower power output. These findings expand on the results of Nadland et al. (18), who examined only the HUT and HOR positions with contractions at 30% MVC, and are consistent with previous studies comparing arm below and above heart level (20, 34). Muscle perfusion pressure increased only slightly during exercise, pointing to the primary role of VC to increase muscle blood flow. For LPOHUT, it was necessary to recruit <20% peak VC to attain steady-state muscle blood flow, while LPOHDT required almost 80% of the peak VC. In HPOHUT and HPOHOR,
there was still VC reserve, indicating that muscle blood flow could have increased a further two to threefold. However, in HPOHDT, the VC reached ~92% of the peak, so further increases in muscle blood flow would depend primarily on increases in muscle perfusion pressure.

The early response of muscle blood flow in the LPOHUT had a small overshoot, but this was exaggerated in HPOHUT, consistent with the study by Nadland et al. (18). For LPOHOR, a small overshoot was detectable (Fig. 1A), but for HPOHOR and both HDT work rates, there was no overshoot of muscle blood flow. It appears that the interactions between metabolic demand affecting vasodilation and mechanical factors associated with the perfusion pressure and muscle pump contributed to the patterns of response. An overshoot in muscle blood flow is characteristic of a relative excess of flow compared with metabolic demand. The muscle pump, which would have been most effective in the HUT and probably ineffective in HDT, is not selective in causing an increase in blood flow to metabolically active vs. inactive regions (11). Therefore, when the initial venous pressure was elevated with HUT, the change in pressure gradient with muscle contractions evokes an effective increase in “virtual conductance” (18, 23, 24, 32) that is greater than required to deliver blood flow to only metabolically active muscle fibers. This is followed by vasoconstriction in inactive regions of the muscle to return VC and muscle blood flow to lower appropriate levels.

At the cessation of exercise, there were overshoots of muscle blood flow in LPOHDT, HPOHOR, and HPOHDT that might have reflected a relative deficiency of blood flow during the exercise. The overshoots were a consequence of removal of the impediment to muscle blood flow related to muscle contraction, as observed in the VC graphs (Fig. 1, B and D). Over the subsequent 5 min of recovery, baseline VC was achieved in all but LPOHDT, HPOHOR, and HPOHDT. That is, those conditions that had overshoots in muscle blood flow following exercise were the same conditions that had not completely recovered after 5 min.

In the lower power output, there were no differences in the EMGindex, while HPOHDT had a significantly elevated EMGindex. These data suggest that, at the lower power output, even though there were differences in the muscle blood flow between body postures, the apparent deficiency in blood flow and O2 delivery that was suggested by the slow postexercise recovery of VC was not sufficient to produce evidence of muscle fatigue. In contrast for the HPOHDT, the increased EMGindex provides evidence that muscle fatigue occurred with an increased requirement for muscle fiber recruitment to maintain muscle power output (2, 14, 35). In the HPOHDT, both the slower adaptation of muscle blood flow and the lower amplitude of response probably contributed to the metabolic imbalance.

In summary, the present study investigated vascular responses to two different intensities of plantar flexion exercise under a wide range of muscle perfusion pressure. We found that peak VC was independent of limb position and muscle perfusion pressure, but that recruitment of VC during exercise was highly dependent on muscle perfusion pressure. High muscle perfusion pressure in the HUT position was associated with rapid attainment of appropriate VC and muscle blood flow, although at the lower work rate, the absolute muscle blood flow was significantly less than in the HOR position. Reduced muscle perfusion pressure in HDT slowed the vascular responses. For the LPOHDT exercise, there did not appear to be a functional consequence to the delayed adaptation of muscle blood flow, at least as reflected by unchanged EMGindex. In contrast for the HPOHDT, the slower adaptation and smaller amplitude increase in muscle blood flow was associated with a marked increase in EMGindex, suggesting fatigue of some muscle fibers and the potential for performance failure. The postexercise sustained hyperemia also supports the notion that muscle fatigue was developing and performance failure was imminent.

ACKNOWLEDGMENTS

The authors are grateful to Danielle Greaves for excellent technical assistance.

GRANTS

This research was supported by the National Sciences and Engineering Research Council (RGPIN 6473) and by the Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES, Brazil).

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

Author contributions: R.V. and R.L.H. conception and design of research; R.V. performed experiments; R.V. and R.L.H. interpreted results of experiments; R.V. prepared figures; R.V. drafted manuscript; R.V. and R.L.H. edited and revised manuscript; R.V. and R.L.H. approved final version of manuscript.

REFERENCES


