Effect of active muscle mass during ischemic exercise on peak lower leg vascular conductance

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Effect of active muscle mass during ischemic exercise on peak lower leg vascular conductance. J Appl Physiol 98: 765–771, 2005. First published October 22, 2004; doi:10.1152/japplphysiol.00468.2004.—Uncertainty exists as to whether a period of passive arterial occlusion (PAO) or ischemic exercise (IE) results in peak lower leg vascular conductance (LVC). This uncertainty is due to the different body positions, active muscle mass, and occlusion times used for PAO or IE. The purpose of this study was to examine whether 10 min of PAO elicits a similar LVC compared with ischemic dorsiflexion (IDF), ischemic plantar flexion (IPF), and ischemic plantar-dorsiflexion (IPDF). Ten subjects (5 women, 27 ± 9 yr, 68 ± 3 kg) were studied on 3 days over 1 wk in a semireclined position with the right foot attached to an isokinetic dynamometer. Mean arterial pressure (Finapres) and lower leg blood flow (LBF, venous occlusion plethysmography) were measured at rest and after PAO and IE. PAO was administered randomly on 1 of the 3 days and before IE. IE protocols consisted of maximal isokinetic dorsiflexion and/or plantar flexion at 120 and 60°/s, respectively. In a second experiment, an additional eight subjects (4 women, 29 ± 12 yr, 77 ± 12 kg) were studied to examine the effect of isokinetic speed during IDF on peak LBF and LVC. Peak LVC (ml·min⁻¹·100 ml⁻¹·mmHg⁻¹) was similar among IPF (0.590 ± 0.16), IPDF (0.532 ± 0.17), and PAO (0.511 ± 0.18), and significantly lower after IDF (0.334 ± 0.15). No differences in peak LBF and LVC were observed after IDF using different isokinetic speeds. We conclude that 10 min of PAO, IPF, and IPDF performed in a similar posture are adequate stimuli to elicit peak LVC.

Skeletal muscle blood flow; peak vascular conductance; reactive hyperemia; vasodilation

PEAK VASCULAR CONDUCTANCE, or the vasodilatory capacity of a limb (i.e., lower leg), is often used to indicate the role of the arterial vasculature in health and disease. Vascular conductance can be assessed by measuring limb blood flow in conjunction with mean arterial pressure (MAP). A commonly used noninvasive technique to measure limb blood flow is venous occlusion plethysmography (12). Studies have examined the relationship between peak lower leg vascular conductance (LVC) and aerobic capacity (5, 15, 21, 26, 34), disease (1, 6, 30, 38), and aging (20, 22), thus emphasizing the importance of obtaining a valid measurement of peak LVC. However, the stimulus that is required to elicit peak LVC remains unknown (15, 30, 34).

Typically, two types of stimuli, passive arterial occlusion (PAO) or ischemic exercise, are used to elicit peak LVC. Studies have examined whether a period (5–10 min) of PAO results in the same peak LVC compared with ischemic exercise. Both differences (15, 34) and no differences (30) have been reported between these two stimuli. Comparison between these previous studies is difficult because of the different body positions used during PAO vs. ischemic exercise (i.e., standing, semireclined, or supine), the differences in muscle mass used during ischemic exercise, and the different occlusion times used for PAO. For example, Snell et al. (34) compared standing ischemic plantar-dorsiflexion (IPDF) (i.e., heel-toe raises to exhaustion) to 5 min of supine PAO, whereas Kosmas et al. (15) compared standing ischemic plantar flexion (IPF) (i.e., standing toe raises to exhaustion) to 10 min of supine PAO. Both studies reported a significantly higher LVC after ischemic exercise. However, Rueckert and Hanson (30) compared IPF with 10 min of PAO while subjects were in a semireclined position for both tests and found no difference in peak LVC.

Thus uncertainty remains as to what stimulus elicits peak LVC. Missing from previous studies is a systematic examination of the effects of active muscle mass during ischemic exercise on peak LVC. Furthermore, with the exception of Rueckert and Hanson (30), different postures have been used during PAO and ischemic exercise. Therefore, the purpose of this study was to examine the effect of active muscle mass during ischemic exercise on peak LVC and compare that with the peak LVC obtained after 10 min of PAO while subjects maintained a similar posture during and after all tests. We hypothesized that using the greatest amount of muscle mass during ischemic exercise would produce the greatest LVC.

METHODS

Subjects

Eighteen healthy subjects (9 men, 9 women) participated in this two-part study. Ten subjects (5 men, 5 women, 27 ± 9 yr, 68 ± 3 kg) were included in experiment 1, and eight (4 men, 4 women, 29 ± 12 yr, 77 ± 12 kg) were included in a second experiment. Four of these latter eight subjects had also participated in experiment 1. Subjects were free from chronic disease (e.g., cardiovascular, neurological, etc.), and physical activity levels ranged from sedentary to recreational-ly active. None of the subjects was competing at local-, state-, national-, or elite-level athletic events. The Institutional Review Board at Marquette University approved this study, and subjects gave written, informed consent before participation.

Experiment 1 Procedures

Subjects were studied three times within a 1-wk period at the same time of day but not on adjacent days. Subjects were studied at least 2 h postprandial and had abstained from caffeine since waking. Resting blood pressure was obtained by manual sphygmomanometry 10 min after subjects were positioned in a Biodex Multi-Joint System 3 Pro

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isokinetic dynamometer (Biodex Medical Systems, Shirley, NY) with the thigh supported and foot attached to a footplate. The right leg was used for all tests. The subjects were in a semireclined position (45° from seated upright position), with a knee angle at 60° of extension and the lower leg 10 cm above heart level. The lateral malleolus was aligned with the center of rotation of the dynamometer. Ankle angle of motion was 40° to −5° of plantar flexion. MAP was obtained continuously by automated finger photoplethysmography (Finapres, Ohmeda, Louisville, CO) and sampled online at 100 Hz (CED model 1401 Plus, Cambridge Electronic Design, Cambridge, UK). Blood pressures were obtained from the right hand, which was supported and positioned at heart level. Each experimental day, resting lower leg blood flow (LBF) was obtained every 15 s for 3 min. After resting LBF measurements, peak LBF was determined after one of four protocols. Three of these protocols were designed to vary the amount of active muscle mass during ischemic exercise. These ischemic exercise protocols are described in detail later. The fourth was 10 min of PAO. On one of the three days and before ischemic exercise, LBF and MAP were measured after PAO. During each visit, LBF and MAP were measured after one of the three ischemic exercise protocols. On the day PAO was administered, at least 15 min of recovery was allowed before ischemic exercise was performed. All ischemic exercise protocols were randomized and counter-balanced as was as the day PAO was administered.

**LBF**

Rest measurements. LBF was measured noninvasively by venous occlusion plethysmography. A double-stranded mercury-in-Silastic strain gauge (40) was positioned around the largest circumference of the calf and connected to an electrically calibrated and self-balancing (8) plethysmograph (EC-6 Plethysmograph, D. E. Hokanson, Bellevue, WA). Data from the plethysmographs were sampled at 100 Hz (CED model 1401 Plus, Cambridge Electronic Design) and stored for offline analysis. A venous occlusion cuff was positioned 5 cm proximal to the knee and connected to a rapid and adjustable cuff inflator air source (E-20 Rapid Cuff Inflator and AG-101 Air Source, D. E. Hokanson). An ankle cuff was manually inflated to 300 mmHg before all LBF measurements to prevent blood flow to the foot (13, 17). LBF was measured repeatedly over 3 min by inflating the venous occlusion cuff to 50 mmHg (7) for 8 s and deflating for 7 s, resulting in 12 resting LBF measurements. Blood pressure was monitored continuously, and the venous occlusion cuff pressure was maintained lower than diastolic blood pressure.

**PAO.** PAO of the lower leg was administered by inflating the occlusion cuff to suprasystolic pressures (220–280 mmHg). Ischemic exercise began within 10 s of cuff inflation. Blood pressure was monitored continuously so that occlusion cuff pressure was at least 20 mmHg higher than systolic blood pressure during each protocol. The three ischemic exercise protocols consisted of (1) ischemic dorsiflexion (IDF) at 120/5 s with passive plantar flexion, (2) IPF at 60/5 s with passive dorsiflexion, and (3) IPDF at 60 and 120/5 s, respectively. A metronome was used to ensure that a muscle contraction (active or passive) occurred every 2 s. During passive plantar flexion or dorsiflexion, the test administrator would manually move the footplate (i.e., foot) through the intended range of motion in time with the metronome. Subjects were instructed to relax between the passive movements. All three ischemic exercise protocols are illustrated below:

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<tr>
<td>IDF</td>
<td>active dorsiflexion</td>
<td>repeat to exhaustion</td>
</tr>
<tr>
<td>IPF</td>
<td>active plantar flexion</td>
<td>passive plantar flexion</td>
</tr>
<tr>
<td>IPDF</td>
<td>active dorsiflexion</td>
<td>passive dorsiflexion</td>
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Subjects were verbally and continuously encouraged to give maximal efforts during contractions. Exercise was terminated when subjects could not maintain the cadence of the metronome. The same test administrator was responsible for terminating each test. Torque (N·m) sampled at 100 Hz for each muscle group and MAP were measured during each ischemic exercise protocol. Endurance time was recorded for all ischemic exercise protocols. The ischemic exercise protocols were designed to last longer than 90 s because it has been reported that peak conductance plateaus after ischemic exercise of 90–120 s (15). On the basis of preliminary work from our laboratory, we chose speeds of 60 and 120/5 s for plantar flexion and dorsiflexion, respectively. These speeds were selected to allow endurance time to be greater than 90 s and to reduce the variability in endurance time among protocols.

**Experiment 2 Procedures**

The use of different isokinetic speeds for dorsiflexion and plantar flexion may have affected the degree of muscle fatigue, which could have led to differences in peak LBF or LVC. To ensure that using a faster speed for dorsiflexion did not affect peak LBF or LVC, a second...
experiment was performed to determine the effect of isokinetic speed during IDF on peak LBF and LVC. Subjects performed IDF at each of 60 and 120°/s (randomized and counterbalanced) on 2 different days, and data were recorded as in the first study.

Statistical Analysis

Dependent variables in this study were LBF, MAP, LVC, endurance time, and torque, and the independent variables were PAO, IDF, IPF, and IPDF. A repeated-measures ANOVA was used to compare dependent variables across protocols. After a significant F value, a Student-Newman-Keuls post hoc test was used to examine differences between groups. Paired t-tests were used to compare resting MAP vs. peak MAP. Paired t-tests were also used to compare dorsiflexor torque during IPDF and IDF and plantar flexor torque during IPDF and IPF.

All data are reported as means ± SD, and the significance level was set at $P < 0.05$.

RESULTS

Resting MAP, LBF, and LVC

Subjects reported to the laboratory on 3 different days; however, because PAO was administered randomly on one of the three visits, resting measurements are reported for all four protocols. There were no significant differences in resting MAP, LBF, and LVC across protocols (Table 1). Manually obtained measurements of resting MAP were not significantly different across protocols and are also presented in Table 1.

Peak LBF and MAP

Results of peak LBF are shown in Fig. 1. Peak LBF after IPF and IPDF was significantly greater compared with IDF and PAO. Furthermore, PAO resulted in a significantly greater LBF compared with IDF. Results of the resting and peak 15-s averaged MAP after all protocols are shown in Fig. 2. As expected, immediately after all ischemic exercise protocols, MAP was significantly elevated above resting values, whereas MAP after PAO was not significantly different from rest. MAP values after IPDF, IPF, and IDF were significantly higher compared with PAO.

Peak LVC

Results of peak LVC after all protocols are shown in Fig. 3. Peak LVC was similar among IPF (0.590 ± 0.16 ml·min⁻¹·100 ml⁻¹·mmHg⁻¹), IPDF (0.532 ± 0.17 ml·min⁻¹·100 ml⁻¹·mmHg⁻¹), and PAO (0.511 ± 0.18 ml·min⁻¹·100 ml⁻¹·mmHg⁻¹), and these were all significantly greater than IDF (0.334 ± 0.15 ml·min⁻¹·100 ml⁻¹·mmHg⁻¹).

Ischemic Exercise Endurance Time

Endurance times for all ischemic exercise protocols are presented in Fig. 4. Despite our efforts to minimize the variability in endurance time for ischemic exercise protocols, all were significantly different from one another.

MAP During Ischemic Exercise

Because differences in endurance time existed among protocols, the MAP at 25, 50, 75, and 100% of endurance time was examined for all ischemic exercise protocols. Fifteen-second averages of MAP were taken at each percentage of endurance time. There were no significant differences in MAP (Fig. 5) at any percentage of endurance time across all ischemic exercise protocols.

Muscle Torque During Ischemic Exercise

Dorsiflexor and plantar flexor torque was analyzed at 10% increments of endurance time for all ischemic exercise proto-

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Table 1. Resting MAP, LBF, and LVC

<table>
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<tr>
<th></th>
<th>Manual MAP, mmHg</th>
<th>Finapres MAP, mmHg</th>
<th>LBF, ml·min⁻¹·100 ml⁻¹</th>
<th>LVC, ml·min⁻¹·100 ml⁻¹·mmHg⁻¹</th>
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<tr>
<td>PAO</td>
<td>86.4 ± 10</td>
<td>80.1 ± 5</td>
<td>2.59 ± 0.6</td>
<td>0.032 ± 0.01</td>
</tr>
<tr>
<td>IDF</td>
<td>90.8 ± 9</td>
<td>82.4 ± 13</td>
<td>2.79 ± 1.1</td>
<td>0.034 ± 0.01</td>
</tr>
<tr>
<td>IPF</td>
<td>86.4 ± 10</td>
<td>80.5 ± 11</td>
<td>2.51 ± 0.7</td>
<td>0.031 ± 0.01</td>
</tr>
<tr>
<td>IPDF</td>
<td>86.9 ± 10</td>
<td>85.8 ± 10</td>
<td>2.59 ± 0.6</td>
<td>0.030 ± 0.01</td>
</tr>
</tbody>
</table>

Values are means ± SD. MAP, mean arterial pressure; LBF, lower leg blood flow; LVC, lower leg vascular conductance; PAO, passive arterial occlusion; IDF, ischemic dorsiflexion; IPF, ischemic plantar flexion; IPDF, ischemic plantar-dorsiflexion. There were no significant differences in manual MAP, Finapres MAP, LBF, and LVC across protocols.
cols. There were no significant differences at any percentage of endurance time between the dorsiflexors during IPDF and IDF and between the plantar flexors during IPDF and IPF (Fig. 6). Thus similar torques were produced by the same muscle group in each ischemic exercise protocol at the same relative time (i.e., dorsiflexors during IPDF and IDF).

Experiment 2

Experiment 2 was performed to examine the effects of isokinetic speed during IDF on peak LBF and LVC. There were no significant differences between 60 and 120°/s with respect to peak LBF (38.8 ± 9 vs. 39.9 ± 8 ml·min⁻¹·100 ml⁻¹, respectively) and peak LVC (0.409 ± 0.10 vs. 0.400 ± 0.12 ml·min⁻¹·100 ml⁻¹·mmHg⁻¹, respectively). Therefore, we conclude that using a faster speed for dorsiflexion in experiment 1 did not affect the results of this study.

DISCUSSION

The significant findings from this study are that, under otherwise similar conditions (i.e., semireclined position), 1) contraction of a small muscle mass (i.e., IDF) during ischemic exercise of the lower leg results in a diminished peak LVC and LBF, but similar MAP, compared with IPF and IPDF, and 2) 10 min of PAO results in a similar peak LVC compared with IPF and IPDF. Subjects maintained a similar posture during and after all protocols while the amount of active muscle mass during ischemic exercise was systematically altered and the peak LVC measures obtained were compared with those after 10 min of PAO.

Studies (15, 34) finding differences in peak LVC have compared standing ischemic exercise (i.e., heel-toe raises or toe raises) to supine PAO. In these previous studies, subjects returned to the supine position after ischemic exercise. It is unknown what effects gravity had on LVC between protocols in these previous studies. A semireclined position was utilized in our study so that one body position was maintained during and after each protocol. This ensured that the first blood flow measurement was obtained quickly (~10 s) after all protocols, and it minimized the risk of altering the position of the strain gauge, which may occur when moving from a standing to supine position. In addition, by employing a similar body position for each protocol, we controlled for any confounding effects that posture might have had in evaluating the effects of PAO and ischemic exercise of varying muscle mass on peak LVC.

LBF significantly increased after PAO and ischemic exercise. The large increase in LBF can be attributed to both metabolic and myogenic-induced vasodilation (2, 4, 11, 14, 16, 18, 19, 32, 35, 39) and the increase in MAP resulting from the muscle reflex-induced sympathetic response originating from type III and IV muscle chemoreceptors (29). However, it has been reported that increased levels of sympathetic activation resulting from type III and IV muscle chemoreceptor stimulation may actually compete with local vasodilation in skeletal muscle (29, 31). Therefore, it can still be questioned whether increasing active muscle mass during ischemic exercise of the lower leg results in a greater LVC and whether ischemic exercise elicits a greater LVC compared with PAO.

Active Muscle Mass During Ischemic Exercise

Rueckert and Hanson (30) reported a similar peak LVC after PAO and ischemic exercise while subjects were in a semireclined position; however, only the plantar flexors were used during ischemic exercise. Others have suggested that IPDF may result in an even higher LVC compared with IPF (15). To our knowledge, no study has systematically examined the effect of active muscle mass during ischemic exercise on eliciting peak LVC.

Our results indicate that activating only the dorsiflexors during ischemic exercise results in a significantly lower LVC (Fig. 3) and LBF (Fig. 1), but similar MAP (Fig. 2), compared with IPF and IPDF. Because MAP was similar after all ischemic exercise protocols, the significantly smaller LVC after IDF was due to a diminished LBF response. This was likely due to activation of a smaller muscle mass during IDF, resulting in a limited potential to increase vasodilation within the lower leg. It is surprising that MAP, an indication of sympathetic drive, was similar during (Fig. 5) and after (Fig. 2) all ischemic exercise protocols. During ischemic exercise, one would expect a higher MAP for a greater amount of active muscle mass (27). The similar MAP among the ischemic exercise protocols in our study could be explained by differences in muscle fiber

Fig. 1. Left: Lower leg vascular conductance after 4 ischemic stimuli. Values are means ± SD. *P < 0.05 compared with IDF. Right: Comparison of peak LVC and LBF (ml·min⁻¹·100 ml⁻¹). LBF significantly increased after PAO and ischemic exercise. MAP also significantly increased after PAO compared with IPF and IPDF. Therefore, it can be concluded that the increased LBF was not due to increased MAP. It is possible that increased levels of sympathetic activation resulting from type III and IV muscle chemoreceptor stimulation may compete with local vasodilation in skeletal muscle. Therefore, it can still be questioned whether increasing the active muscle mass during ischemic exercise of the lower leg results in a greater LVC and whether ischemic exercise elicits a greater LVC compared with PAO.

Fig. 2. MAP and LVC were measured during 3 ischemic exercise protocols. Significant differences existed across all protocols. Values are means ± SD. *P < 0.05 across protocols.

Fig. 3. Peak lower leg vascular conductance after 4 ischemic stimuli. Values are means ± SD. *P < 0.05 compared with IDF.

Fig. 4. Endurance time for the 3 ischemic exercise protocols. Significant differences existed across all protocols. Values are means ± SD. *P < 0.05 across protocols.
type between the anterior and posterior compartments of the lower leg. As a result of the position of the subjects in our study (i.e., knee angle at 60° extension), the soleus was the predominant muscle used during plantar flexion (37). The soleus contains a greater amount of slow, fatigue-resistant, muscle fibers compared with the primary dorsiflexor, the tibialis anterior (10). Studies in animals have indicated that the increase in MAP in response to static contractions is lower in highly oxidative compared with more glycolytic muscle fibers (9, 24, 41). This may explain why utilization of the dorsiflexors during IDF and IPDF resulted in a similar MAP, but significantly shorter endurance time, compared with IPF.

We hypothesized that using the largest amount of muscle mass (IPDF) during ischemic exercise would result in the greatest amount of vasodilation. Contrary to our hypothesis, there was no difference in peak LVC, LBF, or MAP between IPF and IPDF. During IPDF, the plantar flexors may not have had ample time to produce the same vasodilatory stimulus compared with IPF. However, during IPDF, the active dorsiflexors should contribute additional muscle metabolites, thereby increasing the possibility of a similar vasodilatory stimulus within the lower leg.

The results of this study indicate that the relationship between active muscle mass during ischemic exercise of the lower leg and peak LVC is complex. This complexity is likely due to the different muscle fiber types within the lower leg compartments. Active muscle mass during ischemic exercise to exhaustion does not seem to be playing a major role in determining peak LVC, except when a relatively small amount of muscle mass within the lower leg is used. This is further indicated by the fact that 10 min of PAO (no active muscle mass) results in a similar peak LVC compared with IPF and IPDF, as discussed in the next section. The significantly lower LVC after IDF was likely due to activation of a smaller, less oxidative, muscle mass, resulting in a similar vasoconstrictor stimulus, but blunted vasodilatory stimulus, compared with IPF and IPDF.

**Ischemic Exercise vs. Arterial Occlusion**

The results of this study indicate that 10 min of PAO result in a similar peak LVC compared with IPF and IPDF. Although IPF and IPDF resulted in a higher LBF, this occurred at a higher MAP compared with PAO. An elevated MAP after ischemic exercise (Fig. 2) compared with rest is consistent with other studies (30, 34) and was likely the result of metaboreceptor stimulation, because both central command and mechanical stimuli were absent during measurements of LBF and MAP. The similarity in MAP after PAO and rest is consistent with previous studies (28, 30) and suggests the importance of metabolites produced during exercise to elicit a significant pressor response (27). Given that ischemic exercise likely results in a relatively higher concentration of local muscle metabolites, our results suggest that an increased local vasodilatory signal after IPF and IPDF is offset by an increased, metaboreceptor-mediated, sympathetic drive. Therefore, the similar peak LVC after IPF, IPDF, and PAO could result from a similar balance of vasodilation and vasoconstriction within the lower leg arterial vasculature.

Significant research (for review, see Ref. 3) has been devoted to the possibility of vasoconstriction occurring in active muscle during dynamic exercise. The study of peak LVC in humans using venous occlusion plethysmography requires the measurement of LBF immediately after an ischemic stimulus while the muscles are relaxed. The effect of sympathetic tone on peak vasodilation after an ischemic stimulus has not been investigated in the lower leg. However, others have examined whether heightened sympathetic tone, induced via application of ice to the forehead (33), lower body negative pressure (31, 36), or lower leg ischemia after dynamic exercise (31), attenuates peak vascular conductance of the forearm after PAO. Both application of ice to the forehead and leg ischemia after exercise attenuated peak forearm vascular conductance; however, lower body negative pressure, which increases sympathetic output via baroreceptor unloading, had no effect on peak vasodilation of the forearm. These previous studies demonstrate that some sympathoexcitatory pathways are powerful enough to attenuate the peak vasodilatory response in the forearm.

In our study, both vasoconstrictor and vasodilator stimuli were applied to one limb; therefore, we could not assess the
impact of each stimulus individually on eliciting peak LVC. However, given that ischemic exercise should provide a greater vasodilatory stimulus, we hypothesized that peak LVC would be greater after ischemic exercise. However, it appears that the heightened sympathetic drive resulting from metaboreceptor stimulation after ischemic exercise may have opposed the greater vasodilatory signal experienced by the lower leg.

Implications

Both PAO and ischemic exercise have been used extensively to study the arterial vasculature of the lower leg vasculature. However, ischemic exercise may subject high-risk disease populations (e.g., congestive heart failure or hypertension) to greater levels of sympathetic outflow and MAP. Furthermore, ischemic exercise may be difficult to perform in individuals with chronic immune, neurological, or other diseases, such as multiple sclerosis. In the present study, we have shown that PAO, when measured in a semireclined position, results in a similar peak LVC compared with ischemic exercise, but it does so with lower levels of blood pressure and presumably sympathetic activation. In this respect, we confirm the results of a previous study (30) that PAO is a useful technique to assess the arterial vasculature of the lower leg compared with ischemic exercise.

Study Limitations

The use of “maximal,” when referring to vascular conductance, must be used with caution (16); therefore, we chose “peak” to refer to the highest LBF and LVC values obtained after all ischemic stimuli. Subjects were studied in a semireclined position, and we cannot rule out that a standing protocol might have elicited a higher LVC. However, the goal of this study was to systematically examine whether active muscle mass during ischemic exercise affects peak LVC and how these values compared with peak LVC after 10 min of PAO. We did not directly assess sympathetic outflow but used MAP as an indication of sympathetic activity. However, the sustained increase in MAP after exercise, when both central command and mechanical factors are absent, is thought to be primarily due to the stimulation of type III and IV muscle chemoreceptors (29).

The differences in endurance time among the ischemic exercise may have affected peak LVC. Because endurance time was different among protocols, the amount of time a nonactive muscle group was occluded (i.e., the dorsiflexors during IPF vs. the plantar flexors during IDF) may have affected LBF and LVC because of the amount of myogenic or metabolic stimuli experienced by the nonactive muscle.

The results of this study were obtained from healthy nonathletic subjects. Thus we cannot generalize our results to other populations, such as endurance-trained athletes.

It is also possible that the reason we observed a similar peak LVC after PAO, IPDF, and PAO was due to a ceiling effect, such that there was a limited potential for conductance to increase (15).

In conclusion, the study of peak vasodilation of the lower leg remains an important tool to investigate aspects of aging, disease, and mechanisms of vascular control. The results of this study suggest that 10 min of PAO or ischemic exercise consisting of plantar flexion or plantar-dorsiflexion performed in a similar posture result in a similar peak LVC. Further studies are warranted regarding the effects of posture during PAO and ischemic exercise on peak LVC.

ACKNOWLEDGMENTS

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GRANTS

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